ATP-dependent nucleosome remodelling: factors and functions

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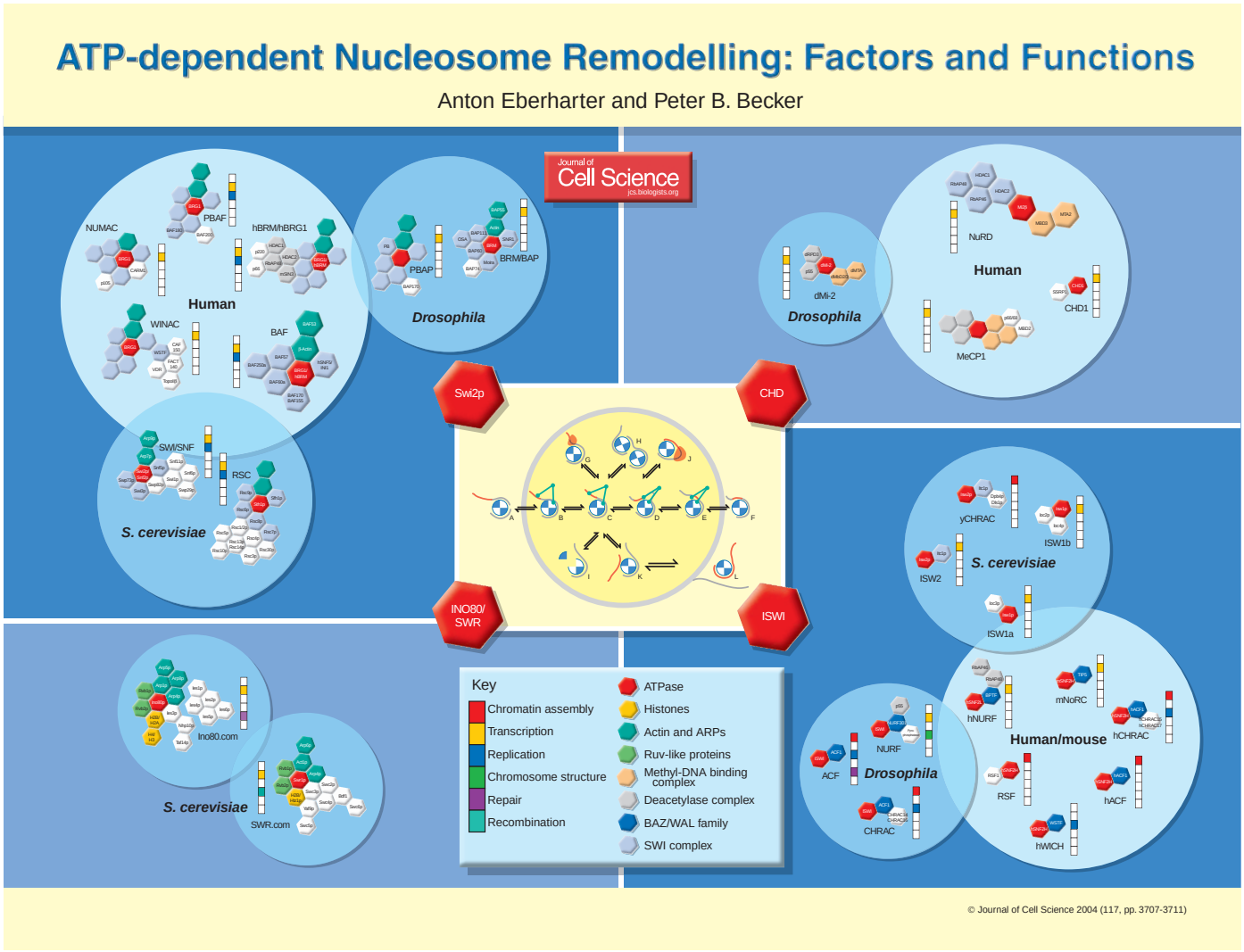
The term ‘nucleosome remodelling’ subsumes a large number of ATP-dependent changes of canonical nucleosome structure brought about by dedicated nuclear enzymes, which are usually part of larger, multifactorial complexes (Becker and Hörz, 2002; Havas et al., 2001; Lusser and Kadonaga, 2003; Neely and Workman, 2002). Collectively, nucleosome remodelling machines endow chromatin with dynamic properties that implement states of ‘plasticity’ or ‘fluidity’ compatible with the function of chromatin as a substrate for processes revolving around DNA metabolism, such as the expression of genes, the duplication of the genome, the repair of DNA damage and the recombination of chromosomes. Controlled energy-dependent nucleosome remodelling is vital for faithful execution of a cell’s proliferation and differentiation programme, as perturbations lead to neoplasia and other diseases (Cairns, 2001). Most importantly, ‘remodelling’ of nucleosomes increases the accessibility of DNA sequence elements to regulatory proteins that scan the genome for target sites. In vivo, the action of ATP-dependent nucleosome remodelling machines may lead to a variety of phenomena, ranging from the complete absence of nucleosomes at regulatory sites (Reinke and Horz, 2003) to shifting nucleosome positions (Belikov et al., 2001; Fazzio and Tsukiyama, 2003; Goldmark et al., 2000; Kent et al., 2001; Lomvardas and Thanos, 2001), increasing the access of DNA on the surface of positioned nucleosomes (Truss et al., 1995) and exchange of H2A variants (Krogan et al., 2003; Mizuguchi et al., 2003).

