Poly(ADP-ribose) polymerase (PARP) is a DNA-binding enzyme that plays roles in response to DNA damage, apoptosis, and genetic stability. Recent evidence has implicated PARP in transcription of eukaryotic genes. However, the existing paradigm tying PARP function to the presence of DNA strand breaks does not provide a mechanism by which it may be recruited to gene-regulating domains in the absence of DNA damage. Here we report that PARP can bind to the DNA secondary structures (hairpins) in heteroduplex DNA in a DNA end-independent fashion and that automodification of PARP in the presence of NAD<sup>+</sup> inhibited its hairpin binding activity. Atomic force microscopic images show that in vitro PARP protein has a preference for the promoter region of the PARP gene in superhelical DNA where the dyad symmetry elements likely form hairpins according to DNAse probing. Using a chromatin cross-linking and immunoprecipitation assay we show that PARP protein binds to the chromosomal PARP promoter in vivo. Reporter gene assays have revealed that the transcriptional activity of the PARP promoter is 4-5-fold greater in PARP knockout cells than in wild type fibroblasts. Reintroduction of the PARP promoter is 4-5-fold greater in PARP knockout cells thus providing support for PARP protein as a potent regulator of transcription including down-regulation of its own promoter.

Poly(ADP-ribose) polymerase (PARP, EC 2.4.2.30) is a chromatin-associated enzyme that catalyzes the transfer of successive units of the ADP-ribose moiety from NAD<sup>+</sup> to itself and other nuclear acceptor proteins (1). PARP is a zinc finger-containing protein, which allows enzyme binding to either double or single strand DNA breaks without any apparent sequence preference (2, 3). The catalytic activity of PARP is strictly dependent on the presence of strand breaks in DNA and is modulated by the level of automodification (4, 5). Data from many studies show that PARP is involved in numerous biological functions, all of which are associated with breaking and rejoining DNA strands, and it plays a pivotal role in DNA damage repair (2, 6-8).

Recent studies have implicated PARP in transcription of eukaryotic genes (9-16). PARP-dependent gene regulation involves poly(ADP-ribose)ylation of transcription factors, which, in turn, prevents their binding to specific promoter sequences (10). The basal transcription factors TFIID and TEF-1 as well as transcription factors TATA box-binding protein, YY1, SP-1, cAMP-response element-binding protein, p53, and NFκB are all highly specific substrates for poly(ADP-ribose)ylation (10, 11, 14, 16). PARP may also interact directly with gene promoters. For instance, recombinant full-length PARP bound the DNA sequences within the MCAT1 regulatory element (11) and to the DP4 protein binding site of the Pax-6 gene neuroretina-specific enhancer (17). Furthermore, PARP involvement in the active transcriptional DNA-protein complex formation on Reg promoter has been recently reported (12). Together these observations suggest that PARP may exert its function in transcription through direct binding to the gene-regulating sequences and through modification of transcription factors by poly(ADP-ribose)ylation. However, total dependence of PARP function on DNA strand breaks (5) does not provide a mechanism by which it may ADP-ribosylate transcription regulators and be recruited to gene-regulating sequences in the absence of DNA damage.

Based on the ability of PARP to interact with partially unwound DNA (18, 19), we reasoned that DNA secondary structures with single-stranded character may provide potential binding sites for PARP in gene-regulating sequences in the absence of DNA strand breaks. In this work we investigated the interactions between PARP protein and DNA structures of different complexity such as DNA heteroduplexes carrying stable secondary structures and superhelical DNA containing PARP promoter sequences. We found that PARP can recognize noncanonical conformations (hairpins) in a DNA end-independent fashion, and it is capable of in vitro binding to the PARP promoter sequences where the dyad symmetry elements may form the cruciforms. Using a chromatin cross-linking and immunoprecipitation assay we show that the human PARP promoter is an in vivo target for PARP protein. Further, we show that PARP protein down-regulates its gene promoter and that DNA binding activity of PARP is essential for its function in transcription.
Plasmid Constructs—The plasmid pPR-PARP was constructed by cloning the 5′-flanking region of the human PARP gene (from −899 to +156) fused to a chloramphenicol acetyltransferase reporter (20) into pCDNA 3.1 (Invitrogen) modified to remove the cytomegalovirus promoter. The 5′-deletion mutant of the parp promoter (pΔPR-PARP) was generated as described previously (20). The expression plasmid pCDNA 3.1 containing human PARP has been described previously (20). pPARP-DDB was constructed by cloning the PCR-generated fragment of cDNA (22) for human PARP-DDB (amino acids 1–303) tagged at its carboxy terminal with a sequence encoding four FLAG epitope tags into pCDNA 3.1. The integrity of all constructs was confirmed by sequence analysis.

