The Affinity of Nuclear Factor 1 for Its DNA Site Is Drastically Reduced by Nucleosome Organization Irrespective of Its Rotational or Translational Position*

(Received for publication, September 12, 1995, and in revised form, October 24, 1995)

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A DNA-bending sequence has been used for in vitro reconstitution of nucleosomes in order to direct a nuclear factor 1 (NF-1) binding site into different nucleosome positions. By this strategy nucleosomes were obtained that had one of two rotational positions of the NF-1 binding site, one oriented toward the periphery and the other toward the histone octamer, translationaly positioned 50 and 45 base pairs, respectively, from the nucleosome dyad. The affinity of partially purified NF-1 for these nucleosomal targets was compared with its affinity for free DNA by dimethylsulfate methylation protection and DNase I footprinting assays. The binding affinity of NF-1 to all nucleosomal targets was reduced 100–300-fold compared with its affinity for free DNA. The two rotational settings of the NF-1 site showed the same binding affinity for NF-1 as did other nucleosome constructs in which the NF-1 binding site was translationally positioned 50 and 45 base pairs from the nucleosome dyad. We conclude that the nucleosomal inhibition of NF-1 binding is an inherent characteristic of NF-1 since another transcription factor, the glucocorticoid receptor, is able to bind to its DNA site in a nucleosome.

The nuclear factor 1 (NF-1) family of DNA binding proteins can act both as transcription factors for RNA polymerase II genes (1) and as initiation factors for viral DNA replication in adenovirus-infected cells (2). NF-1 family members act as transcriptional activators in regulatory regions of several genes of both viral and cellular origin, such as the mouse mammary tumor virus (MMTV) promoter (1, 3), the human papilloma virus type 16 enhancer (4), the adipocyte-specific P2 enhancer (5), the Xenopus laevis vitellogenin B1 gene (6), the proenkephalin gene of human brain (7), the liver-specific serum albumin enhancer (8), and the CYP1A1 gene (9).

The NF-1 protein family binds DNA as dimers, and their binding sites are homologous to the partially palindromic sequence TTGGC(N)5GCCAA (10, 11). Several different forms of NF-1 are found in various differentiated cell types (12). They originate from the expression of four different NF-1 genes (13), different splicing variants (13, 14) and covalent modifications such as glycosylation (15) and phosphorylation (16). The NF-1 dimers are stable and seem to be formed cotranslationally, and different NF-1 protein variants are able to heterodimerize (17). However, it is not known whether heterodimers occur in vivo. The various NF-1 proteins often mediate transcriptional activation but may also participate in the repression of genes in certain cellular contexts (18, 19). The N-terminal domain, sufficient for DNA binding and dimerization, of all NF-1 proteins is highly conserved and contains four cysteine residues that are strictly required for DNA binding (20). This DNA-binding domain is not homologous to any of the other known classes of DNA-binding motifs such as the zinc fingers, leucine zippers, or helix-turn-helix motifs (11, 21).

NF-1 binding sites are often positioned close to the binding site of other transcription factors in the regulatory region of many genes. In the retroviral MMTV promoter, an NF-1 binding site is localized in the −76/−60 DNA segment (1, 3), next to the −185/−79 DNA segment that contains four binding sites for the glucocorticoid receptor (GR) (22). Transcription from the MMTV promoter is controlled by glucocorticoid hormone (23), but the glucocorticoid-induced transcriptional response is reduced about 5-fold by a mutation in the NF-1 binding site (3). This hormone-dependent NF-1 enhancement might be explained by the observation that the NF-1 protein is not bound to its binding site in the silent MMTV promoter and that NF-1 binding depends on hormone activation of the promoter (24). This suggests that the constitutive DNA-binding protein NF-1, which is located in the cell nucleus, is prevented from binding to DNA by the specific chromatin organization in the MMTV promoter. In line with this hypothesis, chromatin remodelling of the MMTV promoter was shown to occur within minutes of hormone stimulation (25) and to involve the rearrangement of a positioned nucleosome covering the −250/−60 DNA segment (26), where GR and NF-1 bind. Further studies of the chromatin structure of the MMTV promoter in different cell lines and at different hormonal states have confirmed a strong correlation between NF-1 promoter occupancy and the degree of chromatin opening, the latter as measured by in situ restriction enzyme access (27, 28).

