The Coactivator CBP Stimulates Human T-cell Lymphotrophic Virus Type I Tax Transactivation in Vitro*

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Tax interacts with the cellular cyclic AMP-responsive element binding protein (CREB) and facilitates the binding of the coactivator CREB binding protein (CBP), forming a trimeric complex on the cyclic AMP-responsive element (CRE)-like sites in the human T-cell lymphotrophic virus type I (HTLV-I) promoter. The trimeric complex is believed to recruit additional regulatory proteins to the HTLV-I long terminal repeat, but there has been no direct evidence that CBP is required for Tax-mediated transactivation. We present evidence that Tax and CBP activate transcription from the HTLV-I 21 base pair repeats on naked DNA templates. Transcriptional activation of the HTLV-I sequences required both Tax and CBP and could be mediated by either the N-terminal activation domain of CBP or the full-length protein. Fluorescence polarization binding assays indicated that CBP does not markedly enhance the affinity of Tax for the trimeric complex. Transcriptional activation of HTLV-I sequences in the human T-cell lymphotrophic virus type I (HTLV-I) promoter (25–28). CBP was first identified as a component of the CREB activation pathway (29, 30) and was shown to interact specifically with the phosphorylated form of CREB. Subsequently, CBP was shown to interact with Tax, leading to the hypothesis that Tax, CREB, and CBP form a trimeric complex on the CRE-like sequences in the HTLV-I LTR (25–28). Interestingly, in the presence of Tax, CBP binding and transactivation of the viral LTR does not require CREB to be phosphorylated (25, 31). Presumably, CBP is recruited through Tax and not through the activation domain of CREB.

The presence of CBP in the HTLV-I transcription complex provides several additional possible mechanisms for gene activation. CBP has been reported to interact with the basal transcription factors TFIIA and TFIID (22–24). The role of CREB in this model is to allow the binding of Tax to DNA rather than to provide a direct transcriptional activation function because nonphosphorylated (inactive) CREB can substitute for the phosphorylated (active) form of the transcription factor. In contrast, the activation of cellular promoters is believed to require phosphorylated CREB, and CREB mutants that cannot be phosphorylated function as dominant negatives.

Recent studies have suggested that the transcriptional coactivator CBP also contributes to Tax-mediated activation of the HTLV-I LTR (25–28). CBP was first identified as a component of the CREB activation pathway (29, 30) and was shown to interact specifically with the phosphorylated form of CREB. CBP was shown to interact with Tax, leading to the hypothesis that Tax, CREB, and CBP form a trimeric complex on the CRE-like sequences in the HTLV-I LTR (25–28). Interestingly, in the presence of Tax, CBP binding and transactivation of the viral LTR does not require CREB to be phosphorylated (25, 31). Presumably, CBP is recruited through Tax and not through the activation domain of CREB.
for gene regulation (29, 30, 39–59).

In this report, we utilized in vitro transcription assays to determine the requirement for CBP in Tax-mediated activation of the HTLV-I LTR. We found that CBP activates both transcriptional initiation and reinitiation. This ability of CBP to activate transcription does not occur simply through the stabilization of Tax binding, but rather through activation properties of CBP itself. These studies also demonstrate that the ability of CBP to activate transcription does not depend entirely on its intrinsic or associated histone acetyltransferase functions.

**Experimental Procedures**

In Vivo Transcription Assays—The G-free DNA templates used in the in vitro transcription assays were pLovTATA and pTRE-1Id (23) and 4 TxRE (a generous gift of Dr. Mark Anderson, Medical College of Georgia). Escherichia coli-expressed Tax protein was purified by ammonium sulfate precipitation as described (60). For the in vitro transcription reactions, preincubation was at 30 °C for 30 or 45 min, followed by the addition of 2 μl of (α-32P)UTP (Amersham Pharmacia Biotech, 400 Ci/mmol), and incubation at 30 °C for 60 min. In experiments containing Sarkosyl, 0.02–0.1% Sarkosyl was added with the [32P]UTP after preincubation. Reactions contained HeLa whole cell extract, 12.5 μg/ml poly(dI:dC), 1.0–1.5 μg supercoiled DNA, 100 ng of Tax protein, 0.75–2.25 μg of CBP (1–682), or 2.5 μg of full-length CBP in a total volume of 50–65 μl. Transcription buffer (30.5 μl/reaction) contained 3 μl of 20% PEG (6000), 3 μl of 50 mM MgCl2, 3 μl of 1 mM dithiothreitol, 1 μl of 0.2 mM creatine phosphate (Boehringer Mannheim), 1.5 μl of 50 mM ATP/CTP, 1 μl of 20 mM 3′-O-methylguanosine 5′-triphosphate (Amersham Pharmacia Biotech), 20 units of RNase T1 (100 units/μl, Boehringer Mannheim), and 15 μl of Buffer D containing a final concentration of 20 mM HEPES (pH 7.9), 100 mM KCl, 12.5 mM MgCl2, 0.1 mM EDTA, 17% glycerol, and 1 mM dithiothreitol. For pulse-chase assays, Tax, CBP (1–682), and whole cell extract were incubated in the presence of [32P]UTP for 15 min. A 10-fold molar excess of cold UTP was added to the reaction, and the polymerase complexes were allowed to elongate for 15–60 min. 3′-O-Methyl-GTP was omitted in these assays to allow pol II elongation. Sarkosyl (0.02%) was added to inhibit reinitiation complexes in duplicates of the 15- and 60-min chase samples.

