Occlusion of RNA Polymerase by Oligomerization of DnaA Protein over the dnaA Promoter of Escherichia coli

(Received for publication, June 25, 1996, and in revised form, September 13, 1996)

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DnaA protein, the initiator protein for initiation of Escherichia coli chromosomal replication, has been shown to repress its own expression from two dnaA promoters, 1P and 2P. The sequence-specific binding of DnaA protein to the DnaA box, located between the two promoters, results in subsequent oligomerization of DnaA protein. Upon increasing the concentration of DnaA protein, the oligomerization proceeds to both dnaA promoters from the DnaA box and inhibits RNA polymerase binding to both promoters. This results in the repression of transcription, suggesting that the extent of oligomerization of DnaA proteins over two dnaA promoters contributes to the autoregulation of expression of the dnaA gene. When the two dnaA promoters were bound and repressed by DnaA protein, the interaction of RNA polymerase with IciA protein, which is a specific inhibitor of initiation of in vitro E. coli chromosomal replication, appeared to dissociate the oligomerized DnaA proteins from the 1P promoter and allowed RNA polymerase to be loaded for its transcription.

DnaA protein is essential for the initiation of Escherichia coli chromosomal DNA replication in vivo and in vitro (1–5). The binding of 20–30 molecules of DnaA protein to oriC (origin of chromosomal DNA replication) containing five DnaA boxes (or 9-mers), which are recognized by DnaA protein, forms an initial complex for the initiation of in vitro oriC plasmid DNA replication (4, 6–8). DnaA protein binds ATP, with a $K_{D}$ of 0.03 μM, and other nucleotides (9). Whereas the ADP- or AMP-bound form of DnaA protein is not active for oriC plasmid DNA replication, the ATP-bound form is active. Phospholipid exchange changes ADP in the ADP form of DnaA protein with ATP. Also, ATP stabilizes DnaA protein. DnaA protein appears to exist as monomeric and aggregated forms in E. coli (2). The inactive and aggregated form of DnaA protein containing phospholipid was converted in an ATP-dependent manner to active monomeric forms by phospholipase or Dnak protein (10). The DNase I footprint of oriC bound by the nucleotide-bound form of DnaA protein was distinct from that bound by the nucleotide-free form of DnaA protein (7, 11). While DNase I cleavages in the footprint with the nucleotide-free form were widely distributed in oriC and its adjacent region, those with the nucleotide-bound form were in the regions containing the DnaA box. The localized binding of DnaA protein to the regions containing the DnaA box was similar to the in vivo footprint pattern of oriC (12).

The dnaA gene, encoding DnaA protein, contains two promoters, 1P and 2P (see Fig. 1). One DnaA box is located between the two promoters. In vivo overproduction of DnaA protein reduced the transcription from both promoters (13). Transformation of E. coli with a plasmid containing several DnaA boxes resulted in an increase of dnaA expression due to the titration out of intracellular DnaA proteins (14). In vitro transcription of the dnaA gene was inhibited by DnaA protein (15). These results supported the autoregulation of expression of the dnaA gene (3, 16, 17). Also, DnaA protein functions as a transcriptional repressor for the expression of other genes including rpoH (18), mica (19), the guaBA operon (20), and wrbB (21), while the expression of the rrd gene appeared to be enhanced by DnaA protein (22).

In the presence of a millimolar level of ATP, DnaA protein unwinds the AT-rich region (containing three 13-mers) of oriC for the next step of initiation of oriC replication. IciA protein has been shown to specifically inhibit this early step of in vitro E. coli chromosomal DNA replication (23, 24). The binding of IciA protein to the AT-rich region blocks the opening of the AT-rich region. Also, IciA protein binds to two sites, IciA I and IciA II, in the dnaA promoter region (25). The IciA I site is located upstream of dnaA promoter 1P, and the IciA II site is downstream of dnaA promoter 2P (see Fig. 1). Among the two dnaA promoters, transcription from the 1P promoter was specifically enhanced by in vivo overproduction of IciA protein or by the addition of IciA protein, regardless of the presence of DnaA protein, in the transcription assay of the dnaA gene in vitro.

