Molecular Cloning and Characterization of a Novel Histone Deacetylase HDAC10*

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The growing number of proteins controlled by reversible acetylation suggests the existence of a large number of acetyltransferases and deacetylases. Here, we report the identification of a novel class II histone deacetylase, HDAC10. Homology comparison indicates that HDAC10 is most similar to HDAC6. Both contain a unique, putative second catalytic domain not found in other HDACs. In HDAC10, however, this domain is not functional. This tandem organization of two catalytic domains confers resistance to the inhibitors trapoxin B and sodium butyrate, which potently inhibit the deacetylase activity of all other HDAC members. Thus, HDAC10 and HDAC6 share unusual structural and pharmacological characteristics. However, unlike HDAC6, which is normally a cytoplasmic deacetylase, HDAC10 resides in both the nucleus and cytoplasm. In the nucleus, when tethered to a promoter, HDAC10 represses transcription independent of its deacetylase activity, indicating that HDAC10 contains a distinct transcriptional repressor domain. These observations suggest that HDAC10 might uniquely play roles both in the nucleus, as a transcriptional modulator, and in the cytoplasm in an unidentified role. Together, our results identify HDAC10 as a novel deacetylase with distinct structure, pharmacology and localization and further expand the complexity of the HDAC family.

It has been known for many years that histone acetylation, a post-translational modification on specific lysine residues, is important for regulating the transcription of many genes (1). It is generally believed that hypo-acetylated histones have an increased charge, allowing chromatin condensation and, consequently, are associated with transcriptionally silent regions. Conversely, hyper-acetylation of histones neutralizes this charge, permitting chromatin decondensation and, thus, is associated with transcriptionally active regions (reviewed by Wade et al. (2)). The acetylation of these lysine residues is catalyzed by histone acetyltransferases, whereas the removal of the acetyl group is carried out by histone deacetylases (HDACs).1 Recent studies, however, have expanded the substrate repertoire of histone acetyltransferases and HDACs, suggesting that the function of acetylation is not limited to histone metabolism or transcription but may regulate a variety of biological processes. Indeed, acetylation has been shown to regulate the DNA binding activity of several transcription factors (3, 4), nuclear import (5), microtubule function (6),2 and protein-protein interactions (7). Additionally, acetylation is an important modification in the pathway that leads to p53 activation and stabilization (8). These results suggest that acetylation is a critical post-translational modification that, like phosphorylation, has diverse biological effects.

Recent studies have identified nine members of the mammalian HDAC family, and they are divided into two classes, originally defined by size and sequence homology to the yeast prototypic HDACs. The class I HDACs, which are about 400–500 amino acids in size, include HDAC1 (9), HDAC2 (10), HDAC3 (11), and HDAC8 (12) and share homology to the yeast protease Rpd3. The larger class II members, all about 1000 amino acids, include HDAC4, HDAC5, HDAC6 (13), HDAC7 (14), and HDAC9 (15) and are homologous to the yeast Hda1p. The complexity of this family is illustrated by the subcellular localization patterns of the two classes. Although the class I HDACs are primarily nuclear, the class II HDAC4 and -5 can shuttle between the nucleus and cytoplasm. Their shuttling is involved in the regulation of myogenesis and controlled by calmodulin-dependent kinase-mediated phosphorylation (16, 17). Although predominantly nuclear, HDAC7 was found to bind the membrane-associated endothelin receptor A, implying that HDAC7 might also have a function in the cytoplasm (18). Unlike any previously identified HDAC, human HDAC6 is a cytoplasmic deacetylase (19).2

The discovery of non-histone acetylated proteins and cytoplasmic localization of the class II HDACs implies that acetylation is an important post-translational modification required for the proper regulation of a variety of biological processes. Indeed, HDAC inhibitors such as trichostatin A (TSA) and suberoylamide hydroxamic acid have anti-tumor effects as they can arrest cell growth (20, 21), induce differentiation of transformed cells in culture (21, 22), and prevent tumor growth in mice models (23). These results suggest that the inhibition of HDACs is a potential therapeutic approach for more potent cancer treatment. The emerging numbers of diverse and important proteins that are reversibly acetylated argue that different members of the HDAC family have a unique set of substrates and regulate different biological processes. Therefore, the identification of all acetyltransferases and deacetylases might hold significant insight into the full picture and complexity of acetylation biology.