DNA Heteroduplex Formation and Isolation—Heteroduplex formation between 301-bp PouI-PouII fragments of pUC8 and a similar fragment of pUC8F14C and isolation of the heteroduplex isomers were performed as described previously (23). Briefly, 10 μl of hybridization mixture containing 1 pmol of each DNA fragment in 100 mM NaCl, 50 mM Tris-HCl, pH 7.9, 1 mM dithiothreitol, 10 mM MgCl2 were incubated stepwise at 100 °C (1 min), 85 °C (10 min), and 70 °C (60 min) and then cooled to room temperature. Hybridization products were run in a 5% native polyacrylamide gel at 90 mM Tris borate (pH 8.3), 2.5 mM EDTA, and bands of heteroduplex fragments, which migrate slower than correctly annealed parental fragments (23), were excised. After an additional purification step using an UltraClean 15 DNA purification kit (MoBio, Solana Beach, CA), isolated heteroduplexes were resuspended in the reaction mixture containing 150 mM NaCl and 1 mM Tris-HCl, pH 7.6, for 1 min, rinsed with deionized water, and dried in a gentle nitrogen flow. The AFM images were obtained using a NanoScope IIIa instrument equipped with E-scanner (Digital Instruments, Santa Barbara, CA) operating in a tapping mode in air as described previously (28). The tapping frequency of the 125-μm silicon cantilever was 300–400 Hz, and the nominal scanning rate was set at 1–2 Hz. No less than 150 uncoupled DNA molecules and 100 PARP-DNA complexes were analyzed in each experiment.

Parp DNA binding reaction product in Mg2+−containing buffer (28) was deposited on an atomically flat mica surface, allowed to adsorb for 1 min, rinsed with deionized water, and dried in a gentle nitrogen flow. The AFM images were obtained using a NanoScope IIIa instrument equipped with E-scanner (Digital Instruments, Santa Barbara, CA) operating in a tapping mode in air as described previously (28). The tapping frequency of the 125-μm silicon cantilever was 300–400 Hz, and the nominal scanning rate was set at 1–2 Hz. No less than 150 uncoupled DNA molecules and 100 PARP-DNA complexes were analyzed in each experiment.

RESULTS AND DISCUSSION

Parp Binds to Hairpins in DNA Heteroduplexes—To investigate the interactions of PARP with DNA, we used AFM, which allows direct visualization of protein and DNA molecules at nanometer resolution (30–32). This approach was preferred to biochemical assays to address the hypothesis that PARP binding to DNA sites other than strand breaks was directed to single strand regions as observed in unwound structures in double-stranded DNA. Alternative DNA secondary structures are not thermodynamically stable in linear DNA fragments and, therefore, are not amenable to investigations of their functional transactions such as protein binding. Accordingly, our experimental approach used model heteroduplex constructs carrying stable DNA secondary structures. We used three-way junction heteroduplexes that contain 106-bp inverted repeats in one DNA strand (25) to form hairpin-like DNA structures (Fig. 1A). A representative AFM image shows that heteroduplex molecules have extrusions of the size expected for the 50-bp hairpin in the B conformation and bends at the junction (Fig. 1B).

After allowing full-length PARP protein to bind to the model hairpin-containing DNA, AFM images revealed a high incidence of DNA-protein complexes (~60% of all DNA molecules) that were divided into two types based on their locations in the heteroduplexes. In complexes of the first type, PARP associated directly to the cell culture medium to a final concentration of 1%, and fixation proceeded at 37 °C for 10 min as described in the Chip assay protocol (Upstate Biotechnology). Immunoprecipitation was performed with rabbit polyclonal anti-PARP antibody (Cell Signaling Technology). Cross-links were reversed by heating to 65 °C for 4 h in the presence of 200 mM NaCl followed by PCR analysis of DNA for the detection of the PARP promoter sequences using upstream (5′-TGTCA ACCCA GAGT GGCAT-3′) and downstream (5′-AACTA CTGGG GAGGC TGAA-3′) PCR primers designed according to the reported sequence data for the PARP 5′-region of the human PARP gene (27). Immunoprecipitation of PARP from cross-linked chromatin was analyzed by immunoblotting with goat polyclonal anti-PARP antibody (1:1000, R&D Systems) as described previously (20).

