Salt-dependent Intra- and Internucleosomal Interactions of the H3 Tail Domain in a Model Oligonucleosomal Array*

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The core histone tail domains are known to be key regulators of chromatin structure and function. The tails are required for condensation of nucleosome arrays into secondary and tertiary chromatin structures, yet little is known regarding tail structures or sites of tail interactions in chromatin. We have developed a system to test the hypothesis that the tails participate in internucleosomal interactions during salt-dependent chromatin condensation, and here we used it to examine interactions of the H3 tail domain. We found that the H3 tail participates primarily in intranucleosomal interactions when the nucleosome array exists in an extended “beads-on-a-string” conformation and that tail interactions reorganize to engage in primarily internucleosomal interactions as the array successively undergoes salt-dependent folding and oligomerization. These results indicated that the location and interactions of the H3 tail domain are dependent upon the degree of condensation of the nucleosomal array, suggesting a mechanism by which alterations in tail interactions may elaborate different structural and functional states of chromatin.

The eukaryotic genome is assembled into vast arrays of nucleosomes, which are in turn condensed into 30-nm chromatin fibers and other secondary chromatin structures (1–3). In vitro evidence indicates that the folding of nucleosome arrays into secondary structures is strongly favored at physiological concentrations of mono- and divalent salts, as is the oligomerization of arrays into higher order, tertiary chromatin structures (2, 4, 5). The stability of secondary and tertiary chromatin structures is influenced by the incorporation of specific non-allelic variants of the core histone proteins, the binding of linker histones, and association of ancillary chromatin proteins (2, 3). It is generally believed that for genomic DNA to be accessible to various enzymatic machineries, the chromatin fiber has to undergo transient decondensation, facilitated by specific posttranslational modifications such as lysine acetylation within the histone tail domains or ATP-dependent chromatin remodeling (6, 7).

The core histone tail domains are essential for compaction of oligonucleosome arrays into secondary chromatin structures and for efficient assembly of oligomeric tertiary structures (1, 2, 8–10). However, the structures and interactions of the histone tail domains and the molecular mechanisms by which they facilitate chromatin compaction remain largely uncharacterized. Evidence indicates that these highly basic domains interact with both protein and DNA targets within chromatin (11–15), and it is generally assumed that the core histone tail domains participate in internucleosomal as well as intranucleosomal interactions within condensed chromatin. Indeed, several crystal structures of nucleosome core particles indicate that the tails project away from the main body of the nucleosome to potentially mediate internucleosomal interactions (2, 16, 17). These interactions may occur in cis, between nucleosomes within the same array, or in trans, mediating long range fiber-fiber interactions between nucleosomes on separate chromatin fibers. However, direct evidence for such interactions has been presented only for cis interactions of the H4 tail domain with a charged protein patch on the surface of adjacent nucleosomes (15).

We have developed a new approach to detect the presence of internucleosome core histone tail interactions during salt-dependent condensation of a model nucleosome array and have used this approach to examine the interactions of the H3 tail. Our data indicate that the H3 tail domain relocates from mainly intranucleosomal interactions, within the fully extended “beads-on-a-string” form of the chromatin fiber in low salt, to primarily internucleosomal interactions upon formation of condensed secondary and tertiary chromatin structures. These results suggest that the essential functions of the histone tails in chromatin condensation stem from their involvement in internucleosomal interactions.

