Mouse BTEB3, a new member of the basic transcription element binding protein (BTEB) family, activates expression from GC-rich minimal promoter regions

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Members of the three-zinc-finger family of transcription factors play an important role in determining basal transcription. We have cloned mouse BTEB3 (mBTEB3), a new member of the basic transcription element binding protein (BTEB) family, which is expressed in a wide variety of tissues. mBTEB3 activates transcription of the simian virus 40 early promoter (4-fold) and

of the tissue-specific SM22 α promoter (100-fold), suggesting that, like BTEB1 and Sp1, mBTEB3 is a basal transcription factor.

Key words: basal transcription element, GC box, SM22 α promoter, SV40 promoter, transcription factor.

INTRODUCTION

The promoters of many eukaryotic genes contain GC-rich sequences within the minimal region required to give promoter activity. These sequences bind members of the zinc-finger family of transcription factors, and binding of these factors is required for maximal promoter activity [1–4]. The number of GC boxes and their spatial arrangement varies significantly between promoters. Several promoters, including nuclear factor 1 [5], contain a single GC box, whereas others, including some housekeeping genes, the gene encoding transforming growth factor β 1 and the simian virus 40 (SV40) early promoter, contain multiple GC boxes in tandem arrays [1,6,7]. The archetypal zinc-finger transcription factor involved in the control of basal transcription is Sp1, which is widely expressed and binds to the consensus sequence 5'-(G/T)(G/A)GGCG(G/T)(G/A)(G/A)-(C/T)-3' [7]. However, Sp1 is not expressed at high levels in all tissues, and the identification of other zinc-finger transcription factors which activate transcription through GC boxes implies that Sp1 is not the only regulator of basal transcription [8]. Other factors which bind to the GC box include BTEB1 and BTEB2, members of the basic transcription element binding protein (BTEB) family, which, like Sp1, contain three C₂H₂ zinc fingers [8,9]. BTEB1 is expressed in most tissues, and the level of BTEB1 expression is inversely correlated with that of Sp1 expression [8]. Like Sp1, BTEB1 activates transcription from GC boxes in tandem repeats. However, in contrast with Sp1, BTEB1 is not active against single GC boxes, and it has been suggested that BTEB1 may repress the activity of Sp1 at these sites [8]. BTEB2 is only expressed in the placenta of the rabbit and the testis of the adult rat [9].

In addition to their role in the activation of widely expressed genes, GC boxes are important elements in the promoters of tissue-restricted genes. For example, Sp1 has been shown to bind

to GC boxes present in the cardiac α -actin promoter and to activate transcription from this promoter [10–12]. In skeletal muscle cells, this activation by Sp1 appears to be regulated by the formation of multi-transcription factor complexes, as Sp1 has been shown to interact with the myogenic basic helix–loop–helix protein, myogenin [13]. The formation of such complexes therefore provides a mechanism by which ubiquitously expressed transcription factors activate the transcription of tissue-restricted genes. The promoters of smooth-muscle-specific genes also contain GC-rich sequences which can bind recombinant Sp1. However, the factors present in vascular smooth muscle nuclear extracts which bind to these sequences do not include Sp1 ([14]; P. R. Kemp, unpublished work). In an expressed sequence tag (EST) database screen to identify mouse zinc-finger proteins, we identified a sequence with no counterpart in the non-redundant database. Here we describe the cloning and analysis of function of this gene.

EXPERIMENTAL

Northern blot analysis

Total RNA was isolated using the guanidine thiocyanate/CsCl procedure with Phaselock gel (Flowgen) according to the manufacturer's instructions. RNA was electrophoresed on a 1.0 % (w/v) agarose/formaldehyde gel and blotted on to nitrocellulose filters (Hybond; Amersham). The filters were hybridized at 60 °C overnight in hybridization buffer [1 M NaCl, 6 % PEG 8000, 0.1 % tetrasodium pyrophosphate, 10 $\times$ Denhardt's solution (1 $\times$ Denhardt's solution contains 0.02 % Ficoll 400, 0.02 % polyvinylpyrrolidone and 0.02 % BSA), 100 μ g/ml denatured salmon sperm DNA, 1 % SDS and 50 mM Tris, pH 7.6] with ³²P-labelled random-primed mouse BTEB3 (mBTEB3) cDNA (bp 544–880 relative to the translation start site), and then washed

