Nucleosome Sliding Induced by the xMi-2 Complex Does Not Occur Exclusively via a Simple Twist-diffusion Mechanism*

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ATP-dependent chromatin remodeling complexes can induce the translocation (sliding) of nucleosomes in cis along DNA, but the mechanism by which sliding occurs is not well defined. We previously presented evidence that sliding induced by the human SWI/SNF complex does not occur solely via a proposed “twist-diffusion” mechanism whereby the DNA rotates about its helical axis without displacement from the surface of the nucleosome (Aoyagi, S., and Hayes, J. J. (2002) Mol. Cell. Biol. 22, 7484–7490). Here we examined whether the Xenopus Mi-2 nucleosome remodeling complex induces nucleosome sliding via a twist-diffusion mechanism with nucleosomes assembled onto DNA templates containing branched DNA structures expected to sterically hinder rotation of the DNA helix on the nucleosome surface. We find that the branched DNA-containing nucleosomes undergo xMi-2-catalyzed sliding at a rate and extent identical to that of nucleosomes assembled on native DNA fragments. These results indicate that both the hSWI/SNF and xMi-2 complexes induce nucleosome sliding via a mechanism(s) other than simple twist diffusion and are consistent with models in which the DNA largely maintains its rotational orientation with respect to the histone surface.

In the nucleus, the eukaryotic genome is packaged in the form of a highly condensed chromatin fiber through its interactions with histones and non-histone proteins (1, 2). To allow efficient progression of nuclear processes, cells have developed several mechanisms to facilitate access of DNA target sites within chromatin by trans-acting factors (3–5). One strategy involves targeted post-translational modifications of histone proteins such as acetylation, methylation, and phosphorylation that may directly alter the biochemical properties of chromatin or signal binding and recruitment of ancillary factors (6–10). A second strategy involves the activity of ATP-dependent chromatin remodeling complexes that couple the energy derived from ATP hydrolysis to alter chromatin structure, facilitating the activity of trans-acting factors (4, 11–13).

All ATP-dependent chromatin remodeling complexes contain a subunit that belongs to the SNF2 superfamily of ATPases (14). There are four main classes within this superfamily, differentiated by homology to the SWI2/SNF2, ISWI, INO80, and Mi-2 ATPase subunits within these complexes (11–13, 15). The molecular mechanism of nucleosome remodeling by the ATP-dependent chromatin remodeling complexes has been under intense study. Remodeling complexes such as the SWI/SNF complex have been shown to cause disruption of histone-DNA interactions as detected by electron energy loss microscopy and atomic force microscopy studies (16, 17). The loss of histone-DNA interactions within the nucleosome is correlated with the increase in activities of various transcription factors, restriction enzymes, and DNase I on nucleosomal DNA (18–21). Evidence indicates that Mi-2 complexes also cause such disruptions in histone-DNA interactions as detected by increase in accessibility to DNase I and restriction enzyme cleavage (22–24).

A number of chromatin remodeling complexes including Dro sophila and Xenopus Mi-2 complexes have been shown to induce nucleosome translocation (sliding) along the DNA in cis for both mononucleosomes and nucleosome arrays (17, 25–31). Nucleosome sliding is thought to be an important outcome of chromatin remodeling, which allows stable exposure of DNA target sites to trans-acting factors involved in chromatin directed activities in vivo (4, 11, 13). There are many possible ways in which chromatin remodeling could result in nucleosome sliding, and several models have been put forward (11, 30, 32). In one model nucleosome sliding is envisioned to occur by twisting of the DNA helix like a screw in a groove on the surface of the histone octamer, without significant displacement of the helix from the nucleosome surface. This model is supported by recent crystal structures of nucleosome core particles in which a turn of DNA within the core contains one less base pair compared with the symmetry-related position, causing an overwinding of the DNA at the former (33, 34). This “twist defect” could stochastically diffuse throughout the nucleosome with the cumulative effect of many such events occurring in one direction resulting in the twisting of the DNA helix along the histone surface (twist diffusion). Nucleosome sliding also may occur via a DNA uncoiling-recapture mechanism, initiated by the unraveling of DNA from one end of the nucleosome surface that is then recaptured to form a loop. The loop would then be propagated through the rest of the nucleosome, leading to change in the position of the histone octamer along the DNA (30, 32, 35). Evidence for such a mechanism during nucleosome sliding induced by the yeast SWI/SNF complex recently has been reported (35). Finally, apparent nucleosome movement may occur upon relaxation of a structurally altered, remodeled nucleosome to a canonical structure (11).

