B-MYB is implicated in cell growth control, differentiation, and cancer and belongs to the MYB family of nuclear transcription factors. Evidence exists that cellular proteins bind directly to B-MYB, and it has been hypothesized that B-MYB transcriptional activity may be modulated by specific cofactors. In an attempt to isolate proteins that interact with the B-MYB DNA-binding domain, a modular domain that has the potential to mediate protein-protein interaction, we performed pull-down experiments with a glutathione S-transferase-B-MYB protein and mammalian protein extracts. We isolated a 110-kDa protein associated endogenously with B-MYB in the nuclei of HL60 cells. Microsequence analysis and immunoprecipitation experiments determined that the bound protein was poly(ADP-ribose) polymerase (PARP). Transient transfection assays showed that PARP enhanced B-MYB transactivation and that PARP enzymatic activity is not required for B-MYB-dependent transactivation. These results suggest that PARP, as a transcriptional cofactor of a potentially oncogenic protein, may play a role in growth control and cancer.

B-MYB is a nuclear transcription factor belonging to the MYB family, which is expressed ubiquitously and is involved in cell growth control, differentiation, and cancer (1, 2). Virtually all proliferating cell lines transcribe the B-MYB gene in the G1/S phase of the cell cycle, although lower but detectable levels of B-MYB protein are also observed during the G0 and G2/M phases of the murine fibroblast cell cycle (3–7). B-MYB, similarly to other members of the family, binds to DNA through the consensus sequence (C/T)AACNG, resulting in transactivation of consensus site-bearing promoters. In practice, however, it has been quite difficult to detect B-MYB-dependent transactivation because of variable response in different cell types (8). The leading hypothesis describes B-MYB as a constitutively repressed molecule that requires post-translational modifications to disclose its activity. In this regard, several groups (9, 10) have shown that phosphorylation of the B-MYB protein induced by the Cdk2/cyclin A kinase results in activation of the B-MYB transactivating function. Activation is also achieved by truncation of the carboxyl terminus of the B-MYB molecule (11, 12). Although cyclin-induced phosphorylation relieves inhibition exerted by the carboxyl terminus, a B-MYB mutant deleted at the carboxyl terminus is activated by enforced expression of Cdk2/cyclin A (11). This suggests that, in addition to relieving intramolecular repression, phosphorylation may enhance B-MYB cross-talk with putative co-activators or may inhibit binding of co-repressors. This hypothesis is corroborated by evidence suggesting that the B-MYB transactivating function depends on the cellular context and that it correlates with the binding of cellular proteins to specific B-MYB domains (8).

Numerous reports have established a fundamental requirement for B-MYB expression during the growth and survival of normal and tumorigenic cell lines, whereas ectopic expression of a B-MYB transgene results in a perturbed cell cycle and differentiative pattern (13–18). These results correlate well with circumstantial evidence suggesting that abnormal B-MYB expression may contribute to tumorigenesis. For example, B-MYB is overexpressed during the progression of human small cell lung carcinoma, and high B-MYB expression is predictive, independently from other markers such as N-MYC, of poor survival of child bearing neuroblastoma tumors (19, 20). A current hypothesis is that B-MYB may activate sets of genes involved in the regulation of the cell cycle and/or DNA synthesis.

Klempnauer and co-workers (21) have shown that the v-MYB DNA-binding domain, which consists of three imperfect reiterations shared among all MYB proteins, interacts with the mammalian transactivator C/EBPβ. C/MYB and MYB-like proteins from mammalian to yeast also interact with regulatory proteins through the conserved tryptophan repeat structure (22–25). As a basis of the present study, we hypothesized that proteins binding to the B-MYB DNA-binding domain may function as regulatory molecules, contributing to B-MYB transcriptional activity. To address this issue, a hybrid protein consisting of GST fused to the B-MYB DNA-binding domain was used as bait and mixed with HL60 cell lysate to isolate binding proteins. A nuclear protein of 110 kDa, which was identified as poly(ADP-ribose) polymerase (PARP),1 bound to the GST-B-MYB fusion protein. PARP is associated with chromatin and catalyzes the transfer of poly(ADP)-ribose units to acceptor proteins. Interestingly, PARP and B-MYB share several characteristics. Both proteins are nuclear, and transcription of their genes is down-regulated during mammalian cell differentiation, and they are both able to suppress in vitro induced differentiation when expressed ectopically in mammalian cells (15, 17, 26, 27). Among other functions attributed to PARP is apoptosis regulation and DNA repair (28, 29). Recent studies have implicated PARP in the regulation of gene transcription through association with specific transcription factors. PARP can behave both as an inhibitor or stimulator of the bound transcription factors, suggesting that its biological role may depend on the availability of specific partners, the cell type, and the differentiative and/or proliferative status of the cell. For example, PARP binds to and inactivates the DNA-binding function of p53, and it is a suppressor of RAR/RXR signaling (30, 31). On the other hand, PARP increases the

