**Direct Interaction between *Escherichia coli* RNA Polymerase and the Zinc Ribbon Domains of DNA Topoisomerase I**

Received for publication, April 2, 2003, and in revised form, May 29, 2003

Published, JBC Papers in Press, June 4, 2003, DOI 10.1074/jbc.M303403200

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**Escherichia coli** DNA topoisomerase I (encoded by the topA gene) is important for maintaining steady-state DNA supercoiling and has been shown to influence vital cellular processes including transcription. Topoisomerase I activity is also needed to remove hypernegative supercoiling generated on the DNA template by the progressing RNA polymerase complex during transcription elongation. The accumulation of hypernegative supercoiling in the absence of topoisomerase I can lead to R-loop formation by the nascent transcript and template strand, leading to suppression of transcription elongation. Here we show by affinity chromatography and overlay blotting that *E. coli* DNA topoisomerase I interacts directly with the RNA polymerase complex. The protein-protein interaction involves the β subunit of RNA polymerase and the C-terminal domains of *E. coli* DNA topoisomerase I, which are homologous to the zinc ribbon domains in a number of transcription factors. This direct interaction can bring the topoisomerase I relaxing activity to the site of transcription where its activity is needed. The zinc ribbon C-terminal domains of other type I topoisomerases, including mammalian topoisomerase III, may also help link the enzyme activities to their physiological functions, potentially including replication, transcription, recombination, and repair.

DNA topoisomerases are ubiquitous enzymes that have functional roles in many vital cellular processes (1, 2). Among different classes of topoisomerases, type IA topoisomerases found in archaea, prokaryotes, and eukaryotes share the mechanistic feature of cutting and rejoining a single strand of DNA via a 5'-phosphotyrosine linkage and homologous amino acid sequences (3). *Escherichia coli* DNA topoisomerase I (encoded by the topA gene) is the most extensively studied example of this class of enzyme. Its most apparent physiological role is the maintenance of steady-state DNA supercoiling (4, 5). During transcription, the movement of the RNA polymerase complex on the DNA template creates local transcription-driven supercoiling and negative supercoiling generated behind the RNA polymerase and positive supercoiling generated ahead of the RNA polymerase (6, 7). DNA gyrase is needed for removing the positive supercoils, and topoisomerase I is responsible for removing the excess negative supercoils. In the absence of topoisomerase I function due to mutation in the topA gene, the accumulation of hypernegative supercoiling can lead to R-loop formation by nascent transcription and template stranding with the consequent suppression of transcription elongation (8, 9). In in vivo studies, Tn5 transposase was found to copurify with *E. coli* DNA topoisomerase I and inhibit the topoisomerase I activity (10). RNA polymerase was also found to copurify with Tn5 transposase, but the copurification was reduced in extracts from a topA mutant strain, suggesting that the interaction between RNA polymerase and DNA topoisomerase I was responsible for the copurification of RNA polymerase with Tn5 transposase (10). The proposed function of topoisomerase I activity in removal of transcription-driven hypernegative supercoiling (8, 9) would be greatly facilitated by direct protein-protein interaction with RNA polymerase. Experiments described here provide direct evidence for such interaction as well as identifying the domain of topoisomerase I and the subunit in RNA polymerase that are responsible for this protein-protein interaction.

**EXPERIMENTAL PROCEDURES**

**Enzymes—*E. coli* DNA topoisomerase I and its subdomains were expressed and purified as described previously (11–14).** Purified *E. coli* RNA polymerase was purchased from Epicentre and USB Corp.

**Maltose-binding Protein Affinity Chromatography—**A PCR fragment encoding the *E. coli* topA gene was generated by *Pfu* DNA polymerase (from Stratagene) and cloned into the XmnI site of pMal-c2X (New England BioLabs) to create a fusion protein with MBP§ linked to the N terminus of topoisomerase I. Expression of MBP-topoisomerase I and MBP in *E. coli* TB1 cells was induced with isopropyl-1-thio-β-D-galactopyranoside. Cells from a 250-ml culture were lysed by lysozyme and lysozyme treatment combined with freeze-thawing in phosphate-buffered saline. The soluble extract obtained after centrifugation was applied to 1 ml of amylase resin (New England BioLabs) equilibrated with column buffer (20 mM Tris, pH 7.5, 200 mM NaCl, 1 mM EDTA). After extensive washing, the proteins bound to the column were eluted with column buffer containing 10 mM maltose.

