Effect of DNA-binding Drugs on Early Growth Response Factor-1 and TATA Box-binding Protein Complex Formation with the Herpes Simplex Virus Latency Promoter*

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Adjacent binding sites for early growth response factor-1 (EGR1) and TATA box-binding protein (TBP) were identified on the herpes simplex virus latency promoter in previous work. The binding of EGR1 to the GC-rich region prevented TBP binding to the AT-rich region. With the simultaneous addition of both EGR1 and TBP, the intercalator nogalamycin prevented EGR1 complex formation, resulting in a dose-dependent increase of the TBP-DNA complex. The minor groove binder chromomycin A₃ inhibited EGR1 complex formation but resulted in a smaller increase of the TBP complex. In contrast, an alkylating intercalator hedamycin strongly inhibited binding of both proteins. The ability of these GC-binding drugs to prevent EGR1-DNA complex formation was in the following order: hedamycin > nogalamycin > chromomycin A₁₂ and the specificity was nogalamycin > chromomycin A₂ > hedamycin. With transcription factor IIA (TFIIA) in the assay, TBP was able to bind the promoter whereas formation of the EGR1-DNA complex was reduced. An AT minor groove-binding drug, distamycin A, disrupted the TBP-TFIIA-DNA complex and restored the EGR1-DNA complex. We conclude that the binding motif and sequence preference of DNA-interactive drugs are manifested in their ability to inhibit the transcription factor-DNA complexes.

DNA-binding drugs have been studied for their ability to disrupt the activity of DNA-processing enzymes including polymerases and topoisomerases (1–7). Recently, transcription factors (TFs)³ that form TF-DNA complexes have been evaluated as potential targets of DNA-binding drugs. For example, the AT minor groove-binding drug distamycin A inhibited the binding of proteins such as OTF-1, NEF-1, and antennapedia homeodomain to their AT-rich regulatory elements (8, 9). Likewise, the GC minor groove-binding drug mithramycin inhibited Sp1 binding to the GC-rich SV40 early promoter and prevented transcription initiation from the c-myc P1 and P2 promoters (10, 11).

Recently, our laboratory undertook to identify characteristics of DNA-binding drugs that were required for inhibition of TF-DNA complex formation (12, 13). Individual TFs that recognized DNA sequences at AT- or GC-rich sites were used to test the specificity of drugs. A number of drugs were evaluated for their ability to block the association of the general transcription factor TBP to its AT-rich binding site in the DNA minor groove. AT minor groove-binding agents such as distamycin A were very effective at both preventing and disrupting TBP-DNA complexes (12). In a subsequent study, we examined the ability of a wide variety of drugs including intercalators and minor groove-binding agents to interfere with the binding of EGR1, a nuclear phosphoprotein with three zinc fingers binding to the DNA major groove, and a panel of other TFs to their consensus DNA binding sites (13–15). The most potent inhibitors at EGR1-DNA complex formation were nogalamycin, hedamycin, and chromomycin A₂, which shared a preference for the GC-rich binding site of EGR1.

To further understand how a drug might specifically affect TFs binding to their consensus binding sites, it would be useful to evaluate drug inhibition of TFs using a DNA fragment composed of multiple factor-binding sites. Recent studies by Tatarowicz et al.² identified a DNA sequence of 5'-TATAAAAGCGGGGG that contained adjacent regulatory binding sites for EGR1 and TBP, on the herpes simplex virus latency (HSV-L) promoter. Our laboratory wished to examine whether AT- and GC-binding drugs could specifically interfere with either EGR1 or TBP or both when they were bound to adjacent sites on the HSV-L promoter. Based upon their DNA sequence preference and mode of binding (e.g. minor or major groove and intercalation), nogalamycin, hedamycin, chromomycin A₁₂, and distamycin A were chosen for comparative study of their ability to inhibit single or multiple TF-DNA complex formation.

Results presented here confirmed that EGR1 prevented TBP from binding to an adjacent site on the HSV-L promoter, and subsequent data showed that TBP, in the presence of TFIIA, could interfere with EGR1-DNA complex formation. Mobility shift assays examined the effect of AT- and GC-binding drugs on the DNA complex formation of individual TFs (i.e. TBP and EGR1) that recognize either AT- or GC-rich DNA binding sites. Certain DNA-binding drugs selectively interfered with one or the other of these TFs that bound to adjacent sites on the HSV-L promoter.

