The Histone Acetyltransferase, hGCN5, Interacts with and Acetylates the HIV Transactivator, Tat*

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Factor acetyltransferase activity associated with several histone acetyltransferases plays a key role in the control of transcription. Here we report that hGCN5, a well known histone acetyltransferase, specifically interacts with and acetylates the human immunodeficiency virus type 1 (HIV-1) transactivator protein, Tat. The interaction between Tat and hGCN5 is direct and involves the acetyltransferase and the bromodomain regions of hGCN5, as well as a limited region of Tat encompassing the cysteine-rich domain of the protein. Tat lysines 50 and 51, target of acetylation by p300/CBP, were also found to be acetylated by hGCN5. The acetylation of these two lysines by p300/CBP has been recently shown to stimulate Tat transcriptional activity and accordingly, we have found that hGCN5 can considerably enhance Tat-dependent transcription of the HIV-1 long terminal repeat. These data highlight the importance of the acetylation of lysines 50 and 51 in the function of Tat, since different histone acetyltransferases involved in distinct signaling pathways, GCN5 and p300/CBP, converge to acetylate Tat on the same site.

Histone acetyltransferases (HATs) are enzymes capable of acetylating specific lysine residues in the N-terminal tails of core histones. This acetylation would modify DNA-nucleosome or nucleosome-nucleosome interactions, and then facilitate gene activation (reviewed in Refs. 1–4). The link between histone acetylation and transcriptional activation has been confirmed by the detection of a HAT associated with transcription factors, some characterized first as cofactors of transcriptional activators (reviewed in Ref. 5). Furthermore, it has been shown that histone acetyltransferases p300, CBP, and P/CAF also possess a factor acetyltransferase (FAT) activity, in that they acetylate non-histone substrates, such as specific and general transcription factors or chromatin-related proteins (reviewed in Ref. 5). Factor acetylation can modify protein-DNA or protein-protein interaction depending on the nature of the substrate. For instance, acetylation of the transcriptional activators GATA-1 by p300 (6), p53 by p300 and P/CAF (7–9), and E2F (10, 11), or TAL1 (12) by P/CAF increases their DNA-binding capacities, whereas acetylation of E1A 12S and TAL1 by P/CAF inhibits their interaction with a co-repressor (12, 13). Finally, the acetylation of the chromatin-associated protein HMG-17 by P/CAF reduces its affinity for nucleosomes (14). Protein acetylation may also regulate other cellular functions such as the stability or nuclear import of proteins (10, 15).

Tat, a viral protein encoded by human immunodeficiency virus 1 (HIV-1), activates viral gene transcription from the proviral long terminal repeat (LTR) by interacting with several cellular factors. One such factor is the Tat-associated kinase CDK9/P-TEFb, which is recruited by Tat on the transcription response RNA (TAR) element, located at the 5’ termini of all viral transcripts. This complex stimulates transcriptional elongation by phosphorylating RNA polymerase II C-terminal domain (Refs. 16 and 17; reviewed in Refs. 18 and 19). Interestingly, it has recently been shown that in addition to cellular kinases Tat can also recruit cellular HATs (20–24). Two nuclear HATs, p300 and P/CAF, were found to interact with and acetylate Tat on distinct lysine residues (25, 26). The acetylation of the activator domain of Tat by P/CAF enhances the binding of Tat to the cellular factor CDK9/P-TEFb, whereas the acetylation of the TAR binding domain of Tat by p300 promotes its dissociation from TAR element during early transcriptional elongation, and both events increase the activation of transcription from the LTR. Thus, HIV-1 appears to develop the capacity to use the cellular acetylation signaling system to enhance its transcription (25, 26) and probably to control various cellular functions (20, 24).