**EXPERIMENTAL PROCEDURES**

Preparation of H2A/H2B Dimers and H3/H4 Tetramers—Recombinant Xenopus H2A/H2B were prepared and purified as preformed dimers as described (18). Coding sequences for Xenopus H3 cysteine substitution mutants H3T6C, H3A15C, H3A24C, and H3V35C containing cysteine substitutions at amino acid positions 6, 15, 24, and 35, respectively, were constructed, based on the wild type H3 sequence, by mutagenesis PCR and cloned into the PET3a expression plasmid. Note that all H3s also contained the mutation C110A to eliminate the possibility of modification this position in the native protein. The notation “C110A” will be omitted in the text for simplicity. *Escherichia coli* BL21 expression stocks were prepared from single colonies after expression tests. Recombinant Xenopus H4 was expressed, and H3/H4 tetramers were prepared as described (19). Briefly, we first determined the mass of either protein within each bacterial cell culture and then mixed cultures containing roughly equimolar amounts of H4 and each of the H3 mutant proteins. A pellet containing both proteins (from 1 combined liter of culture) was suspended in 100 ml of P1 buffer (50 mM Tris, 10 mM EDTA, 100 μM MgCl₂/RNase, pH 8.0), and then cells were lysed with an equal volume of P2′ buffer (0.2 M NaOH, 0.4% Triton X-100) in the presence of 0.4 mM phenylmethylsulfonil fluoride, 8 mM dithiorthitol at room temperature for an hour. The lysate was dialyzed against 2 M NaCl, TE buffer (pH 8.0, 10 mM β-mercaptoethanol) at 4 °C for at least 3 days. The abbreviations used are: TE, Tris-EDTA; N1, nucleosome 1; APB, azidophenacyl bromide.

* This work was supported by National Institutes of Health Grants GM52426 (to J. J. H.) and GM45916 (to J. C. H.). The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

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Internucleosomal Interactions of the H3 Tail in Chromatin

Radioactive Labeling and Azidophenacyl Bromide (APB) Modification of H3 Cysteine Substitution Mutants—H3/H4 tetramers (500 μg) containing mutant H3 proteins were mixed with 5 μg of Aurora-A kinase pEG2 (a generous gift from Drs. D. Angelov and S. Dimitrov), 7 mM Mg2+, 10 mM dithiothreitol, and 0.55 mM of [γ-32P]ATP in 1 ml of 2 M NaCl-TE buffer and were then incubated at 37 °C for 2 h. Histones were recovered by addition of 140 μl of a 50% slurry of 100–200 mesh Biorex 70 resin, along with 30 μl of 1 M dithiothreitol and 2.83 ml of TE buffer to bring the total volume to 4 ml. The mix was slowly rotated at 4 °C for 2 h and then poured into a micro column, and the column was washed with 4 ml of 0.6 M NaCl-TE. Labeled tetramers were eluted with 2 M NaCl in TE buffer, and the 100-μl fractions containing H3/H4 tetramers were sonicated to reduce the size of contaminating nucleic acids, and the Bio-Rex resin purification was repeated. The fractions containing H3/H4 tetramers were mixed with 50 μl of hydroxyapatite resin/ml (Bio-Rad, 50% slurry) for 30 min at 4 °C, and the resin was removed by centrifugation. This process was repeated three times to eliminate final traces of contaminating nucleic acids. The proteins were checked by SDS-PAGE and trial reconstitutions.

Preparation of Ligated 13-Mer Oligonucleosome Array—The proteins were dialyzed at 4 °C against 2.0 liters of 1.0 M NaCl-TE for 4 h and 0.75 M NaCl-TE for 3 h. The final dialysis was done overnight against TEN (10 mM Tris, 0.25 mM EDTA, 2.5 mM NaCl (pH 8.0)). The sedimentation profiles in TEN and 2 mM MgCl2 were analyzed by ultracentrifugation using either a Beckman XL-A or XL-1 Ultracentrifuge to confirm full saturation of the array as described (10, 23). Reconstituted nucleosome arrays were stored at 4 °C until used. Gradient-purified nucleosome N1 from one reconstitution was concentrated 10-fold using Microcon YM-50 filter concentrators as described (19). The ligation reactions typically contained 10 μl (~5 μg) of nucleosome N1, 30 μl (~26 μg) of 12-mer array, 4,000 units of T4 DNA ligase (New England Biolabs), 100 μg/ml bovine serum albumin, 1 mM ATP, 2.5 mM Mg2+, 0.25 mM EDTA, and 10 mM Tris (pH 8.0) in a total volume of 100 μl. The ligation was incubated at room temperature in the dark for 1 h. Usually about 60–80% of the 12-mer array was ligated into a 13-mer.

