The Role of the Minor Groove Substituents in Indirect Readout of DNA Sequence by 434 Repressor*

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The sequence of non-contacted bases at the center of the 434 repressor binding site affects the strength of the repressor-DNA complex by influencing the structure and flexibility of DNA (Koudelka, G. B., and Carlson, P. (1992) Nature 355, 89–91). We synthesized 434 repressor binding sites that differ in their central sequence base composition to test the importance of minor groove substituents and/or the number of base pair hydrogen bonds between these base pairs on DNA structure and strength of the repressor-DNA complex. We show here that the number of base pair H-bonds between the central bases apparently has no role in determining the relative affinity of a DNA site for repressor. Instead we find that the affinity of DNA for repressor depends on the absence or presence of the N2-NH2 group on the purine bases at the binding site center. The N2-NH2 group on bases at the center of the 434 binding site appears to destabilize 434 repressor-DNA complexes by decreasing the intimacy of the specific repressor-DNA contacts, while increasing the reliance on protein contacts to the DNA phosphate backbone. Thus, the presence of an N2-NH2 group on the purines at the center of a binding site globally alters the precise conformation of the protein-DNA interface.

It is well established that the sequence-specific binding of proteins to DNA involves specific contacts between DNA bases and protein side chains in a process known as “direct-readout.” The precise alignment of DNA and protein can be specifically modulated by the sequence of DNA bases in the binding site that are not directly contacted by the protein. This phenomenon is known as indirect readout. In indirect readout, the affinity or specificity of a protein-DNA complex depends on sequence-dependent alterations in the conformation and/or conformational flexibility of the noncontacted bases in the DNA site.

Since sequence-dependent DNA structural differences play a role in mediating protein-DNA complex formation, this implies that a protein binds only to a distorted DNA conformation. Noncontacted bases may be envisioned to affect the formation of a protein-DNA complex in any of three ways: 1) altering the deformability of DNA; 2) altering the structure of the DNA in the bound complex, thereby changing the strength of particular protein-DNA contacts; or 3) altering the structure of the unbound DNA, thereby eliminating or requiring the imposition of energetically costly large-scale DNA conformational changes. Although indirect readout is part of the sequence recognition mechanism of many sequence-specific DNA binding proteins, little is known about the physical basis of how base sequence and/or the functional groups on the bases contribute to the sequence dependence of such DNA deformations. In addition, the mechanisms by which sequence-dependent differences in DNA conformation influence the strength or specificity of protein-DNA complexes is also unclear.

434 repressor does not contact the functional groups on bases at the center of its binding site (1, 2; see also Fig. 1). Nonetheless, changing the sequence of these bases remarkably influences the affinity of DNA for repressor (3). Biochemical and crystallographic studies show that, in complex with protein, the noncontacted bases at the center of the 434 binding site are overwound and the minor groove in this region of the binding site is narrower than in canonical B-DNA (1, 3–6). Binding sites with higher intrinsic twists bind 434 repressor with higher affinity than do those with lower twist (5, 6). Also, sites that bear central sequences or modifications that increase twisting flexibility bind repressor with higher affinity than those that do not (3, 7). Hence, the sequence of the noncontacted central bases in the 434 binding site affects its affinity for repressor by regulating both the structure of the unbound binding site and the ease with which the site adopts the overtwisted conformation necessary to bind repressor.

Despite knowing how sequence-dependent differences in DNA twist influence the affinity of a binding site for repressor, we still do not precisely know how sequence determines DNA twist and/or twisting flexibility. Two observations focus our attention on the N2-NH2 group of the purine bases present at the center of the 434 repressor binding site (positions 6–9). First, introducing this group at the center of the binding site by changing the noncontacted central base sequences from one that is A/T-rich to one that is G/C-rich decreases the affinity of the DNA for repressor by 60-fold. Second, binding sites bearing I/C base pairs at the central positions bind repressor as well as do binding sites bearing A/T-rich sequences at their centers. Inosine is identical to guanine, except that it lacks the N2-NH2 group on the minor groove surface of the base and thereby resembles A/T base pairs in the minor groove. Hence, the presence of the N2-NH2 group appears to decrease the affinity of a binding site for 434 repressor.

