Identification of Diazepam-binding Inhibitor/Acyl-CoA-binding Protein as a Sterol Regulatory Element-binding Protein-responsive Gene*

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Diazepam-binding inhibitor/acyl-CoA-binding protein (DBI/ACBP), a highly conserved 10-kDa polypeptide, has been implicated in various physiological processes including γ-aminobutyric acid type A receptor binding, acyl-CoA binding and transport, steroidogenesis, and peptide hormone release. Both in LNCaP prostate cancer cells and 3T3-L1 preadipocytes, the expression of DBI/ACBP is stimulated under conditions that promote lipogenesis (treatment with androgens and insulin, respectively) and that involve the activation of sterol regulatory element-binding proteins (SREBPs). Accordingly, we investigated whether DBI/ACBP expression is under the direct control of SREBPs. Analysis of the human and rat DBI/ACBP promoter revealed the presence of a conserved sterol regulatory element (SRE)-like sequence. Gel shift analysis confirmed that this sequence is able to bind SREBPs. In support of the functionality of SREBP binding, coexpression of SREBP-1a with a DBI/ACBP promoter-reporter gene resulted in a 50-fold increase in transcriptional activity in LNCaP cells. Disruption of the SRE decreased basal expression and abolished SREBP-1a-induced transcriptional activation. In agreement with the requirement of a co-regulator for SREBP function, transcriptional activation by SREBP-1a overexpression was severely diminished when a neighboring NF-Y site was mutated. Cholesterol depletion or androgen treatment, conditions that activate SREBP function in LNCaP cells, led to an increase in DBI/ACBP mRNA expression and SRE-dependent transcriptional activation. These findings indicate that the promoter for DBI/ACBP contains a functional SRE that allows DBI/ACBP to be coregulated with other genes involved in lipid metabolism.

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The nucleotide sequence(s) reported in this paper has been submitted to the GenBank®/EBI Data Bank with accession number(s) X94563.

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1 The abbreviations used are: DBI, diazepam-binding inhibitor; ACBP, acyl-CoA-binding protein; SRE, sterol regulatory element; SREBP, sterol regulatory element-binding protein; FAS, fatty acid synthase; HMG, 3-hydroxy-3-methylglutaryl; LPDS, lipoprotein-deficient serum; CT-FCS, charcoal-treated fetal calf serum; PSA, prostate-specific antigen; wt, wild type; HMG, hydroxymethylglutaryl.
In agreement with its expression in various tissues and cell types and its postulated roles in many different biological processes, the promoter of the DBI/ACBP gene displays all the hallmarks of a typical housekeeping gene (24, 25) but may also allow controlled activation related to specific regulatory pathways, including hormonal stimulation (26). Hormones that have been shown to stimulate the expression of DBI/ACBP include insulin and androgens. Insulin regulation of DBI/ACBP expression has been observed in 3T3-L1 preadipocytes (27). Regulation by androgens has been found in various male accessory sex organs (28) and in the human prostate cancer cell line LNCaP (25, 29). Interestingly, both conditions promote lipogenesis and involve the activation of sterol regulatory element-binding proteins (SREBPs) (30–36). SREBPs are cholesterol-regulated transcription factors that are synthesized as inactive membrane-bound precursors (see Ref. 36 for review). Proteolytic activation results in release and translocation to the nucleus. In the nucleus, SREBPs bind to specific sterol-responsive elements (SREs) and in cooperation with the generic transcription factors SP-1 or NF-Y (37–46), they coordinately modulate the transcription of a wide array of genes involved in cholesterol and fatty acid metabolism (36).

In view of the postulated role of DBI/ACBP in fatty acid and cholesterol metabolism and its induction under conditions that involve SREBP activation, we explored whether DBI/ACBP is directly controlled by SREBPs. Both LNCaP and HepG2 cells were used as experimental paradigm.