In a previous study, we tested whether hSWI/SNF-depend-
ent nucleosome sliding involves a twist-diffusion mechanism by using nucleosomes assembled on branched and nicked DNAs that would sterically hinder rotation of the DNA on the histone surface and inhibit retention of torsional stress within the DNA. Remodeling of these nucleosomes showed that SWI/SNF-induced nucleosome sliding does not occur principally via a simple twist-diffusion pathway or rotation of the DNA on the histone surface for this complex (31). In addition, we found that the presence of a nick, which could relieve torsional stress generated by the hSWI/SNF complex, did not affect the nucleosome sliding process (31) in agreement with results obtained with Drosophila ISWI (36).

In this study, we tested whether Xenopus Mi-2-induced nucleosome sliding occurs via a simple twist-diffusion mechanism using nucleosomes containing branched DNA structures expected to sterically hinder rotation of the DNA helix on the histone surface. Nucleosomes with very homogenous translational positions were used, allowing comparison of the rate of sliding between nucleosomes assembled on native and branched DNA templates. Our results indicate that Mi-2 induces nucleosome sliding on both types of templates at identical rates (31) in agreement with results obtained with Drosophila Mi-2-induced nucleosomes. Nucleosomes used for the cross-linking experiment were dialyzed for 3 h against a buffer containing 10 mM Tris-Cl, pH 8.0, prior to UV irradiation.

Mapping of Nucleosome Positions by Restriction Enzyme-Translational Gel Assay—Nucleosomes reconstituted on native DNA were digested with 20 units of BamHI, RsaI, or HhaI restriction enzyme in a 20-μl reaction for 15 min in 10 mM HEPES, pH 7.5, 0.2 mM EDTA, 5 mM MgCl2. The reactions were then stopped with 10 mM EDTA, and excess calf thymus DNA (0.75 μg) was added to compete the restriction enzymes away from the nucleosomes. The reactions were loaded on a polyacrylamide translational gel, and electrophoresis carried out as described above.

The %2d detergent Roy induced nucleosome sliding via a mechanism other than simple twist diffusion or rotation of DNA on the histone surface.

Materials and Methods

DNA Fragments—All three DNA templates are based on the 215 bp EcoRI-DdeI fragment containing a Xenopus borealis somatic SS RNA gene derived from the plasmid pXP-10 (37). The native 215-bp DNA fragment encompassing positions from -78 to +137 in the SS sequence was radiolabeled at 5’ end of the EcoRI site and purified on 6% native polyacrylamide gels as described (37). The related hairpin-containing template was generated as described previously (31) (see Fig. 1). The continuous hairpin substrate was prepared in two steps. First, a continuous strand extending from positions -78 to +137 in the SS sequence was generated as described previously (31). The top strand of the continuous hairpin substrate was generated by ligating 450 pmol each of the primer (SS primer -78 to +12) AAT TCG AGC TCG CCC CGG ATG CGG GCC CCC CCC CCA GA, (SS primer -12 to +18) AGG CAG AAC AAG AAG AAA AGT CGA GAC CAG GAG GGG TTT TTC CCC TCC TGG TCA GAG TCT GGT GCC CTA CGG CCA TAC CAC C, (SS primer +18 to +75) CTT AAA GTG CCC GAT ATC GTC TGA CCG AAG CCA AGG AGG GCT GGG CTT GTG TAG, and (SS primer +75 to +137) TAC TTG GTG AGG AGA CCC CCT GGG AAT ACC ACG TGT GAG CTT CTG CAT CCT TGG CTT GAT CCT. These complementary oligonucleotides were annealed with 900 pmol each of three bridging oligonucleotides, GGC GGT TTC CCA TCC AAG TAC TAA CCA GGC CCC ACC CTC C, AGC ATA TCG GCC AGT TTC AGG GTG TTA TGG CGC TAG GGC A, and TTC CTC CCC TTG TGC TCT GTC CTG GGG GGG CCC CAC C, and then ligated with T4 DNA ligase (Invitrogen) as described previously (31). The 245-mol ligated product was isolated on preparative denaturing polyacrylamide gels (8%) after visualization of the bands by ethidium bromide staining. Finally, double-stranded continuous hairpin substrate was generated by annealing the 5’-radiolabeled ligated 247-mer strand with the 215-mer bottom strand prepared as described above. These oligonucleotides were mixed in annealing buffer (10 mM Tris-EDTA, 50 mM NaCl, 1° C to 95 °C for 10 min), and cooled slowly by turning off the heating block. The annealing reaction created a 215-bp double-stranded SS DNA with a hairpin (12-bp stem and a 5-nucleotide loop) with 2 nucleotide hinges at the beginning and at the end of the stem at the -12 position (Fig. 1).