UV-induced Cross-linking and Identification of Cross-linking Bands—At the end of the ligation reaction, the buffer was exchanged to 200 μl of TEN via microfiltration as described (19). Nine-μl fractions of the sample were rapidly mixed with 1 μl of NaCl or MgCl2 solutions and placed into the bottom of a Falcon 5-ml polystyrene tube, which was inside a 15-ml Pyrex 9820 glass tube. Then the samples were irradiated with 365 nm UV light generated by a VMR LM-20E light box for 1 min. Afterward, the samples were mixed with 2 μl of NEB III, 2 μg of bovine serum albumin, and 20 units each of DraIII and HhaI to produce a total volume of 20 μl at 37 °C for 3 h. One sample was diluted to 20 μl, mock-digested under the same conditions, and used as the total cross-linking control. The samples were then typically mixed with gel loading solution containing SDS, loaded onto 0.7% agarose gels, and run at 100 V for 2.5 h before EtBr staining and drying for PhosphorImager analysis.

RESULTS

We wished to determine whether the H3 tail domain contacts the DNA of neighboring nucleosomes and whether these interactions are dependent upon condensation of the nucleosome array. Thus, we employed a well defined model nucleosome array in which the degree of condensation of the array can be modulated by altering the concentrations of mono- and divalent salts in solution. In low salt conditions (~10 mM Tris buffer), the nucleosome arrays used in our studies adopted an extended beads-on-a-string conformation (9, 24). With increasing salt concentrations (0.5–2.0 mM MgCl2), arrays condense into more tightly packed secondary chromatin structures with the same hydrodynamic shape as a fully compacted 30-nm chromatin fiber (2). At higher salt concentrations, (~5 mM MgCl2) arrays oligomerize into tertiary chromatin structures.

Our experimental scheme to investigate internucleosomal interactions during salt-dependent condensation of the model nucleosome array is depicted in Fig. 1. First, a mononucleosome (N1) containing a site-specifically modified H3 tail domain was ligated to the end of a 12-mer nucleosome array preassembled with native chicken erythro-
Internucleosomal Interactions of the H3 Tail in Chromatin

To detect internucleosome cross-linking, a portion of the sample was digested with restriction enzymes to remove nucleosome N1 before separation on the SDS-agarose gels. Any radioactivity remaining associated with the digested 12-mer template must arise from internucleosome cross-linking (Fig. 1A).

We next used this approach to investigate whether the N-terminal region within the H3 tail participates in internucleosomal interactions within the nucleosomal array and whether these internucleosomal interactions are dependent on salt-dependent condensation of the array. Arrays were prepared as described above with the UV-activatable cross-linking agent attached to the sixth residue in the H3 tail domain of the N1 nucleosome (see "Experimental Procedures" and Fig. 1B). After adjusting MgCl₂ concentrations to cause various extents of array condensation, we briefly irradiated the N1-containing arrays with UV light and analyzed cross-linking by SDS-agarose gels and autoradiography. We observed that the total amount of H3 tail cross-linked to the 13-mer DNA template (both intra- and internucleosomal cross-linking; see Fig. 1) was maximal at the lowest salt concentrations, and this amount decreased about 60% upon increasing Mg²⁺ concentrations from 0 to 5 mM (Figs. 3A and 4A). Control experiments confirmed that our nucleosome arrays were extended in 0 mM MgCl₂, folded in 1–2 mM MgCl₂, and oligomerized in higher MgCl₂, as documented previously (results not shown; see Ref. 2).

