Identification of SWI-SNF Complex Subunit BAF60a as a Determinant of the Transactivation Potential of Fos/Jun Dimers*

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Taiji Ito, Mai Yamauchi, Mitsue Nishina, Nobutake Yamamichi, Taketoshi Mizutani, Motoyasu Ui, Masao Murakami, and Hideo Iba‡

From the Department of Gene Regulation, Institute of Medical Science, University of Tokyo, 4-6-1 Shirokanedai, Minato-ku, Tokyo 108-8639, Japan

Fos family proteins form stable heterodimers with Jun family proteins, and each heterodimer shows distinctive transactivating potential for regulating cellular growth, differentiation, and development via AP-1 binding sites. However, the molecular mechanism underlying dimer specificity and the molecules that facilitate transactivation remain undefined. Here, we show that BAF60a, a subunit of the SWI-SNF chromatin remodeling complex, is a determinant of the transactivation potential of Fos/Jun dimers. BAF60a binds to a specific subset of Fos/Jun heterodimers using two different interfaces for c-Fos and c-Jun, respectively. Only when the functional SWI-SNF complex is present, can c-Fos/c-Jun (high affinity to BAF60a) but not Fra-2/JunD (no affinity to BAF60a) induce the endogenous AP-1-regulated genes such as collagenase and c-met. These results indicate that a specific subset of Fos/Jun dimers recruits SWI-SNF complex via BAF60a to initiate transcription.

Transcription factor AP-1, which plays pivotal roles in cell growth, differentiation, development, and tumor formation, is composed of Fos family proteins (Fos; c-Fos, Fra-1, Fra-2, and FosB) and Jun family proteins (Jun; c-Jun, JunB, and JunD). The members of both families form dimers through a leucine zipper structure; Jun family members can form low-affinity homodimers and high affinity heterodimers with the Fos family (1, 2). However, members of the Fos family do not form stable homodimers. Although these hetero- and homodimers bind to similar sites on DNA (TGAC/GTCA, AP-1 binding sites) through the basic domains of both proteins, each dimer has a distinct transcriptional regulatory function that can be modulated either positively or negatively (3). For example, transcriptional activity of the c-Fos/c-Jun dimer is much higher than the Fra-2/c-Jun dimer. Although functional interactions between some members of the Fos/Jun family of proteins and adaptor proteins such as CREB-binding protein (CBP) or TATA-binding protein (TBP) have been reported (4, 5), crucial proteins that recognize the dimer specificity and/or facilitate the induction of transcriptional initiation were largely unknown. Therefore, we proposed here to isolate such a crucial protein using a yeast two-hybrid system and have demonstrated that BAF60a (6), a component of the SWI-SNF chromatin remodeling complex, can select specific Fos/Jun dimers and function as a determinant of transcriptional activation via AP-1 binding sites.

EXPERIMENTAL PROCEDURES

Plasmid Construction—The Gal4DBD-c-Jun (25–187) fusion construct (pDBLeu-cJ(25–187)) was generated by inserting the 0.49 kilobase pair EheI fragment of the rat c-jun gene into the MacI restriction endonuclease cleavage site within the open reading frame of Gal4DBD. For the construction of template DNA for in vitro translation, we first generated a starter plasmid from pGEM2–475Jun-D, the translation initiation site of which has a Kozak consensus sequence with a unique Ncol site that is preceded by a fragment 475 sequence for an efficient translational initiation (2). The 1.0-kilobase pair NcoI-BamHI fragment of pGEM2–475/Jun-D encoding JunD was excised and substituted with a synthetic DNA fragment carrying multiple cloning sites to generate pGEM2–475/Met. A series of c-jun or c-fos deletions was inserted into frame into pGEM2–475/Met. Full-length BAF60a cDNA was constructed by inserting a PCR-generated DNA fragment encoding the N-terminal region into the original partial BAF60a clone, which encodes BAF60a, amino acids 139–475, and was isolated by yeast two-hybrid screening. Glutathione S-transferase (GST)-BAF60a was constructed by inserting the full-length DNA into the pGEX4T vector (Amersham Pharmacia Biotech). For the construction of a retrovirus vector expressing HA-tagged proteins, a synthetic oligo DNA fragment coding the HA sequence was inserted into the unique BamHI site of pBabe-IREs puro (7) to generate pBabeHA-IREs puro. The DNA fragment was generated by annealing the following two nucleotides: 5′-gctaactagctactatgtgttccagattgctagcctcgcctcgagtggccgacagcgtctc-3′ and 5′-gataatacactgtacctgctggctggcgagagcgggtgactcggcgcgaagcgtctc-3′.