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1 The abbreviations used are: HDAC, histone deacetylase; NaB, sodium butyrate; PID, p53 target protein in the deacetylases complexes; TPX-B, trapoxin B; TSA, trichostatin A; NLS, nuclear localization signal; PBS, phosphate-buffered saline.

2 C. Hubbert, A. Guardiola, R. Shao, A. Ito, A. Nixon, M. Yoshida, X. F. Wang, and T.-P. Yao, submitted for publication.
lase, HDAC10. Our analysis indicates that HDAC10 is most similar to HDAC6. Both HDAC10 and -6 contain two spaced catalytic domains. Although the second catalytic domain in HDAC10 is not functional, its presence confers resistance to the inhibitors trapoxin B (TPX-B) and sodium butyrate (NaB). Despite their similar structural and pharmacological characteristics, HDAC10 and HDAC6 localize to different subcellular compartments. Unique among all the HDACs identified so far, HDAC10 resides in both the nuclear and cytoplasmic compartments. In the nucleus, HDAC10 is capable of repressing transcription via a deacetylase-independent mechanism. Therefore, our results suggest that HDAC10 is a novel deacetylase that could have both nuclear and cytoplasmic functions.

EXPERIMENTAL PROCEDURES

Cloning—The amino acid sequence of HDAC6 catalytic domains (amino acids 138–792) was used in a protein-protein BLAST search of the NCBI database for novel class II HDACs. A putative deacetylase was found in a bacterial clone of chromosome 22q13.13–13.33 (GenBank accession number AL022328). Sequence-specific primers were used to generate a C-terminal FLAG-tagged HDAC10 by PCR from a K562 library (PCR primers: 5’ primer, 5’-CCCTTCTCAACTTTCCATCGAACGTTTCTTATGGCTCAAGTATCGCACCAAGCAGTTGAGGATGG-3’ and 5’-GGGCTCTTATCACTCAATCATT-1’), and inserted into the XhoI and EcoRI sites of pCMX-PL2. The FLAG tag was incorporated into the reverse primer. The Gal4-HDAC10-MLS construct was generated by PCR using the same 5’ primer and a 3’ primer including the SV40 NLS (5’-GAAGATCTAACATTTTCTTCTTGTGTTATCATCATTCAATCTTTTATGTC-3’). The HDAC10pCMX-PL2 H135A mutant was generated using the QuikChange site-directed mutagenesis kit (Stratagene).

Tissue Culture and Transfection—U2OS cells were maintained in Dulbecco’s modified Eagle’s medium supplemented with 10% fetal clone serum, penicillin/streptomycin, and 25 μg/ml aprotinin, 1 μg/ml aprotinin, 1 μg/ml leupeptin). Equal amounts of lysate (2 μg of protein) were precleared by incubating with protein A-agarose for 30 min at 4°C. Precleared lysates were immunoprecipitated as described above. HDAC assays were performed in a 200-μl volume containing immunoprecipitated HDACs, 10 μM Tris, pH 8, 10 μM NaCl, 1 mM EDTA, 0.5% Nonidet P-40) for 15 min at 4°C and then centrifuged by centrifugation at 14,000 × g for 10 min. 750 μg of total protein were immunoprecipitated with the anti-FLAG (M2) antibody (Sigma), 2 μl of rabbit anti-mouse IgG (Jackson ImmunoResearch), and 25 μl of protein A-agarose for 3–5 h. The proteins were separated by SDS-PAGE and subjected to Western blot analysis.