To determine the fraction of this cross-linking attributable to internucleosomal interactions of the H3 tail domain, we cut the irradiated 13-mer arrays with DraIII and HhaI restriction enzymes to remove N1 nucleosomes (see "Experimental Procedures") (Fig. 3B, lanes 4–9). Thus, any radioactivity remaining associated with the digested 12-mer DNA template after treatment with SDS must arise from internucleosomal interactions. We found that nearly all of the radioactively labeled H3 that was cross-linked to the array DNA template in low salt buffer (0 mM MgCl₂) was lost upon removal of nucleosome N1 (Fig. 3B, compare lanes 3 and 4). Quantification revealed that only about 1% of the total cross-links were internucleosomal in low-salt (0 mM MgCl₂) conditions, when the array was fully extended. Interestingly, as the concentration of MgCl₂ was increased above 2 mM to induce folding and oligomerization of the arrays, we observed that the intensity of the band corresponding to internucleosome cross-linking of the H3 tail increased >10-fold (Fig. 3B, lanes 5–9, and Fig. 4B). Controls showed that no internucleosome cross-links were detected if N1 was not ligated to the array before irradiation (results not shown). Moreover, we found that that in 4–5 mM MgCl₂, internucleosome cross-linking by the H3 tail approached 90% of the total cross-links (Fig. 4C). These results indicate that nearly all cross-links formed in low salt are internucleosomal, whereas at 4–5 mM MgCl₂ the majority of cross-links formed by the H3 tail domain are internucleosomal. Thus interactions of the H3 tail domain apparently undergo significant rearrangement upon salt-dependent chromatin condensation.

We then determined whether moving the cross-linker to a position close to the histone fold domain altered the extent of internucleosomal cross-linking. The cross-linker was attached to residue 35 within the H3 tail, near where the H3 tail exits the nucleosome, by incorporating H3V35C-APB into N1 (Fig. 1B). This N1 nucleosome was then ligated to the 12-mer array, and the experiment was repeated. Similar to the previous results, we observed a band indicating that H3V35C-APB efficiently cross-linked to the ligated 13-mer (Fig. 5A), signifying “total” cross-linking (Fig. 1). Likewise, upon removal of N1, the cross-linking signal was reduced to near background, indicating that most of these cross-links were intranucleosomal when the array was fully extended in 0 mM MgCl₂ (Fig. 5A, compare lanes 2 and 3). However, in contrast to the results obtained when the cross-linker was located near the N terminus of the H3 tail domain (Fig. 4), no increase in the internucleosome
cross-linking signal was evident as the MgCl₂ concentration was increased from 0 to 5 mM (Fig. 5A, lanes 3–8). Thus, the region of the H3 tail nearest the histone fold domain does not appear to participate in internucleosomal interactions, even when the array is condensed in high MgCl₂ concentrations.

In addition, we tested two intermediate positions in the H3 tail for internucleosome cross-linking by substitution of alanines at positions 15 and 24 with cysteine to generate H3A15C and H3A24C (Fig. 1B). Each of these proteins was radiolabeled and modified with APB; then each was incorporated into N1 as described above, and the experiment

FIGURE 2. Detection of cross-linking within ligated nucleosome arrays. A, H3T6C/H4 tetramers were radiolabeled with ³²P and then modified by APB as described under “Experimental Procedures.” Samples were analyzed by 15% SDS-PAGE, and the gel was imaged by Coomassie Blue staining (left) and PhosphorImager (right). B, nucleosome N1 containing radiolabeled and APB-modified H3T6C was incubated with the 12-mer oligonucleosome array in the absence (lane 2) or presence (lane 3) of T4 ligase for 1 h at room temperature; then products were analyzed by electrophoresis in a 0.7% agarose nucleoprotein gel. Left, ethidium bromide-stained gel; right, phosphorimage of the same gel. Lane 1 contains a 1-kb-plus DNA marker. The positions of the cross-linked 13-mer, the cross-linked (cl) mononucleosome N1 template, and the free H3 are shown.