Yeast Two-hybrid Screening—The yeast strain MaV203 (MATa, leu2–3,112, trp1–901, his3Δ200, ade2–101, gal1ΔA, gal10ΔA, SPAL10: URA3, GAL1: lacZ, HIS3 gal10ΔA HIS3 gal10ΔA HIS3 gal10ΔA, can1Δ, cyh2Δ) harboring pDBLeu-cJ (25–187) (Fig. 1A) was transformed by the lithium acetate method with a mouse brain cDNA fusion library inserted into the activation domain vector, pC86 (Life Technologies, Inc.). Transformants were selected with a HIS3 reporter containing the Gal4 DNA binding sites by seeding onto SC/Leu−/Tryp−/His+·3AT (50 μM) plates. After incubation for 60 h at 30 °C, the plates were replicated for the reduction of false-positive clones according to the manufacturer’s protocol and further incubated at 30 °C for 44 h. The positive clones were confirmed by another promoter system that induces β-galactosidase expression through the same DNA binding domain. β-Galactosidase activity in yeast was assayed according to the manufacturer’s protocol.

Cell Culture, Transfection, and Virus Transduction—Virus-packaging cell line BOSC23 and the adenocarcinoma cell line SW13 were grown in Dulbecco’s modified Eagle’s medium supplemented with 10% fetal calf serum. NIH3T3 cells were grown in Dulbecco’s modified Eagle’s medium supplemented with 10% fetal calf serum. BOSC23 cells were transfected with pBabeHA-BAF60a-IREs puro, and NIH3T3 cells were

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‡ To whom correspondence should be addressed. Tel.: 81-3-5449-5730; Fax: 81-3-5449-5449; E-mail: iba@ims.u-tokyo.ac.jp.

1 The abbreviations used are: PCR, polymerase chain reaction; RT-PCR, reverse transcription-PCR; GSH, glutathione; GST, glutathione S-transferase; CAT, chloramphenicol acetyltransferase; GAPDH, glyceraldehyde-3-phosphate dehydrogenase; GR, glucocorticoid receptor.

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Recruitment of SWI-SNF Complex by Fos/Jun Dimers

**RESULTS**

To identify the molecules involved in the transactivation of AP-1, we first proposed to isolate proteins that specifically bind with rat c-Jun N-terminal amino acids 25–187 (Fig. 1A), which reportedly contain transactivation domains (10). Using the yeast two-hybrid system, 23 positive clones were obtained upon screening $5 \times 10^5$ colonies of a mouse brain cDNA library. DNA sequence analysis revealed a single cDNA clone encoding the C-terminal half of mouse BAF60a, which is a subunit of the mouse SWI-SNF complex. The SWI-SNF complex is a 2-MDa, multi-subunit, DNA-dependent ATPase that is thought to displace repressive chromatin structure (11). Because interaction of c-Jun with the SWI-SNF complex seemed likely to be important for regulation of transcription in vivo, we isolated a cDNA containing the entire open reading frame of BAF60a, which had the same nucleotide sequence as a clone reported previously (8). Escherichia coli-produced, histidine-tagged c-Jun amino acids 1–225 (9) bound with GST-BAF60a fusion protein but not with GST (data not shown), demonstrating that BAF60a interacts directly with c-Jun. By synthesizing truncation mutants of c-Jun in reticulocyte lysates, we showed that N-terminal amino acids 25–187 of c-Jun were sufficient for binding, but further truncation at either the N-terminal or C-terminal regions totally abolished binding (Fig. 1A).

To examine whether BAF60a binds with other members of the Jun family, JunB and JunD proteins were synthesized in reticulocyte lysates and assayed for binding with a recombinant GST-BAF60a fusion protein. In comparison with BAF60a binding with c-Jun, BAF60a displayed weaker binding with JunB and no binding with JunD (Fig. 1C). This result was somewhat surprising because the BAF60a binding domain of c-Jun includes regions conserved in both JunB and JunD (conserved regions 1–3; Fig. 1A). Even more surprising was the finding that GST-BAF60a fusion protein bound with in vitro translated, full-length c-Fos (Fig. 1D); c-Fos and c-Jun share little sequence similarity outside of their leucine zipper domains. Among the Fos family proteins, this binding is specific for c-Fos (Fig. 1D). Analysis of c-Fos truncation mutants indicated that C-terminal amino acids 237–380 are sufficient for BAF60a binding (Fig. 1B). Binding of BAF60a with v-Fos, which is derived from FBJ-MuSV (12), was not detected (Fig. 1B). The C-terminal amino acids of v-Fos and c-Fos are divergent because of a frameshift mutation starting at c-Fos amino acid 333 (13) (Fig. 1B). Therefore, c-Fos C-terminal amino acids 333–380 are likely to be critical for binding with BAF60a.