The molecular mechanism of the DnaA protein-dependent transcriptional repression has not been reported. In this report, we show that the repression is due to the occlusion of RNA polymerase caused by oligomerization of DnaA proteins over two dnaA promoters. The binding of IciA protein to the IciA site was also addressed to enhance the binding of RNA polymerase to dnaA promoter 1P covered by DnaA protein.

EXPERIMENTAL PROCEDURES

Reagents and Proteins—Sources were as follows: [$\gamma$-32P]ATP and [$\alpha$-32P]UTP (4000 Ci/mmole), Amersham Corp.; poly(dI)poly(dC) and Fast Q. Pharmacia Biotech Inc.; calf intestinal alkaline phosphatase, Boehringer Mannheim; Long Ranger polyacrylamide, AT Biochem; T4 polynucleotide kinase, New England Biolabs Inc.; and restriction and cloning enzymes, Promega. Unless otherwise indicated, other reagents were purchased from Sigma.

Monomeric DnaA protein from MC1061(pDS596) (10), IciA protein from MC1061(pJSC1) (26), and the $\alpha^{59}$ subunit of RNA polymerase from BL21(pGEMD) (27) were purified as described previously (28), except that Fast Q chromatography replaced Mono Q chromatography at the final step of purification. RNA polymerase holoenzyme was reconstituted by mixing the purified RNA polymerase with a 3.5-fold molar excess of the $\alpha^{59}$ subunit.

* This work was supported in part by a grant for genetic engineering research from the Ministry of Education, Republic of Korea. The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

† Supported by a young researchers grant from the Ministry of Education, Republic of Korea.

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FIG. 1. Physical map of the dnaA promoter region. Two dnaA promoters and neighboring regions were as described previously (38, 39), except that the positions of the nucleotide sequence are based on the transcription start point of dnaA promoter 1P as nucleotide +1. Nucleotide +1 corresponds to nucleotide 651 of Refs. 38 and 39. Promoters are as follows: rpmH 1P, rpmH promoter 1P (−116 to −143); dnaA 1P, dnaA promoter 1P (−4 to −34); and dnaA 2P, dnaA promoter 2P (+51 to +79). Nucleotide −35 and −10 from the transcription start point of each promoter are indicated. The direction of transcription is indicated by the arrows beneath. The DNA box (+18 to +26) indicates DnaA protein recognition sequence. IciA I and IciA II are IciA protein-binding sites IciA I (−50 to −63), determined by OP-CuII footprinting (Y. S. Lee and D. S. Hwang, unpublished data)) and IciA II (−179 to −224, previously determined by DNase I footprinting (25)), respectively. The restriction sites (from the right) are HindIII, BamHI (from plasmid pBF1509), BglII, HindII, HindIII, and EcoRI, located at nucleotides −247, −108, +52, +174, +227, and +296, respectively.

Bacterial Strains and Plasmid DNAs—The E. coli strains W3110 (λ−, IN[rrnD-rrnE]) and DH5α (29) were previously described. E. coli DH5α was used for isolation of plasmid DNAs. The plasmid DNAs pBfA1/dnaA (30), pBF1509 (31), pSC1 (26), pYS1 (25), and pBlue-IH (BAS1000). The abbreviations used are: bp, base pair(s); OP-CuII (OP-CuII footprinting was performed as described previously (32) with minor modifications. A gel-shift assay with 21.5 fmol of the 228-bp footprinting was performed as described previously (32) with minor modifications. A gel-shift assay with 21.5 fmol of the 228-bp footprinting was performed as described previously (32) with minor modifications. 18

Gel-shift Assay—To end-label DNA fragments for gel-shift and footprinting assays, DNA fragments were dephosphorylated with calf intestinal alkaline phosphatase and radioactively labeled with T4 polynucleotide kinase and [γ-32P]ATP. Gel-shift assays were performed as described previously (23) with minor modifications. 18 μl of gel-shift assay buffer (20 mM HEPES/KOH (pH 8.0), 5 mM magnesium acetate, 60 mM KCl, 1 mM EDTA, 4 mM dithiothreitol, 0.5 mg/ml bovine serum albumin, 10 μM ATP, and 10% glycerol) contained 1 μg of poly(dI-poly(dC)) and 1 μM DNA fragment, which was isolated from plasmid pBF1509, with the Klengow fragment was followed by insertion into the EcoRV site of vector pBlueScript SK.