FIGURE 3. MgCl₂-dependent internucleosome cross-linking by the N-terminal region of the H3 tail domain. Nucleosome N1 was prepared containing radiolabeled H3T6C-APB and was then ligated to the 12-mer array; total and internucleosome cross-linking was analyzed as described in the text. A, salt dependence of total cross-link formation. Ligated arrays in TEN buffer were UV-irradiated in the presence of increasing MgCl₂ concentrations, mixed with SDS, and run on a 0.7% SDS agarose gel; the gel was visualized by ethidium bromide (EB) staining (left) and PhosphorImager (right). Lane 1 contained the 1-kb-plus DNA marker; lane 2, no UV irradiation; lanes 3–8 were UV-irradiated in the presence of 0, 1, 2, 3, 4, and 5 mM MgCl₂, respectively. B, internucleosome cross-linking. The ligated arrays were UV-irradiated in the presence of different MgCl₂ concentrations and digested with DraIII and HhaI, and cross-linking was analyzed as described in A. Left, ethidium bromide-stained gel; right, phosphorimage of the gel. Lane 1, 1-kb-plus DNA marker; lane 2, no UV irradiation; lanes 3–9 were UV-irradiated in the presence of 0, 0, 1, 2, 3, 4, and 5 mM MgCl₂, respectively. Lanes 2 and 3 were not digested by restriction enzymes, and lanes 4–9 were digested to remove nucleosome N1. The positions of the cross-linked 12- and 13-mer are indicated.

FIGURE 4. Quantification of internucleosome cross-linking by H3T6C-APB. Data including that shown in Fig. 3, A and B, were quantified using ImageQuant software. A, total cross-linking (to the 13-mer template; see Fig. 3A) at each MgCl₂ was determined relative to total cross-linking at 0 mM MgCl₂ and plotted. B, bands corresponding to internucleosome cross-linking (see Fig. 3B) were quantified and normalized to data at 0 mM MgCl₂. C, the fraction of total cross-links comprised by internucleosomal cross-links was calculated at each MgCl₂ concentration and plotted. Error bars represent ±1 S.D., n = 3.
was repeated. Similar to the results of H3T6C-APB, we observed an increase in internucleosome cross-linking in the presence of ≥2 mM Mg\textsuperscript{2+}, evident after removal N1 by digestion with DraIII and HhaI restriction enzymes (Figs. 5, B and C). Quantification of the level of cross-linking for all four sites in the H3 tail is shown in Fig. 5D. Thus the majority of the H3 tail participates in internucleosomal interactions upon MgCl\textsubscript{2}-dependent condensation of the array.

To further define at what condensation step H3 rearrangement occurs, we examined cross-linking in monovalent salts. In contrast to divalent cations, monovalent salts such as NaCl can induce only intermediate levels of fiber condensation but not maximally folded secondary chromatin structures or oligomerization of arrays into tertiary chromatin structures (2). We therefore tested whether internucleosome cross-linking was observed in NaCl. We found that increasing NaCl concentrations to levels known to induce intermediate levels of condensation did not lead to a detectable increase in internucleosome cross-linking, regardless of the position of the cross-linker in the H3 tail domain (Fig. 6). These results support the conclusion that only maximally folded states and/or oligomerization of the arrays, induced in the presence of ≥2 mM MgCl\textsubscript{2}, can lead to internucleosome cross-linking by the H3 tail domain.

**DISCUSSION**

The core histone tail domains are key regulators of eukaryotic chromatin structure. The functions of these tail domains likely involve complex intra- and internucleosomal histone-DNA interactions, yet little is known about either the structures or interactions of these domains within chromatin fibers. Here we have developed a method for examining internucleosomal interactions of the tail domains in a model oligonucleosomal system that allows internucleosome cross-links to be distinguished unambiguously from intranucleosome cross-links at various levels of chromatin condensation. We provide evidence that the H3 tail makes predominantly internucleosomal interactions when the array is in a fully extended beads-on-a-string conformation in low salt but relocates to predominantly internucleosomal interactions as the array folds and oligomerizes in elevated MgCl\textsubscript{2}. In addition, the initial step in array folding involving close approach of neighboring nucleosomes (1, 25) does not result in internucleosome cross-links by the H3 tail (Fig. 6), consistent with results regarding interactions of the H4 tail (26). Our experiments also demonstrate that three positions in the outer three-fourths of the H3 tail domain participate in internucleosomal interactions (Figs. 3–5), whereas a position in the tail near the histone fold domain does not appear to be in close proximity to the DNA of neighboring nucleosomes (Fig. 5A). These results are consistent with a model in which the H3 tail transverses the distance from its own nucleosome and contacts DNA binding sites on other nucleosomes as the fiber reaches a state of full condensation and/or oligomerization.

