

托仰翳学院 科技活动月研究成果展

PCBP1 interacts with the HTLV-1 Tax oncoprotein to potentiate NF-kB activation

背景介绍

Human T-cell leukemia virus type 1 (HTLV-1) is the etiologic agent of adult T-cell leukemia (ATL). The continuous activation of NF-KB signaling by the HTLV-1 Tax protein is essential for ATL occurrence. Despite extensive study of Tax, how Tax interacts with host factors to regulate NF-kB activation and HTLV-1-driven cell proliferation is not entirely clear. PCBP1 is closely associated with the occurrence of cancer and viral replication. However, there have been no studies on the relationship between PCBP1 and HTLV-1 to date, which presents a major obstacle for understanding the function and molecular mechanisms of PCBP1 in HTLV-1 infection. In this study, we investigated the function and the molecular mechanism of PCBP1 on Tax activation of NF-KB.

研究方法

- 1. Western blotting
- 2. Luciferase reporter assays
- 3. Co-immunoprecipitation
- 4. Immunofluorescence
- 5. CCK8
- 6. Flow cytometry

作者简介

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Tax-mediated activation of NF-kB Tax-induced IKK-NF-kB signaling

Fig 1. PCBP1 upregulates HTLV-1 Fig 2. Overexpression of PCBP1 potentiates



Fig 4. Knockdown of PCBP1 promotes apoptosis and inhibits proliferation in HTLV-1-transformed cells



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In sum, the present study identified that PCBP1 as a novel Taxinteracting protein is recruited to the Tax/IKK complex to regulate Tax-mediated NF-kB activation. We demonstrated that PCBP1 plays an essential role in promoting proliferation and inhibiting apoptotic cell death of HTLV-1-transformed cells. Therefore, PCBP1 may represent an important regulatory mechanism of HTLV-1 Taxmediated NF-kB activation and cell survival.

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代表作

结

论

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3. Su R, et al. Featured interactome of homocysteine-inducible endoplasmic reticulum protein uncovers novel binding partners in response to ER stress. Comput Struct Biotechnol J. 2023; 21:4478-4487. (2, IF:6.0)

4. Su R, et al. PCBP1 interacts with the HTLV-1 Tax oncoprotein to potentiate NF-κB activation. Front Immunol. 2024; 15:1375168. (2, IF:7.3)