Protein Electroporation Assays—Wild-type and Tax mutant M47 protein were electroporated with the HTLV-I LTR-CAT or HIV-LTR-CAT plasmid into Jurkat cells as described previously (61).

Expression and Purification of Full-length CBP from SF9 Cells—Two copies of the FLAG epitope were added to the C terminus of full-length CBP subcloned into pFAST Bac Life Technologies, Inc.) The pFAST-Bac-CBP-2x FLAG vector was then transformed into DH10Bac competent cells. The recombinant bacmid DNA was prepared from the positive colonies and transfected into SF9 cells. The expression of full-length CBP in SF9 cells was confirmed by immunoprecipitation using anti-CBP and anti-FLAG antibodies. The FLAG-tagged CBP was purified on a FLAG affinity column (Kodak) and eluted with FLAG peptide. The mobility of the FLAG-tagged CBP determined on a 6% polyacrylamide electrophoresis gel was identical to that of CBP isolated from HeLa cells.

Tax, CBP, and CREB Expression Vectors and Protein Purification—Tax protein was overexpressed in bacteria and purified as described previously (60). CBP (1–682), CBP-(451–682), and CBP-(509–682) were expressed as polyhistidine fusion proteins by inserting polymerase chain reaction fragments into pET-15b (Novagen). Proteins were purified by nickel-affinity chromatography (Qiagen), followed by cation-exchange fast protein liquid chromatography (HiTrap SP, Amersham Pharmacia Biotech) (50). Full-length FLAG-tagged CBP was expressed in SF9 cells and purified by affinity chromatography (Qiagen).

Fluorescence Polarization Assays—Association of Tax with the CREB-TxRE complex was measured by fluorescence polarization as described previously (25, 27). Binding reactions (1 ml) contained 25 mM Tris-HCl (pH 7.5), 50 mM NaCl, 5 mM MgCl2, 0.5 mM EDTA, 1 mM dithiothreitol, 5% glycerol, 5 μg/ml poly(dI:dC), 50 μg/ml bovine serum albumin. Fluorescence anisotropy measurements utilized a fluorescence-tagged duplex oligonucleotide derived from the promoter proximal Tax-responsive sequence (F-TxRE) (25). Binding reactions containing 5 nM F-TxRE and 30 nM CREB341 were titrated with increasing concentrations of purified Tax in the absence or presence of either 2.1 μM CBP-(451–682) or 2 μM CBP-(1–682). The Kb for the association of Tax with CBP under these conditions is approximately 90 nM (25, 27), thus all Tax added to the binding reactions is effectively bound to CBP prior to association with the CREB-TxRE complex. Binding affinity parameters were determined by nonlinear regression (SigmaPlot) as described previously (25, 27).