**Experimental Procedures**

**Cell Culture—** LNCaP and HepG2 cells were obtained from the American Type Culture Collection (ATCC, Rockville, MD) and were cultured as described previously (32, 47). To assess the impact of cholesterol on gene expression, cells were incubated with lipoprotein-deficient serum (LPDS) (Perimmune, Rockville, MD) in the absence or presence of 10 μg/ml cholesterol and 1 μg/ml 25-hydroxycholesterol added from stock solutions in ethanol. Control cultures received similar amounts of ethanol only. In experiments assessing the effects of steroids, fetal calf serum was pretreated with dextran-coated charcoal (CT-FCS) to reduce the background levels of steroids. The synthetic androgen R1881 (methyltrienolone), purchased from Calbiochem-Novabiochem, was dissolved in absolute ethanol and added to the cultures. Final ethanol concentrations did not exceed 0.2%. All experiments involving LNCaP cells were carried out with cells passages 30 to 75.

**Electrophoretic Mobility Shift Assay—** Recombinant 6×His-tagged SREBP-1a (amino acids 1–490) was expressed in Escherichia coli BL2 (DE3) as described previously (25). The plasmid pET-26b(+) containing the human DBI/ACBP gene, kindly provided by Dr. T. Osborne, Department of Molecular Biology and Biochemistry, University of California, Irvine, USA (37) and was purified by means of TALON metal affinity chromatography (CLONTECH). Complementary single-stranded oligonucleotides corresponding to nucleotides 137 to 113 of the published human DBI promoter sequence (25) were end-labeled with [γ-32P]ATP and T4 polynucleotide kinase and annealed. The probe (50,000 cpm) was incubated with recombinant SREBP-1a in a solution containing 10 mM Tris-HCl pH 7.6, 50 mM NaCl, 0.05 mM EDTA, 2.5 mM MgCl₂, 8.5% glycerol, 1 mM dithiothreitol, and 0.5 μg/ml poly(dI-dC). After a 20 min incubation at room temperature, the DNA-protein complex was observed when recombinant SREBP-1a, and plasmid pSV-ARo containing the androgen receptor expression vector (pSV-ARo) (48) and incubated with 10−8 M R1881 or with ethanol vehicle in medium containing 5% CT-FCS. In experiments assessing the effects of steroids, cells were incubated in medium containing 5% LPDS in the absence or in the presence of 10 μg/ml cholesterol and 1 μg/ml 25-hydroxycholesterol, or ethanol vehicle. One day after treatment, cells were washed with phosphate-buffered saline and harvested in 500 μl of reporter lysis buffer (Promega, Madison, WI). Aliquots of 10 μl of cleared lysate were assayed for luciferase activity using a luciferase reporter assay kit from Promega and a Berthold Microlumat LB 960 luminometer. The activity of β-galactosidase was used to normalize for transfection efficiencies.

**Northern Blot Analysis—** Total RNA was prepared using a modified guanidinium/CsCl ultracentrifugation method as described previously (29). Equal aliquots of total RNA (20 μg) were denatured and subjected to electrophoresis in a 1% agarose gel containing formaldehyde. The RNA was transferred to Biotrans membranes (ICN Pharmaceuticals, Inc., Costa Mesa, CA), prehybridized and hybridized with DBI, FAS, and 18 S probes as described before (29, 31). Blots were autoradiographed by exposure to Amersham Hyperfilm-MP or to Kodak Biomax film (Amersham International, Buckinghamshire, UK). Hybridization signals were quantitated using PhosphorImager screens (Molecular Dynamics, Sunnyvale, CA) and normalized for differences in RNA loading.