Abbreviations used: AMV-RT, avian myeloblastosis virus reverse transcriptase; BTE, basic transcription element; BTEB, basic transcription element binding protein; CAT, chloramphenicol acetyltransferase; DMEM, Dulbecco's modified Eagle's medium; EST, expressed sequence tag; EMSA, electrophoretic mobility shift assay; mBTEB3, mouse BTEB3; α -MEM, α -minimal essential medium; RT-PCR, reverse transcription-PCR; SV40, simian virus 40; TIEG, transforming growth factor β -induced early gene.

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The nucleotide sequence data reported will appear in the EMBL, GenBank and DDBJ Nucleotide Sequence Databases under accession number AJ245644.

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AACTTGCAGGGGTCTGTCCTAACGTGCGCTCCGCTCGACGTCCCTAAGA
CAGGCCGACTCTGACGCCGACGCGGACCCTCCTTACTGGAGCCCCCATGCGCTCACCC
TCCAGTGGCCGGCCAGGCGGACGCGGAGCCGCGCGGTGACGGCAGAAGCGGCTACGCGC
CCAGCCTAGCCCAGCCCAGCCCAGCCGAGAAGAGGGCGCGCCGCCCCGCCCCCGGC
CGCTCTCCGGAGCCGCTGGGTGCGGATGCGCCGCTGACGACTCCTGCGAGAGCACCGCGC
GCCCCAGCCCGCAGCATGGCAGCCGCCCTATGTGGACCACTTTGCCGCCGAGTGCCTC
      M A A A A Y V D H F A A E C L
GTGTCCATGTCCAGCCGCGCAGTCTGTCACGAGCCGCGGAAGGACCTCGGCCCGGCC
V S M S S R A V V H E P R E G P R P R P
GAGGGCGCGGCCCGCCGCCCTCAGCTGCCCCGCTTGACGAGCGCCGCGACGGCAAG
E G A A A A A L T L P R V D E R R D G K
GACAGCGCCTCGCTTTTCGTGGTGGCTCGGATCCTAGCGGACCTCAACCAGCAGGCGCCG
D S A S L F V V A R I L A D L N Q Q A P
GCGCCCGCCCCCGCGAAGCAGAGAAGGGGCGCTGCGCGCAAGGCGAGGACCCCTGC
A P A P A E R R E G A A A R K A R T P C
CGCCTGCGCCTGCGCCCCCTGCGCCGCCACCCGCCCCAGAGCCCGCTCCCCGGGACAA
R L P P A P P A P P P G P E P A S P G Q
GCAGGCGCGCCGCGCGCCCCCAGCCCCGCTGGAGCGAGCCCGAGGCGGCATTGGAG
A G A P A A P P S P A W S E P E A A L E
CAGGAACCCGCCCCGCGGGGAGCGGCGAGCCTGGCCTCAGACAAAGGGTTCGGCGAGGC
Q E P G P A G S G E P G L R Q R G R R G
CGGAGCGCGCGGACCTCGAGTCCCCGAGAGGAAGCACAAGTGCCACTACGCGGGCTGC
R S R A D L E S P Q R K H K C H Y A G C
GAGAAAGTTTACGGGAATCTTCGCACCTCAAGGCGCACCTGAGAACTCACACAGGTGAG
E K V Y G K S S H L K A H L R T H T G E
AGGCCTTTGCGCTGCAGCTGGCAGGAGTGCAACAAGAAGTTCGCACGCTCGGACGAGCTG
R P F A C S W Q E C N K K F A R S D E L
GCACGCGCACTATCGCACGCACACGGGCGAGAAGAAGTTACGCTGCCCCATCTGTGAGAAG
A R H Y R T H T G E K K F S C P I C E K
CGCTTCATGCGGAGCGACCACCTGACGAAGCACGCACGCCGCCACGCCAACTTCCACCCA
R F M R S D H L L T K H A R R H A N F H P
GGCATGCTGCAGCGGCGCGGGGGCTCGAGGACCGGCTCGCTCAGCGACTACAGCCGC
G M L Q R R G G G S R T G S L S D Y S R
TCCGATGCCAGCAGCCCCACCATCAGCCCGGCCAGCTCACCTGA
S D A S S P T I S P A S S P *