In vitro studies involving the reconstitution of the GR and NF-1 binding DNA segment of the MMTV promoter into a nucleosome demonstrated that the DNA was rotationally positioned into a preferred rotational frame on the histone octamer surface. These studies showed that purified glucocorticoid receptor protein could bind to its target sequences in the nucleosome (29–31) while NF-1 was unable to bind to its nucleosomal target (30, 31). In these studies the NF-1 binding DNA segment was rotationally positioned such that its two consecutive major grooves containing the recognition sequence for NF-1 faced the histone octamer (30). Thus, there remain two explanations for the absence of NF-1 binding in the nucleosonally reconstituted...
MMTV promoter (32); (i) the NF-1 protein is incapable of binding to its site in nucleosomal DNA irrespective of the nucleosomal positioning, or (ii) the inability of the NF-1 protein to bind is due to the particular position of the NF-1 site on the histone octamer in the MMTV promoter (see above). These two explanations have implications for how chromatin organization may restrict access of certain transcription factors. This restriction in access may in turn influence how gene induction is triggered by an inducible transcription factor such as GR and a constitutive DNA binding transcription factor such as NF-1. Here we address this issue by use of in vitro reconstituted nucleosomes in which a single NF-1 binding site is held in various well defined rotational and translational positions relative to the histone octamer. This positioning is achieved by placing the NF-1 binding site within a segment of DNA-bending sequence (33).

Our results show that the NF-1 protein binds with at least a 100-fold lower affinity to a nucleosomal DNA site than to a corresponding site on free DNA. The rotational and translational positioning of the binding site on the nucleosome had no effect on the affinity of NF-1 for DNA. This is in contrast to GR, which binds to a nucleosomal glucocorticoid response element (GRE) with a high affinity if held in certain translational (34) and rotational (35) positions. Thus, the lack of NF-1 binding to a nucleosome is an intrinsic property of the NF-1-DNA complex. These findings suggest how the organization of DNA into nucleosomes can be exploited in the cell to create DNA binding hierarchies for various classes of transcription factors.

MATERIALS AND METHODS

DNA Constructs—The construction of various plasmids containing a single transcription factor binding site within repeats of DNA-bending sequences has been described previously (34). Briefly, the plasmid pNo4 was constructed by consecutive cloning of a 30-bp DNA segment containing an NF-1 binding site, followed by four 20-bp DNA segments consisting of a DNA-bending sequence (referred to as the TG motif (33)) into the asymmetric Aval site of pGem-Q2 (34). Another construct, pNi4, differed from pNo4 in that the NF-1 binding site was moved 5 bp relative to the periodicity of the DNA-bending sequence (Fig. 1). No and Ni oligonucleotides contain the NF-1 site from the mouse mammary tumor virus promoter, at positions 77 to 63 (3), and they contain 15 bp of DNA-bending sequence (33). We generated plasmids containing a 161-bp EcoRI/HindIII DNA insert, which was used for nucleosome reconstitution. This insert contained 157 bp of double-stranded DNA and four nucleotides of 5'-protruding single-stranded DNA at each end. The first nucleotide in the top strand, the EcoRI site, was given number 1.2

Nucleosome Reconstitution—DNA labeling and fragment isolation were carried out as previously (34). Both nucleosome reconstitution with salt dilution and the following purification by glycerol gradient centrifugation were performed as described previously (29) with one modification, namely the final NaCl concentration after salt dilution was 0.13 M.