Sample Preparation and Imaging with AFM—DNA samples or PARP-DNA binding reaction product in Mg2+−containing buffer (28) were deposited on an atomically flat mica surface, allowed to adsorb for 1 min, rinsed with deionized water, and dried in a gentle nitrogen flow. The AFM images were obtained using a NanoScope IIIa instrument equipped with E-scanner (Digital Instruments, Santa Barbara, CA) operating in a tapping mode in air as described previously (28). The tapping frequency of the 125-μm silicon cantilever was 300–400 Hz, and the nominal scanning rate was set at 1–2 Hz. No less than 150 uncoupled DNA molecules and 100 PARP-DNA complexes were analyzed in each experiment.

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Transfections and Reporter Assays—Mouse embryonic fibroblasts deri-
(301-bp fragment of pUC8 and 401-bp fragment of pUC8F14C), thus indicating the specificity of PARP binding to hairpin-containing regions in double-stranded DNA. This finding presents a challenge to the generally accepted view that PARP binds only to strand breaks in DNA. In the presence of NAD\(^+\)/H\(_{11001}\), PARP bound to DNA strand breaks undergoes auto(ADP-ribosyl)ation, acquiring a high negative charge. Due to the charge repulsion the protein rapidly dissociates from DNA (4, 33, 34). Therefore, we next tested the ability of PARP to bind hairpin-containing DNA under conditions conducive to PARP automodification. Similar to our previous observations of PARP binding to DNA ends (28), NAD\(^+\) significantly decreased PARP affinity to the hairpins. Reversal of this effect was observed in the presence of 3-aminobenzamidine (Fig. 1F), a potent inhibitor of PARP catalytic activity. The relatively low yield of hairpin-protein complexes suggests that PARP has higher affinity to DNA ends than to hairpins in DNA fragments. These observations indicate that (i) PARP is capable of binding to certain secondary structures (e.g. hairpin-containing regions) in double-stranded DNA independently of the presence of DNA ends and (ii) NAD\(^+\)-dependent automodification of PARP results in inhibition of its hairpin binding activity.

**PARP Protein Binds to the 5’-Flanking Region of the PARP Gene**—Accumulating evidence supports the involvement of DNA secondary structures such as hairpins and cruciforms in transcription (34–38). We reasoned that PARP affinity for stem-loops in DNA might influence regulation of transcription in undamaged cells by binding to such domains in promoter regions. To test this hypothesis, we investigated interaction of the PARP protein with the 5’-flanking region of the PARP gene (20). Structurally, the PARP gene promoter is TATA-deficient and G + C-rich, typical of promoters that contain dyad symmetry elements with high propensity to form secondary structures such as cruciforms (39). Secondary structures are favored when DNA is negatively supercoiled and are not thermodynamically stable in linear DNA fragments (40). Therefore, we examined the PARP interactions with supercoiled (\(\sigma = -0.050\)) and topologically relaxed (\(\sigma = 0\)) pPR-PARP plasmids (Fig. 2, A and B). PARP binding reactions were performed using the same DNA to protein molar ratio (4:1) as in experiments with...
unidentified sites in the promoter region that are recognized by the promoter sequence by the computer algorithm MFOLD (Fig. 2). Several imperfect inverted repeats have been identified in the PARP promoter region (nt from 899 to +1). The position of dyad symmetry elements (DSE) in the promoter sequence and the hairpin free energies calculated by the MFOLD program are indicated in the boxed area. Putative P1 nuclease-sensitive sites are shown with arrows. B, pPR-PARP (topoisomers with superhelical density (σ) ranging from 0 to −0.111 were treated with P1 nuclease. The promoter-containing fragment (1.1 kb) was isolated and analyzed by alkaline agarose gel electrophoresis. The products of P1 nuclease digestion are denoted on the right. Topoisomer fractions 0−7 numbered at the bottom had the average σ of 0, −0.019, −0.031, −0.050, −0.065, −0.080, −0.094, and −0.111, respectively.

Hairpin-containing DNA heteroduplexes. AFM imaging of DNA-protein interactions revealed that PARP is capable of binding to supercoiled plasmid in a DNA end-independent fashion. Further, a quantitative evaluation of the AFM images revealed a 3−4-fold higher yield of DNA-protein complexes on a supercoiled plasmid compared with topologically relaxed DNA. These data suggest that the preferential binding of PARP to supercoiled plasmid is attributable to the formation of recognition sites for PARP in torsionally stressed DNA.