1 The abbreviations used are: PARP, poly(ADP-ribose) polymerase; GST, glutathione S-transferase; wt, wild type; DBD, DNA-binding domain.
on-rate binding of nuclear proteins to the PAX-6 gene enhancer, resulting in increased gene transcription, and it is a co-activator of the AP2 transcription factor (32, 33). Furthermore, PARP associates with OCT-1, enhancing its recruitment onto DNA (34). Here we show that endogenous B-MYB and PARP co-immunoprecipitate from nuclear extracts of HL60 cells. Overexpression of PARP and B-MYB results in synergistic activation of a MYB-responsive promoter, suggesting that PARP is a B-MYB-specific co-activator.

MATERIALS AND METHODS

Cell Lines—The human osteosarcoma tumor cell line SAOS2 was obtained from ATCC and was cultured in Dulbecco’s modified Eagle’s medium supplemented with 15% fetal calf serum. The human hematopoietic HL60 cell line was cultured in RPMI 1640 medium with 10% fetal calf serum.

Plasmid Constructions—An EcoRI-BamHI fragment (900 base pairs) containing the DNA-binding domain of human B-MYB, was cloned into pGEX2T (Amersham Pharmacia Biotech) and cut with EcoRI-BamHI to generate pGST-B-MYB-DBD. This construct encodes the glutathione S-transferase protein (GST) in-frame with the B-MYB DNA-binding domain. The construction was verified by sequencing.

Recombinant Protein Production and Purification—Bacteria strain BL21 was used for producing the GST-B-MYB-DBD fusion protein. After induction for 2 h with 0.1 mM isopropyl-β-D-thiogalactopyranoside, the bacteria were harvested and resuspended in phosphate-buffered saline. The cells were treated by several cycles of sonication on ice and centrifuged. The supernatant, containing soluble recombinant protein, was incubated with glutathione-agarose beads (Amersham Pharmacia Biotech) for 1 h at 4 °C, washed three times with phosphate-buffered saline, and used directly for protein binding assays.

Metabolic Labeling and Protein Binding Assay—5 × 10^9 HL60 cells were cultured for 3 h in methionine- and cysteine-free RPMI medium supplemented with 10% dialyzed fetal bovine serum (Life Technologies, Inc.) containing pro-Mix (20 mM KOH, 1 mM EDTA, 5 mM MgCl₂, 0.5 M NaCl, 100 mM NaOH, 0.1 M MgCl₂, 0.5 mM NaN₃, 20% glycerol, protease inhibitors) for 10 min at 4 °C and 5% dialyzed fetal bovine serum, and used directly for protein binding assays.

Protein Microsequencing—A total of 1 × 10^9 HL60 cells were lysed. After pre-clearing with GST-coated beads, nuclear extracts were adsorbed with GST-B-MYB-DBD beads. After washings, the bound proteins were eluted with SDS sample buffer and analyzed on a 10% SDS-polyacrylamide gel. PARP protein was translated in vitro from the pSPARP plasmid (a kind gift of Dr. Perry Kannan) with the TNT kit according to manufacturer’s instructions (Promega).

Immunoprecipitation/Western Blot—Nuclear HL60 cell lysates were pre-cleared with 50 μl of protein A-Sepharose and immunoprecipitated with PARP polyclonal or monoclonal antibody (Santa Cruz Biotechnology or Biomol), B-MYB (10), or control antibody (c-FGR, Santa Cruz Biotechnology) for 1 h at 4 °C in a buffer containing 10 mM Tris-HCl, pH 7.2, 150 mM NaCl, 0.1% Nonidet P-40, 30 μl of protein A-Sepharose was added, and the samples were rocked for an extra hour at 4 °C. After five washes, samples were run on an SDS-polyacrylamide gel and transferred to nitrocellulose. Western blot was performed with B-MYB or PARP antibody in Blotto (5% dry-milk in phosphate-buffered saline) plus 0.1% Nonidet P-40. After washings and incubation with secondary antibody coupled to peroxidase, the filter was developed using a chemiluminescent substrate according to manufacturer’s instructions (ECL, Amersham Pharmacia Biotech).

