Curriculum Vitae

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Education

Date	Degree	Institution	Subject
1992	Habilitation (Venia Legendi)	Free University of Berlin	Molecular Biology
1984	Ph.D. (summa cum laude)	Philipps University of Marburg	Biochemistry
1982	Diploma (Grade 1.0)	Philipps University of Marburg	Chemistry
1976-1982		Philipps University of Marburg	Chemistry

Appointments/Affiliations

Date	Title	Institution	City
1994-Present	Research Group Leader, Head of Laboratory	Max-Delbrück-Center for Molecular Medicine	Berlin
1992-Present	Affiliate Professor (Privatdozent)	Free University	Berlin
1989-1994	Independent Research Group Leader	Max-Planck-Institute for Molecular Genetics	Berlin
1986-1989	Postdoctoral Fellow	The Rockefeller University	New York
1984-1985	Research Assistant	Philipps-University	Marburg

NF-κB/IKK and AP-1 pathways as molecular targets in human lymphomas

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Transiently activated IκB kinases (IKK) and NF-κB transcription factors are central regulators of immune responses and differentiation. However, under various pathological conditions, aberrant persistent activation, refractory to normal homeostatic controls, contributes to disease progression. NF-κB proteins provide a broad pathogenetic potential, based on the diverse processes controlled. Subgroups of lymphoma, such as Hodgkin's lymphoma (HL) and other neoplastic diseases reveal constitutive activation of NF-κB and of the IκB kinase (IKK) complex. Activation involves the classical p65-p50 heteromer, as well as the p100 NF-κB-precursor pathway. Constitutively activated NF-κB results in resistance to apoptosis, enhanced G1-S phase progression and tumorigenicity. A genome-wide identification of NF-κB regulated genes in lymphoma cells with constitutive activation and in lipopolysaccharide stimulated cells by high density microarrays has been undertaken to dissect IKK and NF-κB dependent pro-oncogenic and innate immune response gene networks. In addition to IKK/NF-κB, HL tumor cells bear a MAP kinase independent superactivation of transcription factor AP-1 (c-Jun/JunB), which distinguishes classical Hodgkin's disease from other B or T cell malignancies. AP-1 supports cell cycle progression and cooperates with NF-κB to co-stimulate expression of cyclin D2 and the lymphocyte homing receptor CCR7. These data suggest an important role for a cooperation between IKK/NF-κB and AP-1 in Hodgkin lymphoma pathogenesis.

Constitutive activation of IKK/NF-κB, but not of AP-1 in HL cells is abrogated by drugs which inhibit Hsp90. This chaperone Hsp90 is generally required for both constitutive and inducible IKK and NF-κB activity and controls IKKs at two levels, inducibility of enzymatic activity and biogenesis. Further modes of pharmacological interference will be discussed.