Peter Becker

Chromatin dynamics and gene regulation

Introduction

Research in the Becker lab focuses on the molecular mechanisms by which dedicated modulators of chromatin structure regulate the activity of chromosomal loci. We strive to understand the principles that allow the targeting of regulators to specific sites in chromatin, the nature of the structural changes provoked by these factors, as well as the molecular mechanisms through which alterations of chromatin structure affect enzymes involved in nucleic acid metabolism.

Lab members (in 2004)

Peter B. Becker
Professor of Molecular Biology; Adolf-Butenandt-Institute, Ludwig Maximilians Universität , Schillerstr. 44, D80336 München, Germany
Tel: +49-89-21875-427, Fax: +49-89-21875-425
Email: pbecker@med.uni-muenchen.de

Postdoctoral fellows:
Cristina Chioda
Ina Dahlsveen
Anton Eberharter
Roger Ferreira
Gregor Gilfillan
Sonja Münchow
Matthias Prestel
Catherine Regnard
Tobias Straub

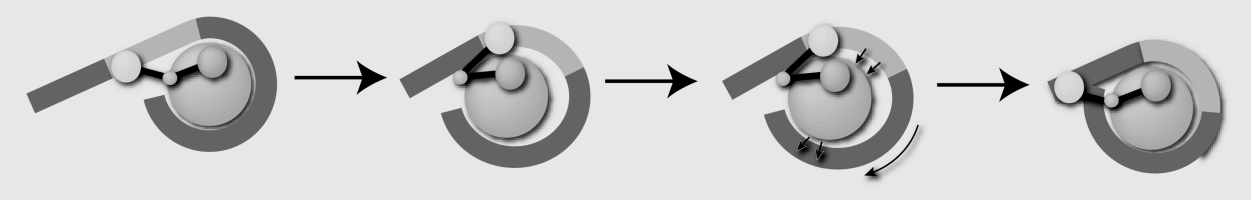
PhD students:
Felix Hartlepp
Annalisa Izzo

Diploma / MD students:
Angelika Kitziltas
Conny Koenig
Verena Maier

Technical assistance
Rosemarie Lamm
Christa Schwarzlose
Heike Mitlöhner
Irene Vetter

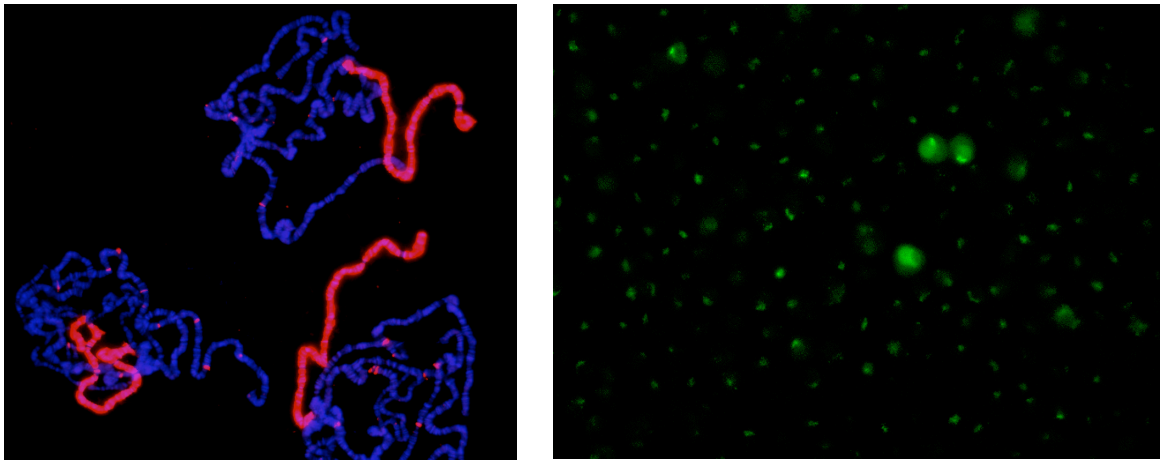
ATP-dependent nucleosome remodelling

One long-standing aim of research is to understand the mechanism and regulation of ATP-dependent nucleosome remodeling by the ISWI containing CHRomatin Accessibility Complex (CHRAC) and its functional significance in vivo. CHRAC was discovered in the lab and published in 1997. In CHRAC, ISWI is associated with Acf1, a protein featuring a bromodomain and PHD fingers in addition to a variety of other conserved domains, and two small histone fold subunits.



Dosage compensation in Drosophila

A second broad theme is to understand the principles that underlie the process of dosage compensation in Drosophila. Dosage compensation in fruit flies involves enhancing the transcription of the majority of genes on the single X male chromosome by two-fold. The acetylation of histone H4 at lysine 16 by the acetylase MOF is causally involved in this increased transcription. MOF is targeted to the X chromosome as part of a dosage compensation complex (DCC) consisting of at least 4 other proteins (MSL1, MSL2, MSL3 and MLE), whose loss-of-function leads to male specific lethality. The complex also requires incorporation of non-coding roX RNA for efficient targeting and coating of the X chromosome. Using a variety of biochemical, cell biological and genetic methods we are characterising the protein-protein, protein-DNA and protein-RNA interactions that define DCC and its association with X chromosomal chromatin.



Interaction of dosage compensation complex with the X chromosome
in polytene tissue and diploid cells