Transient Transfections and Luciferase Assay—SAOS2 cells were transfected in 30-mm plates according to the calcium-phosphate precipitation method, as described (10). pCMV-B-MYB (14), pPARP31(35), pGL2-MIM (36), pDNA3-3-PARPwt, pDNA3-C908R, or M890V (31) were transfected at a 1:1 ratio (1 μg each) and were incubated with nuclear (N) or cytoplasmic (C) cell lysates obtained from HL60 cells that were metabolically labeled with [35S]methionine. To isolate cellular proteins interacting with the fusion proteins, the reaction mix was incubated with glutathione-Sepharose resin, as described under “Materials and Methods.” After washings, the Sepharose beads were washed onto a 10% polyacrylamide gel, and the cellular proteins bound to the fusion proteins were revealed by autoradiography. The arrows indicate nuclear and cytoplasmic proteins consistently interacting with the fusion proteins in several independent experiments.

RESULTS

A Nuclear Protein of 110 kDa Binds to the B-MYB DNA-binding Domain—In order to identify proteins interacting with the amino terminus of the B-MYB protein, HL60 cells were metabolically labeled with [35S]methionine, and nuclear or cytoplasmic protein lysates were prepared. Protein lysates were incubated with GST-B-MYB fusion protein, and cellular proteins binding to GST-B-MYB were analyzed by SDS-PAGE and autoradiography. Several cytoplasmic and nuclear proteins bound to the bait, and a nuclear protein of approximately 110 kDa (p110) was consistently eluted in multiple independent experiments. The B-MYB/p110 interaction was specific, since p110 bound the A-MYB DNA-binding domain, which is 75% identical to the B-MYB domain, with much lower affinity, and it did not bind to GST (Fig. 1, panel N). Several cytoplasmic proteins bound the fusion proteins, and a 22-kDa protein was consistently co-eluted with both A-MYB and B-MYB (Fig. 1, panel C). The identity of this cytoplasmic protein was not investigated further.

![Image](http://www.jbc.org/)

**FIG. 1.** A 110-kDa protein binds to the B-MYB-DNA-binding domain. Fusion proteins (GST-A-MYB-DBD and GST-B-MYB-DBD) or GST alone were incubated with nuclear (N) or cytoplasmic (C) cell lysates obtained from HL60 cells that were metabolically labeled with [35S]methionine. To isolate cellular proteins interacting with the fusion proteins, the reaction mix was incubated with glutathione-Sepharose resin, as described under “Materials and Methods.” After washings, the Sepharose beads were washed onto a 10% polyacrylamide gel, and the cellular proteins bound to the fusion proteins were revealed by autoradiography. The arrows indicate nuclear and cytoplasmic proteins consistently interacting with the fusion proteins in several independent experiments.

PARP antibody in Blotto (5% dry-milk in phosphate-buffered saline) plus 0.1% Nonidet P-40. After washings and incubation with secondary antibody coupled to peroxidase, the filter was developed using a chemiluminescent substrate according to manufacturer’s instructions (ECL, Amersham Pharmacia Biotech).

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![Diagram](http://www.jbc.org/)

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in vitro translated PARP binds to GST-B-MYB-DDBD. A, binding reactions were carried out with nuclear lysates from HL60 cells and GST-B-MYB or, as a control, GST alone. Proteins were mixed with glutathione-Sepharose beads that were washed several times before being loaded onto a SDS-mini gel. Proteins were transferred to a nitrocellulose filter and subjected to Western blot analysis with a PARP-specific antibody. 1st lane, HL60 nuclear lysate; 2nd lane, GST eluate; 3rd lane, GST-B-MYB eluate. B, PARP protein was translated in vitro in the presence of [35S]methionine and mixed with either 5 μg of GST-B-MYB or 5 μg of GST. After binding to glutathione resin, the proteins were eluted in sample buffer and loaded onto a 10% SDS gel. Lower panel shows the Coomassie Blue-stained gel to evaluate loading of GST and GST-B-MYB proteins.

The 110-kDa Protein Interacting with the B-MYB-DNA-binding Domain Is PARP—A preparative gel was loaded with the eluate from GST-B-MYB protein mixed with nuclear lysates from 10⁸ HL60 cells. Protein microsequencing determined that the 110-kDa protein was PARP. To identify positively PARP as the interacting protein, the eluted material was loaded onto an SDS-PAGE mini-gel and subjected to Western blot analysis with a PARP-specific monoclonal antibody. Indeed, eluate from GST-B-MYB, but not from GST alone, contained PARP protein that was recognized by the PARP antibody (Fig. 2A). To corroborate the hypothesis that PARP and B-MYB are able to form a stable heterodimer in vitro, we mixed [35S]methionine-labeled PARP with GST-B-MYB fusion protein. In vitro translated PARP bound efficiently to B-MYB, but not to GST, as detected by pull-down experiments (Fig. 2B). The B-MYB/PARP interaction is existing naturally in the living cell, since Western blot analysis showed that the PARP antibody, but not the control antibody, was able to co-immunoprecipitate B-MYB protein from HL60 nuclear extracts (Fig. 3A). Consistent with this result, a B-MYB-specific polyclonal antibody was able to co-immunoprecipitate PARP protein from HL60 cells (Fig. 3B).

