double-bromodomain of the CCG1 subunit in the TFIID complex (DBD(CCG1)). This interaction should be a key to connecting the histone acetylation and the nucleosome structure change. Here we report the crystal structure of the CIA-DBD(CCG1) complex at 3.3 Å resolution. Although we crystallized the complex by mixing equimolar amounts of CIA and DBD(CCG1), the crystal structure showed that one DBD(CCG1) molecule binds two CIA molecules in two distinct sites. The GST pull-down assay with point mutants of DBD(CCG1) at either binding site also suggested that DBD(CCG1) has two binding sites for CIA in solution. The crystal structure of the CIA-DBD(CCG1) complex suggested that an acetylated lysine residue can interact with DBD(CCG1) in the complex. In addition, the superposition of the CIA/ASF1 in the CIA-histone-H3-H4 complex [Natsume *et al.*, Nature (2007)] onto the CIA in the present complex showed that the histone H3-H4 dimer competes with DBD(CCG1) for CIA through steric hindrance. These observations suggest that (i) the recruitment of the CIA-DBD(CCG1) complex to the acetylated histone and (ii) the histone acetylation-induced nucleosome structure change are mediated by interactions among CIA, DBD(CCG1), and acetylated histones.

Keywords: histone chaperone, bromodomain, nucleosome assembly/disassembly

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Structural studies of human PCNA mutant, REV6-1

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All cells are continually exposed to a large variety of external and internal agents that damage their DNA. DNA replication in eukaryote is a highly coordinated process involving many proteins that work cooperatively to ensure the accurate and efficient replication of DNA. Although replicative DNA polymerases δ/ε synthesize genomic DNA in normal condition, the replicative polymerases are not able to synthesize DNA using damaged nucleotide as a template because of their high fidelity. In that situation, translesion synthesis (TLS) polymeases alternatively synthesize DNA by error-free or error-prone manners. PCNA, a member of sliding clamp family and ring-shaped homotrimer, not only stimulates processive DNA synthesis by the replicative polymerase, Polδ, but also provides the central scaffold to which TLS polymerases bind to gain access to the replicative machinery stalled at the lesion site and to execute lesion bypass synthesis. Actually, PCNA is characterized as one of *REV* genes' products affecting UV-induced mutagenesis in *Saccharomyces cerevisiae*. Furthermore, the *rev6-1* mutant strain produces a G178S substitution in PCNA (REV6-1) and shows deficiency in TLS. Although the side chain of substituted serine appears to show an unfavorable steric interaction because the G178 residue is located in a β-sheet at the interface between the monomers composing trimer, structural features of REV6-1 are still unclear. To clarify the influence to oligomerization of PCNA by only one-residue-substitution, we investigate the difference of structural and functional features between human PCNA and REV6-1 by several methods including crystallography, and discuss the comparisons of their behaviors in solution and crystal.

Keywords: DNA replication, structural biology, protein crystallography

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Uranyl mediated photocleavage in proteins

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Uranyl ions absorb light in the UV-visible region and have been found to bind to and cause photo-cleavage in biomolecules such as proteins and nucleic acids. In DNA, the uranyl ion binds to the phosphate backbone of the molecule and can potentially cleave the molecule between any base pair along the chain. In proteins, this binding and thereby the induced structural changes, are much more specific. One of many uranyl-binding proteins is porcine pancreatic elastase (PPE). In this study, elastase-crystals have been soaked in a uranyl-containing buffer and several data sets have been collected in house and at the synchrotron at both room temperature and at 100K. The uranyl binds with high occupancy in a single position close to the well-known calcium/sodium binding-site. Only small conformational changes compared to the native structure are seen upon ligand binding. These changes originate from amino acids moving either to accommodate space for (Tyr71) or to co-ordinate the ligand (Glu59, Glu69). However, the sodium ion is not moving from its native site. In addition, two acetates bind to the uranyl. After irradiation (400nm, 2h), the observed structure is still that of the native structure. However the density of one amino acids in the direct proximity of the uranyl ion, Asn63, has completely disappeared. The densities of the acetates have also degraded and only the positions of the coordinating oxygens are visible.

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Water-soluble chlorophyll-binding protein from *Chenopodium album*

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Water-soluble chlorophyll-binding proteins (WSCPs) have been found in some terrestrial plants. WSCPs can be categorized into two classes according to photoconvertibility. The absorption spectra of class I WSCPs change drastically on exposure to visible light, whereas those of class II WSCPs do not change at all. The amino acid sequences of classes I and II WSCP show no similarity to each other. The physiological function of WSCPs is not known although it is supposed to be a chlorophyll (Chl) carrier. So far, only the structures of class II WSCP have been determined because of high sensitivity