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Figure 1 cDNA and protein sequences of mBTEB3

The nucleotide and amino acid sequences of mBTEB3 are shown. The DNA-binding domain containing the basic sequence and the three zinc fingers is underlined.

twice at 60 °C for 30 min in 1 × SSC/0.1 % SDS (1 × SSC contains 150 mM NaCl and 15 mM sodium citrate). The blots were exposed to a Phosphorimager screen overnight and the data were analysed using Image Quant software (Molecular Dynamics).

Reverse transcription, PCR and cloning

A portion of 1 µg of total RNA in 11 µl of water was denatured for 10 min at 65 °C and then chilled on ice. To the denatured RNA was added 4 µl of 5 × avian myeloblastosis virus reverse transcriptase (AMV-RT) buffer (Promega), 0.5 µl (2 units) of AMV-RT (Promega), 2 µl of 2 mM dNTPs, 1 µl of 100 mM dithiothreitol, 0.5 µl of RNasin (Promega) and 1 µl of random hexamer primers (Pharmacia). The samples were incubated at 42 °C for 2 h and placed on ice to terminate the reaction.

For PCR amplification, 2 µl of DNA was mixed with 4 µl of 10 × *Taq* polymerase buffer (Pharmacia), 5 µl of 2 mM dNTPs, 2.5 µl of each primer (10 pmol/µl), 2.5 µl of DMSO and 21.5 µl of water. The reaction was hot-started by heating to 95 °C for 30 s, then reducing the temperature to 80 °C and adding 10 µl of reaction mixture containing 1 µl of 10 × *Taq* polymerase buffer, 0.5 µl (2.5 units) of *Taq* polymerase (Pharmacia) and 8.5 µl of water. The thermal cycling profile was 1 min at 95 °C (dissociation), 1 min at 65 °C and 2 min at 72 °C for 35 cycles. The

final cycle was followed by an additional extension time of 5 min at 72 °C. The forward primer was 5'-CCACAACCTTGCAAGG-GGTCTGCTGCTAAC-3' and the reverse primer was 5'-TCA-GGGTGAGCTGGCCGGGCTCATG-3', giving a 1.2 kbp product. The 1.2 kbp mBTEB3 PCR product was cloned into *EcoRV*-linearized pCDNA3 to which T overhangs had been added.

Electrophoretic mobility shift assay (EMSA)

mBTEB3 protein was made using a rabbit reticulocyte lysate *in vitro* coupled transcription translation kit (Promega) according to the manufacturer's instructions. BTEB3 or nuclear extracts were added to ³²P-labelled synthetic oligonucleotides or PCR products in the presence or absence of unlabelled competitor in a binding buffer [50 mM Tris, pH 8.0, 100 mM KCl, 12.5 mM MgCl₂, 1 mM EDTA, 1 µg of poly(dI-dC) and 1 mM dithiothreitol in 20 % (v/v) glycerol] at 25 °C for 30 min. Samples were run on a 6 % (w/v) acrylamide non-denaturing gel and visualized using Image Quant software (Molecular Dynamics).