**RNA Polymerase Affinity Chromatography—**Polyol-responsive monoclonal antibodies against *E. coli* RNA polymerase β subunit (NT73) was purchased from Neoclonal and coupled to cyanogen bromide-activated Sepharose according to published procedures using 0.9 ml of the antibodies (15). The NT73 affinity matrix was mixed with 1 ml of extracts of *E. coli* BL21 cells prepared from 400 ml of culture expressing intact topoisomerase I (11) or its subdomains (12–14) as described for affinity purification of RNA polymerase (15). After extensive washing, the bound proteins were eluted with 1 ml of 40% ethylene glycol with 0.5% NaCl.

**Blotting of RNA Polymerase with Digoxigenin (DIG)-labeled Topoisomerase I and Its Subdomains—**Purified RNA polymerase (0.8 μg) was electrophoresed in SDS-polyacrylamide gel to separate the subunits. The proteins were transferred onto either support nitrocellulose

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1 The abbreviations used are: MBP, maltose-binding protein; DIG, digoxigenin; NEM, N-ethylmaleimide.
membrane for chemiluminescence detection or polyvinylidene difluoride membrane for color detection. Topoisomerase I and its subdomains were labeled with DIG using the labeling kit from Roche Applied Science. Each DIG-labeled protein was incubated with the membrane at 25 °C for 1 h. Anti-DIG antibodies linked to peroxidase were used for detection by the ECL Plus system (Amersham Biosciences). Anti-DIG antibodies linked to alkaline phosphatase were used for color detection with the nitro blue tetrazolium/5-bromo-4-chloro-3-indolyl phosphate substrate (Roche Applied Sciences). After color detection, the position of the topoisomerase I-binding signal was marked and photographed before staining of the nylon membrane by Coomassie Blue.

**In Vitro Transcription—In vitro transcription with E. coli RNA polymerase (3 units) was carried out with procedures similar to those described previously (16). The reaction (30 µl) contained 40 mM Hepes/KOH (pH 7.6), 11 mM magnesium acetate, 100 mM potassium glutamate, 1 mM dithiotreitol, 4 mM ATP, 0.5 mM each of GTP, CTP, and UTP, 0.5 mM of negatively supercoiled plasmid pBR322. Transcription was carried out at 37 °C for 10 min. After deproteinization by phenol extraction, the DNA samples were analyzed by electrophoresis in 1% agarose gel with 0.5% TPE (90 mM Tris, 90 mM phosphate, 2 mM EDTA, pH 8.0) buffer with the indicated chloroquine concentrations for detection of positively supercoiled DNA (17). One-dimensional agarose gels were stained with ethidium bromide and photographed. When analyzed by two-dimensional agarose gel electrophoresis, DNA was visualized by hybridization to 32P-labeled probes as described previously (17).

**Induction of Synthesis of the 14-kDa C-terminal Fragment in Vivo—** DNA coding for the 14-kDa C-terminal fragment of E. coli DNA topoisomerase I was generated by PCR using the Plu DNA polymerase and inserted into the pBAD-thio-TOPO expression vector (from Invitrogen). The resulting plasmid pBAD14K has the 14-kDa C-terminal fragment inserted into the pBADThio-TOPO expression vector (from Invitrogen). DNA topoisomerase I and any associated cellular proteins were then concentrated and applied to the gel to visualize the eluted proteins.

**RESULTS**

**Direct Interaction between E. coli DNA Topoisomerase I and RNA Polymerase—** Extract of E. coli cells expressing recombinant E. coli DNA topoisomerase I with MBP fused to its N-terminal end was applied to amylose resin. The bound topoisomerase I and any associated cellular proteins were then eluted with buffer containing maltose. Identical procedures were carried out with elution of recombinant MBP from amylose resin. When the maltose eluate fractions were analyzed by Western blotting, RNA polymerase α and β subunits were detected in the eluate fractions from cells expressing MBP-topoisomerase I but not in the eluate fractions prepared from cells expressing MBP (Fig. 1). This demonstrates that the linkage of DNA topoisomerase I to MBP is required for binding of RNA polymerase to the amylose resin.
The Zinc Ribbon Domains of Topoisomerase I Are Responsible for the Interaction with RNA Polymerase—An RNA polymerase affinity column was prepared using polyclonal responsive monoclonal antibodies against the β’ subunit of *E. coli* RNA polymerase (15). Soluble extracts of *E. coli* BL21 cells expressing DNA topoisomerase I (11) or its subdomains (12–14) were applied individually to the affinity column in separate experiments carried out with identical procedures (Fig. 2). Topoisomerase I could be detected in the bound proteins eluted from this affinity column with buffer containing 40% ethylene glycol and 0.75 M NaCl. In similar experiments carried out separately with lysates expressing an individual topoisomerase I subdomain, it was determined that the 67-kDa N-terminal trans-esterification domain (Top67) did not bind to the affinity column but the 20-kDa (ZD2) and 14-kDa C-terminal fragments could bind to the RNA polymerase affinity column. These two fragments contain three and two copies of zinc ribbon domains, respectively, and are homologous to the zinc ribbon domains in transcription regulators including RPB9, TFIIS, and TFIIIB (21). However, only the three zinc ribbon domains on ZD2 have Zn(II) bound to tetracysteine motifs (13). Binding of the RNA polymerase subunits to this affinity column was confirmed by both Coomassie Blue staining of the eluted proteins and Western blot analysis using antibodies against the α, β, and β’ subunits (Fig. 2c).