The biochemical analysis of nucleosome remodelling led to the description of a plethora of deviant ‘nucleosomoid’ structures that are generated by various remodelling enzymes under certain



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experimental conditions. At first sight, the diversity of these structures is puzzling (see centre of the poster). They include the peeling of significant segments of DNA off the ends of nucleosome particles, the interaction of DNA-binding proteins with nucleosomal DNA at the edge or more central parts of the nucleosome, the formation of particles with 'dimer' characteristics, the distortion of DNA on the nucleosome surface to follow an 'altered path', the extraction of histone H2A/H2B from the nucleosome and, finally, the relocation of entire histone octamers to close-by DNA in cis or in trans. Many of these structures have been generated from model mononucleosomes and it is still not clear to what extent corresponding structural transitions are permitted in nucleosomal arrays.

The inner part of the poster presents an attempt to integrate these various findings into a 'unified', hypothetical model for nucleosome remodelling (Längst and Becker, 2004). Accordingly, nucleosome remodelling machines target a nucleosome by contacting the histone moiety and the DNA at the nucleosomal edge (B). A conformational change lifts a segment of DNA off the nucleosome surface (C). Available DNA may contact the bare histone surface during dimer formation (H) or initiate the process of trans-displacement (K,L). Alternatively, a histone H2A/H2B complement may be extracted from the particle (I). The accessible DNA may be contacted by non-histone proteins (G) or histones during dimer formation (H). It may also be recaptured by the histone surface in a shifted manner, such that a segment of DNA is distorted into a small loop or bulge protruding from the nucleosome surface (D). Propagation of this bulge around the histone octamer by disruption of histone-DNA interactions at the front and reformation of equivalent interactions in the wake of propagation (E) will eventually lead to relocation of the histone octamer to a neighbouring DNA segment in cis (F). Accumulation of such distortions without release will lead to disruption of histone-DNA interaction through an extended segment of DNA, which then becomes available for contact by non-histone proteins. There is relatively little evidence for an in vivo correlate of any of these reactions, owing to the inherent difficulty of monitoring the state of single nucleosomes in vivo. However, there is evidence for ISWI-catalysed nucleosome sliding (E,F) (Fazzio and Tsukiyama, 2003) and for the involvement of the SWR complex in replacing nucleosomal H2A by the H2A.Z variant (I) in yeast (Krogan et al., 2003; Mizuguchi et al., 2003).

The presentation of 'remodelling' intermediates relies on the assumptions that the variety of nucleosome remodelling complexes displayed act along a more or less common mechanistic pathway and that the different experimental observations during the analysis of different remodelling enzymes are related to quantitative, kinetic differences between remodelling enzymes, which affect the various equilibria displayed in the centre of this poster differently. However, because in vitro remodelling by SWI/SNF- and ISWI-containing complexes leads indeed to very different results [the latter catalyses nucleosome sliding exclusively whereas the former is also accessible to DNA on the nucleosome surface (Fan et al., 2003)] in careful side-by-side comparison, these assumptions have been challenged by arguments for fundamental mechanistic differences between different classes of nucleosome remodelling machine (Fan et al., 2003; Narlikar et al., 2001).

All nucleosome remodelling ATPases belong to the SNF2 family of ATPases (Eisen et al., 1995; Lusser and Kadonaga, 2003). They can be further divided into seven subfamilies according to characteristic domain features. We restrict this overview to the four most prominent subfamilies, about which most is known in terms of function and complex formation: the SWI2, ISWI, CHD and Ino80 subfamilies. Nucleosome remodelling has also been described for members of the Cockayne Syndrome protein B (CSB), Rad54 and DDM1 subfamilies (Alexeev et al., 2003; Alexiadis and Kadonaga, 2002; Brzeski and Jerzmanowski, 2003; Citterio et al., 2000; Jaskelioff et al., 2003).

We focus our overview on remodelling complexes isolated from three key organisms: *Saccharomyces cerevisiae*, *Drosophila melanogaster* and human/mouse. We refer to primary literature and reviews that support the identity of the complexes and their subunit composition depicted on the poster and highlight selected functional aspects. For more detailed and comprehensive information we refer the reader to review articles. Functionally related subunits are labeled with a unique colour code – for example, the ATPases in red, actin and actin-related proteins (arps) in green, and histone deacetylase modules in grey. Identical positions of subunits in homologous complexes identify functionally equivalent proteins.