HDAC Assays—Cells were lysed in low stringency lysis buffer (50 mM Tris-Cl, 120 mM NaCl, 0.5 mM EDTA, 0.5% Nonidet P-40, 0.5 mM phenylmethylsulfonyl fluoride, 2 μg/ml aprotinin, 1 μg/ml leupeptin). Equal amounts of lysate (2 μg of protein) were precleared by incubating with protein A-agarose for 30 min at 4°C. Precleared lysates were immunoprecipitated as described above. HDAC assays were performed in a 200-μl volume containing immunoprecipitated HDACs, 10 μM Tris, pH 8, 10 μM NaCl, 10% glycerol, and 10,000 cpm [3H]H4 peptide. As determined by Western analysis of cell lysates, HDAC4, -6, -10, and the HDAC10 H135A mutant were expressed at similar levels (Fig. 2A). HDAC10 was observed at about 70 kDa, in agreement with the predicted molecular mass. As shown in Fig. 2B, HDAC10 exhibited deacetylase activity.
although HDAC6 activity was higher than both HDAC4 and -10, presumably because of its two independent catalytic domains. Importantly, HDAC10 enzymatic activity was sensitive to 400 nM TSA, a potent HDAC inhibitor (Fig. 2B). Furthermore, the HDAC10 H135A mutant had no activity, demonstrating that the second partial catalytic domain of HDAC10 is not a functional deacetylase domain (Fig. 2B).

Although all mammalian deacetylases are inhibited by TSA, a recent report has shown that HDAC6 is resistant to the HDAC inhibitor TPX-B (26). As shown in Fig. 2C, HDAC6 activity is indeed resistant to TPX-B. Furthermore, we have found that HDAC6 is also resistant to NaB. Because of the sequence similarity between HDAC6 and -10, we wanted to determine whether HDAC10 also exhibits this pharmacological property of HDAC6. As shown in Fig. 2C, HDAC1 activity was inhibited by all three inhibitors tested. In contrast, the
activity of HDAC10 was much more resistant to TPX-B and NaB. Thus, similar to HDAC6, HDAC10 also shows resistance to TPX-B and NaB.

At least two possibilities can explain this unique pharmacological property of HDAC6 and -10. First, their common N-terminal catalytic domain might specifically confer resistance to TPX-B and NaB. Alternatively, the presence of the second complete (in HDAC6) or incomplete (in HDAC10) catalytic domain renders this subfamily resistant to TPX-B and NaB. Because both of the catalytic domains of HDAC6 are functional (13), we assayed a single point mutant that inactivates the first catalytic domain of HDAC6, H216A, to distinguish between these two possibilities. As shown in Fig. 2D, the HDAC6 H216A mutant was completely resistant to these inhibitors, suggesting that the common N-terminal catalytic domain is not responsible for this resistance. To directly address the second hypothesis, we assayed an HDAC10 mutant that lacks the C-terminal catalytic domain. This HDAC10 C-terminal deletion mutant displayed similar sensitivity to TPX-B and NaB as to TSA, suggesting that the presence of two catalytic domains is necessary to confer this resistance (Fig. 2D). Taken together, these results further support the idea that HDAC6 and HDAC10 comprise a subclass within the class II deacetylases since their similar pharmacological properties are unlike any other known HDAC.

Nuclear and Cytoplasmic Localization of HDAC10—The varied subcellular localization of the class II deacetylases reflects their roles in regulating distinct cellular processes. To begin to identify the subcellular localization of HDAC10, a FLAG-tagged HDAC10 construct was transfected into U2OS cells, and the localization of HDAC10 was determined by immunostaining. In contrast to HDAC6, which exhibits an exclusively cytoplasmic staining pattern (Fig. 3C), HDAC10 localized both to the nucleus and the cytoplasm (Fig. 3A). The nuclear staining pattern appeared to exclude the nucleoli. In most cells, the nuclear staining was much more intense than the cytoplasmic signal. The cytoplasmic staining of HDAC10, however, is most likely not an artifact of overexpression, because the staining pattern remained the same despite decreasing amounts of transfected plasmid, and it was observed in every cell that we have examined (data not shown). Interestingly, despite the presence of three putative nuclear export signals, the cytoplasmic staining of HDAC10 remained constant, suggesting that the nuclear import machinery is not responsible for this localization.
mic localization of HDAC10 was not sensitive to leptomycin B treatment, an inhibitor of Crm1-mediated export, suggesting that the cytoplasmic localization of HDAC10 is not a consequence of nuclear export (Fig. 3B). The difference in HDAC6 and HDAC10 immunostaining patterns, therefore, suggests that these class II HDACs are involved in different cellular processes.