Nucleosome Protection and NF-1 Binding—Exonuclease III, DNase I footprinting, and dimethylsulfate methylation protection were performed as described (Refs. 34, 29, and 35, respectively). Quantification of NF-1 binding was done with PhosphorImager™ analysis and ImageQuant™ software version 3.3 (Molecular Dynamics).

Preparation of NF-1 Protein—Recombinant NF-1 protein was isolated from approximately 10⁸ HeLa cells grown in roller flasks and infected with vaccinia virus that contained a full-length clone for NF-1 (36) with six histidines fused to the N terminus (constructed and kindly provided by Drs. Jacky Schmitt and Hendrik Stunnenberg at EMBL, Heidelberg). The cells were harvested in phosphate-buffered saline (PBS) containing 5 mM MgCl₂, 1 mM phenylmethylsulfonyl fluoride, 5 mM β-mercaptoethanol, 0.5 mM dithiothreitol, and 0.2 mM EDTA. The supernatant was applied onto a 50-ml Sephacryl S-500 column (Pharmacia) and was eluted with buffer D containing 0.15 M KCl, pH 7.9. The protein concentration in this preparation was less than 1 µg/ml.

Results and Discussion

Rotational and Translational Positioning of an NF-1 Binding Site in Mononucleosomes Reconstituted in Vitro—Based on previous experience (34) we constructed DNA fragments that were 161 bp long and contained a single NF-1 binding site. The NF-1 site was placed within a synthetic DNA-bending sequence having a 10-bp periodicity of (A/T)_7(N/G/C)_7(N), referred to as the TG motif (33) (Fig. 1). This DNA sequence is known to direct rotational setting of DNA on a histone octamer such that A/T segments are located at sites of minor groove compression and G/C segments at sites of major groove compression (33). We used this bending sequence to direct the rotational positioning of the 15-bp NF-1 binding DNA segment into two opposite rotational frames: No4, with the two consecutive major grooves
of the two NF-1 half-sites facing toward the periphery of the nucleosome, and Ni4, with the two major grooves of the two half-sites facing toward the histone octamer protein surface (compare No and Ni in Fig. 1A). These DNA fragments contain the NF-1 binding sites (15 bp), 95 bp of TG-bending sequence, and 51 bp of flanking vector sequence (Fig. 1B).

The rotational positioning of the NF-1 site in each reconstituted nucleosome was determined by DNase I footprinting. DNase I is known to cleave the DNA in the minor groove with higher efficiency where DNA is bent away from the enzyme cleavage site. Rotationally positioned nucleosomal DNA will thus generate a 10-bp DNase I ladder (40, 41). When we compared nucleosomal No4 and Ni4 DNA with their free counterparts there was a 10-bp periodicity of DNase I cutting with intervening segments of protection in the nucleosomal DNA (Fig. 2A). The staggering of DNase I cutting between the two strands was 2–4 bp, as expected for DNA that is wrapped around a histone octamer (42). Both No4 and Ni4 gave the same histone-induced 10-bp periodicity, indicating that they adopt the predicted rotational position: the major grooves of the NF-1 half-sites in No4 face the periphery, while the major grooves of NF-1 half sites in Ni4 face the histone octamer surface (Fig. 2B). The only differences between the DNase I cutting pattern of nucleosomal No4 and that of nucleosomal Ni4 were seen in the region containing the NF-1 binding site. These differences are due to different DNA sequences being located in the minor groove facing the periphery.