The SWI/SNF family

The distinguishing feature of ATPases of the SWI2/SNF2-type is a bromodomain (Horn and Peterson, 2001; Martens and Winston, 2003). The SWI/SNF complex from yeast is involved in transcriptional activation and repression of certain yeast genes (Cairns et al., 1999; Sudarsanam and Winston, 2000) with fluctuations throughout the cell cycle (Krebs et al., 2000). An additional function for activation of weak replication origins has been documented (Flanagan and Peterson, 1999). The highly related RSC complex (remodels the structure of chromatin) is essential in yeast (Cairns et al., 1996) and, similarly to SWI/SNF, functions in activation and repression of transcription (Ng et al., 2002). In human cells several remodelling complexes form around either of the two remodelling ATPases hBRM and BRG1. With regard to subunit composition, the BAF complex is related to SWI/SNF whereas PBAF and RSC represent each other (Wang, 2003). Depending on the isolation procedure, variants of a basic BAF (Brg1-associated factors) complex with additional subunits are observed (Kitagawa et al., 2003; Sif et al., 2001; Underhill et al., 2000; Xu et al., 2004). The *Drosophila* homolog of Swi2p/Snf2p is Brahma (BRM). BRM resides in two closely related complexes that share many subunits: the BAP (Brahma-associated proteins) complex, which is related to human BAF/yeast SWI/SNF (Martens and Winston, 2003; Wang, 2003); and the recently isolated PBAP (polybromo-associated BAP)

complex (Mohrmann et al., 2004), representing human PBAF/yeast RSC. Both *Drosophila* complexes, BAP and PBAP, are trithorax group activators of homeotic gene expression and thereby antagonize the repressive function of polycomb complexes (Mohrmann et al., 2004; Vazquez et al., 1999; Wang, 2003). Finally, cell-type-specific remodelling factors have also been reported (Lemon et al., 2001). SWI/SNF-type complexes are able to slide histone octamers, but they can also distort the path of DNA within the nucleosome without nucleosome relocation (Fan et al., 2003; Havas et al., 2000; Saha et al., 2002).

The ISWI family

ISWI-type remodellers are characterised by C-terminal SANT-like domains (Grüne et al., 2003). A number of complexes have been described in a variety of species; all of these function in vitro predominantly by relocating histone octamers without disruption (Längst and Becker, 2001). All three fly complexes known (Ito et al., 1997; Tsukiyama et al., 1995; Varga-Weisz et al., 1997) have homologs in humans (Barak et al., 2003; Bochar et al., 2000; LeRoy et al., 2000; Poot et al., 2000). In mammals two ISWI-homologs exist, SNF2H and SNF2L. While most of the complexes contain SNFH, SNF2L so far has only been found in the human NURF complex (Barak et al., 2003). Common to these and other ISWI-containing remodelling complexes (Bozhenok et al., 2002; Strohner et al., 2001) are subunits related to Acf1 (labeled blue in the poster), which contain plant homeo (PHD)- and bromodomains.

Like humans, *S. cerevisiae* expresses two forms of ISWI, Isw1p and Isw2p. Complexes resembling yeast CHRAC and ACF contain Isw2p (Goldmark et al., 2000; Iida and Araki, 2004). Isw1p resides in two functionally distinct complexes (Vary et al., 2003). Functions for ISWI-containing complexes are diverse, ranging from transcriptional regulation of RNA polymerase II (Morillon et al., 2003; Tsukiyama et al., 1999), RNA polymerase I (Santoro et al., 2002) chromatin assembly (Deuring et al., 2000; Fyodorov and Kadonaga, 2002; Loyola et al., 2003) and replication (Bozhenok et al., 2002; Collins et al., 2002).

The CHD family

Members of the CHD family are characterised by a chromodomain (Delmas et al., 1993; Kelley et al., 1999). The most prominent member is the ATPase Mi-2, which resides in NuRD complexes that have histone deacetylase activity (Feng and Zhang, 2003). In some isolation procedures NuRD copurifies with a second SNF2H-cohesin complex (Hakimi et al., 2002), but further fractionation separates the two entities (Y. Zhang, personal communication). NuRD associates with MBD2 and p66/68 to form the MeCP1 complex, which mediates effects of DNA methylation (Feng and Zhang, 2001). A multi-subunit Mi-2 complex has recently been isolated from *Drosophila melanogaster* (K. Bouazoune and A. Brehm, personal communication). In *S. cerevisiae* CHD1 appears to work as a monomer (Tran et al., 2000). Mi-2-containing complexes catalyse nucleosome sliding in vitro (Brehm et al., 2000).

Split ATPase domain enzymes

Ino80p and Swr1p share several interesting properties. Their ATPase domains contain an insert, which splits this conserved region into two segments. So far, multi-subunit complexes of this class of ATPase have only been isolated from yeast (Krogan et al., 2003; Mizuguchi et al., 2003; Shen et al., 2000). Ino80.com and SWR.com share several subunits, including the Rvb1p and Rvb2p enzymes, histones, actin and actin-related proteins (ARPs). The association of Rvb1 and Rvb2 proteins, which are related to the bacterial RuvB helicase within such complexes, implicates a role for this type of remodeller in DNA repair (Shen et al., 2000). The SWR complex (Krogan et al., 2003; Mizuguchi et al., 2003) represents a novel type of chromatin remodelling activity that can exchange canonical H2A within nucleosomes for H2A variants. Although the SWR complex has so far only been purified from yeast, orthologs of the SWR1 gene are known throughout the eukaryotic kingdom and hence similar complexes may be widespread.

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