MATERIALS AND METHODS

Drugs—Chromomycin A₁₂ purchased from Sigma was prepared in dimethyl sulfoxide. Distamycin A from Sigma was made in distilled water. Hedamycin (NCI, National Institutes of Health) was dissolved in

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¹ The abbreviations used are: TF, transcription factor; TFIIA, transcription factor IA; EGR1, early growth response factor-1; HSV-L promoter, herpes simplex virus latency promoter; TBP, TATA box-binding protein; MES, 2-(N-morpholino)ethanesulfonic acid.

² W. A. Tatarowicz, A. S. Pekosz, S. L. Madden, F. J. Rauscher III, S.-Y. Chiang, T. A. Beerman, and N. W. Fraser, manuscript in preparation.
The preparation of proteins, EGR1, TBP, and TFIIA was described previously (12, 13). Briefly, EGR1, a 13.5-kDa truncated form of the full-length protein, was expressed in Escherichia coli as a histidine-tagged fusion protein and purified through a nickel-chelate affinity column. After elution with 6 M guanidine hydrochloride, EGR1 was dialyzed against 25 mM Hepes-KOH, pH 7.5, 100 mM KCl, 10 mM ZnSO4, 5% glycerol, 0.1% Nonidet P-40, and 2 mM dithiothreitol. A similar procedure was used for TBP preparation, in which bacteria were transformed with plasmid pDS56-hTBP, a gift from T. Kerpolla and T. Curran (Roche Institute of Molecular Biology). Expressed TBP fused to six histidine residues at the NH2 terminus of the protein was purified through a nickel column, dialyzed with 25 mM Mops, pH 6.5, 5% glycerol, 1 mM dithiothreitol, and 1 or 0.1 M guanidine hydrochloride and then in the same buffer without guanidine hydrochloride. The TFIIA was a gift from M. Schmidt (University of Pittsburgh Medical School). The purity of proteins determined by SDS-polyacrylamide gel electrophoresis/Coomasie Blue staining was greater than 90%. The quantity of proteins was measured by using Bio-Rad protein assay.

Mobility Shift Assays—Gel mobility shift assays were utilized to measure the ability of proteins to bind to DNA that contained the protein consensus binding sites. Based upon previous studies, 3 ng of EGR1 and 5 ng of TBP were used in assays (12, 13). The equilibrium binding of either protein to the HSVL oligonucleotides was performed at 30°C. Formation of the protein-DNA complex was examined at intervals of 1–120 min. The results showed that protein-DNA complex reached equilibrium by 5–10 min after incubation at 30°C and stayed consistent for 30 or 120 min for TBP and EGR1-DNA complexes, respectively. Moreover, for optimizing assay conditions, radioisotope-labeled probe was titrated in the presence of constant amounts of EGR1 or TBP to maximize DNA-protein complex formation. Experimentally, both 3 ng of EGR1 and 5 ng of TBP prepared in the binding buffer (20 mM Hepes-KOH, pH 7.9, 25 mM KCl, 2 mM MgCl2, 0.1 mM EDTA, 100 μg/ml bovine serum albumin, 0.5 mM dithiothreitol, 0.8 mM spermidine, 10% glycerol, and 0.025% Nonidet P-40) were incubated at 30°C for 30 min with labeled probe (DNA containing the consensus binding sites for both TFs at the final concentration of 0.5–1.0 nM). Samples were electrophoresed in a 4% native polyacrylamide gel at room temperature with a running buffer of 45 mM Tris-base, 45 mM boric acid, and 1 mM EDTA. Autoradiography was performed by exposing dried gels to Kodak film, and results were quantitated by a computing laser densitometer (Molecular Dynamics, Sunnyvale, CA). In the competition test, EGR1 and TBP were preincubated with 8 nM unlabeled DNA (HSV L oligonucleotides) at 30°C for 30 min, prior to adding the radiolabeled probe. With TFIIA, a modified assay condition was designed, in which a mixture of 3 ng of TBP and 0.2 μg of TFIIA was first incubated with 1 nM radiolabeled probe. After incubation at 30°C for 30 min, 0.3 ng of EGR1 was added to the reaction for an additional 30 min. Electrophoresis, autoradiography, and quantitation were carried out as described above.