Nucleosome Reconstitution—Recombinant Xenopus H2A and the cytosine-substituted H2A used in this complex were expressed as preformed dimers, and the latter was modified with 4-azidophenacyl bromide (APB, Sigma) as described (37, 38). Histone H3/H4 tetramers were prepared from chicken erythrocyte nuclei, and nucleosomes were reconstituted with either native H2A/H2B or H3A/H2B2G26C-APB. Reconstitution with the 215-bp SS DNA fragment yields a majority of nucleosomes in which the dyad axis of symmetry is located near position -3.

The abbreviations used are: APB, 4-azidophenacyl bromide; EcoIII, exonuclease III.
cleosomes. Exonuclease III (Exo III, 0.5 units; New England Biolabs) was added, and the reactions were incubated for 15 min and then stopped by the addition of 0.1% SDS, 10 mM EDTA stop solution. DNA in the samples was then ethanol-precipitated, and cleavage products were analyzed as described (31). Note that for the Exo III digests the nicked hairpin substrate was radiolabeled at the base of the hairpin (at −12) (Fig. 1 and Fig. 6B), thus precluding BamHI selection of nucleosomes on this template.

RESULTS
 Mi-2 complexes from Drosophila and Xenopus have been shown to catalyze nucleosome sliding in vitro (25, 26). Previous work by Guschin et al. (25) demonstrated that the xMi-2 complex catalyzes the sliding of nucleosomes toward the center of a 250-bp DNA fragment from the Xenopus thyroid hormone receptor βA gene as detected by translational gel assays. To determine whether xMi-2-induced nucleosome sliding could be observed on another DNA sequence, nucleosomes were reconstituted onto a 215-bp 5S DNA fragment, and the distribution of translational positions was examined on native polyacrylamide gels (44). Prior to xMi2 remodeling, this method revealed at least 5 translational positions (Fig. 2, lane 1). The approximate location of each of these translational positions was determined by cleavage of the nucleosomes with either RsaI, HhaI, or BamHI, before separation on the translational gel (Fig. 2, lanes 2, 3, and 5, respectively). We find that ~70% of the nucleosomes occupy the upstream end of the 215-bp 5S DNA fragment (thick oval, Fig. 2). This result closely agrees with chemical and nuclease mapping of the Xenopus 5S nucleosome (38, 40, 45).

