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Non-Rel components of NF-κB as novel targets for selective inhibition of NF-κB in human cancer cells

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CD25, the alpha chain of interleukin-2 receptor (IL-2R), is inducibly expressed and required for formation of high affinity IL-2R. Elevated expression of CD25 has been detected in T cells in an array of autoimmune diseases and a large variety of cancers including both hematopoietic cancers and solid tumors. Moreover, soluble CD25 shed from the cell surface has been proposed as a prognostic indicator in cancer patients, with high plasma levels correlating with poor survival rates, in particular adult T-cell leukemia (ATL) caused by human T-lymphotropic virus type I (HTLV-I). In elucidating the mechanisms that govern the selective transcription of CD25, we identified the Src-associated substrate during mitosis of 68 kDa (Sam68) as a novel non-Rel component in the nuclear factor-kappaB (NF-κB) complex that confers CD25 transcription, and discovered that Sam68 plays an essential role in the induction and maintenance of CD25 in T cells. It thus revealed the important roles of KH domain-containing components in determining the promoter selectivity and transcriptional specificity of NF-κB. Given that aggressive leukemia and lymphoma cells are characterized by constitutively activated NF-κB signaling and elevated cell surface CD25 and that CD25-based therapies are used for ATL treatment, the potential to kill ATL cells through selective inhibition of NF-κB by targeting the non-Rel component Sam68 will be discussed.

Biography

Fengyi Wan completed his Ph.D. from the Chinese Academy of Sciences, China, in Biophysics and did his postdoctoral training at the National Institutes of Health, Maryland, USA. He is currently an Assistant Professor at the Johns Hopkins Bloomberg School of Public Health. He has published about 20 papers in reputable journals and book chapters and serves on the editorial board of several journals.

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