Sequence-specific DNA binding of DnaA protein to the dnaA promoter region (Fig. 1) was studied in detail using a combined gel-shift and chemical footprinting assay to determine the extent of DNA binding to the region flanking the consensus DNA-binding site, the dnaA box. The 32P-labeled 228-bp XbaI/XhoI DNA fragment used for in vitro DNA binding reactions was derived from plasmid pHJ4 and contains the dnaA promoter, the dnaA promoter 1P region, and a truncated region of dnaA promoter 2P (Fig. 2).

At the lowest level of DnaA protein added to the gel-shift assay, one DnaA protein-DNA complex predominated (complex A) (Fig. 2, second lane). Increasing amounts of DnaA protein added to the reactions produced more slowly migrating complexes (B, C, and D), which are presumably formed by binding of increasing numbers of DnaA molecules to the DNA fragment.

To analyze each individual protein-DNA complex in more detail, in situ footprinting was performed using the OP-CuII complex as a chemical DNA cleavage agent (32). After electrophoresis of the protein-DNA complexes, the polyacrylamide gel was treated with OP-CuII. The DNA in each band was isolated and subjected to electrophoresis through a 5% Long Range polyacrylamide sequencing gel containing 7 M urea. The gel was dried and visualized by autoradiography.

RESULTS

Oligomerization of DnaA Protein on the dnaA Promoter—Sequence-specific DNA binding of DnaA protein to the dnaA promoter region (Fig. 1) was studied in detail using a combined gel-shift and chemical footprinting assay to determine the extent of DNA binding to the region flanking the consensus DNA-binding site, the dnaA box. The 32P-labeled 228-bp XbaI/XhoI DNA fragment used for in vitro DNA binding reactions was derived from plasmid pHJ4 and contains the dnaA box, the dnaA promoter 1P region, and a truncated region of dnaA promoter 2P (Fig. 2).

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DNase I Protection Assay—The standard reaction (25 μl) contained 40 mM HEPES/KOH (pH 7.6), 50 mM potassium chloride, 10 mM magnesium acetate, 0.1 mM ATP, 2.5 μg of bovine serum albumin, 10% glycerol, 20 fmol of the indicated 32P-end-labeled DNA fragments, and the indicated amounts of proteins. After incubation at 37 °C for 10 min, DNase I (5 ng in 1.5 μl of H2O) was added and incubated for 30 s, and the reaction was stopped by the addition of 27 μl of 0.6 M sodium acetate, 0.4% sodium dodecyl sulfate, 25 mM EDTA, and 2.5 μg of yeast tRNA. Proteins were removed by phenol/chloroform extraction. DNA was precipitated with ethanol, followed by a 70% ethanol wash. DNA was subjected to electrophoresis through a 5% Long Range polyacrylamide sequencing gel containing 7 M urea. The gel was dried and visualized by autoradiography.

Run-off Transcription Assay—Run-off transcription assays were performed as described previously (25).

To analyze each individual protein-DNA complex in more detail, in situ footprinting was performed using the OP-CuII complex as a chemical DNA cleavage agent (32). After electrophoresis of the protein-DNA complexes, the polyacrylamide gel was treated with OP-CuII. The DNA in each band was isolated and subjected to electrophoresis through a 5% denaturing sequencing gel, followed by autoradiography (Fig. 3A) or by scanning the gel with a FUJIX Bio-Imaging Analyzer (BAS1000) (Fig. 3B). OP-CuII cleavage of complex A revealed that the protection by DnaA protein is limited to a region encompassing −20 bp within the dnaA box and to a region encroaching upon dnaA promoter 2P (Fig. 3A, lane 2).

In complexes B to D, which were produced at higher amounts of DnaA protein, the protected regions became more extended from the dnaA box toward the two dnaA promoter start sites. As the DnaA protein used in this report was monomeric and the binding reactions were performed in the presence of ATP (see “Experimental Procedures”), the possibility of binding of a contaminating aggregated form of DnaA protein can be excluded. These results suggest that the initial binding of DnaA protein
to the DnaA box promotes cooperative binding of additional monomers extending ultimately over the two dnaA promoters.