**Transcription Repression Mediated by HDAC10**—The nuclear localization of HDAC10 prompted us to ask if it has transcription repression activity. To ensure nuclear localization of HDAC10, an expression plasmid was generated that incorporated the SV40 NLS signal as a C-terminal fusion to FLAG-tagged Gal4-HDAC10. The effect of HDAC10 expression on a Gal4 binding site-driven luciferase reporter was then determined by co-transfection of this reporter with the Gal4-HDAC10-NLS expression plasmid. As shown in Fig. 4, similar to a known co-repressor HDAC4, Gal4-HDAC10-NLS repressed transcription in a dose-dependent manner, supporting the idea that HDAC10 can function as a transcriptional regulator. Unexpectedly, the Gal4 fusion of the deacetylase-inactive mutant, Gal4-H135A-NLS, also possessed repression activity, suggesting that HDAC10 contains an active transcription repression domain independent of its deacetylase activity.

**Coimmunoprecipitation of HDAC10**—As a nuclear protein, HDAC1 exists in several co-repressor complexes including the NuRD and Sin3 complexes (reviewed in Knoepfler and Eisenman (27)). To determine whether HDAC10 represses transcription by interacting with factors of either the NuRD complex or Sin3, a series of coimmunoprecipitations was probed with antibodies to three factors unique to the NuRD complex, Mi-2 (CHD4), MTA-2 (PID) as well as mSin3A. As expected, all of these factors appeared to associate with FLAG-tagged HDAC1 (Fig. 5B, lane 3). Interestingly, despite its likely role in transcription repression, none of these proteins were apparent in the HDAC10 immunoprecipitations, indicating that HDAC10 is in a biochemically distinct complex from HDAC1 in vivo.

**DISCUSSION**

With the identification of HDAC10, there are four class I deacetylases and now six class II HDACs in addition to at least one more HDAC, HDAC11, which will likely represent a class III deacetylase. Sequence analysis shows that HDAC10 is most closely related to HDAC6, a unique member of the class II HDACs. Phylogenetic tree analysis indicates that HDAC10 and HDAC6 form a subclass within the class II enzymes since they are grouped in a branch separate from the other known class II HDACs, HDAC4, -5, -7, and -9. Indeed, unlike any other class II members, these two HDACs share a similar domain structure in that they contain two spaced catalytic domains, with HDAC10 having only a partial second domain.

Although all the known mammalian deacetylases are sensitive to TSA, they exhibit different sensitivity to TPX-B and NaB. Furumai et al. (26) have previously reported that HDAC6

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3 A. Ito, K. Wei, and T.-P. Yao, unpublished observation.
is resistant to the inhibitor TPX-B. In this report, we demonstrate for the first time that HDAC6 is also resistant to NaB. Our analysis of HDAC10 shows that it is also highly resistant to these two inhibitors (Fig. 2), in contrast to all other deacetylases, which are sensitive to TPX-B and NaB. This unique pharmacological property of both HDAC6 and HDAC10, therefore, further supports the notion that they comprise a subclass of the class II deacetylases.