Cell culture, transfection and chloramphenicol acetyltransferase (CAT) assay

Mouse P19 embryonic carcinoma cells were maintained in α-minimal essential medium (α-MEM) supplemented with 10 %

(v/v) fetal calf serum, and were subcultured by trypsinization (1:10) every 48 h. At 24 h before transfection, cells were seeded into six-well plates at a cell density of 2×10^5 cells/well. A 10 μ l aliquot of Lipofectin (Gibco) reagent in 100 μ l of OptiMEM (Gibco), prepared as described by the manufacturer, was mixed with a total of 2 μ g of plasmid DNA {1.5 μ g of either pCAT-Control (Promega) or pCB83 [15] and 0.5 μ g made up of pCBTEB3 and pCDNA3 in varying ratios} in 100 μ l of OptiMEM and incubated at room temperature for 15 min. The cells were washed with serum-free α -MEM before the Lipofectin/DNA mixture was added. After 18 h the medium was changed to α -MEM supplemented with 10% (v/v) fetal calf serum. The cells were harvested 48 h after transfection. Cells were lysed by freeze/thawing and centrifuged (13000 g, 5 min) to obtain supernatant fractions.

C2C12 cells were grown in Dulbecco's modified Eagle's medium (DMEM) supplemented with 10% (v/v) fetal calf serum and subcultured 1:8 every 2–3 days by trypsin treatment. For transfection, C2C12 cells were seeded at 3×10^4 cells/ml into 12-well plates and grown overnight. The cells were washed with serum-free DMEM and then incubated with a mixture of 940 ng of DNA (made up of 700 ng of pCB83 and 240 ng of either pCDNA3 or pCBTEB3) with 5.6 μ l of lipofectamine (Gibco) in OptiMEM prepared as described by the manufacturer. Cells were incubated with the lipid/DNA mixture for 5 h before the medium was replaced with DMEM supplemented with 10% (v/v) fetal calf serum.

CAT assays were performed by incubation of the cell extract with [14 C]chloramphenicol and n-butyryl-CoA overnight at 37 °C. Butyrylated chloramphenicol was extracted from the reaction mixture with xylene, the xylene phase was back-extracted twice with 100 μ l of 250 mM Tris, pH 7.5, and radioactivity was determined by liquid scintillation counting.

RESULTS AND DISCUSSION

Identification and cloning of mBTEB3

To identify putative GC-box-binding transcription factors, we screened the mouse EST database with a protein sequence derived from the BTEB/TIEG (transforming growth factor β -induced early gene) family of three-zinc-finger proteins. This screen identified 11 clones for a cDNA sequence with no comparable sequence in the non-redundant database. PCR primers were designed using the consensus sequence from these ESTs, and reverse transcription-PCR (RT-PCR) analysis showed that these primers produced a single product of the appropriate size from RNA isolated from the mouse heart. The PCR product was cloned and sequenced to confirm that it corresponded to the EST sequences in the database. The sequence obtained was screened through the full EST database. Mouse, rat and human sequences identified by an iterative screening process were assembled to generate a full-length coding sequence, so that primers capable of amplifying a full coding sequence could be designed. RT-PCR from mouse heart RNA using these primers gave a 1.2 kbp product which was cloned and sequenced. The cDNA derived by this process encoded a 289-amino-acid protein which had three C_2H_2 zinc fingers at the C-terminus of the protein (Figure 1). The zinc-finger sequence shows 81% and 72% similarity to those of mBTEB1 and mouse Sp1 respectively, and residues thought to be important for base recognition by this family of proteins are conserved (Figure 2a). In addition to the three zinc fingers, this region of the protein also contains a basic region which has been shown to be required for binding to DNA in other zinc-finger transcription factors. Given the similarity of

Figure 2 Comparison of mBTEB3 domains with those of other C_2H_2 -zinc-finger proteins

(a) Comparison of the zinc fingers of the mouse proteins BTEB1, BTEB3, TIEG1, Sp1 and GLKF (gut-enriched Kruppel-like factor). Cysteine residues and histidine residues thought to coordinate zinc are aligned and shown in **bold**. (b) Comparison of transactivation region A in BTEB1 from different species with transactivation region A of mBTEB3. Conserved residues are shown in **bold**.

the zinc-finger sequence to those of mBTEB1 and human BTEB2 (66%), we have named this protein mBTEB3.

The N-terminal 164 amino acids of the protein show little significant similarity with the mBTEB1 sequence, but were rich in alanine (34/164), proline (28/164) and serine (11/164) residues. Protein domains rich in these amino acids form the activation domains of a number of transcription factors.