To assess the ability of xMi-2 to catalyze sliding of nucleosomes, glycerol gradient purified nucleosomes were incubated with Mi-2 in the presence or absence of ATP followed by addition of excess calf thymus DNA to promote dissociation of Mi-2 complexes from the nucleosomes. The reactions were then analyzed on translational gels to determine whether remodeling resulted in alterations in the distribution of nucleosome positions (Fig. 3). Incubation of nucleosomes with Mi-2 in the presence of ATP caused most of the major fast-migrating species (the darkest band in Fig. 3, lanes 2 and 3) to shift to a slower migrating species (compare Fig. 3, lanes 3 and 4). This indicates that xMi-2 remodeling results in the accumulation of nucleosomes positioned near the center of the DNA fragment, in agreement with the previous study (25). Interestingly, in contrast to nucleosomes remodeled by the Mi-2 complex, remodeling with the hSWI/SNF complex alters the original distribution of translational positions to faster migrating species (Fig. 3, lane 5). This is in agreement with previous work (27, 31) showing the propensity of yeast and human SWI/SNF to catalyze nucleosome movement toward the edge of DNA fragments.

To determine whether Mi-2 induced nucleosome sliding occurs via a twist-diffusion mechanism, nucleosomes were assembled on templates containing DNA hairpin structures. The hairpins consist of a 12-bp stem and a 5-nucleotide loop placed near the center of the nucleosome positioning sequence within the 5S DNA fragment. The location of the junction was chosen so that the hairpin will extend out away from the histone surface after reconstitution (Fig. 1). If xMi-2-induced nucleosome sliding occurs by a simple twist-diffusion mechanism, then the presence of a DNA hairpin is expected to result in

**Fig. 1. DNA fragments used for nucleosome reconstitution.** The (nicked) hairpin and continuous hairpin substrates contain the same sequences as the 215-bp native 5S fragment. The positions of the hairpins, relevant restriction enzyme sites, and the main translational position of the nucleosome assembled on the native 5S DNA fragment (oval) are indicated relative to the start site of transcription of the 5S gene (+1). Detail of the continuous hairpin sequence is shown at the bottom. nt, nucleotide.

**Fig. 2. Analysis of nucleosome translational positions assembled on the native 215-bp 5S DNA fragment.** Nucleosomes were digested with the indicated restriction enzymes for 15 min and then loaded on a 5% native polyacrylamide gel that allows discrimination of translational positions along the DNA. Lanes 1–3 show nucleosomes labeled at the 5′ end of the bottom strand before and after RsaI and HhaI digestion, respectively. Lanes 4 and 5 show nucleosomes labeled at the 5′ end of the top strand before and after digestion with BamHI, respectively. Approximate translational positions of the nucleosomes corresponding to each of the bands on the gel are shown on the left. The predominant position is indicated by the thick oval.
severe steric clash during rotation of the DNA on the histone surface. Two different types of hairpin structures were used in this study. One contains a nick at the hairpin junction (Fig. 1, hairpin), whereas the other construct does not contain a nick and forms a continuous top strand with a hairpin (Fig. 1, continuous hairpin). The continuous and nicked hairpin substrates were designed to ascertain whether the generation of torsional stress during remodeling, as demonstrated previously (46) for a number of ATP-dependent chromatin remodeling complexes, affects nucleosome sliding efficiency.

To generate a population of uniformly translationally positioned nucleosomes prior to remodeling, nucleosomes assembled on native, hairpin, and continuous hairpin DNAs were subjected to BamHI digestion. BamHI digestion selects for nucleosomes positioned at the 5’ end of the 5S DNA fragment because cleavage of these nucleosomes is inhibited, whereas nucleosomes occupying more downstream positions have BamHI sites exposed and are rapidly cleaved, resulting in the loss of the radiolabel (Fig. 2, lane 5). Purification of the BamHI-selected nucleosomes by glycerol gradient from the free DNA and subnucleosomal species yields a population of homogeneously positioned nucleosomes for subsequent analysis (Fig. 4). The vast majority of these nucleosomes are located at the 5’ end of the 5 S DNA templates (see below).