**Oligomerized DnaA Protein-DNA Complexes Occlude RNA Polymerase from the dnaA Promoter**—DnaA protein has been shown to repress the transcription of the dnaA gene in vivo (13) and in vitro (15). However, the mechanism of the repression has not been addressed. Two possible mechanisms involve either an inactivation of RNA polymerase-DNA initiation complexes through direct protein-protein interactions or the inhibition of RNA polymerase binding to the promoter. The effect of DnaA protein on the stable binding of RNA polymerase to dnaA promoter 1P was examined using a gel-shift assay with the 32P-labeled 228-bp XbaI/XhoI fragment from plasmid pHJ4 (Fig. 4).

RNA polymerase bound stably to the 1P promoter and shifted ~30% of the input DNA (Fig. 4). With increasing amounts of DnaA protein added to the reaction, RNA polymerase binding was inhibited at levels that also inhibited in vitro run-off transcription. These results suggest that competition for promoter binding is responsible for DnaA protein inhibition of transcription.

Small amounts of DnaA protein (38 ng or less) did not significantly inhibit the binding of RNA polymerase to the dnaA promoter, nor did these low levels of DnaA protein inhibit in vitro transcription (Fig. 4). At 19 ng of DnaA protein (Fig. 2), the ratios of free DNA and complex A over the input DNA were 0.54 and 0.38, respectively. At 38 ng of DnaA protein, the ratios of free DNA and complexes A, B, C, and D were 0.20, 0.36, 0.20, 0.19 and 0.05, respectively. At these lower levels of DnaA protein, the major form of DnaA protein is in complexes A and B (Fig. 2), where the binding of DnaA protein was limited to the DnaA box (Fig. 3). The significant inhibition of RNA polymerase binding to the 1P promoter was only apparent at 75 ng or more of DnaA protein, where the formation of DnaA protein-DNA complexes C and D was proportional to the rate of inhibition of RNA polymerase binding to the dnaA promoters (Fig. 4). These observations indicate that the inhibition of binding of RNA polymerase to the dnaA promoters is dependent upon the oligomerization of DnaA protein to regions flanking the DnaA box and that the binding of one or two DnaA monomers to the DnaA box is not sufficient for occlusion of RNA polymerase from the dnaA promoters.

The inhibition of the binding of RNA polymerase to dnaA promoters 1P and 2P was also confirmed using a DNase I protection assay (Fig. 5). The region bound by RNA polymerase alone was localized within promoters 1P and 2P. As the amounts of DnaA protein increased, the protection of both promoters from DNase I cleavage by RNA polymerase was reduced dramatically, and the DNase I footprinting pattern of the two promoters became similar to that for protection by DnaA protein alone. The less efficient binding of RNA polymerase to the dnaA promoter in gel-shift assays compared with the binding in DNase I protection assays was caused by the presence of poly(dI)-poly(dC) in the gel-shift assays.
amount of poly(dI)-poly(dC) used abolished the nonspecific binding of RNA polymerase to DNA fragments, but did not significantly affect the binding of DnaA protein to the DNA fragments containing the DnaA box.

IciA Protein Counteracts the Repression of DnaA Protein by Stabilizing RNA Polymerase DNA Binding to dnaA Promoter 1P—IciA protein binds to two sites in the dnaA promoter region, which are located upstream of dnaA promoter 1P (IciA I) and downstream of dnaA promoter 2P (IciA II) (Fig. 1) (25). Between the two dnaA promoters, transcription from the 1P promoter was specifically activated by IciA protein in vivo and in vitro. The binding of two dimers of IciA protein to the IciA I site is responsible for the activation of dnaA promoter 1P.2 When the two dnaA promoters were repressed by DnaA protein, IciA protein was able to restore transcription from the 1P promoter with little effect on transcription from the 2P promoter (25). The mechanism of IciA protein stimulation of transcription from dnaA promoter 1P in the presence of inhibitory amounts of DnaA protein was examined.

Using a gel-shift assay (Fig. 6A), RNA polymerase-DNA binding activity (lane 2) was inhibited by the addition of DnaA protein (lane 3). However, the binding of RNA polymerase to the DNA fragment in the presence of DnaA protein was restored by the addition of IciA protein (lanes 4–6).