To examine PARP protein-promoter interactions in vitro, bound proteins were cross-linked to superhelical plasmid (σ = −0.050) with 0.5% glutaraldehyde, and the 1.1-kb fragment containing the promoter region was isolated and examined by AFM. An average of 1.2 protein molecules were bound to the promoter-containing DNA duplex, indicating that PARP recognizes certain relatively infrequent sites in the promoter region (Fig. 2C). Although the PARP binding site(s) in its own promoter is yet to be identified, our data might conceivably reflect polymerase interaction with the regions of single-stranded character that can be formed in superhelical DNA. One potential option is the formation of cruciform-like structures since several imperfect inverted repeats have been identified in the promoter sequence by the computer algorithm MFOLD (Fig. 3A). In support of this, we observed the appearance of yet unidentified sites in the promoter region that are recognized by the single strand-specific nuclease P1. These sites are generated by unwinding torsional stress in supercoiled DNA with a threshold value of superhelical density σ = −0.050 (Fig. 3B) and were not detected in relaxed covalently closed plasmid DNA. Based on the size of P1 nuclease-generated fragments, the positions of the putative unwound sites correspond to imperfect inverted repeat (nt −325−290) or an AT-rich region with dyad symmetry (nt −418−403) in the PARP promoter sequences. Although these data suggest that the 5′-flanking region of the PARP gene has the ability to adopt unwound or alternatively base-paired structures, further studies are required to assess functional transactions between PARP protein and such structures and to map PARP binding sites on the promoter.

To analyze the PARP protein-DNA interactions at the human PARP promoter in vivo we performed formaldehyde cross-linking and immunoprecipitation experiments. This approach permits analysis of DNA-binding proteins in eukaryotic cells under physiological conditions (41, 42). We observed that antibody immunoprecipitation effectively immunoprecipitated endogenous PARP protein and the 5′-flanking region of the PARP gene promoter (Fig. 4) from Ewing’s sarcoma cells that constitutively express PARP protein (20). This observation indicates that PARP protein is recruited to the human PARP promoter sequences in vivo. It remains to be determined whether PARP protein binds to the promoter sequences as a monomer or forms a heterodimer with yet to be identified transcriptional regulator(s). In support of the latter possibility, the physical association of PARP with transcription factors TEF-1, B-MYB, and AP-2 and its involvement in the active transcriptional DNA-protein complex on Reg and Pax-6 promoters have been recently demonstrated (11, 12, 17, 43, 44).

Transcriptional Autoregulation of the Human PARP Gene— The functional significance of PARP interactions with its gene promoter was evaluated by transient transfection assays using immortal fibroblasts (PARP+/−) derived from PARP knockout mice (29). We found that the transcriptional activity of the PARP promoter was 4−5-fold greater in PARP−/− cells than in wild type (PARP+/+) fibroblasts (Fig. 5A). Introduction of plasmid pCD12 carrying PARP cDNA into PARP−/− cells conferred transcriptional down-regulation of the PARP gene promoter (Fig. 5B). These data are in accord with the previously reported observations that inducible PARP expression in PARP-producing cells also inhibited PARP promoter activity (45), thus suggesting intrinsic autoregulation of PARP expression. Next we observed that deletion of the −899 to −95 region from the PARP promoter sequences alleviated PARP-mediated transcriptional inhibition (Fig. 5C) thus indicating that at least some of the functional sites that are required for PARP-mediated down-regulation of transcription may reside upstream of the minimal PARP promoter (nt from −95 to +156). This suggestion agrees with our earlier observations that the PARP promoter region (nt −420−290), harboring two putative unwound sites (at nt −418−403 and −325−290) (Fig. 3), is involved in negative control of the PARP promoter in cells.
naturally overexpressing PARP protein (20). To address the question whether catalytic activity of PARP is required for transcriptional down-regulation, the amino-terminal fragment of human PARP (amino acids 1–303) encompassing the region that encodes two zinc fingers of the enzyme and the proximal (amino acids 200–220) helix-turn-helix motif (22) was transiently expressed in PARP−/− cells. Co-transfection of the reporter gene (pPR-PARP) and a vector (pPARP-DDB) expressing a truncated PARP mutant (that contains the DNA-binding domain but lacks catalytic activity) resulted in transcriptional down-regulation of the PARP promoter in cells with a PARP−/− phenotype (Fig. 5B), indicating that PARP-mediated inhibition of transcription was independent of PARP catalytic activity. Together these data demonstrate that PARP protein is a potent repressor of transcription when targeted to the PARP promoter and up-regulates transcription in repressed cells. This alleviates the PARP-mediated block on the promoter and up-regulates transcription of its own and other genes involved in the DNA damage response. PARP-dependent inhibition of transcription elongation by RNA polymerase II in undamaged cells and up-regulation of mRNA synthesis in response to DNA damage have been recently demonstrated both in vitro and in vivo (13). Studies testing this hypothesis are underway.

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Transcriptional Repression by Binding of Poly(ADP-ribose) Polymerase to Promoter Sequences
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