How might the N2-NH2 group exert this deleterious effect on affinity? Here we test two ideas. First, since purine bases bearing an N2-NH2 group form a third base pair hydrogen bond between the base it is possible that this “extra” hydrogen bond renders binding sites bearing G/C base pairs at their center less readily deformable than those containing A/T (or I/C) pairs at these positions. Alternatively, the bulky N2 amino group of guanine may sterically or electrostatically oppose the repressor-induced DNA overwinding of this region of the DNA.

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Circular Dichroism—All circular dichroism (CD) measurements were performed using a Jasco J715 spectropolarimeter (Pharmaceutical Sciences Instrumentation Facility, University at Buffalo). Data were acquired from 220 to 10 nm at 0.5-nm intervals, scanned at 20 nm/min. The final spectrum represents an average of three separate scans. CD measurements for each sample were performed at 2 or 50 mK concentrations to ensure that CD features were not derived from oligomerization of the DNA.

Hydroxyl Radical Cleavage—Hydroxyl radical cleavage experiments were performed essentially as described previously (10). Cleavage of DNA in the absence of repressor was performed to give, on average, one cleavage per DNA molecule. Following cleavage, the reaction product was phenol/chloroform-extracted and fractionated on 12% denaturing DNA in the absence of repressor was performed to give, on average, one

FIG. 1. Schematic representation of the N-terminal DNA binding domain of 434 repressor in complex with its binding site (1). Indicated are the positions of the bases that are or are not directly contacted by 434 repressor. Base numbering is from the 5’ end of the consensus 14 base pair-long binding site. The bases indicated by X7 and Y8 correspond to positions substituted by noncanonical bases as described in Fig. 2.

forms a base pair hydrogen bond with the carbonyl O2 on the opposing pyrimidine base. The third base pair hydrogen bond contributed by the N2-NH2 group on purines may decrease the affinity of repressor for DNA by resisting the repressor-induced change in propeller twisting (3). Alternatively, the N2-NH2 group on a purine at the center of the binding site may decrease the affinity of repressor for DNA by sterically (3) or electrostatically (11) hindering overwinding or narrowing of the minor groove.

If the N2-NH2 group influences repressor binding by resisting propeller twist, we anticipate that the affinity of repressor for DNA would decrease with an increase in the number of hydrogen bonds between the bases at the center of the binding site. However, there is no correlation between the number of base pair hydrogen bonds between the bases at the center of the binding site and the affinity of repressor for DNA (Fig. 2). Instead, repressor has lower affinity for binding sites containing an N2-NH2 group on the bases at positions 7 and 8 than it does for sites that lack this functional group. For example, despite having one more hydrogen bond between its central bases than does T7/AP8, the T7/AP8 site binds repressor with a higher affinity than does T7/AP8. This effect is seen independent of the number of base pair hydrogen bonds between the bases at the center of the binding site. For example, the AP7/C8 site contains an N2-NH2 group at position 7 and the central base pairs are joined by a single H-bond, whereas the I7-C8 site lacks the N2-NH2 group at position 7 and the I7-C8 base pair is joined by two H-bonds. Nonetheless, repressor binds to the AP7/C8 with a lower affinity than does T7/AP8. This effect is seen independent of the number of base pair hydrogen bonds between the bases at the center of the binding site. For example, the AP7/C8 site contains an N2-NH2 group at position 7 and the central base pairs are joined by a single H-bond, whereas the I7-C8 site lacks the N2-NH2 group at position 7 and the I7-C8 base pair is joined by two H-bonds. Nonetheless, repressor binds to the AP7/C8 with a lower affinity than does the I7-C8 site. Repressor does not closely approach the N2 position of the bases at positions 7 and 8 (1, 3). Thus, the presence of the N2-exocyclic NH2 group appears to interfere

The abbreviations used are: CD, circular dichroism; DAP, diaminopurine; AP, amino purine.

DNA Synthesis and Preparation—All oligonucleotides used in the studies were purchased from the AMBRI Nucleic Acid Facility (University at Buffalo). The binding sites were synthesized as a self-complementary single DNA strand 50 bases long (8). These single-stranded DNAs were purified from denaturing gels and resuspended in buffer (25 mM NaP04, pH 6.8, 25 mM NaCl). All purified DNAs were heated to 70 °C and allowed to self-annel by cooling slowly to room temperature.