Fos/Jun heterodimers composed of c-Fos and c-Jun, c-Fos and JunD, Fra-2 and c-Jun, or Fra-2 and JunD were assayed for binding with GST-BAF60a by precipitation with glutathione-Sepharose beads. The bound proteins were boiled in SDS and separated by SDS-10% polyacrylamide gel electrophoresis. A fraction (12.5%) of the total labeled Jun or Fos proteins was also analyzed on the same gel (Fig. 1A).
mixtures were analyzed by precipitation with GSH-Sepharose-bound min at 37 °C to complete the dimer formation as described (3). The immediately after synthesis in reticulocytes lysates and incubated for 30 min at 37 °C to complete the dimer formation as described (3). The mixtures were analyzed by precipitation with GSH-Sepharose-bound GST-BAF60a.

![Fig. 2](image)

**Fig. 2. BAF60a binds with a specific subset of Fos/Jun dimers in vitro.** The interactions between GST-BAF60a and c-Fos/c-Jun (cF + cJ) (A), c-Fos/JunD (cF + cJD) (B), Fra-2/c-Jun (F2 + cJ) (C), and Fra-2/JunD (F2 + cJD) (D) dimers are shown. Equimolar amounts of 35S-labeled c-Fos (or Fra-2) and c-Jun (or JunD) were mixed immediately after synthesis in reticulocyte lysates and incubated for 30 min at 37 °C to complete the dimer formation as described (3). (A) The mixtures were analyzed by precipitation with GSH-Sepharose-bound GST-BAF60a.

one (GSH-Sepharose beads. GST-BAF60a precipitated 4% of c-Fos alone. Precipitation of c-Fos with BAF60a increased to 7.5% of the total c-Fos when preincubated with an equimolar amount of c-Jun (Fig. 2A). Because the molar amount of c-Jun recovered in the precipitate was nearly equal to the amount of c-Fos, it seems that binding of BAF60a with the c-Fos/c-Jun heterodimer would be preferential to binding with c-Fos alone. Fra-2 alone, JunD alone, and the Fra-2/JunD heterodimer displayed only marginal binding with BAF60a (Fig. 2D). JunD alone displayed marginal binding with GST-BAF60a, whereas precipitation of the c-Fos/JunD heterodimer with GST-BAF60a resulted in the recovery of 1.5% of the total JunD added to the reaction mixture.

These results suggest that BAF60a can simultaneously bind restricted members of Fos family proteins (c-Fos alone) and Jun family proteins (c-Jun, and probably to a lesser extent JunB but not JunD) using different interfaces present in the BAF60a molecule. These results also suggest that the Fos/Jun dimer formation via the leucine zipper region is compatible with formation of a ternary complex composed of Fos, Jun, and BAF60a. A c-Fos/JunD heterodimer bound with GST-BAF60a, but the binding was less than that observed with c-Fos alone (Fig. 2B), suggesting that dimerization of c-Fos and JunD did not appreciably contribute to BAF60a binding. Similarly, dimerization of Fra-2 and c-Jun did not seem to contribute to BAF60a binding (Fig. 2C). The binding affinity of each heterodimer examined here correlated positively with their transactivating activity when determined in F9 cells by transient expression with a reporter gene containing a single AP-1 binding site derived from the human collagenase gene (Table I). c-Fos/c-Jun had the highest transactivating activity, and other dimers consisting of either c-Fos or c-Jun had lower activity. Dimers that contained neither c-Fos nor c-Jun had only marginal activity.

To examine whether BAF60a affects the specific DNA binding activity of c-Fos/c-Jun heterodimer, electrophoretic mobility shift assays were performed with 32P-labeled probe containing the AP-1 DNA binding site of the human collagenase gene. Gel mobility shift was not affected upon addition of GST alone, but a dramatic enhancement of the shifted band was detected upon the addition of GST-BAF60a (Fig. 3A). These results indicate that BAF60a stimulates the specific DNA binding activity of the c-Fos/c-Jun heterodimer. When the Fra-2/JunD dimer was assayed, the addition of GST-BAF60a caused only a slight enhancement of the shifted band (data not shown).