Drug Studies—For chromomycin A3, hedamycin, and nogalamycin, drug at the indicated dilution was incubated with labeled probe prior to the addition of a mixture of EGR1 and TBP. All samples were analyzed by gel mobility shift assays. Results were quantitated by a densitometer and expressed as the percentage of inhibition of EGR1 and increase of TBP complexes compared with controls that included each individual TF without drug treatment. Alternatively, when TFIIA was used in the assay, constituents in the assay were added in a different order. First, labeled probe was incubated with TBP-TFIIA and followed by treatment with distamycin A and then EGR1. All incubations were at 30°C for 30 min. Similarly, after electrophoresis and autoradiography, the formation of TF-DNA complex was quantitated and expressed as the percentage of inhibition of TBP-TFIIA and restoration of EGR1 complexes compared with the reaction containing all three proteins.

RESULTS

Characterization of the Binding of TFs on the HSVL Promoter—Certain drugs have been previously shown to interfere with formation of either EGR1 or TBP-DNA complexes (12, 13). In this study, we investigate how these drugs specifically interfere with EGR1 or TBP binding to adjacent sites on the HSVL promoter. Prior to examining the ability of drugs to inhibit the binding of TFs to HSVL promoter, characterization of each TF-DNA complex was undertaken. With the simultaneous addition of both EGR1 and TBP, the TBP complex formation was reduced dramatically compared to that with TBP.
alone (Fig. 2, lanes 2 and 4). On the other hand, the EGR1 complexes were similar in the presence or absence of TBP (Fig. 2, lanes 3 and 4). A ternary complex of EGR1-TBP-DNA (i.e. both proteins simultaneously binding to adjacent sites at HSVL promoter) was not formed, nor did EGR1-DNA and TBP-DNA complexes exist concomitantly under a saturating assay condition.

The binding activities of the two TFs in combination were further examined by incubating a serial dilution of EGR1 and a fixed amount of TBP with unlabeled DNA prior to adding labeled probe. If the DNA binding activities of EGR1 were stronger than that of TBP, it might be possible to bind EGR1 to an unlabeled DNA, resulting in free radiolabeled probe for TBP binding. As shown in Fig. 3, in the presence of unlabeled DNA, the radiolabeled EGR1-DNA complex was diminished in a concentration-dependent manner, and the DNA binding of TBP was observed (lanes 4, 6, and 8). In contrast, in reactions without unlabeled DNA, 3 and 1.5 ng of EGR1 complexed to the DNA, whereas no TBP complex was formed (lanes 3 and 5). When less EGR1 (0.75 ng) was used, both individual EGR1-DNA and TBP-DNA complexes were present (lane 7). This result indicated that the stronger DNA binding activity of EGR1 precluded TBP complex formation.

If the binding activity of TBP could be strengthened, it might be possible to obtain DNA complexes with both EGR1 and TBP or to inhibit EGR1 binding. It is known that TFIIA, while not binding directly to DNA, enhances TBP association with DNA (12, 17–19). When TFIIA and TBP were incubated with DNA prior to the addition of EGR1, more TBP-DNA complex was formed (Fig. 4, lanes 2 and 3). Quantitative assessment of the TF-DNA complex revealed that concomitantly with the enhancement of the TBP-DNA complex by TFIIA, the EGR1-DNA complex was reduced by 50% (Fig. 4, lanes 5 and 6). Thus, the TFIIA-enhanced TBP binding competes with EGR1 for association with DNA. It is assumed that a ternary complex of EGR1-TBP-DNA is not formed, since a third band is not observed (Fig. 4, lane 6).

Schemes to depict the interaction of EGR1 and TBP-TFIIA with the HSVL promoter are presented in Fig. 5. Schemes I and II present models of individual TFs, EGR1 and TBP, binding to their consensus GC- and AT-rich binding sites, respectively. Scheme III represents the motif of both DNA-binding proteins on the HSVL promoter and shows that EGR1 binding to its GC-rich sequences prevents TBP binding to its adjacent AT-rich binding site. Scheme IV shows that TBP can compete with EGR1 for DNA binding in the presence of TFIIA.
to the HSVL promoter when both proteins were added simultaneously.