**RESULTS**

**Identification of a SREBP-binding Site in the DBI/ACBP Promoter—** Examination of the nucleotide sequences of the human DBI/ACBP promoter for potential SREBP binding sites revealed a sequence CTCGC CCCGA G at positions −127 to −118 of the published sequence (25), resembling the 10-base pair SREs found in other SREBP-regulated genes (Fig. 1). This sequence is homologous to the SRE of the farnesyl-diphosphate synthase gene (57) and differs only at two positions. In agreement with the requirement for a coregulator for SREBP function (SP-1 or NF-Y) (37–46), the putative SRE is closely positioned to a reverse CCAAT box, which is a potential binding site for the specific heterotrimeric transcription factor NF-Y. Interestingly, both SRE and NF-Y sites are perfectly conserved in the otherwise less homologous rat DBI/ACBP gene (Fig. 1A).

To determine whether the putative SRE is able to bind SREBP's, we performed electrophoretic mobility shift assays with recombinant SREBP-1a and a radio-labeled wild type (wt) DNA fragment corresponding to a 25-base pair DBI/ACBP promoter region encompassing the putative SRE. A single-shifted DNA-protein complex was observed when recombinant SREBP-1a was added to the binding reaction mixture (Fig. 2). A 50-fold excess of unlabeled homologous competitor fragment displaced SREBP binding to the labeled DNA. Oligonucleotides linked to a luciferase reporter gene have been described previously (25) and are here referred to as “wt.” Mutations or deletions within the promoter were created using the Quikchange mutagenesis kit (Stratagene, La Jolla, CA). The constructs mutSRE and delSRE contain the same mutation and deletion, respectively, as the oligonucleotides described under “Electrophoretic Mobility Shift Assay.” In construct mutNF-Y, the A at position −142, which is part of a putative NF-Y site, was replaced by C. In construct +4A, 4 A residues were inserted at position −131, which is located between the NF-Y and the SRE-like sequence. Plasmid pPASLuc, a fatty acid synthase promoter-reporter construct, and plasmid pSV-ARo, expressing the androgen receptor, have been described previously (32, 48). Plasmid pPA-T, a luciferase-based plasmid with the prostate-specific antigen (PSA) promoter (49), was kindly provided by Dr. J. Trapan (Erasmus University, Rotterdam, The Netherlands).

**Transient Transfections and Reporter Gene Assays—** LNCaP cells were seeded in 6-cm dishes in Dulbecco's modified Eagle medium containing 10% fetal calf serum at a density of 7 × 10⁵ cells. On the next day, the medium was replaced with Dulbecco's modified Eagle's medium with 2% CT-FCS. Cells were transfected with 5 μg of the indicated luciferase reporter constructs, the indicated amounts of pIRES1neo or pSREBP-1a−140, and a plasmid encoding β-galactosidase. After 4 h of incubation with DNA, cells were subjected to a glycerol shock and washed with phosphate-buffered saline. To explore potential effects of androgens, transfected cells were cotransfected with an androgen receptor expression vector (pSV-ARo) (48) and incubated with 10−8 M R1881 or with ethanol vehicle in medium containing 5% CT-FCS. In experiments assessing the effects of steroids, cells were incubated in medium containing 5% LPDS in the absence or in the presence of 10 μg/ml cholesterol and 1 μg/ml 25-hydroxycholesterol, or ethanol vehicle. One day after treatment, cells were washed with phosphate-buffered saline and harvested in 500 μl of reporter lysis buffer (Promega, Madison, WI). Aliquots of 10 μl of cleared lysate were assayed for luciferase activity using a luciferase reporter assay kit from Promega and a Berthold Microlumat LB 960 luminometer. The activity of β-galactosidase was used to normalize for transfection efficiencies.