Filter Binding Assays—Approximately 0.5 μg of each hairpin was 5’ end-labeled by incubating the DNA with 20 μCi of [γ-32P]ATP in the presence of T4 polynucleotide kinase (Invitrogen) for 30 min at 37 °C in a buffer containing 50 mM Tris, pH 8.0, and 10 mM MgCl2. The labeled DNA was ethanol-precipitated and used in filter-binding experiments as previously described (9). Values of the dissociation constant (Kd) were determined by nonlinear squares fitting of the filter binding data using Prism 3.0 software (GraphPad Software Inc.). Each dissociation constant was determined from at least eight replicate measurements.

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RESULTS AND DISCUSSION

Affinity of Synthetic 434 Repressor Binding Sites for 434 Repressor Depends on Base Substitutions at Positions 7 and 8—434 repressor binding sites that differ in their central sequence base composition were synthesized to test the importance of minor groove substituents (the N2-NH2 group) and/or hydrogen bonding in binding affinity to repressor (see Fig. 2). In a design that mimics that used in studies of trp repressor hydrogen bonding in binding affinity to repressor (see Fig. 2).

Indicated are the positions of the bases that are or are not directly contacted by 434 repressor. Base numbering is from the 5’ end of the consensus 14 base pair-long binding site. The bases indicated by X7 and Y8 correspond to positions substituted by noncanonical bases as described in Fig. 2.

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with high affinity binding of repressor by opposing repressor-induced changes in DNA twist or minor groove geometry.

Salt Concentration Dependence of the Affinity of 434 Repressor for Position 7 and 8 Variant 434 Binding Sites—To determine how the N2-NH2 group influences the affinity of repressor for DNA, we examined the effect of changing salt concentration on the affinity of repressor for binding sites the do or do not contain an N2-NH2 group on the purines at positions 7 and 8. We analyzed DNA sites that differ only in the absence or presence of the N2-NH2 group at positions 7 (G7-C8 and I7-C8) or 8 (T7-A8 and T7-DAP8).

The affinities of 434 repressor for binding sites that do not have an N2-NH2 group on the purine at positions 7 or 8 are relatively unaffected by salt concentration (Fig. 3). In comparison, the affinities of repressor for sites bearing an N2-NH2 group on the purines at positions 7 and 8 are much more dependent on salt concentration (Fig. 3). The increased salt sensitivity of the affinities of repressor for binding sites bearing an N2-NH2 group at positions 7 and 8 suggests that the stabilities of these repressor-DNA complexes are more dependent on ionic interactions between protein and DNA than are repressor complexes with DNAs that lack this functional group on the central bases.

Extrapolating the salt concentration dependence of affinity data to 1 M KCl allows determination of the salt-insensitive component of the binding affinity (12). This number, in part, reflects the strength of specific protein-DNA contacts. This analysis shows that the strength of the salt-insensitive protein-DNA contacts is decreased between 102 and 103-fold in complexes between repressor and the sites bearing the N2-NH2 group on the bases at their centers, relative to those complexes that lack this functional group on these bases (Fig. 3). Together, these data suggest that the N2-NH2 group on bases at positions 7 and 8 destabilizes 434 repressor-DNA complexes by decreasing the intimacy of the specific repressor-DNA contacts while increasing the reliance on protein contacts to the DNA phosphate backbone.

Probes of 434 Repressor Binding Site Structure—The salt concentration dependence studies imply that the conformation of repressor-DNA complexes vary with the absence or presence of an N2-NH2 group on the purines at positions 7 or 8. We examined whether the N2-NH2 group also affects the conformation of unbound DNAs by CD spectroscopy. The CD spectrum of each sequence is characteristic of a right-handed DNA double helix (Fig. 4, A and B). However, the relative intensities of the positive CD peak at -275 nm and the negative peak at -250 nm are much lower for the binding sites that lack the N2-NH2 group at the central positions than for those that do have this group (Fig. 4, A and B, compare the spectrum of G7-C8 with I7-C8 and T7-A8 with T7-DAP8). The spectra of the heat-denatured DNAs are not affected by the presence or absence of the N2-NH2 group (data not shown). Thus, the base composition-dependent changes in CD intensities are not due to variation in the extinction coefficients between bases that do or do not contain the N2 group. Hence, the differences in CD spectra between the sites that do or do not contain the N2-NH2 group can be attributed to base type-dependent changes in DNA conformation.

