



Molecular Mechanism of Hsp70 and Hsp90 Chaperones

by

Matthias Mayer

ZMBH, Heidelberg

Abstract:

The 70 kDa heat shock proteins (Hsp70) are without doubt the most versatile of all molecular chaperones. They are involved in de novo folding of nascent polypeptides and refolding of denatured proteins into the native state, they prevent aggregation of misfolded proteins and, together with Hsp100 or Hsp110 proteins, solubilize and refold aggregated proteins, they drive polypeptide transport across biological membranes, and they assist assembly and disassembly of oligomeric protein complexes. Hsp70s together with Hsp90s form a chaperone machinery that directly controls stability and activity of a large number of natively folded protein clients including receptors, protein kinases and transcription factors, many of which are key components of essential signal transduction pathways and regulatory circuits. In general these clients only become responsive to upstream activating signals after they interacted with the chaperone machinery.

Using biochemical and biophysical techniques we investigate the molecular mechanism of Hsp70 and Hsp90 chaperones by analyzing their dynamics and how their conformation is regulated by nucleotides and cochaperones. We ask the question what makes a native protein to be a client of Hsp70 and Hsp90 while closely related proteins are not so much dependent on them and what happens to the client when they interact with the chaperones.

Friday, May 3rd, 2013, 13:00

Room PH 127