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Transcriptional Regulation of DBI/ACBP by SREBP

A

Fig. 1. Nucleotide sequence alignment of the promoter region of the human and rat DBI/ACBP gene (a) and comparison of SREBP sites from known sterol-responsive genes with the putative SRE site of the DBI/ACBP gene (b). The nucleotide sequences of the human (h) and rat (r) DBI/ACBP genes, as reported previously (24, 25) are aligned in panel A. The sequences corresponding to the putative binding sites for NF-Y, SREBP and TATA-like sequences are boxed. In panel B, the sequences of the SREBP sites of the hamster (ham), rat (r), frog (f), human (h), and mouse (m) LDL receptor, of the rat fatty acid synthase gene (rFAS), of the LDL receptor, of the human (h) and rat (r) ACBP genes are aligned. The putative SRE sites of the DBI/ACBP gene are underlined. Asterisks indicate residues that are conserved in all aligned sequences.

Transcriptional Regulation of DBI/ACBP Promoter-Reporter Genes by Coexpressed SREBP—In order to determine whether SREBP binding is functional, we transiently transfected LNCaP cells with a DBI/ACBP promoter-reporter construct harboring the SRE site (pDBI-264lac, here referred to as wt), together with a plasmid encoding β-galactosidase and increasing amounts of pSREBP-1a1–460, a plasmid encoding transcriptionally active SREBP-1a. Two days after the transfection, the luciferase activity was measured and the values were corrected for any differences in transfection efficiency, as determined from the β-galactosidase assay. As Fig. 3A shows, the transcriptional activity of the DBI/ACBP promoter was elevated with increasing amounts of co-transfected pSREBP-1a1–460. Maximal effects were reached at 20 ng of pSREBP-1a1–460. In order to determine that the stimulation of transcriptional activity by SREBP-1a overexpression is mediated by the SRE-like site, we generated DBI/ACBP promoter-reporter constructs in which the SRE site is mutated (mutSRE) or deleted (delSRE) (Fig. 3B). Transfection experiments were carried out as described above with maximally effective amounts of pSREBP-1a1–460 (20–50 ng). In support of the involvement of the SRE site in SREBP-induced transcriptional activation, stimulation of luciferase activity was severely decreased when the SRE site was mutated or deleted (Fig. 3C). Additionally, the basal transcriptional activity of the mutant and control constructs was 5–10-fold lower than that of the wild type construct, demonstrating the importance of the SRE site for the transcriptional activity of the DBI/ACBP gene (Fig. 3D).

Requirement of NF-Y Binding for SREBP Activation of DBI/ACBP Transcription—SREBPs are weak activators of transcription in isolation and are known to function more efficiently when a co-regulatory factor (SP-1 or NF-Y) binds to a neighboring site (37–46). The SRE in the DBI/ACBP promoter is preceded by a potential NF-Y site. To test whether this latter site is important for SREBP-induced activation of DBI/ACBP transcription, we generated a DBI/ACBP promoter-reporter construct in which the NF-Y site is mutated (Fig. 4A). In another construct, we increased the distance between the NF-Y and the SRE sites by insertion of four deoxyadenosine residues. Transient co-transfection of these constructs with pSREBP-1a1–460 revealed that the stimulatory effect of SREBP overexpression as observed with the wild type construct is severely diminished when the NF-Y site is mutated or when the distance between the NF-Y and the SRE sites is modified (Fig. 4B). Basal transcriptional activities, however, remained high. Mutation of the NF-Y site even led to a 3–4-fold increase in basal transcription. Insertion of four bases between the NF-Y and the SRE site caused a 2-fold decrease in luciferase activity (Fig. 4C).

Regulation of DBI/ACBP Expression by Sterols—Having demonstrated that overexpression of SREBPs leads to SRE-dependent activation of the transcriptional activity of DBI/ACBP promoter-reporter genes, we examined whether these con-
structs are also responsive to physiological changes in endogenous SREBP levels. One of the main and universal physiological signals that trigger SREBP processing resulting in increased nuclear levels of SREBPs is cholesterol depletion. Fig. 5A shows that the luciferase activity of LNCaP cells that were transiently transfected with DBI/ACBP promoter-reporter constructs was 2-fold higher in cells that were deprived of cholesterol as compared with sterol-treated cells. Similar