Nogalamycin, a GC intercalator that strongly affected the formation of either EGR1 or TBP-DNA complexes, was studied to determine whether it would be an equally effective inhibitor of the binding of TFs to adjacent sites on the HSVL promoter (13, 20, 21). DNA was treated with nogalamycin prior to the addition of both proteins. A representative mobility shift assay was shown in Fig. 6. As represented in Fig. 5, scheme III, when both proteins were added to the labeled probe, only the EGR1-DNA complex was observed. Upon the addition of 10 μM drug, the formation of the EGR1-DNA complex was inhibited completely, and the TBP-DNA complex became evident (Fig. 6, lane 5). With lower concentrations of nogalamycin (5 and 2.5 μM), both EGR1-DNA and TBP-DNA complexes were observed (Fig. 6, lanes 6 and 7). Reducing drug concentrations to 0.5 and 0.05 μM allowed formation of the EGR1-DNA complex with no evidence of the TBP-DNA complex (Fig. 6, lanes 8 and 9), which was similar to the pattern found in untreated sample (Fig. 6, lane 4). The drug response curves for both complexes demonstrated a dose-dependent inhibition of EGR1 accompanied by an appearance of the TBP-DNA complexes (Fig. 7). Approximately 2.6 μM of nogalamycin inhibited the formation of EGR1-DNA complex by 50%, and 10 μM of drug reduced the complex about 90%, while within the same dose range, a 3–6-fold increase in the complex formation of TBP was observed.

Like nogalamycin, hedamycin is a GC intercalator, but it also alkylates DNA at deoxyguanosine residues (22–24). Previous results have shown it to be a strong inhibitor of both EGR1 and TBP complex formation (12, 13). The pattern of hedamycin inhibition of EGR1 and TBP binding to the HSVL promoter was tested. As shown in Fig. 8, a hedamycin concentration of 0.58 μM was sufficient to inhibit EGR1-DNA complex formation by 50% and increased the TBP-DNA complex by 2-fold. Doses of 1 μM prevented the EGR1-DNA complex by 60%, but rather than allowing further TBP binding to DNA they also blocked TBP complex formation. At higher drug concentrations, EGR1-DNA complex formation was reduced by more than 90%, and the TBP-DNA complex formation also was undetectable.

Chromomycin A₂ is a GC minor groove binder that was found to be effective at preventing EGR1 and Wilms' tumor suppressor protein-1 binding to the GC-rich sites and preventing the negative regulator of interleukin-2, NIL-2A, from binding to its mixed sequence site (13, 25, 26). Chromomycin A₂ is also an inhibitor of TBP-DNA complex formation. We found that chromomycin A₂, like nogalamycin, inhibited the complex formation of EGR1, resulting in a dose-dependent increase of TBP-DNA complex formation (Fig. 9). For example, a 50% reduction of the EGR1-DNA complex with 3.1 μM chromomycin A₂ resulted in a 2-fold increase in the TBP complex. Using 14 μM drug, the EGR1-DNA complex was inhibited by 85%, and the TBP-DNA complex formation was increased to 4-fold.

The Effect of an AT-binding Drug on TF-DNA Complex Formation at the HSVL Promoter—The data to this point have demonstrated how GC-binding drugs with different interactive mechanisms disrupted the EGR1-DNA complex and resulted in various degrees of increase of TBP binding. An alternative situation where an AT-binding drug targeting the TBP binding
Drug Effects on DNA-Protein Complexes at the HSVL Promoter

We first determined the effectiveness of distamycin A at disrupting preformed TBP-TFIIA-DNA complex formation. Interference with complex formation by distamycin A occurred in a dose-dependent manner. At 0.25 μM distamycin A, complex formation was inhibited by 50%, whereas 1 and 2.5 μM of distamycin A disrupted 83 and 92% of the complex, respectively (Fig. 10). As shown in Fig. 4, TBP and TFIIA bound to the DNA, resulting in a reduction of EGR1-DNA complex by 50%. In subsequent assays, preformed TBP-TFIIA-DNA complex was treated with distamycin A, and then EGR1 was added. Under conditions where distamycin A disrupted the TBP-TFIIA-DNA complex, the EGR1-DNA complex formation was increased. For example, 0.25 μM distamycin A, which inhibited TBP-TFIIA-DNA complex to 50% of control, increased the EGR1 complex formation from 50 to 67% of control. A 92% inhibition of TBP-DNA complex formation by distamycin resulted in an increase to 80% of the EGR1-DNA complex (Fig. 10).