GCN5 is one of a number of well characterized nuclear HATs that acetylate histones H3 and H4 at specific residues (27, 28). GCN5 is a conserved protein from yeasts to humans (27, 29–31). Human GCN5 (hGCN5) exists in two forms resulting from alternative splicing (30, 31). The longer form (813 amino acids) shares strong homologies with P/CAF. The shorter form (476 amino acids) exhibits a size similar to yeast GCN5 and corresponds to the C-terminal part of the long hGCN5.

In contrast to its homologue P/CAF, GCN5 has been described as a poor transcriptional coactivator in mammals. Additionally, it is not known whether hGCN5, like p300, CBP, and P/CAF, uses its acetyltransferase activity to modulate transcription factors by acetylation.

In this report we investigated the ability of hGCN5 to acetylate and modulate the activity of the HIV transactivator, Tat. Data presented here show that hGCN5 interacts directly with Tat. The domains involved in this interaction were mapped on both proteins and the site of acetylation on Tat characterized. hGCN5 was found to acetylate Tat in vitro on lysine 50 and 51.
In agreement with these findings, hGCN5 was shown to stimulate Tat-dependent transcriptional activation of the HIV-1 LTR. These results point to hGCN5 as a novel “FAT” that can take part in the activation of HIV gene expression by acetylating the viral transactivator Tat.

**MATERIALS AND METHODS**

**Plasmid Constructs**—Expression vectors for short hGCN5, wild type (wt), and mutants were constructed as follows. Coding sequences for hGCN5 and different mutants were produced by PCR with appropriate primers and forward primers: 5’-GCGTCTAGAGATCCGCAGAGCAAGTCAGTCA-3’ for GATC for wt and mutants 1–388 and 1–110, 5’-GCGTCTAGAGATCCGCAGAGCAAGTCAGTCA-3’ for 389–476 and 252–476, and 5’-CTCGTCTAGAGATCCGCAGAGCAAGTCAGTCA-3’ for mutants 1–110; 5’-GGGATCCCAAGCTATAGGGAGG-3’ for wt and mutants 1–110, 252–276, and 389–476; 5’-GGGATCCAACTGCT-3’ for reverse primers. Ten micrograms of each sample were used to perform a Southern blot analysis. The blot was hybridized with a plasmid containing 0.5% SDS, and digested with 0.1 mg/ml proteinase K. Ten micrograms of GST pulldown assay were performed with purified recombinant His-tagged hGCN5 as described. Results show that the interaction of Tat with one domain may occur independently with the other domains of hGCN5, without any cellular intermediates.

**RESULTS**

**Tat and GCN5 Interact Directly in Vitro**—GST-Tat pull-down assays were set up to investigate the ability of Tat to interact with hGCN5: GST-Tat (Tat86, HXB2 strain) fusion protein and GST (as a control) were produced in *E. coli*, immobilized on glutathione-Sepharose beads, and incubated with HeLa nuclear extracts expressing flag-tagged hGCN5 (f-hGCN5; Fig. 1A). A Western blot using an anti-Flag antibody revealed that flag-tagged hGCN5 is efficiently retained on GST-Tat beads, but not on GST. hGCN5 present in the HeLa nuclear extracts could therefore specifically interact with Tat. To show direct interaction between hGCN5 and Tat, GST-Tat pulldown assays were performed with purified recombinant His-tagged hGCN5 protein, followed by Western blotting using an anti-His antibody for detection of bound tagged hGCN5. Tat from two different viral strains, Tat 86 (HXB2 strain) and Tat 101 (SF2), were produced in fusion with GST. Fig. 1 (B and C) shows that recombinant hGCN5 is specifically retained on Tat 86 as well as on Tat 101. We can conclude from these experiments that hGCN5 is capable of interacting directly with both forms of Tat, without any cellular intermediates.