The translational positions of the NF-1 sites in the two mononucleosomes were determined by exonuclease III protection analysis. Nucleosomal No4 and Ni4 gave similar exonuclease III patterns, showing that they adopt the same translational position (Fig. 3A). Full-length probe (position 161) was partially protected from exonuclease III digestion of the top strand in reconstituted nucleosomes for both No4 and Ni4. The first protected base on the bottom strand of nucleosomal No4 was at position 18, giving a 144-bp segment of protected DNA, as expected for nucleosomal DNA located in the strongly preferred translational position. The additional exonuclease III protections are caused by the previously described capacity of
exonuclease III to digest DNA within a nucleosome, which gives rise to the characteristic 10-bp ladder (43) seen in Fig. 3A. In nucleosomal Ni4 the first stop in the bottom strand occurred at position 19. We conclude that nucleosomal No4 and Ni4 adopt the same rotational and translational position of the DNA-bending segment. The dyads of the NF-1 sites are positioned 50 and 45 bp from the nucleosome dyads in No4 and Ni4, respectively (Fig. 3B), in opposite rotational settings (Fig. 2B).

Low Affinity of NF-1 for Its Nucleosomal Binding Site—Binding of NF-1 to DNA was measured by its ability to protect the N-7 position of two guanosines from methylation by dimethylsulfate. The binding affinity of NF-1 for an NF-1 site on free DNA was compared with its nucleosomal counterpart in both rotational orientations, No4 and Ni4. NF-1 protects a double G located 4 bp from the dyad on both strands of the NF-1 site from methylation by dimethylsulfate (10) (Fig. 1A). Considerably higher concentrations of NF-1 protein were required to give partial protection from methylation for nucleosomal No4 and Ni4 than were required for free No4 and Ni4 (Fig. 4A).

Only the bottom strand analysis is shown, but the result was the same for the top strand. Quantification of the dimethylsulfate methylation protection by PhosphorImager analysis (Fig. 4B) showed that a 100-fold higher NF-1 concentration was required to obtain 50% protection of the nucleosomal NF-1 site than was required for same site located in free DNA. NF-1 protects nucleosomal No4 and Ni4 to the same extent (Fig. 4B, compare nNo4 to nNi4).

DNase I footprinting was also used to assay binding of NF-1 to free and nucleosomal DNA. A distinct footprint of NF-1 bound to free DNA was seen both for No4 and Ni4 (Fig. 5A, lanes 2–5 and 11–14). NF-1 bound to the corresponding nucleosomal DNA also gave protections over the NF-1 binding site both in No4 (Fig. 5A, lanes 7–9) and Ni4 (Fig. 5A, lanes 16–18). Quantification by PhosphorImager analysis of the NF-1-dependent DNase I protection showed that an approximately 300-fold higher concentration of NF-1 was required to obtain...
50% protection of a nucleosomal NF-1 site than was required to obtain 50% protection of a binding site located on free DNA. In agreement with the dimethylsulfate methylation protection data, there was no difference in NF-1 binding affinity between nucleosomal N04 and N14 (Fig. 5B, compare nN04 with nN14). DNase I footprinting analyses of nucleosomal DNA also showed that NF-1 induced protection and hypersensitivity outside of the NF-1 binding region. This is probably caused by nonspecific interactions between protein and DNA since it also occurred in control experiments using nucleosomal DNA lacking an NF-1 site.

The results described above were obtained using recombinant NF-1 protein expressed in vaccinia virus. A series of similar binding experiments were also performed using NF-1 protein that was partially purified from rat liver nuclear extracts. These extracts showed the same relative difference in affinity between a nucleosomal NF-1 site and a free NF-1 site (data not shown).

Effect of Translational Position on NF-1 Binding to Nucleosomal DNA—The dyad of the NF-1 site in N04 is located 50 bp from the dyad of the nucleosome. We investigated whether moving the NF-1 site relative to the nucleosome dyad would have any influence on NF-1 affinity for nucleosomal DNA. An additional four DNA segments were constructed and reconstituted in vitro into nucleosomes. The position of the NF-1 site and the affinity of NF-1 for the site were measured as described above. These constructs and their nucleosomal organization are shown in Fig. 6: 1N03, where the dyad of the NF-1 site was located 30 bp from the nucleosome dyad; 2N02, where the dyad of the NF-1 site was located 10 bp from the nucleosome dyad; 3N01, where the dyad of the NF-1 site was located 20 bp from the nucleosome dyad; and 4N0, where the NF-1 site was located either 40 or 30 bp from the nucleosome dyad (two alternative translational positions with the same rotational position). In all of these constructs the same extent of nucleosome-induced inhibition of NF-1 binding was observed as was observed for N04 and N14 (data not shown).