**FIG. 3.** Transcriptional activation of DBI/ACBP promoter-reporter constructs by coexpression of SREBP-1a. A, triplicate dishes of LNCaP cells were transiently cotransfected with a DBI/ACBP promoter luciferase construct harboring the SRE-like site (pDBI-264luc, here referred to as wt), together with a plasmid encoding β-galactosidase and increasing amounts of pSREBP-1a,1–460 plasmid expressing a transcriptionally active form of SREBP-1a. Analogous control transfections were carried out with increasing amounts of pIRES1neo, the empty expression vector. Two days after transfection, the luciferase activity was measured, corrected for any differences in transfection efficiency as determined from the β-galactosidase assay, and expressed relative to the control transfections with the empty expression vector (pIRES1neo). The results shown are representative of two independent experiments. B, schematic representation of the wt DBI/ACBP promoter-reporter construct and those with a mutated (mutSRE) or deleted SRE (delSRE). The putative binding site for NFY is indicated. The sequences of the wild type and mutated SRE-like site are shown. C, LNCaP cells were transiently cotransfected with the DBI/ACBP promoter-reporter constructs shown in panel B, together with a β-galactosidase-encoding plasmid and a maximally effective

**FIG. 4.** Requirement of NF-Y binding for SREBP activation of DBI/ACBP transcription. A, schematic representation of the wt DBI/ACBP promoter-reporter construct, mutNF-Y, in which the A in the NF-Y site is mutated to a C, and construct +4A, in which 4A residues were inserted in between the NF-Y and the SRE site. B, triplicate dishes of LNCaP cells were transiently cotransfected with the DBI/ACBP promoter-reporter constructs shown in panel A together with a plasmid encoding β-galactosidase and 50 ng of pSREBP-1a,1–460 or the empty pIRES1neo vector. Two days after the transfection, luciferase activity was measured, normalized for β-galactosidase activity and expressed as -fold stimulation relative to control transfections with pIRES1neo. Values represent the means ± S.E. The results are representative of three independent experiments. C, basal promoter activities of the DBI/ACBP promoter-reporter constructs shown in panel A were measured as luciferase activities and were compared relative to the normalized luciferase activity of the wt construct. Values represent the means ± S.E. The results are representative of three separate experiments.

amount of pSREBP-1a,1–460 (20–50 ng) or with pIRES1neo. Luciferase activity was measured, corrected for any differences in transfection efficiency, and expressed as -fold stimulation relative to the control transfection with pIRES1neo as described in A. Values represent the means ± S.E. of triplicate dishes. The results are representative of three separate experiments. D, basal promoter activities of the DBI/ACBP promoter-reporter constructs shown in panel B were measured as luciferase activities and were compared relative to the normalized values of the wt construct. Values represent the means ± S.E. of triplicate dishes. Similar
results were obtained with a luciferase reporter construct harboring the promoter of the fatty acid synthase gene (31), a well known sterol-regulated gene (38, 54, 60). The promoter activity of a reporter construct containing a promoter fragment of the PSA gene (a gene encoding a prostate-secreted protein that is not directly related to cholesterol and fatty acid metabolism) (49), was not affected by sterols, indicating that the effects of cholesterol were specific. In support of the involvement of SREBP1a in the effects of cholesterol depletion on DBI/ACBP gene transcription, no effects of sterols were observed when LNCaP cells were transfected with DBI/ACBP constructs in which the SRE-site was mutated or deleted (Fig. 5A).

To determine whether also the endogenous DBI/ACBP gene is under the control of cholesterol we cultured LNCaP cells for 24 h in media containing 5% LPDS either in the absence or in the presence of sterols, and analyzed the mRNA expression of DBI/ACBP by Northern blot analysis. As Fig. 5B illustrates, DBI/ACBP mRNA expression was 2-fold higher in cells that were deprived of sterol as compared with sterol-treated cells. Similar results were obtained when the same blot was hybridized with a FAS probe. mRNA levels for PSA were only marginally affected. Similar results were obtained when HepG2 cells were cultured in the presence of the cholesterol synthesis inhibitor mevastatin and then incubated in the absence or in the presence of sterols (Fig. 5C).