The functional domains of the BTEB1 activation domain have been determined by deletion analysis [16]. These studies have shown that two hydrophobic regions, i.e. amino acids 13–27 (region A) and amino acids 58–68 (region B), are important in transcriptional activation and can act independently of each other. The sequences of these regions of BTEB1 are conserved across species, and BTEB2 has a hydrophobic region similar to BTEB1 region B. In spite of the overall low degree of similarity between the BTEB1 activation domain and the mBTEB3 N-terminus, a hydrophobic region similar to BTEB1 region A is present in mBTEB3 (Figure 2b), suggesting that mBTEB3 acts as a transcription factor.

Expression of mBTEB3 in the adult mouse and during development

The pattern of mBTEB3 expression in a range of adult mouse tissues was determined by Northern analysis. A 5 kb mBTEB3 mRNA was detectable in all tissues examined, and also in RNA

Figure 3 Tissue distribution of mBTEB3 expression

(a) Northern analysis of mBTEB3 expression in different tissues. Total RNA was isolated from adult mouse tissues as described in the Experimental section and fractionated on a 1% (w/v) agarose gel. After transfer to a nylon membrane, the blot was probed with a 336 bp fragment of mBTEB3, washed and exposed to a phosphor screen. (b) RT-PCR analysis of mBTEB3 expression in embryos. Portions of 1 μ g of total RNA isolated from embryos between days 9 and 14.5 were reverse-transcribed and amplified using primers to nucleotides 44 and 880 of mBTEB3.

extracts from cultured vascular smooth muscle cells (Figure 3a). RT-PCR analysis of mRNA isolated from mouse embryos at embryonic days 9.0, 10.0, 13.5 and 14.5 showed that mBTEB3 is expressed in the developing as well as in the adult mouse (Figure 3b). However, mBTEB3 was not detected by screening a cDNA library from mouse embryo at day 7.5 or by RT-PCR of RNA isolated from P19 embryonic carcinoma cells.

mBTEB3 is a transcriptional activator which binds to a consensus basic transcription element (BTE)

To determine whether mBTEB3 is a transcriptional activator, the cDNA for mBTEB3 was cloned into pCDNA3. The construct was co-transfected with pSVControl (in which the CAT gene is

Figure 5 mBTEB3 binding to a BTE

(a) pCBTEB3 was transcribed and translated as described in the Experimental section, then analysed on SDS/15%-PAGE. (b) EMSA analysis of *in vitro* generated mBTEB3. Lane 1, BTE sequence probe alone; lane 2, probe incubated with mBTEB3 protein; lane 3, as for lane 2 but with a 100-fold excess of unlabelled competitor mutant BTE oligonucleotide (GATCGA-GAATTAGGCGTGGCCAAC); lane 4, as for lane 2, but with a 100-fold excess of unlabelled competitor BTE oligonucleotide.

under the control of an SV40 promoter and enhancer) into P19 embryonic carcinoma cells. CAT assays of extracts prepared 48 h after transfection showed that mBTEB3 caused a dose-dependent increase in the CAT activity present in the cell extracts, with maximal stimulation of approx. 4-fold (Figure 4a), indicating that mBTEB3 is a transcriptional activator. This increase in activity of an SV40 promoter is similar to that observed with both BTEB1 and BTEB2 [8,9].

Both BTEB1 and BTEB2 have been shown to bind to the sequence GATCGAGAAGGAGGCGTGGCCAAC [8,9]. To determine whether mBTEB3 may bind to the same sequence, mBTEB3 protein was made using *in vitro* transcription and translation. The synthesis of a protein of the predicted size (40 kDa) by the *in vitro* system was confirmed by synthesizing a ³⁵S-labelled protein that was analysed by SDS/PAGE (Figure 5a). Unlabelled mBTEB3 protein was generated using the same