Similar results were also observed using DNase I footprinting (Fig. 7). RNA polymerase binding to the region containing dnaA promoter 1P (lane 3) was inhibited by DnaA protein (lane 4). Under these conditions, IciA protein restored RNA polymerase binding to dnaA promoter 1P (lanes 5 and 6). As all three proteins were present in the same reaction and as the promoter binding of both IciA protein and DnaA protein remained the same in the absence or presence of each other (lanes 2 and 7), these results suggest that IciA protein may establish protein-protein contacts with RNA polymerase that are dominant over the occlusion and inhibition promoted by DnaA protein.

DISCUSSION

Physically and functionally heterogeneous complexes containing DnaA protein bound to the dnaA promoter were isolated and analyzed in order to address the mode of binding of DnaA protein to DNA containing a single DnaA box and how this binding inhibits transcription by RNA polymerase. First, by recognition of its consensus sequence, a DnaA protein monomer specifically binds to the dnaA box. Second, either through the intrinsic aggregation property of DnaA protein (2, 10) or by random and nonspecific nucleation to DNA surrounding the first dnaA protein, cooperative binding of DnaA protein monomers surrounds the DNA box and extends to regions containing promoters 1P and 2P (Fig. 3). Although we did not accurately define the number of DnaA protein molecules present at the dnaA promoter, a monomer is the minimal stable active unit for sequence-specific binding to a DnaA box (33). The
formation of up to four major protein-DNA complexes at 150 ng of DnaA protein (Fig. 2) suggests that four monomers bind. This number of DnaA proteins per one DnaA box is within the range of 20–30 molecules of DnaA protein bound to the oriC region containing five DnaA boxes, the number that was deduced from the electron microscopic structure of the initial complex (7, 31, 34).

Previous genetic studies have suggested that RNA polymerase and DnaA protein may form direct complexes (35, 36), although biochemical evidence for this does not exist. We investigated whether DnaA protein and RNA polymerase can coexist at the dnaA promoter by gel-shift and footprinting analyses and whether a direct protein-protein interaction between DnaA protein and RNA polymerase is the mechanism of inhibition of transcription. Our results indicate that the repression of transcription from the two dnaA promoters (1P and 2P) by DnaA protein is promoted by the oligomerization of DnaA protein over dnaA promoters 1P and 2P and that this binding directly prevents RNA polymerase binding to the two promoters. The extent of oligomerization of DnaA protein at its own promoter, which depends upon the concentration of DnaA protein, could determine the rate of binding of RNA polymerase to the dnaA promoters and transcription of the dnaA gene.

IciA protein is a dimer of a single polypeptide (26). The binding of two dimers of IciA protein to the IciA I site, which is located −50 to −63 nucleotides from the transcription start site of dnaA promoter 1P, is required to stimulate RNA polymerase binding to the 1P promoter. IciA protein may directly interact with the α-subunit of RNA polymerase to stimulate transcription. The presumed interaction enhances the capability of RNA polymerase to be loaded onto the 1P promoter, resulting in the activation of transcription from dnaA promoter 1P. Other transcriptional activators including CRP (cAMP receptor protein), OxyR, Ada, and OmpR, which bind to the −40 to −60 region from the transcription start site, have been shown to interact with the α-subunit of RNA polymerase to enhance the binding of RNA polymerase to the corresponding promoters (reviewed in Ref. 37). Our previous experiments have shown that when both dnaA promoters 1P and 2P were repressed by DnaA protein, IciA protein specifically activated transcription from dnaA promoter 1P, while dnaA promoter 2P was still repressed (25). Analysis of the IciA and DnaA protein promoter-binding sites was combined to determine the mechanism of this activation, and it was found that the interaction of RNA polymerase with IciA protein is sufficient to dissociate oligomerized DnaA proteins from the 1P promoter for the activation of transcription. Once RNA polymerase is bound to the 1P promoter, the oligomerized DnaA proteins over the 2P promoter do not block the movement of RNA polymerase for transcription from the 1P promoter, while the oligomerized DnaA proteins inhibit the binding of RNA polymerase to the 2P promoter.

Acknowledgments—We thank Dr. Theodore R. Hupp for critical reading of the manuscript and Dr. Akira Ishihama for the gift of plasmid pGEMD.
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J. Biol. Chem. 1997, 272:83-88.
doi: 10.1074/jbc.272.1.83

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