**Fig. 5. Regulation of DBI/ACBP gene expression by sterols.** A, LNCaP cells were transiently cotransfected with the indicated promoter-reporter constructs and then incubated in medium supplemented with 5% lipoprotein-deficient serum in the absence (−) or in the presence (+) of sterols (10 μg/ml cholesterol and 1 μg/ml 25-hydroxycholesterol). One day after treatment, luciferase activity was measured. Values represent the means ± S.E. of three individual experiments and are expressed relative to the values obtained in the absence of sterols (10 μM mevastatin in the absence (−) or in the presence (+) of 10−8 M amount of the synthetic androgen R1881). One day after treatment, luciferase activity was measured. Values represent the means ± S.E. of five individual experiments and are expressed relative to the values obtained in the absence of R1881.

**Fig. 6. Involvement of the SRE site in the regulation of DBI/ACBP expression by androgens.** LNCaP cells were transiently cotransfected with the indicated DBI/ACBP promoter-reporter constructs, and then incubated in medium supplemented with 5% CT-FCS in the absence (−) or in the presence (+) of 10−8 M amount of the synthetic androgen R1881. One day after treatment, luciferase activity was measured. Values represent the means ± S.E. of five individual experiments and are expressed relative to the values obtained in the absence of sterols.

**DISCUSSION**

The current experiments provide evidence that DBI/ACBP is a SREBP-responsive gene. A SRE-like sequence was found in the human DBI/ACBP promoter by visually scanning the DNA sequence. This site resembles the SREs of other SREBP-responsive genes and is present also in the promoter of the rat homologue. The SRE-like site is functional; it binds purified SREBP-1a and mediates transcriptional activation by overexpressed SREBP-1a in cotransfection experiments. Like the genes encoding farnesyl diphosphate synthase, HMG-CoA syn-
thase, squalene synthase, SREBP-2, and glycerol-3-phosphate acyltransferase (41–46). Regulation by SREBPs requires a neighboring binding site for the generic transcription factor NF-Y. Furthermore, physiological conditions that are known to activate SREBP processing and stimulate their nuclear translocation (such as cholesterol depletion or androgen treatment of LNCaP cells) increase the expression of DBI/ACBP promoter-reporter genes and of the endogenous gene. Consistent with this finding is the report by Hansen et al. (27) that DBI/ACBP expression is stimulated during insulin-induced lipogenesis in 3T3-L1 preadipocytes, another physiological condition that involves changes in SREBP-mediated DBI/ACBP expression (35). Together with our finding that a functional SRE is important also for basal DBI/ACBP gene transcription, the current experiments illustrate the importance of SREBPs in the regulation of DBI/ACBP gene expression.

Regulation of DBI/ACBP by SREBPs is consistent with the postulated role for DBI/ACBP in fatty acid metabolism and allows DBI/ACBP to be coregulated with other proteins and enzymes involved in lipid metabolism. Our finding that DBI/ACBP is under the control of cholesterol may be of special importance of SREBPs in the regulation of DBI/ACBP gene expression (39). It is postulated role for DBI/ACBP in fatty acid metabolism and allows DBI/ACBP to be coregulated with other proteins and enzymes involved in lipid metabolism. Our finding that DBI/ACBP is under the control of cholesterol may be of special importance of SREBPs in the regulation of DBI/ACBP gene expression (39). It is postulated role for DBI/ACBP in fatty acid metabolism and allows DBI/ACBP to be coregulated with other proteins and enzymes involved in lipid metabolism. Our finding that DBI/ACBP is under the control of cholesterol may be of special importance of SREBPs in the regulation of DBI/ACBP gene expression (39).

Reference(s)
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