The β’ Subunit of RNA Polymerase Interacts with Topoisomerase I—*E. coli* RNA polymerase was expected to bind to the affinity matrix as a multisubunit complex. To determine which subunit was responsible for the interaction with DNA topoisomerase I, the RNA polymerase subunits were electrophoresed in a 7% SDS-polyacrylamide gel and transferred onto membrane. Overlay blotting was carried out using DIG-labeled topoisomerase I, DIG-labeled Top67, DIG-labeled ZD2, and DIG-labeled 14-kDa fragment. All of these DIG-labeled proteins, except DIG-labeled Top67, gave a positive chemiluminescence signal with peroxidase-linked anti-DIG antibody at a position where RNA polymerase β and β’ subunits would migrate in the 7% SDS-polyacrylamide gel (Fig. 3a). This result was in agreement with the data in Fig. 2b and showed that DNA that might have been present in the *E. coli* protein extract during binding to the affinity columns was not required for interaction between DNA topoisomerase I and RNA polymerase. There was also no signal when the RNA polymerase subunits on the membrane were blotted with DIG-labeled bovine serum albumin.

To determine whether the β or β’ subunit was interacting with topoisomerase I, a 5% polyacrylamide gel was used for SDS-gel electrophoresis of the RNA polymerase subunits to better separate these two high molecular weight subunits. After transfer, the membrane was incubated with DIG-labeled ZD2 followed by alkaline-phosphatase linked anti-DIG antibodies. The signal of the DIG-topoisomerase I fragment bound to the nylon membrane was developed with the nitro blue tetrazolium/5-bromo-4-chloro-3-indolyl phosphate color reagent. The position of the signal was marked on the membrane and photographed alongside a ruler. The nylon membrane was then stained with Coomassie Blue to visualize both β and β’ in the same lane. Blotting with DIG-topoisomerase I according to this procedure gave the same results. The Zinc Ribbon Domains of Topoisomerase I Are Responsible for the Interaction with RNA Polymerase—An RNA polymerase affinity column was prepared using polyclonal responsive monoclonal antibodies against the β’ subunit of *E. coli* RNA polymerase (15). Soluble extracts of *E. coli* BL21 cells expressing DNA topoisomerase I (11) or its subdomains (12–14) were applied individually to the affinity column in separate experiments carried out with identical procedures (Fig. 2). Topoisomerase I could be detected in the bound proteins eluted from this affinity column with buffer containing 40% ethylene glycol and 0.75 M NaCl. In similar experiments carried out separately with lysates expressing an individual topoisomerase I subdomain, it was determined that the 67-kDa N-terminal trans-esterification domain (Top67) did not bind to the affinity column but the 20-kDa (ZD2) and 14-kDa C-terminal fragments could bind to the RNA polymerase affinity column. These two fragments contain three and two copies of zinc ribbon domains, respectively, and are homologous to the zinc ribbon domains in transcription regulators including RPB9, TFIIS, and TFIIIB (21). However, only the three zinc ribbon domains on ZD2 have Zn(II) bound to tetracysteine motifs (13). Binding of the RNA polymerase subunits to this affinity column was confirmed by both Coomassie Blue staining of the eluted proteins and Western blot analysis using antibodies against the α, β, and β’ subunits (Fig. 2c).

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was also added to the in vitro transcription reaction. The 14-kDa C-terminal fragment could compete with topoisomerase I for interaction with E. coli RNA polymerase, and the formation of positively supercoiled DNA template was found to be inhibited (Fig. 4). In a relaxation reaction in the absence of RNA polymerase, the 14-kDa fragment has no significant effect on the relaxation of negatively supercoiled DNA by DNA topoisomerase I; thus so the 14-kDa fragment does not inhibit the catalytic activity of DNA topoisomerase I (Fig. 4c).

