

Seminar-Ankündigung

Titel: "NF-kB and STAT1 are negatively regulated by acetylation of STAT1"

Vortragender: Dr. Oliver H. Krämer

**Institut für Biochemie und Biophysik
Friedrich-Schiller-Universität Jena**

Center For Molecular Biomedicine

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Acetylation of signaling molecules can lead to apoptosis or differentiation of carcinoma cells. The molecular mechanisms underlying these processes and the biological role of enzymes mediating the transfer or removal of an acetyl-group are currently under intense investigation. Our study shows that Stat1 is an acetylated protein. Stat1 acetylation depends on the balance between Stat1-associated histone deacetylases (HDACs) and histone acetyltransferases (HATs) such as CBP. Remarkably both inhibitors of HDACs and the cytokine interferon α alter this equilibrium and induce Stat1 acetylation. The analysis of Stat1 mutants reveals Lys 410 and Lys 413 as acetylation sites. Experiments with Stat1 mutants mimicking either constitutively acetylated or nonacetylated states show that only acetylated Stat1 is able to interact with NF- κ B p65. As a consequence, p65 DNA binding, nuclear localization, and expression of anti-apoptotic NF- κ B target genes decrease. These findings show how the acetylation of Stat1 regulates NF- κ B activity and thus ultimately apoptosis.

Alle Interessierten sind zu diesem Vortrag herzlich eingeladen.