To study whether c-Fos/c-Jun dimer associates with BAF60a in vivo, NIH3T3 cells expressing HA-tagged BAF60a were growth-stimulated (G1) to induce endogeneous c-Fos and c-Jun. Cell lysates were mixed with a Ni2+ beads bound with histidine-tagged c-Jun. Proteins that bound with histidine-tagged c-Jun were recovered and analyzed by Western blotting. They contained not only HA-tagged BAF60a but also Brm (14), the catalytic subunit (DNA-dependent ATPase) of the SWI-SNF complex (Fig. 3B). When cell lysates of growth-stimulated (G1) NIH3T3 cells were precipitated with anti-c-Jun antisera and analyzed by Western blotting, the immunoprecipitates contained Brm and SWI-SNF complex subunit INI1 (Fig. 3C) (14). Both proteins disappeared when the anti-c-Jun antisera was preabsorbed by the antigen. Similar results were obtained when immunoprecipitations were performed with anti-c-Fos antisera. No Brm or INI1 proteins were detected when experiments were performed in growth-arrested (G0) cells, which express no Fos protein (Fig. 3C). Taken together, it was shown that c-Fos and c-Jun associate with the SWI-SNF complex through BAF60a in vivo.

These results indicate that BAF60a has a versatile protein interface for c-Fos, c-Jun, and the other SWI-SNF complex subunits. Mutational analysis (Fig. 4) of GST-BAF60a revealed the location of a large domain spanning amino acids 129–366, which is responsible for binding with either c-Fos or c-Jun synthesized in reticulocyte lysate. Next, we located domains within the primary structure of BAF60a that bind with other SWI-SNF complex subunits. GST-BAF60a truncation mutants were mixed with cellular lysates prepared from NIH3T3 cells, subsequently precipitated with GSH-Sepharose, and analyzed by Western blotting with anti-Brm antisera. As expected, Brm was precipitated with the full-length BAF60a. Moreover, the same centrally located domain of BAF60a that binds with c-Fos or c-Jun also binds with the other subunits of the SWI-SNF complex. We speculate that these 238 amino acids form a versatile pocket structure and that any truncation of this region perturbs the three-dimensional structure of the domain, which leads to a simultaneous loss of binding with the c-Fos, c-Jun, and SWI-SNF subunits that associate directly with BAF60a.

We next asked whether the transactivating activity of c-Fos/c-Jun, the dimer that binds BAF60a with the highest affinity, is modulated by the functional SWI-SNF complex in vivo. Assays were performed with human adenocarcinoma cell line SW13 because it expresses undetectable levels of Brm and BRG1 (14), which are critical catalytic (DNA-dependent ATPase) subunits of the SWI-SNF complex. In mammalian cells, each SWI-SNF complex contains either Brm or BRG1 but
not both (6). Therefore, the introduction of Brm or BRG1 into SW13 would enable the formation of functional SWI-SNF complex. Transfection of an expression plasmid encoding either c-Fos, BRG1, or Brm into SW13 cells did not transactivate a CAT reporter plasmid. Although each RNA contained similar amount of GAPDH mRNA as judged by RT-PCR, semiquantitative RT-PCR indicated that the collagenase gene was inducible through AP-1 binding sites in cells originating from adrenal cortex. Total RNA was isolated from SW13 cells transfected with the same set of expression vectors but not with the CAT reporter plasmid. Although each RNA contained similar amount of GAPDH mRNA as judged by RT-PCR, semiquantitative RT-PCR indicated that the collagenase gene was inducible, dependent on transfection of expression vectors for c-Fos, c-Jun, and Brm (or BRG1) (Fig. 5B). These results indicate that unlike the results of CAT analysis, BRG1 is equally efficient for the induction of native collagenase gene expression. As for the c-met gene, induction is dependent upon the expression of c-Fos, c-Jun, and BRG1 (Fig. 5B). Brm has only marginal effects on transactivation of this gene, suggesting that the Brm or BRG1 subunit has distinct target specificity to facilitate gene activation. Brm- or BRG1-dependent transactivation was marginally detected when fra-2 and junD were transfected instead of c-fos and c-jun (Fig. 5B).

**DISCUSSION**

We demonstrated here that SWI-SNF complex subunit BAF60a binds with distinct affinities to different Fos/Jun dimers by interacting with the N-terminal region of c-Jun and the C-terminal region of c-Fos (Fig. 1). BAF60a binds the c-Fos/c-Jun heterodimer with the highest affinity and the other dimers containing either c-Fos or c-Jun with lower affinity. Moreover, deletions in c-Fos and c-Jun that contain neither c-Fos nor c-Jun have no binding activity (Fig. 2). The binding affinity of each Fos/Jun heterodimer to BAF60a is correlated strongly with the respective transactivating potential of each heterodimer in F9 cells (Table I). These observations indicate that the SWI-SNF complex is a major determinant of transactivation potential of Fos/Jun dimers. Indeed, in SW13 cells, which lack functional SWI-SNF complex, the transacti-
vating activity of Fos/Jun dimers is kept at basal levels. However, transactivation by c-Fos/c-Jun heterodimer but not by Fra-2/JunD was strongly enhanced by supplying Brm or BRG1 into SW13 to recover the functional SWI/SNF complex (Fig. 5).