PARP Enhances the Transactivating Activity of B-MYB—Since PARP is binding to the B-MYB DNA-binding domain, it is likely that the interaction may affect B-MYB transactivating function. To address this point, we co-transfected a luciferase reporter plasmid, containing a MYB-binding site, with expression vectors encoding for PARP and B-MYB. Transfection of PARP alone decreased basal transcription, perhaps due to its suppressive activity on RNA polymerase II (37). In sharp contrast, co-expression of PARP and B-MYB resulted in synergistic activation of the MYB-responsive promoter (Fig. 4A). To investigate whether the enzymatic activity of PARP is required in the co-activating function, we took advantage of two enzyme-dead mutants (C908R and M890V) carrying an amino acid substitution at the catalytic domain (38). Although at different levels, both PARP mutants were able to cooperate with B-MYB suggesting that poly(ADP)-ribosylation activity is not required for the co-activating function of PARP (Fig. 4B).

DISCUSSION
Growing experimental evidence suggests that transcription factors are regulated by physical association with co-activators and co-repressor molecules interacting with multifunctional protein domains. The DNA-binding domain of MYB proteins, the so-called tryptophan repeat structure, has the potential of mediating contact with both DNA and proteins. In this report we show that PARP binds to the B-MYB-DNA-binding domain and enhances its transactivating activity. Interestingly, another member of the MYB family, A-MYB, binds PARP with less efficiency, suggesting that PARP may selectively modulate B-MYB, but not A-MYB, activity (Fig. 1). Although the c-MYB and A-MYB proteins show a 90% amino acid identity in the DNA-binding domain region, homology between A-MYB and B-MYB is only 75%, perhaps explaining the different affinity for PARP displayed by the two proteins (2). Several reports have suggested that PARP can behave as an inhibitor or stimulator of transcription (reviewed in Ref. 39). It has been shown that poly(ADP)-ribosylation is required for both inhibition and stimulation of activated transcription and that PARP enzymatic activity is greatly enhanced by DNA strand breaks (30, 31, 39). PARP participates to DNA-damage surveillance by association with the DNA polymerase-α-primase complex, and PARP is required for DNA polymerase activity (40). In the present study we provide evidence that PARP co-activates B-MYB and that poly(ADP)-ribosylating activity is not required for this function, since PARP proteins devoid of enzymatic activity retain the ability of cooperating with B-MYB (Fig. 4B). Collectively, existing data suggest a model in which, in the presence of DNA damage, PARP is activated and brought to DNA, favoring repair and gene silencing (41-42). On the other hand, during the normal cell cycle, PARP may be recruited by transactivator proteins, such as B-MYB, to regulate positively gene transcription. Since PARP enzymatic activity does not play a role, it is likely that the physical interaction occurring between PARP and the B-MYB DNA-binding domain is essential for the synergistic effect. Truncation of the carboxyl terminus of B-MYB activates B-MYB transactivating activity,
suggesting that B-MYB is a constitutively repressed molecule (1). B-MYB transactivating function also can be enhanced by cyclin A/CDK2 kinase-induced phosphorylation (1). Repressor molecules bound to different B-MYB-domains (or negative intramolecular interactions) may be neutralized by phosphorylation of B-MYB protein and/or by interaction with cofactor molecules such as PARP, resulting in a conformational change and increased transactivating capacity. Also, cofactor molecules may favor the cross-talk between B-MYB and the basal transcription machinery. In preliminary experiments we have been able to show that the PARP/B-MYB interaction is increased in the presence of an oligonucleotide containing an MYB-binding site (data not shown). This suggests that a multiprotein complex containing PARP and B-MYB may cooperatively bind to the MYB-binding site at target promoters resulting in synergistic gene activation. The mechanistic interactions occurring between B-MYB and PARP are worthy of further investigations to define the exact role of PARP in activated transcription regulation. Studies have suggested that abnormal expression of the B-MYB gene may be implicated in the progression of human neoplasias, perhaps through its ability to stimulate transcription of genes related to cell cycle progression and/or apoptosis resistance (1, 19, 20). It would be of interest to study B-MYB effects on cells with a disrupted PARP gene to elucidate further the relationship linking regulation of gene transcription with cellular functions such as proliferation, differentiation, and apoptosis.

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Poly(ADP-ribose) Polymerase Is a B-MYB Coactivator

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