**DISCUSSION**

In the present study, we have used the HSVL promoter as a model system to evaluate whether DNA-binding drugs can selectively interfere with the binding of TFs. First, we confirmed that under conditions where EGR1 and TBP could bind to the HSVL promoter only the EGR1-DNA complex was observed. Preincubation of EGR1 and TBP with unlabeled DNA resulted in a reduced amount of radiolabeled EGR1-DNA complex with a concomitant increase in radiolabeled TBP-DNA complex formation. These data were in agreement with the report of Tatarowicz et al.2 and showed that complex formation by one protein excluded the other.

We found no ternary complex of EGR1-TBP-DNA when both proteins were added simultaneously to the DNA, suggesting that EGR1's presence sterically prevented TBP from binding to its cognate site. The reverse was not true, that the addition of EGR1 resulted in dissociation of preformed TBP-DNA complex (data not shown). Thus, EGR1 bound the HSVL promoter with higher affinity than TBP. A further confirmation that the prevalence of the EGR1-DNA complex was due to the higher binding affinity of EGR1 compared with TBP came from studies in which TFIIA was included. Our previous results and the results of others have shown that TFIIA stabilizes TBP binding to DNA (12, 17–19). In the presence of TFIIA, TBP competed with EGR1 for DNA binding, resulting in reduced EGR1-DNA complex formation (Fig. 4). Schemes for describing the binding relationships of EGR1, TBP, and TFIIA on the HSVL promoter are provided in Fig. 5.

The present work differs from previous studies in which drugs were evaluated as inhibitors of single TFs binding to their consensus binding sites (12, 13). Having confirmed binding patterns of EGR1 and TBP in the HSVL promoter (Fig. 5), we further examined the effect of drugs on both proteins binding to adjacent sites. We found that the GC intercalator nogalamycin selectively inhibited the EGR1-DNA complex formation (Fig. 6). The finding of Williams et al. (20) that nogalamycin unwound the DNA by 11° at the site of intercalation suggested that the drug could induce localized unwinding on the HSVL promoter. The lack of drug effect on the DNA...
conformation of flanking sequences (e.g. AT-rich region) might account for the fact that a dose-dependent inhibition of the EGR1-DNA complex by nogalamycin resulted in a concomitant appearance of TBP-DNA complex.

Hedamycin affected EGR1 and TBP complex formation differently from nogalamycin. Although both drugs preferentially bind to GC-rich sites through a mechanism of threading intercalation, hedamycin was more effective than nogalamycin at inhibiting formation of the EGR1-DNA complex. It has been demonstrated that hedamycin alkylates the N-7 of guanine within the major groove to form an irreversible drug-DNA complex (23, 24, 27–29), and this irreversible drug-DNA complex might account for stronger inhibition of TFs binding by hedamycin compared with nogalamycin. Similarly, CC-1065, an AT minor groove binder that formed an irreversible complex by bonding to the N-3 of adenine, was shown earlier to be almost 200-fold more potent compared with other reversible minor groove-binding drugs at inhibiting TBP binding to the adenoavirus-2 major late promoter (12).

Hedamycin resulted in a 2-fold increase of the TBP-DNA complex at drug concentrations that inhibited the EGR1-DNA complex by <50%. At higher concentrations (2–10 μM), TBP-DNA complexes were suppressed completely. In contrast, further suppression of the EGR1-DNA complex by increasing concentrations of nogalamycin resulted in a 3–6-fold increase in the TBP-DNA complex (Figs. 7 and 8). One explanation for the difference between these drugs with regard to TBP-DNA complex formation on the HSVL promoter was that hedamycin binding to GC-rich sequence might alter DNA conformation complex formation on the HSVL promoter was that damycin inhibited the formation of EGR1-DNA complex in a dose-dependent manner, accompanied by parallel formation of the EGR1-DNA complex. In contrast, chromomycin A₃ inhibited the formation of EGR1-DNA complex to allow DNA complex formation.

These data demonstrated the importance of sequence preferences concerning the specificity of drugs as inhibitors of TF-DNA complexes. Moreover, our results indicated that drugs affected TFs binding to their individual binding sites differently from that to adjacent sites, suggesting that the modes of drug interaction with DNA played a role in determining the activity and specificity of drugs. This work can be viewed as a model to study sequence-specific DNA-binding drugs for their ability to selectively effect binding of TFs. Additionally, the results provide a guide for development of future drugs as potent and specific inhibitors of TF-DNA complexes.

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