Although we did not address the biological function of BAF60a intensively, NIH3T3 cells expressing HA-BAF60a at high levels by retrovirus transduction showed no clear effects on cellular growth. These cells did not acquire anchorage-independent growth at all, indicating that simply a high level expression of BAF60a is not sufficient to induce cellular transformation in NIH3T3 cells (data not shown).

Although several functional models have been presented for the yeast or mammalian SWI/SNF complex (11), our observations on AP-1 and recent reports on glucocorticoid receptor (GR) (17–19), c-Myc (20), GCN4 (21, 22), and C/EBPβ (23) support a model in which transcription factors recruit the SWI/SNF complex to target genes (24). In these previous works, IN11 (associates with c-Myc), Brm (associates with C/EBPβ), and Brm or BRG1 (associates with GR) but not BAF60a were identified as the SWI/SNF complex subunits that bind directly to transcription factors. These results indicate that the 2-MDa, multi-subunit SWI/SNF complex has many different interfaces that interact with various transcription factors and thereby function as a global transcriptional regulator. Because the transactivating function of GR in yeast is known to require Swp73p (18), the yeast homologue of BAF60a, we can speculate that AP-1 (especially c-Fos/c-Jun) and GR compete for most of the integral subunits of the limited numbers of SWI/SNF complex present in the cell (25) for their transactivating activity. Therefore, competition between AP-1 and glucocorticoid receptor transcription factors for SWI/SNF complex would support in part the molecular mechanisms for the mutual repression of transcription frequently observed between these two factors (26–28).

It is quite interesting that c-Fos and c-Jun are recognized specifically by BAF60a among Fos family proteins and Jun family proteins, respectively. Both c-fos and c-jun were isolated originally as cellular counterparts of oncogenes carried by natural RNA tumor viruses (FBJ-MuSV (12) and ASV17 (29), respectively), and their products are known to be the strong transactivators among the family of proteins. Because c-fos and, to a lesser extent, c-jun are immediate early genes, the c-Fos/c-Jun dimer would be formed most promptly after extracellular stimuli and would start to interact with the AP-1 binding site (30, 31). Initially, the chromatin structure would be in an inactive context for transactivation of AP-1-regulated genes. High affinity binding of c-Fos/c-Jun with BAF60a efficiently recruits SWI/SNF complex and thereby induces remodeling of the adjacent chromatin. Subsequently, basal transcriptional machinery and transactivator proteins such as CBP and p300 (4, 5) are recruited for the initiation of transcription.

Because the SWI/SNF-remodeled conformation of the nucleosome in some experimental systems has been shown to persist after detachment of the SWI/SNF complex (32), it is possible that the promoters within the modified chromatin remain accessible to regulation by other Fos/Jun dimers that are induced in later stages, even if these dimers cannot recruit SWI/SNF complex. In summary, the mechanistic link between heterodimeric Fos/Jun transcription factors and chromatin remodeling complexes provided here is essential for understanding the underlying mechanisms that explain the astonishing diversity of AP-1 transcription factor functions in growth, differentiation, development, and tumor formation.

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FIG. 5. c-Fos/c-Jun but not Fra-2/JunD recruits SWI/SNF complex to transactivate AP-1 target genes. A, transactivating activity determined by CAT analysis. SW13 cells (3 × 10⁵ cells/60-mm dish) were transfected with LipofectAMINE reagent (Life Technologies, Inc.) with plasmids (3.0 μg total) containing cDNAs (0.6 μg each) encoding c-Fos, c-Jun, Fra-2, JunD, Brm, or BRG1 and a reporter CAT plasmid (0.6 μg) containing a single AP-1 binding site (1× TRECAT) (3). The total amount of DNA for each transfection was adjusted by adding the empty vector, pRSV-2B. Cells were disrupted for determination of CAT activity 45 h after transfection as described previously. The CAT activity in cell lysates with the equal amount of total protein was determined and normalized by assigning a value of 1 to c-Fos plus c-Jun activities. The columns are the averages of four independent experiments, and the bars represent the S.D. B, expression of endogeneous collagenase and c-met genes. Total RNAs were extracted from SW13 cells transfected with the same set of expression vectors but without the CAT reporter plasmid. Total RNAs were analyzed by RT-PCR with pairs of primers specific for amplification of collagenase and c-met genes as well as the GAPDH gene, which served as an internal control.
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