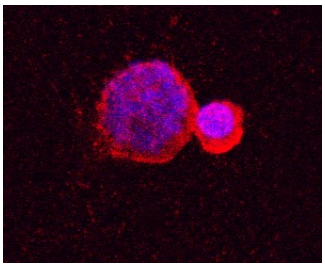

The IL-2 inducible kinase ITK and its role for HIV-1 replication in human T-cells

Prof. Dr. Carsten Münk

AIDS Research Group
Clinic for Gastroenterology, Hepatology and Infectiology
University Hospital Düsseldorf
Heinrich Heine University Düsseldorf
Universitätsstr. 1
40225 Düsseldorf

Phone: +49 211 81-10887

Email: Carsten.Muenk@med.uni-duesseldorf.de

Human immunodeficiency virus type 1 (HIV-1) infection relies on various cellular host factors, among them the IL-2 inducible kinase (ITK), a member of the Tec family of tyrosine kinases. ITK is selectively expressed in T-cells and mast cells and is involved in signaling pathways downstream of the T-cell receptor and chemokine receptors which induce PLC-1 activation, Ca^{2+}-release, transcription factor mobilization and actin rearrangements. Thus ITK is an important factor for T-cell activation, which is required for productive HIV-1 infection. Since HIV-1 cannot replicate in ITK deficient T-cells, it is hypothesized that HIV-1 stimulates ITK specific signaling pathways through binding to the chemokine receptor (CXCR4 or CCR5) to create an environment which is susceptible for infection. Aim of this project is to further characterize the processes in viral replication that are affected by ITK and to identify signaling pathways and proteins that are involved.