The BamHI-selected nucleosomes assembled with continuous hairpin and native DNA templates were analyzed by hydroxyl radical footprinting (Fig. 5). Nucleosomes reconstituted on continuous hairpin substrates have rotational positioning that is indistinguishable from those of nucleosomes reconstituted on native DNA (Fig. 5, compare lanes 3 and 4). The characteristic 10-bp hydroxyl radical cleavage patterns for both the hairpin and control nucleosomes align perfectly in the lower part of the gel (from 43 to 23 on the 5S sequence, Fig. 5). However, with the continuous hairpin nucleosome, the hydroxyl radical cleavage pattern is shifted up on the gel by 33 bp, the precise length of the hairpin sequence (Fig. 5). We note a lack of hydroxyl radical cleavage near position –12 in the continuous hairpin nucleosome pattern that is present in native nucleosomes (Fig. 5). This inhibition of hydroxyl radical cleavage is most likely due to the proximity of this position to the hairpin/template junction, as observed in previous studies of branched DNA structures (31, 47). In addition, the hairpin structure itself is susceptible to hydroxyl radical cleavage, as expected (Fig. 5, lane 4), and a similar pattern can also be seen in the hydroxyl radical cleavage of the naked continuous hairpin DNA (Fig. 5, lane 3). The cleavage pattern of the hairpin structure (horizontal bars on the right-hand side of the gel in Fig. 5) shows robust cleavage for most of the stem region of the hairpin, but cleavage is inefficient within the single-stranded region of the hairpin (Fig. 5, compare S1, S2 to H-1, LP, and H-2), as expected (47).

We next determined whether the presence of either of the branched DNAs within the nucleosome inhibited xMi-2-catalyzed sliding by Exo III analysis. Exo III digests double-stranded DNA in a 3’–5’ direction until the progression of the enzyme is impeded by the edge of a nucleosome (29, 48) and has been used to identify the downstream edge of the 5S nucleosome (31). BamHI-selected nucleosomes reconstituted onto both the native and continuous hairpin DNAs yield strong stops at approximately +75, which places the nucleosomes at the upstream end of the DNA fragment, as expected (Fig. 6, A and C, −Mi-2). Exo III digestion of the nicked hairpin nucleosomes resulted in a strong pause sites at approximately +75 and +125 (Fig. 6B, −Mi-2), due to the inability to carry out BamHI selection with this template (see “Materials and Methods”). Exo III also detects a small fraction of nucleosomes with downstream edges at approximately +125 in the continuous hairpin nucleosomes that was not completely removed by BamHI selection process, but this does not affect the analysis (Fig. 6B, −Mi-2). After remodeling by the xMi-2 complex, the distribution of translational positions detected by Exo III digestion is clearly altered. In all three cases, stops near +75 were severely diminished, and several new Exo III pause sites were detected near the downstream end of the DNA (Fig. 6, A–C, +Mi-2), indicating movement of nucleosomes from the upstream edge toward the downstream edge of the DNA fragment upon remodeling by xMi-2. Importantly, Mi-2-induced nucleosome movement does not appear to be inhibited by the presence of the hairpin structures.

We also determined whether the hairpin structures inhibited xMi-2-induced nucleosome sliding by a FokI site protection assay. The FokI site is about 20 bp beyond the edge of main translational position selected by BamHI digestion (Fig. 1), and thus about 90% of the DNA in the native nucleosome sample was rapidly digested before remodeling with Mi-2. However, protection of this site was drastically increased upon Mi-2 remodeling such that only about 45% of the DNA was digested by FokI after 30 min of remodeling (data not shown; see below). This result is consistent with sliding of nucleosomes over the FokI cleavage site upon Mi-2 remodeling (27, 31). Importantly, a similar increase in protection of the FokI site was observed with the continuous hairpin and nicked hairpin nucleosomes after 30 min of Mi-2 remodeling (data not shown; see below).