In general, lower CD intensity at 275 nm is indicative of higher helical twist (13). Thus, the presence of an N2-NH2 group on the purines at positions 7 and 8 of the 434 binding site decreases the intrinsic twist of these sites, relative to the sites that lack this feature. These findings are consistent with our previous biochemical and NMR results showing that the DNA at the center of the G7-C8 site is underwound and the minor groove in this region is wider than that of the identical region of the T7-A8 site (5). The 434 repressor prefers to bind DNA sites that are overwound as opposed to those that are underwound (6). Since I7-C8 and T7-A8 both exhibit the highest affinity for repressor within their respective sequence contexts, the CD results indicate that the presence or absence of the N2-NH2 group at positions 7 or 8 may affect the DNA affinity of 434 repressor by altering DNA twist.

The salt dependence data (Fig. 3) suggest that the structure of the repressor-DNA complex also varies with the presence or absence of the N2-NH2 group on the central bases. To explore
this possibility, we used ‘OH radial cleavage to examine the effect of the N2-NH2 group on the conformation of the unbound and bound DNAs. ‘OH cleaves DNA by accessing the sugar phosphate backbone through the minor groove and large N2-NH2 group-dependent changes in the geometry of the minor groove of the binding sites would be reflected in changes in cleavage intensity (10). Similarly, repressor-DNA complex formation will protect the DNA from cleavage by making the minor groove less accessible to the ‘OH radical (10). Differences in the ‘OH cleavage patterns of the 434 binding sites in the presence of saturating concentrations of repressor will reflect differences in the conformations of the repressor-DNA complexes.

In the absence of repressor, the intensity of ‘OH radical cleavage at each base in T7-A8 and T7-DAP8 does not differ significantly, regardless of salt concentration (Fig. 5A; note that the apparent effect of salt on cleavage at position 2 is due to a salt effect on electrophoretic mobility of this fragment). Similarly, the ‘OH radical cleavage patterns of I7-C8 and G7-C8 determined at 50 and 150 mM KCl in the absence of repressor are also indistinguishable from each other (Figs. 5B and 6B). These finding suggests that neither salt nor the presence of the N2-NH2 group at positions 7 and 8 in these binding sites significantly affects the width of the minor groove either at the site of substitution or globally throughout the binding site DNA. This conclusion suggests that the N2-NH2 group-dependent twist differences in these DNAs seen by CD spectroscopy (Fig. 4) are not detectable by ‘OH radial or do not translate into large alterations in the minor groove geometry of the unbound DNA. Based on our NMR data (not shown), we favor the former interpretation.

The cleavage pattern of the T7-A8-repressor complex is remarkably different from that of the repressor-T7-DAP8 complex. This N2-NH2 group-dependent difference in cleavage pattern is seen at both 50 and 150 mM KCl (Figs. 5 and 6). Marked differences in the cleavage patterns of the G7-C8 and I7-C8-repressor complex are also seen, regardless of salt concentration. Control experiments show that the N2-NH2 group-dependent changes in ‘OH cleavage pattern are not due to differences in repressor occupancy of the binding sites. Thus, the differences in ‘OH cleavage patterns must reflect N2-NH2 group-dependent differences in the conformations of the repressor-DNA complexes.

N2-NH2 group-dependent changes in ‘OH cleavage pattern are seen throughout the binding site (Figs. 5 and 6), but the most striking differences are seen at the center of the binding site. Specifically, repressor more efficiently protects positions 9 and 10 of the sites that lack the N2-NH2 group at positions 7 or 8 (T7-A8 and T7-DAP8) than it does on sites bearing this functional group at these positions (T7-DAP8 and G7-C8). For example, at 50 mM KCl, repressor binding to T7-A8 reduces the ‘OH cleavage intensity at positions 9 and 10 8-fold and 4-fold, respectively (Fig. 6A). In contrast, under the same conditions, repressor binding to T7-DAP8 reduces the cleavage intensity at these positions by at most 2.5-fold. Even larger differences in protection efficiency occur between the G7-C8 and I7-C8 sites (Figs. 5B and 6B).

In complex with repressor, the minor groove of the bases at positions 9 and 10 faces the repressor (see Fig. 1). The N2-NH2 group on positions 7 and 8 is located in this groove. Repressor makes no direct contacts to the bases in this region of the binding site, interacting only with the DNA backbone (1). The decreased efficiency of protection of the DNA in this region in the repressor-T7-DAP8 and G7-C8 complexes suggests that repressor is less intimately associated with the DNA in this region of the binding site than in the repressor complex with T7-A8 and I7-C8.