**Mapping the Tat-interacting Domains of GCN5**—To determine the domains of hGCN5 involved in interaction with Tat, we constructed several hGCN5 deletion mutants, corresponding to regions encompassing amino acids 1–110, 111–251 (contains the HAT domain), 252–388, and 389–476 (contains the bromodomain), or combinations of two or three adjacent domains (Fig. 2A). 35S-Labeled mutants were generated in vitro and tested for their interaction with Tat in a GST-Tat pull-down assay. The N-terminal 1–110 part of hGCN5 as well as the 252–388 region were unable to interact with Tat (Fig. 2, B (lanes 2 and 6) and D). Two regions of hGCN5, encompassing amino acids 111–151 and 389–476 (corresponding to HAT and bromodomain, respectively) efficiently interacted with Tat (Fig. 2, B (lanes 4 and 8) and D). All the hGCN5 fragments used in the experiment presented on Fig. 2 (C and E) contained either the HAT or the bromodomain and consequently interacted with Tat.

We conclude from these results that the HAT domain and the bromodomain of hGCN5 are two Tat-interacting modules and that the interaction of Tat with one domain may occur independently of the interaction with the other domain.

**Mapping hGCN5-interacting Domain of Tat**—We then mapped Tat domains necessary for hGCN5 interaction. GST-Tat pulldown assays were carried out with different C-terminal truncated forms of Tat 101 (Fig. 3A) fused to GST, immobilized on glutathione-Sepharose beads, and incubated with purified recombinant hGCN5 as described. Results show that the hGCN5-Tat interaction is maintained for a Tat fragment containing the 48 first residues (Fig. 3B, M3 mutant, lane 5). However, the interaction is completely abolished for the M4 mutant (amino acids 1–22) (Fig. 3B, lane 6). These results suggested that the Tat domain responsible for interaction...
acting with hGCN5 was localized between amino acids 22 and 48. In order to confirm this hypothesis, we generated a new Tat mutant containing only the amino acids 21–48 region of the protein fused to GST (M5 mutant). Interestingly, hGCN5 interacted with this Tat mutant as efficiently as with the wild type protein (Fig. 3B, lanes 8 and 9). This experiment suggests that the amino acids 21–48 region of Tat is necessary and sufficient for the interaction of Tat-hGCN5. This region contains the cysteine-rich and core domains of Tat and is located in the minimal activation domain of the protein.

hGCN5 Acetylates Tat on Residues K50 and K51—Recently, it has been discovered that p300 and P/CAF acetyltransferases modulate Tat transcriptional activity by directly acetylating the protein on specific and distinct lysines (25, 26). We examined if hGCN5 was also capable of acetylating Tat. Purified GST-Tat or GST (as a negative control) was incubated with increasing amounts of purified recombinant hGCN5, in the presence of 14C-labeled acetyl-CoA. Proteins were then electrophoresed on a denaturing gel, and the acetylation of Tat revealed by autoradiography. We observed that the incubation of Tat with increasing amounts of hGCN5 led to the appearance of two major radiolabeled bands corresponding to GST-Tat and hGCN5, respectively (Fig. 4A, right panel). This experiment also showed that, in addition to acetylating Tat, hGCN5 is also capable of undergoing auto-acetylation. In the control experiment, using GST as a substrate, only auto-acetylation of hGCN5 was observed (Fig. 4A, left panel). We concluded from these experiments that hGCN5 specifically acetylates Tat in vitro.

To define which lysines of Tat are the site of acetylation by hGCN5, similar experiments as above were performed using the wild type Tat 101 or Tat deletion mutants as substrate (Fig. 4B). hGCN5 could efficiently acetylate wild type Tat, and M1 and M2 mutants, but could not acetylate Tat M3 and M4 mutants (Fig. 4C, lanes 5 and 6). The region of Tat acetylated by hGCN5 is therefore located between amino acids 48 and 60 and corresponds to the basic domain of Tat. This region contains only two lysines at positions 50 and 51 (Fig. 4B). Point mutations of either Lys-50 or Lys-51 were then generated to determine which lysine was the target of hGCN5. In both cases, mutation of a single lysine reduced the acetylation of Tat (Fig. 4C).
HIV LTR transcriptional activity was further enhanced, although to a lesser extent than that of the non-integrated LTR, by its coexpression with hGCN5 (Fig. 5B). Here again, although Tat Lys-50/Lys-51 mutant efficiently stimulated the HIV-1 LTR transcription, it was not as efficient as the wild type protein in cooperating with hGCN5.