**Results**

Tax and CBP Stimulate Transcription from the HTLV-I 21 Base Pair Repeats—Fig. 1A shows a schematic of the HTLV-I LTR with the Tax-responsive 21-bp repeats (hatched boxes) is shown. For in vitro transcription assays, three templates were utilized. 4TxRE contains four copies of 21-bp repeat 1 inserted upstream of the HTLV-I TATA and promoter (−52 to −1) and G-free cassette (provided by Dr. Mark Anderson). pTRE-1g was constructed by inserting two copies of the 21-bp pair repeat upstream of the chicken ovalbumin TATA box in the G-free cassette plasmid pLovTATA. B, in vitro transcription with pLovTATA, pTRE-1g, and 4TxRE (1.5 μg each) in the presence of purified Tax (100 ng), CBP-(1–682) (760 ng), or Tax plus CBP-(1–682) were carried out as described under “Experimental Procedures.” Reactions were preincubated for 45 min at 30 °C. [32P]UTP (20 μCi) was added and incubation continued for 1 h at 30 °C. [32P]RNA was purified and analyzed on a denaturing acrylamide urea gel. Lanes 1–4, pLovTATA alone, plus Tax, plus CBP, plus Tax and CBP; lanes 5–8, pTRE-1g alone, plus Tax, plus CBP, plus Tax and CBP; lanes 9–12, 4TxRE alone, plus Tax, plus CBP, plus Tax and CBP. The position of the 380- and 390-base G-free transcripts are indicated by the arrows.

![Fig. 1. Tax and CBP (1–682) stimulate in vitro transcription from templates containing the HTLV-I 21-bp repeats upstream of a G-free cassette. A, a schematic of the HTLV-I LTR with the Tax-responsive 21-bp repeats (hatched boxes) is shown. For in vitro transcription assays, three templates were utilized. 4TxRE contains four copies of 21-bp repeat I inserted upstream of the HTLV-I TATA and promoter (−52 to −1) and G-free cassette (provided by Dr. Mark Anderson). pTRE-1g was constructed by inserting two copies of the 21-base pair repeat upstream of the chicken ovalbumin TATA box in the G-free cassette plasmid pLovTATA. B, in vitro transcription with pLovTATA, pTRE-1g, and 4TxRE (1.5 μg each) in the presence of purified Tax (100 ng), CBP-(1–682) (760 ng), or Tax plus CBP-(1–682) were carried out as described under “Experimental Procedures.” Reactions were preincubated for 45 min at 30 °C. [32P]UTP (20 μCi) was added and incubation continued for 1 h at 30 °C. [32P]RNA was purified and analyzed on a denaturing acrylamide urea gel. Lanes 1–4, pLovTATA alone, plus Tax, plus CBP, plus Tax and CBP; lanes 5–8, pTRE-1g alone, plus Tax, plus CBP, plus Tax and CBP; lanes 9–12, 4TxRE alone, plus Tax, plus CBP, plus Tax and CBP. The position of the 380- and 390-base G-free transcripts are indicated by the arrows.](http://www.jbc.org/)
A

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\begin{array}{c}
\text{CBP/p300} \\
\text{CREB binding domain} \\
\text{Zinc Finger} \\
\text{CBP} (1-682) \\
\text{CBP} (451-682) \\
\text{CBP} (509-682) \\
\text{Tax} \\
\end{array}
\]

B

\[
\begin{array}{c}
\text{CBP (509-682)} \\
\text{CBP (451-682)} \\
\text{CBP (1-682)} \\
\text{Tax} \\
\end{array}
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Fig. 2. CBP expression vectors and in vitro transcription assays. A, CBP-(1–682), CBP-(451–682), and CBP-(509–682) were expressed as polyhistidine fusion proteins by inserting PCR fragments into pET-15b (Novagen). The His-tagged fusion proteins were purified by nickel-affinity chromatography (Qiagen), followed by cation-exchange fast protein liquid chromatography (HiTrap SP, Amersham Pharmacia Biotech) (51). B, in vitro transcription assays with template pTRE-IId (1.5 μg) and equal amounts (750 ng) of CBP-(1–682), CBP-(451–682), or CBP-(509–682) in the presence of 100 ng of Tax (lanes 3–5), or Tax alone (lane 2). Lane 1 reflects the basal level of transcription from template pTREIId in the absence of Tax and CBP-(1–682). The position of the 360-base transcript is indicated by the arrow.

In vitro transcription from pTRE-IId (Fig. 1B, lanes 5–8) and 4TxRE (Fig. 1B, lanes 9–12). Addition of Tax or CBP-(1–682) alone failed to activate transcription from pTRE-IId and only minimally activated 4TxRE. The template that lacked the 21-bp repeats, pLovTATA, was not transactivated by the addition of Tax, CBP-(1–682), or Tax and CBP-(1–682) (Fig. 1B, lanes 1–4), demonstrating that transcriptional activation depends upon the TRE-1 sequences.