In order to verify these results and to eliminate the possibility that hairpin-containing nucleosomes undergo Mi-2-induced sliding via an alternative pathway, we examined the rate of sliding as determined by the FokI assay. BamHI-selected nucleosomes reconstituted on native, hairpin, and continuous hairpin substrates were subjected to Mi-2 remodeling for var-
Nucleosome Sliding Does Not Occur Solely by Twist Diffusion

Fig. 4. Translational gel analysis of BamHI-selected nucleosomes. Nucleosomes assembled on native, hairpin, and continuous hairpin DNAs were incubated BamHI for 15 min and purified by glycerol gradient sedimentation, and then translational positions were analyzed on a 5% native polyacrylamide gel as in Fig. 2. A–C show native, hairpin, and continuous hairpin nucleosomes, respectively. The lanes from left to right in each panel contain nucleosomes prior to BamHI digestion, nucleosomes after BamHI cleavage, and BamHI-selected nucleosomes after gradient purification. The filled and open arrows indicate the positions of the naked DNA templates (FD) and the main nucleosome translational position selected by the procedure, respectively.

Fig. 5. Hydroxyl radical footprinting analysis of nucleosomes containing native and continuous hairpin DNA. Nucleosomes were subjected to hydroxyl radical cleavage and then incubated with BamHI before loading onto a preparative nucleoprotein gel to separate the nucleosomes from free DNA. The cleavage patterns of the nucleosomal and naked DNAs were analyzed by sequencing gel electrophoresis and PhosphorImager. Lane 1 shows G-specific reaction markers from the native 5S DNA; lanes 2 and 3 show hydroxyl radical cleavage pattern of native naked and nucleosomal 5S DNA, respectively; lanes 4 and 5 show cleavage pattern of continuous hairpin nucleosome and naked DNA, respectively. Peaks in the nucleosomal cleavage pattern for both templates are indicated according to the standard numbering scheme for the 5S sequence. Cleavage bands corresponding to the hairpin are indicated to the right, with bases designated H for hinge, S for stem, and LP for loop, also shown in the schematic of the top strand of the continuous hairpin DNA (bottom).
in exposing target DNA sites within chromatin for trans-acting factors, thereby facilitating various nuclear processes (11, 13). Many of the ATP-dependent chromatin remodeling complexes have been shown to be able to promote nucleosome sliding along a DNA fragment in vitro, but the mechanism(s) by which sliding occurs are not well understood (17, 25–29).

In a previous study (31), we employed nucleosomes containing branched DNA structures to demonstrate that hSWI/SNF-induced nucleosome sliding does not occur solely via a twist-diffusion mechanism. We wished to assess if these observations could be extended to a different ATP-dependent remodeling complex, the xMi-2 complex, which also has robust nucleosome sliding activity (25). We used a nucleosome with hairpin structures placed near the dyad to determine whether xMi-2-dependent nucleosome sliding occurs by the twist-diffusion mechanism. If nucleosome sliding occurs by the twisting of the DNA helix 1 bp at a time on the surface of the histone octamer, the hairpin structure will sterically hinder any twisting motion of the DNA, greatly inhibiting sliding.

Our results indicate that xMi-2-catalyzed nucleosome sliding occurs to the same extent and at the same rate in the presence of a hairpin structure compared with native nucleosomes using two independent assays. These results indicate that similar to the hSWI/SNF complex, xMi-2-dependent nucleosome sliding does not proceed via a simple twist-diffusion mechanism. Moreover, our results support models in which the DNA maintains its rotational orientation with respect to the histone surface and/or partially dissociates from this surface. Importantly, the rate of nucleosome sliding catalyzed by Mi-2 was found to be identical when comparing the native and hairpin-containing nucleosomes (Fig. 7). This is important because if the primary sliding pathway is blocked by the hairpin structure and the nucleosomes undergo sliding via an alternative pathway, then the rate of sliding is expected to be slower due to the energetic cost of adopting the alternative pathway. However, in the case of xMi-2-induced sliding, this clearly is not the case. This implies that sliding of nucleosomes reconstituted on DNAs with hairpin structures occurred via a pathway identical to that of native nucleosomes. Thus it is highly unlikely that sliding induced by both the Mi-2 complex and the hSWI/SNF complex involves a simple twist-diffusion mechanism.

The actual mechanism of xMi-2-dependent nucleosome sliding may be very complicated, involving aspects of twist diffusion.
Nucleosome Sliding Does Not Occur Solely by Twist Diffusion

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