The percentage of control as a function of the logarithmic concentration of NF-1 protein in units. Each point in the diagram is the mean of three experiments. Symbols are as in Fig. 4B.
DISCUSSION

We have shown for the first time that NF-1 cannot bind to its binding site within a nucleosome irrespective of the rotational and translational position of the binding site. The inability of NF-1 to bind to nucleosomal DNA is characteristic of NF-1. GR is perfectly capable of binding to a single GRE in the same nucleosome context as we have used in this study (34, 35). The GR binding affinity to a nucleosomal GRE is often 2-3-fold lower than its free counterpart. There are, however, certain nucleosomal GRE positions where GR cannot bind. These positions are well defined, and the effects depend on the topology. For example, a GRE at the nucleosome dyad has high affinity for GR when oriented toward the periphery but becomes inaccessible when moved 5 bp relative to the TG motif (35). In the latter position, the major groove is rotated into the opposite direction, i.e. oriented toward the histone octamer. The inhibition of NF-1 binding to a nucleosomal binding site is in sharp contrast to GR binding to a nucleosomal GRE.

Our binding data do not allow us to exclude the possibility that the nucleosome structure is locally perturbed during the NF-1 binding reaction. Thus it remains to be shown whether NF-1 is at all able to form a specific ternary complex with an intact nucleosome. Experiments that examine the protection from dimethylsulfoxide methylation have the advantage of detecting close protein-DNA contacts, and are thus highly specific. However, these experiments do not allow insight into the nucleosomal structure of the bound complex since dimethylsulfoxide methylation is unaffected by histone-DNA interaction. Conversely, the DNase I footprinting experiments reveal the typical 10-bp DNase I ladder, which is induced by the nucleosome structure, which should allow the parallel evaluation of specific NF-1 binding and nucleosome structure. However, the high concentration of NF-1 protein, which is required for DNase I protection in a nucleosomal NF-1 site also affects DNase I digestion outside of the NF-1 binding site. This is probably due to nonspecific binding of NF-1 to DNA and possibly the binding of other contaminating proteins present in our partially purified NF-1 preparations. This results in a progressively increased DNase I protection at the NF-1 site and a concomitant fading of the nucleosome-induced DNase I pattern. Thus it is not possible to determine whether the nucleosomes that generate the 10-bp ladder also contain one specifically bound NF-1 protein dimer. Nevertheless, we can still conclude that NF-1 has an at least 100-fold lower affinity for a nucleosomal NF-1 site than it has for an NF-1 site in free DNA. If the DNA organization in the nucleosome must be perturbed for NF-1 binding to occur, then the stability of each nucleosome, as determined by its DNA sequence, would be expected to affect NF-1 binding affinity.

Although translational nucleosome positioning has been described in several promoters (for a review see Ref. 41), little is known about the precision of this positioning at the base pair level (44). Our finding that the NF-1 protein cannot bind to a nucleosomal target 10-50 bp from the dyad shows that a precise nucleosome arrangement is not needed for the binding inhibition to occur. However, our experiments do not determine where the inhibition occurs relative to the nucleosome border. Attempts to position the NF-1 site further than 50 bp away from the nucleosome dyad failed due to problems with variability in translational position of the histone octamer caused by use of longer TG motif DNA.