We subsequently tested two fragments of CBP-(1–682), designated CBP-(451–682) and CBP-(509–682) (Fig. 2A). In the absence of exogenous CBP-(1–682), Tax modestly activated transcription from the TRE-IId template (Fig. 2B, lanes 1 and 2). The addition of Tax and CBP-(1–682) stimulated the appearance of the 360-base transcript by 10-fold (Fig. 2B, lane 3). Addition of CBP-(451–682) or CBP-(509–682), which retain the Tax interaction domain, failed to activate transcription (Fig. 2B, lanes 4 and 5), suggesting that the CBP N terminus is essential for Tax activity. Swope et al. (32) have reported that CBP-(1–400) interacts with the basal transcription factor TBP. Thus, our results suggest that the interaction of CBP-(1–682) with TBP (TFIID) may be important for Tax-dependent CBP activation.

As additional controls to demonstrate the specificity of the in vitro transcription assays, antibody blocking experiments were performed. Addition of either CBP or Tax antibodies, but not control antibodies, inhibited the in vitro transcription (data not shown).

The wild-type Tax protein, which can activate both the CREB and NF-kB-responsive promoters (62), was tested for its ability to activate either CREB-dependent or NF-kB-responsive promoters. The addition of wild-type Tax and CBP-(1–682) significantly increased transcription from the template containing two copies of the 21-bp repeats (Fig. 3A, lanes 1–4). In contrast, the M47 Tax protein failed to activate transcription in the presence of CBP-(1–682) (Fig. 3A, lanes 5–7).

It could be argued that the M47 protein becomes inactivated during purification. To demonstrate the biological activity of the Tax M47 mutant protein, we electroporated the wild-type and mutant Tax protein along with a CREB-dependent or NF-kB-dependent reporter plasmid. This assay takes advantage of the fact that M47 Tax has a mutant phenotype for CREB-dependent transcription, but retains a wild-type activity for transcriptional activation through the NF-kB element (62).

Equal amounts of the wild-type and M47 mutant protein were electroporated with either pU3R-CAT (HTLV-I LTR; CREB-responsive) or LTR-CAT (HIV LTR; NF-kB-responsive) (61). The wild-type Tax protein, which can activate both the CREB and NF-kB pathways, transactivated both the HTLV-I LTR and HIV LTR (Fig. 3B, lanes 2 and 5). In contrast, the M47 mutant protein only minimally transactivated the CREB-responsive pathway.
HTLV-I LTR, but transactivated the NF-kB-responsive HIV LTR normally (Fig. 3B, lanes 3 and 6). These experiments demonstrate that the M47 protein was biologically active.

The Combination of Tax and CBP-(1–682) Facilitates Transcription Initiation and Reinitiation.—To distinguish the effects of CBP on transcriptional initiation and reinitiation, we performed in vitro transcription assays in the presence of low concentrations of the detergent Sarkosyl, which inhibits reinitiation and limits transcription to a single round (63, 64). Polymerase II preinitiation complexes were formed on the 4TxRE template in the presence or absence of Tax and CBP-(1–682), and initiation was allowed to proceed before Sarkosyl was added to prevent transcription reinitiation. The addition of 0.1% Sarkosyl to the reactions inhibited transcription (Fig. 4A, lanes 7 and 8), indicating that a relatively small number of active templates are generated by the addition of Tax and CBP-(1–682) in the initial round of transcription. PhosphorImager™ quantitation of gel indicates that each initiation template supports an average of 25–50 rounds of transcription.

To visualize transcripts arising from reinitiation, 3'-O-methyl-GTP was added to the in vitro transcription assays. As described by Szentirmay and Sawadogo (65), in the presence of 3'-O-methyl-GTP, the first round of pol II elongation complexes remain at the end of a G-free region, blocking the elongation of pol II complexes that result from reinitiation. This results in successive polymerases stacking up from the end of the cassette, thus producing shorter transcripts representing rounds of reinitiation. In vitro transcription reactions were set up as described above. Following preincubulation for 30 min, Sarkosyl was added to a final concentration of 0.02–0.04%. Following a 60-min incubation in the presence of [32P]UTP, RNA was isolated and analyzed on a denaturing acrylamide-urea gel. CBP-(1–682) (750 ng) and/or Tax (100 ng) were added to the reactions as indicated. 32P-RNA was purified and separated on a denaturing acrylamide urea gel. The position of the full-length transcript (360 base) and reinitiation transcripts of 265, 230, 195, 165, 150, and 130 bases were indicated by arrows. Arrows with dashed lines indicate less abundant reinitiation products. D, Sarkosyl inhibits reinitiation complexes.
HAT-independent CBP Coactivator Function with HTLV-I Tax