These results strongly suggest that acetylation of Tat on Lys-50 and Lys-51 by hGCN5 plays an important role in the control of the activity of the protein.

**DISCUSSION**

The HIV transactivator Tat interacts with several histone acetyltransferases, such as Tip60 (24, 33), hTAFII 250 (20), p300, CBP, and P/CAF (21–23). We report here that hGCN5 is a novel Tat-interacting HAT. This interaction occurs directly,
Acetylation of Tat by the HAT hGCN5

Fig. 5. hGCN5 enhances the activation of the HIV LTR by Tat. A, HeLa cells were co-transfected with 50 ng of reporter plasmid pLTR-Luc (luciferase gene under LTR control), 0 or 10 ng of the Tat (wt or K50/S1) double mutant) expression vector pSGTat, and 0 (−) or 400 ng (+) of the flag-tagged hGCN5 expression vector pf-gCN5, as indicated. Total amounts of DNA for transfections were maintained constant by addition of empty control vector. Luciferase activity was measured with a luminometer and normalized with respect to the plasmid uptake as indicated under “Materials and Methods.” Mean values of at least three independent assays are represented. B, HLT2T1 cells, containing an integrated LTR-CAT reporter gene, were co-transfected with 10 ng of pSGTat wild type or K50/S1 mutant and 0 (−) or 400 ng (+) of pf-gCN5. Transfections were performed as described in A. Quantification of the CAT protein was carried out by an enzyme-linked immunosorbent assay and mean values of three independent experiments were represented as in A.

via residues 21–48 of Tat, a region contained in the minimal transactivating domain of Tat. This finding is in agreement with the fact that the amino acid 30–45 region of Tat was required for recruitment of HAT activity from nuclear cell extracts (21). Interestingly, the Tat hGCN5-interacting domain appears to differ from domains involved in interaction with other HATs. For instance, the basic domain of Tat has been shown to be required for Tip60 and p300 interaction, whereas the Tat C-terminal domain is necessary for hTAF1250 interaction (20, 22–24). Otherwise, similar to p300/CBP and P/CAF, hGCN5 is capable of stimulating Tat-dependent LTR transcriptional activation and of acetylating Tat on specific lysines. Tat-Tip60 and Tat-TAF1250 interactions do not, however, affect transcription from the LTR but repress transcription of cellular genes such as the manganese superoxide dismutase gene (24) and the major histocompatibility class I genes (20). Thus, although the targeting of HATs would be a general mechanism of Tat activity, the HAT-interacting domain of Tat and possibly the functional consequences of these interactions could be different for each HAT.

In contrast to P/CAF or p300, only a few reports show a direct recruitment of GCN5 by transcriptional factors in mammals. Long hGCN5 is known to be recruited by c-Myc to activate transcription, but this recruitment was shown to be indirect, and to require the cofactor TRRAP (34). A direct interaction has, however, been described between short hGCN5 and the ubiquituous transcription factor, NF-Y. The interaction was mapped to the N-terminal domain of hGCN5 and to the histone-like DNA binding domain of NF-Y (35). Therefore, the recruitment of hGCN5 by Tat, reported here, constitutes a new example of recruitment of hGCN5 by a transcription factor.

Acetylation of non-histone substrates by GCN5 has also been, to our knowledge, poorly described. Acetylation of E1A by yGCN5 and c-Myb by hGCN5 has been recently reported, but the effect of GCN5 on these transcription factors was not further characterized (13, 36). In this report, we have identified Tat as a new substrate of GCN5 FAT activity. It is therefore very probable that GCN5 (like p300 and P/CAF), acetylates different transcription factors, and more generally other cellular and viral proteins (not necessarily involved in transcription) to modulate their activity.