The significance of the RNA polymerase-topoisomerase I interaction for removal of transcription driven supercoils was also demonstrated in vivo. The 14-kDa C-terminal domain was expressed in E. coli as a thioredoxin fusion protein via the tightly regulated P_{BAD} promoter (22). Accumulation of hypernegative supercoils in the plasmid DNA was observed when synthesis of the 14-kDa C-terminal fragment by pBAD14K was induced by addition of 0.005% arabinose to the culture (Fig. 5a). The induced 14-kDa C-terminal domain was expected to compete with topoisomerase I for interaction with RNA polymerase, impeding the removal of transcription-driven negative supercoils by DNA topoisomerase I. The level of 14-kDa C-terminal domain synthesis and resulting accumulation of hypernegatively supercoiled DNA was also demonstrated in vivo. The 14-kDa C-terminal domain was also added to the in vitro transcription reaction. The 14-kDa C-terminal fragment could compete with topoisomerase I for interaction with E. coli RNA polymerase, and the formation of positively supercoiled DNA template was found to be inhibited (Fig. 4). In a relaxation reaction in the absence of RNA polymerase, the 14-kDa fragment has no significant effect on the relaxation of negatively supercoiled DNA by DNA topoisomerase I; thus so the 14-kDa fragment does not inhibit the catalytic activity of DNA topoisomerase I (Fig. 4c).

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was higher than the transformant expressing Top67 but still about 100-fold lower than that of the transformant expressing the full-length topoisomerase I. Although not required for viability of E. coli under optimal laboratory growth conditions, the absence of the 14-kDa fragment in topoisomerase I thus affected the function of topoisomerase I in stress response significantly.

**DISCUSSION**

The results presented here support a role of the C-terminal zinc ribbon domains in E. coli DNA topoisomerase I for interacting with the β’ subunit of RNA polymerase, so that negative DNA supercoiling formed during transcription can be removed immediately. The rapid removal of transcription-driven negative supercoils would prevent the formation of R-loops (8, 9). This suppression of R-loops during transcription has been proposed to be an essential function for E. coli DNA topoisomerase I (24).

Sequence analysis shows that the number of zinc ribbon domains in type IA DNA topoisomerases found in different organisms varies from zero to five (20). If present, these zinc ribbon domains can potentially interact with RNA polymerase in these organisms during transcription. Other DNA topoisomerases have also been shown previously to be involved directly in transcription. Human topoisomerase I, a type IB topoisomerase, has been shown to be a cofactor of RNA polymerase II transcription (25, 26), whereas topoisomerase IIα is required for RNA polymerase II transcription on chromatin templates (27).

We have observed previously that a recombinant plasmid expressing the 14-kDa C-terminal domain of E. coli DNA topoisomerase I under the T7 promoter was unstable in E. coli BL21DE3 (12). Attempts to construct a recombinant plasmid expressing the 14-kDa C-terminal domain as a MBP-fusion protein in pMal-c2X were unsuccessful. The results showing the interference of E. coli DNA topoisomerase I function during transcription by the 14-kDa C-terminal domain would account for the instability of these recombinant plasmids. It may be possible for topoisomerase I function or DNA supercoiling to be modulated via targeting of the protein-protein interactions between topoisomerase I and its partners in E. coli.

Besides Tn5 transposase and RNA polymerase in E. coli, cellular proteins in other organisms may also interact with type IA topoisomerases. This is particularly intriguing for mammalian type IA topoisomerases. There are two type IA topoisomerases (TOP3α and TOP3β) present in both human and mouse (28–32) with four zinc ribbon domains present in each of these enzymes (21). Human TOP3α has been shown to interact with the Bloom Syndrome helicase, BLM (33, 34), whereas both TOP3α and TOP3β interact with human RecQ5 helicase (35). There is evidence that interaction between Bloom Syndrome helicase and human TOP3α is important for genomic stability (36). The domains in TOP3α and TOP3β responsible for the interactions with the RecQ family of helicases have not been identified experimentally. Multiple transcripts from alternative splicing with tissue specific expression pattern give rise to variant forms of TOP3β that have different numbers of zinc ribbon domains (31). This can affect the interaction of TOP3β with other cellular proteins. The potential involvement of these zinc ribbon domains in protein-protein interactions may play the important role of directing different
forms of mammalian type IA topoisomerases to complexes involved in replication, transcription, recombination, or DNA repair.

Acknowledgments—We thank Richard Burgess for suggestions on the RNA polymerase affinity chromatography, Victor Fried for suggestions on the β′ subunit identification, and Marc Drolet for helpful discussions.

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*J. Biol. Chem.* 2003, 278:30705-30710.
doi: 10.1074/jbc.M303403200 originally published online June 4, 2003

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