Cordingley et al. (24) have observed that NF-1 occupancy of the MMTV promoter in vivo depends on the glucocorticoid hormone. This dependence can be simply explained by our results, which show that nucleosomal DNA is selectively inaccessible for NF-1 but not for GR (34, 35). The modes of DNA binding of NF-1 and GR relative to histone/DNA arrangements in a nucleosome may explain the difference in selectivity. GR is known to form specific DNA contacts with two consecutive major grooves on the same side along the DNA axis (45-47), apparently without any effect on the DNA structure (47). The NF-1 protein also forms specific DNA contacts with two consecutive major grooves along one side of the DNA length axis (10). However, in contrast with GR, NF-1 binding to DNA has been reported to enhance a preformed bend in the DNA segment that flanks its binding site in the adenovirus terminal repeat (48). Whether it is the NF-1-induced DNA structure, steric hindrance of the NF-1 protein as such, or some other feature of the NF-1-DNA complex that inhibits NF-1-nucleosome interaction remains to be investigated. We note that the TATA box binding protein is unable to bind DNA in a nucleosome (49). In that case the reason may be the requirement of TATA box binding protein to form a sharp kink in DNA upon binding (50).

Nucleosomal Inhibition of NF-1 Binding

Since NF-1 binding sites occur in many viral and cellular regulatory DNA segments, it is likely that the inhibition of NF-1 binding that is induced by the nucleosome is of functional significance also in other inducible promoters. One such case might be the CYP1A1 gene, where an Ah receptor, which binds dioxin, induces chromatin opening and NF-1 binding (9). As described previously for the MMTV promoter (26), the uninduced CYP1A1 promoter also contains positioned nucleosomes, some of which are perturbed by the dioxin-induced promoter activation (51). Another example is the liver-specific serum albumin enhancer, which contains three specifically positioned nucleosomes in liver cells where it is active (8). One of the three nucleosomes is structurally perturbed and hypersensitive to DNase I. This perturbed nucleosome contains an NF-1 site and binding sites for liver-specific factors such as HNF3. The albumin enhancer is only occupied by NF-1 in liver cells where the enhancer is active, even though NF-1 is present in many tissues. McPherson et al. (8) suggest that liver-specific factor(s) might induce the chromatin structure required for NF-1 to gain access to its binding site.

A role of chromatin in gene regulation has been demonstrated in yeast cells by genetic interruption of histone H4 synthesis. This resulted in derepression of several previously silent genes (52). Likewise, titration of the histone pool in Xenopus oocytes by injection of competitive DNA resulted in a 10-fold decrease in nucleosome density of injected DNA. A parallel transcriptional activation of the MMTV promoter occurred independent of the glucocorticoid receptor (53). These results suggest that one function of chromatin is to keep constitutively DNA binding transcription factors, such as NF-1 and TATA box binding protein, away from their targets in promoters that should be kept silent. Such a chromatin-dependent effect might be important for two reasons: (i) to avoid transcriptional leakage in promoters that should be inactive, and (ii) to reduce protein-DNA interaction in general and thereby direct the constitutive DNA binding transcription factors to the active promoters. This repressive function of chromatin suggests that induction of a previously silent promoter would require another class of transcription factors. This other class of factors would, in contrast to NF-1, possess the capacity to bind nucleosomal targets and to mediate chromatin remodelling. The remodelling would allow entry of the constitutively DNA binding factors. In this model, a gene that is reversibly regulated between a silent and an active state requires that the nucleosome binding activity of the inducing transcription factor be controllable, for example by ligand binding. This control mechanism has been seen for the GR and the Ah receptor.
Acknowledgments—We thank Ulla Björk for skilful technical assistance. We are grateful to Drs. Jacky Schmitt and Hendrik Stunnenberg (EMBL, Heidelberg) for kindly providing recombinant vaccinia strains. We are indebted to Kristina Nordström and Dr. Björn Vennström for the help and facilities for growing and harvesting HeLa cells infected with vaccinia virus.

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J. Biol. Chem. 1996, 271:153-159.
doi: 10.1074/jbc.271.1.153

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