What is the basis for this unique pharmacological property? Because TPX-B is an irreversible inhibitor that binds and alkylates the enzyme, Furumai et al. (26) suggest that HDAC6 is resistant to TPX-B presumably because TPX-B fails to alkylate it (26). However, because HDAC6 is also resistant to NaB, a short chain fatty acid that does not inhibit HDACs by alkylation, this explanation is less plausible. Our observation that the HDAC6 mutant with an inactive N-terminal catalytic domain remains resistant to these inhibitors (Fig. 2D) demonstrates that it is unlikely that the intrinsic structure of the N-terminal catalytic domains of HDAC6 or -10 is responsible for the TPX-B or NaB resistance. Rather, the presence of a second catalytic domain, regardless of whether it is functional, is the basis for this resistance. Indeed, the deletion of the second catalytic domain of HDAC10 causes sensitivity to TPX-B and NaB (Fig. 2D). We suspect that the two catalytic domains likely interact to form a pocket that would allow tight binding to TSA but not TPX-B or NaB. If true, this hypothesis would argue that although the second catalytic domain may not be directly involved in catalysis, as in the case of HDAC10, it would contribute to the overall structure and, consequently, to the function of HDAC10 and HDAC6. With the structure of the single catalytic domain of histone deacetylase-like protein already solved (25), a structure of the HDAC6 or HDAC10 catalytic domains should provide detailed and useful information to verify this prediction.

Intriguingly, in contrast to the closely related HDAC4 and HDAC5, which work in concert to regulate myogenesis, the
idea that the highly homologous HDAC6 and HDAC10 function in similar biological processes because HDAC6 does not exhibit deacetylase activity toward an HDAC6 substrate. Their subcellular localization pattern is also different (Fig. 3). Furthermore, these two deacetylases do not appear to associate in vivo (data not shown). Thus, despite similarity in sequence and pharmacological property, HDAC6 and HDAC10 will likely have different functions in vivo, further expanding the functional repertoires of the HDAC family.

The diverse functions of the class II HDACs are reflected by their subcellular localization since they all exhibit either induced or constitutive cytoplasmic localization. Consistent with this observation, ectopically expressed HDAC10 is found both in the nucleus and in the cytoplasm within the same cell, in contrast to the shuttling HDAC4 and HDAC5 or, exclusively, cytoplasmic HDAC6. Furthermore, HDAC10 cytoplasmic localization is not affected by leptomycin B. Because HDAC10 has three putative nuclear export signals, these results imply that either these are nonfunctional signals, or once exported, HDAC10 is a stable protein and is sequestered in the cytoplasm. Additionally, this subcellular localization suggests that HDAC10 may play several roles, possibly participating in transcription repression as well as in an unidentified role in the cytoplasm.

Indeed, when tethered to DNA, HDAC10 has transcription repression activity (Fig. 4). Interestingly, in contrast to HDAC1, which requires catalytic activity for its transcription repression activity (28), HDAC10-mediated repression is independent of its catalytic activity (Fig. 4). This deacetylase activity-independent repression has also been observed for HDAC4, which contains several repressor domains that are independent of its catalytic domain. It is formally possible that the HDAC10-mediated repression is due to an associated HDAC because deacetylases have been shown to associate with each other (13). This scenario is unlikely, however, because no apparent deacetylase activity corepressorniprecipitated with the HDAC10 catalytically inactive mutant (Fig. 2B). Consistent with this idea, TSA has a minimal effect on HDAC10-mediated repression (data not shown). Regardless, the HDAC10-mediated repression activity is not likely to directly involve either the mSin3A or NuRD complexes (Fig. 5B). Importantly, the absence of a clearly defined nuclear localization signal implies that the nuclear localization of HDAC10 is most likely mediated by interaction with another protein, possibly a transcription factor. Identification of potential partners of HDAC10 in both the nucleus and cytoplasm will be essential to elucidating its function.

Previous studies show that HDAC inhibitors have anti-proliferative activity in tissue culture cells and in mice (29–32). However, it remains unclear whether all the HDACs or only selected members of the deacetylases are targets of the anti-proliferative activity. This report along with that by Furumai et al. (26) provides evidence that it is now possible to develop molecules that selectively inhibit only a subclass of the HDAC family. These molecules not only will be critical tools to distinguish the unique functions of a given HDAC but also might have the potential to function as highly specific cancer therapeutic drugs with much reduced toxicity.

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