Figure 4 mBTEB3 activation of the SV40 and SM22 α promoters

(a) P19 embryonic carcinoma cells were transfected as described in the Experimental section with 1.5 μ g of pCAT-Control and various amounts of pCBTEB3 and pCDNA3 to keep the total amount of plasmid DNA at 2 μ g. At 48 h after transfection, cells were harvested and CAT activity was assayed as described in the Experimental section. (b) P19 cells were transfected as described above with a CAT gene under the control of a minimal region of the SM22 α promoter (pCB83 [15]) replacing pCAT-Control. (c) C2C12 cells were transfected as described in the Experimental section with a CAT gene under the control of a minimal region of the SM22 α promoter (pCB83 [15]) and pCBTEB3. Data presented are means $\pm$ S.E.M. for triplicate samples. Each experiment was repeated three times.

system, and this protein was used in EMSAs with a ^{32}P -labelled oligonucleotide containing the consensus BTE. This protein formed a complex with the BTE oligonucleotide, indicating that mBTEB3 binds to the BTE sequence (Figure 5b). Complex-formation was inhibited by the presence of a 100-fold excess of an unlabelled competitor BTE oligonucleotide, but not by the presence of a similar excess of a mutant BTE oligonucleotide (GATCGAGAATTAGGCGTGGCCAAC; Figure 5b), showing that the interaction is sequence specific.

Both BTEB1 and BTEB2 have been reported to be weak activators of the Rous sarcoma virus long terminal repeat promoter, a promoter that does not contain a consensus GC box [(G/T(G/A)GGCG(G/T)(G/A)(G/A)(C/T)] [8,9]. This activation of a promoter that lacks a GC box is in contrast with the observation that removal of the GC boxes present in the *Cyp11A1* promoter prevented BTEB2-dependent activation of this promoter [17]. Taken together, these data imply that the BTEBs can bind to other sequences that deviate from the consensus GC box sequence. However, the sequence within the Rous sarcoma virus long terminal repeat promoter to which the BTEBs bind, and the ability of mBTEB3 to bind to and activate this promoter, remain to be determined.

mBTEB3 activates the minimal region of the SM22 α promoter

Basal transcription factors of the three-C₂H₂-zinc-finger family have been shown to play a significant role in the expression of tissue-specific genes. For example, Sp1 forms part of a multi-transcription factor complex involved in the expression of cardiac α -actin in skeletal muscle, and Sp1 binding has also been shown to be important in the activation of the *GLUT1* glucose transporter gene in cardiac myocytes. We have previously shown that a minimal region of the SM22 α promoter (–118 bp to +65 bp) is sufficient to drive the expression of a reporter gene in vascular smooth muscle cells, but not in Rat-1 fibroblasts [15]. We have also found that, whereas this region can bind purified Sp1, Sp1 is not one of the factors present in vascular smooth muscle cell nuclear extracts which binds to these regions (P. R. Kemp, unpublished work). Similar data have been obtained for a minimal region of the smooth muscle α -actin promoter [14]. To determine whether mBTEB3 could activate transcription from a smooth-muscle-specific promoter, mBTEB3 was co-transfected with a CAT gene under the control of bases –118 to +65 of the SM22 α promoter (pCB83 [15]) into P19 cells. mBTEB3 caused a dose-dependent increase in CAT activity present in the cell extracts 48 h after transfection (up to 100-fold; Figure 4b).

To determine whether mBTEB3 could activate transcription from the SM22 α promoter in other cell types, C2C12 cells were

transfected with the mBTEB3 expression construct and pCB83. The basal activity of pCB83 was higher in the C2C12 cells than in the P19 cells (approx. 20-fold; results not shown) in the absence of mBTEB3. However, co-transfection of mBTEB3 with pCB83 resulted in a 3-fold increase in CAT activity from this promoter fragment (Figure 4c), indicating that transcriptional enhancement by mBTEB3 is not restricted to P19 cells.

Taken together, our results indicate that mBTEB3 is a powerful activator of transcription which is expressed in a wide range of tissues. mBTEB3 can activate transcription from the minimal region of genes that are expressed in all tissues (e.g. SV40) and from tissue-restricted genes (e.g. SM22 α). Our findings therefore indicate that mBTEB3, like BTEB1 and Sp1, is a basal transcription factor.

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