Since c-Myb and Tat are acetylated in vitro by GCN5 and P/CAF, and since both HATs are closely related, it can be speculated that hGCN5 and P/CAF have redundant FAT functions. However, our data show divergences between P/CAF and hGCN5 activity on Tat. First, unlike hGCN5, P/CAF does not stimulate Tat transcriptional activity by itself; instead, it enhances the stimulating effect of p300. Indeed, it has been suggested that Tat, p300/CBP, and P/CAF associate in a ternary complex, and that in this complex, p300-P/CAF interaction is required for a stimulating effect of p300 on Tat activity (21). In our experiments we used the short form of hGCN5, which is unable to interact with p300/CBP (37). Therefore, the short hGCN5 would then act on Tat by a different and more direct mechanism, which does not require the simultaneous recruitment of p300. Finally, functional differences between hGCN5 and P/CAF are confirmed by the fact that they target different lysines within Tat; P/CAF acetylases Tat on lysine 28 (25), whereas hGCN5 targets lysines 50 and 51. This observation suggests that, despite strong homology and preferential acetylation of the same lysine (Lys-14) within the same histone (H3) (28, 38), these HATs could in certain cases recognize different lysines in the same substrate.

Our data show that, although divergent in sequence and belonging to different families, hGCN5 and p300/CBP acetylate Tat on the same lysines. The use of different cellular HATs by Tat to acetylate the same site highlights the importance of this post-translational modification for the activity of Tat. In support of this hypothesis, the acetylation of lysine 50 has been correlated to a lower affinity of Tat for the TAR sequence (25). Thus, the acetylation of Tat would increase the rate of its dissociation from TAR element. The consequence of this acetylation would be a faster recycling of each molecule of Tat, and an increased transcription of the HIV LTR. Data presented here show that hGCN5 is also involved in the acetylation-dependent control of Tat activity. However, the role of hGCN5 and other HATs capable of acetylating Tat in activating the LTR transcription in the context of HIV-infected cells remains to be determined.

Interestingly, the hGCN5-interacting domain of Tat (amino acids 21–48) is different from the acetylated region. Moreover, the recruitment of hGCN5 can occur independently of Tat acetylation, as showed by the M3 Tat mutant, which, although not acetylated by hGCN5 (Fig. 4C), interacted with this HAT (Fig. 3B). Thus, besides the control of Tat acetylation state, the hGCN5-Tat interaction may also target hGCN5 to the HIV-1 LTR to acetylate histones and then displace nucleosomes at the initiation or during the elongation steps of the transcription. Therefore, one may separate the role of Tat-hGCN5 interaction...
on HIV-1 LTR chromatin remodeling from its role on Tat acetylation.

Our data show also that the bromodomain of hGCN5 is capable of efficiently interacting with Tat. Bromodomains are conserved sequence motifs probably involved in protein-protein interactions (reviewed in Refs. 39 and 40). This domain is found in transcription-related proteins and especially in the Tat-interacting HATs p300/CBP, P/CAF, and TAFII250 (besides hGCN5). Recently, it has been shown that bromodomains of P/CAF, TAFII250, and GCN5 directly and specifically interact with acetylated histones (41–43). Moreover, in the case of hGCN5, the bromodomain is necessary for nucleosome remodeling by the Swi/Snf complex (44). By contacting the bromodomain of hGCN5, Tat could modify the action of hGCN5 on chromatin. It would therefore be interesting to know if such an interaction could perturb either recruitment of hGCN5 on acetylated histones or the activity of the Swi/Snf complex. Tat-hGCN5 interaction would then have consequences on the transcription of cellular genes. Furthermore, although the involvement of p300, P/CAF, and hTAFII250 bromodomains in the direct interaction with Tat has not been demonstrated, the Tat-bromodomain interaction could be a general mechanism of modulation of chromatin targeting by bromodomain-containing proteins.

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