The ‘OH cleavage pattern at symmetrically related positions

FIG. 4. Circular dichroism spectra of 2 mM of DNAs bearing modified bases at their central positions. Spectra were acquired at 50 mM KCl, 25 °C (see also “Experimental Procedures”). The spectra of T7-A8 (○) and T7-DAP8 (●) are shown in A. B displays the spectra of I7-C8 (○) and G7-C8 (●).
1–4 and 11–14 also differs in the complexes between repressor and sites that do or do not contain an N2-NH2 group on the central bases. For example, at 50 mKCl repressor protects the bases at positions 11–14 in T7-DAP8 binding to a greater extent than in the T7-A8 site (Fig. 6, A and B). Differences are also seen at positions 1–4 (Fig. 6, A and B). A similar set of N2-NH2 group-dependent differences in repressor-mediated protection of the outer bases is seen between the G7-C8 and T7-C8 sites (Fig. 6, C and D). Since repressor makes contacts to the DNA only from the major groove side of the bases near positions 1–4 and 11–14 (Fig. 1) and since ‘OH gains access to the phosphate backbone through the minor groove, repressor binding must affect the ‘OH reactivity of this region indirectly. Thus, we assert that the differences in cleavage efficiency at positions 1–4 and 11–14 between the repressor-DNA complexes that do or do not contain an N2-NH2 group on the purine bases at position 7 and 8 report an N2-NH2 group-mediated difference in specific protein-DNA contacts. This finding is consistent with the large difference in the strength of the salt-insensitive component of the binding affinity between sites that do or do not contain the N2-NH2 group on the purine bases at positions 7 and 8 (Fig. 3).

Our findings show that placing an N2-NH2 group on the purine bases at positions 7 or 8 globally alters the conformation of the DNA and repressor-DNA complexes. Also, we know that repressor binds with higher affinity to DNAs bearing central sequences that are relatively flexible and/or overwound than it does to those that are “stiffer” and/or relatively underwound (3, 5–7). How might the N2-NH2 group exert its effects on the repressor-DNA complex and/or DNA structure? Here, we consider two models. In a “twist/flex model”, the N2-NH2 group of guanine may directly impact the DNA helical twist and/or twisting flexibility (5) by mechanical occlusion (steric hindrance) (6). According to this idea, the effects/interactions of the N2-NH2 are through space, are not mediated by other species, and are therefore intrinsic to the DNA. An alternative viewpoint advocates that the N2-NH2 alters DNA conformation and flexibility via electrostatic interactions of this group with its environment (11), influencing distributions of protein-based or solution-derived cations within the major and minor groove leading to sequence-dependent variation in groove width, DNA twist, and axial bending (14). According to this model, the N2-NH2 group interactions depend on factors extrinsic to DNA.

Although extrinsic forces do play a role in 434 repressor-DNA interactions,3 evidence suggests that the effect of the N2-NH2 group on the central bases on repressor-DNA complex stability is independent of the electrostatic contributions of this group. First, the results of mutagenesis studies (3) suggest that the partial positive charge on the N2-NH2 group (11) is unimportant to the ability of repressor to recognize the noncontacted base sequence at positions 7 and 8. Second, the presence of a partial positive charge in the minor groove should cause DNA to overwind (15). Since our data indicate the N2-NH2 group containing DNAs are underwound (16) with respect to DNAs lacking this group (Fig. 4 and Ref. 5),2 we favor the idea that N2-NH2 groups sterically oppose repressor-induced DNA distortions needed for stable repressor-DNA complex formation. Structural (17–19), biochemical (20–22), and molecular dynamic studies (23) are consistent with this idea.

Regardless of whether the steric or electrostatic “bulk” of the N2-NH2 group on the central bases of the binding site is responsible for its deleterious effects on repressor-DNA complex stability, our results allow us to develop a picture of how the N2-NH2 group influences strength and conformation of the repressor-DNA complex. In agreement with our earlier results (5), the CD data (Fig. 4) show that DNAs bearing the N2-NH2 group at position 7 and 8 are underwound with respect to sites lacking this group. Thus to form a complex, the repressor must overwind these DNAs. The decreased twisting flexibility of the N2-NH2 group containing DNAs contributes to the lower affinity of repressor for the G7-C8 and T7-DAP8 sites (5, 23). In addition, the presence of an N2-NH2 group on the central bases of the binding site may also inhibit the minor groove compression that normally accompanies repressor binding. Failure to compress the minor groove in this region compromises the

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3 G. Koudelka and S. Mauro, manuscript in preparation.
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