Our previous efforts to identify internucleosomal interactions employed a dinucleosome system (20). It has been reported that dinucleosomes undergo salt-dependent compaction in a manner that
Internucleosomal Interactions of the H3 Tail in Chromatin

reflects the salt-dependent folding of nucleosome arrays (27–29). However, we were unable to detect internucleosomal interactions of the H3 tail domain in dinucleosomes. Although the detailed arrangement of nucleosomes within the 30-nm chromatin fiber remains undefined, our results suggest that the dinucleosome is not able to recapitulate internucleosomal interactions found in the native chromatin fiber. In addition, our results indicate there are fundamental differences with regard to internucleosomal interactions of the H3 tail between moderately folded and maximally condensed states of nucleosome arrays. These results raise the possibility that different chromatin states may be characterized by distinct sets of tail interactions, which in turn may be dictated by specific patterns of posttranslational modifications within these domains.

As part of our new methodology, we radiolabeled APB-modified H3 in our experiments by phosphorylation of serine 10 with Aurora-A kinase pEg2 isolated from Xenopus egg extracts (30). However, it is unlikely that the folding behavior of our ligated oligonucleosome array is affected by the H3 phosphorylation. First, the phosphorylation is limited to only one nucleosome at the end of the array. The effect of the negative charge of the phosphate is equivalent to a single acetylated lysine in the tail and is thus expected to be negligible compared with large number of positively charged lysines and arginines. Second, phosphorylation of H3 in vivo may serve as a signal to recruit non-histone factors to induce concerted changes leading to chromatin decondensation, and such factors are absent from our purified system. Third, and most importantly, preliminary results indicate that similar internucleosomal H3 tail-DNA interactions are found in experiments in which the H3 is labeled by other means (results not shown).

Increasing Mg²⁺ simultaneously causes both condensation of nucleosome arrays to fully compacted secondary chromatin structures such as the 30-nm chromatin fiber and oligomerization of fibers into higher order tertiary chromatin structures (2). Thus the internucleosomal interactions of the H3 tail domain we have detected may be relevant to either or both of two distinct structural transitions. First, these interactions may occur during the transition from an intermediately folded, “zig-zag” oligonucleosomal structure to a fully compacted 30-nm fiber and thus could contribute a significant positive force, driving formation of this fully condensed structure. Nucleosome arrays in solutions containing 2–3 mM MgCl₂ are in equilibrium between maximally folded and oligomeric states (2), and thus further elevating MgCl₂ levels may simply shift the equilibrium toward a greater proportion of maximally folded states, increasing H3 internucleosome, intrafiber interactions. Alternatively, internucleosomal interactions of the H3 tail could be relevant to the oligomerization of fibers into higher order structures. In this regard it is important to note that the internucleosomal interactions we detect could be intra (cis)- or inter (trans)-array. In the former case, cis internucleosomal interactions could be involved in the formation of a form of the condensed chromatin fiber that is required for oligomerization, whereas in the latter case, trans interactions of the H3 tail would directly mediate oligomerization by providing bridging interactions between two or more arrays. Experiments to distinguish between these possibilities are under way.

Acknowledgments—We thank Dr. Cheeptip Benyajati for construction of the p12-SS-C1 plasmid and Drs. D. Angelov and S. Dimitrov for the kind gift of Aurora kinase.

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J. Biol. Chem. 2005, 280:33552-33557.
doi: 10.1074/jbc.M507241200 originally published online August 2, 2005

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