CSP Does Not Stabilize the Association of the CREB-Tax Complex with DNA—Previous studies by Lenzmeier et al. (28) suggest that CBP association with Tax may stabilize the association with the Tax, assayed by enhancement of the Tax-

DISCUSSION

The Tax protein of HTLV-I utilizes a novel mechanism for activating gene expression, facilitating the formation of a multimeric CBP-Tax-CREB complex on the Tax-responsive DNA element. Tax facilitates the binding of the cellular transcription factor CREB to the atypical CRE-like sequences within the viral LTR. Recent data indicate Tax and CREB both contact DNA, Tax interacting with GC sequences in the minor groove, and CREB associating with the CRE-like sequences in the major groove (13, 28). In this configuration, only the B-ZIP domain of CREB is required for formation of the ternary Tax-CREB-DNA complex. Consequently, nonphosphorylated CREB, as well as the negative modulator CREMα, are competent to mediate Tax binding. This model differs significantly from an earlier one which suggested that Tax functioned primarily by promoting CREB dimerization (15).
The exact role of CBP in the process of Tax-mediated gene activation has been puzzling. Despite the fact that CBP was originally identified through its ability to interact specifically with phosphorylated CREB, the CREB activation domain appears to be dispensable for Tax-mediated induction of the HTLV-I LTR. The synergistic interaction of Tax and CREB is believed to recruit the co-activator CBP in a manner that does not require CREB phosphorylation probably because, in this context, CBP interacts with Tax rather than CREB. The mechanism of gene activation could depend upon Tax, which has been shown to interact with TFIIA and TFIID (22–24), or CBP, which interacts with TBP, TFIIB, RNA polymerase holoenzyme, and histone acetyltransferases (32, 35–37, 66), or both.

Our anisotropy studies show that CBP-(1–682) probably does not function simply by stabilizing Tax binding to the TRE sequences. Rather, a more direct role of CBP in gene activation is likely. While the histone acetyltransferase function of CBP and associated proteins could participate in the activation of the HTLV-I LTR in vitro, our studies show that CBP can also strongly activate transcription in the absence of chromatin. Our studies demonstrate for the first time that the N-terminal activation domain of CBP, in the presence of Tax, stimulates transcription of a Tax-responsive transcriptional template. Deletion of the TBP binding domain (32) abolishes the activity of CBP-(1–682) in vitro, suggesting that transcriptional activation is due, in part, to interaction with the basal transcription factors.

Our results do not imply that CBP/p300 histone acetyltransferase (HAT) activity is not important for the transcription of some genes (36, 37). For example, steroid receptor induction of gene transcription has been proposed to involve two independent functions of coactivators, chromatin remodeling and enhanced stabilization of the preinitiation complex (67). Moreover, Kraus and Kadonaga (68) have reported that the CBP homologue p300 mediates estrogen receptor-activated gene induction in vitro only in the context of a chromatin template. Acetylated histones are a characteristic feature of transcriptionally active chromatin. Because CBP acetylates histones (HAT activity), it is likely that the coactivator increases transcription of some genes through remodeling of chromatin structure. In addition, the acetyltransferase domain may contribute to transcriptional activation through other mechanisms. For example, Gu and Roeder (69) have demonstrated that CBP increases p53 DNA binding through acetylation of the p53 C-terminal regulatory domain. In addition, Imhof et al. (70) recently demonstrated that p300 acetylates TFIIE p56 and TFIIF (FAT activity). The effect of acetylation on the function of these basal transcription factors remains to be determined.

Our data further suggest that Tax and CBP-(1–682) facili-
tate both transcriptional initiation and reinitiation. The CREB-Tax-CBP complex may facilitate the recruitment of basal transcription factors into the initiation complex and, subsequently, stabilize the promoter complex from dissociation, thereby facilitating entry of pol II into the reinitiation complex. Zawel et al. (71) have analyzed the recycling of general transcription factors during RNA polymerase II transcription. All of the basal factors are found in the mature initiation complex, but following nucleotide addition, the complex becomes disrupted. TFIID remains bound to the promoter, and following release, TFIID reassociates with TFIIID (DB complex), forming the RNA pol II docking site for reinitiation. In agreement with Giebler et al. (26), we have found that the CREB-Tax-CBP complex may facilitate the recruitment of basal transcription factors.

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