Comparative Studies of Human Recombinant 74- and 54-kDa L-Histidine Decarboxylases

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We have expressed and characterized human recombinant 74-kDa (rHDC74) and 54-kDa (rHDC54) L-histidine decarboxylases (HDCs) in SF9 cells. By immunoblot analysis, rHDC74 and rHDC54 were shown to be localized predominantly in the particulate and soluble fractions, respectively. rHDC74 exhibited histamine-synthesizing activity equivalent to that of rHDC54. The existence of 74- and 54-kDa HDCs was also confirmed in the particulate and supernatant fractions of the cell lysate, respectively, from the human basophilic leukemia cell line KU-812-F. The ratio of HDC activity to immunoreactivity was similar for the two forms of the enzyme. The specific activity of purified rHDC54 (1.12 μmol/mg/min) was comparable to those of HDCs from other mammalian tissues or cells. The purified rHDC54 was eluted as a monomer form from a Superdex-200 column; the molecular mass of the enzyme was approximately 54 kDa on SDS-polyacrylamide gel electrophoresis without 2-mercaptoethanol. The HDC activity of rHDC54 significantly decreased on dialysis against buffer without pyridoxal 5'-phosphate; addition of pyridoxal 5'-phosphate to the dialysate readily increased the enzyme activity to the original activity. Taken together, these results suggest that human HDC functions as both 74- and 54-kDa forms having equivalent HDC activity, which are localized in the particulate and soluble fractions, respectively, and that the latter form exhibits its activity as a monomer form.

Histamine plays a pivotal role in a wide variety of reactions, such as inflammation, gastric acid secretion, neurotransmission, and cell growth (Kahlson and Rosengren, 1968; Beaven, 1978; Woolley, 1993). L-Histidine decarboxylase (HDC, EC 4.1.1.22) is the only enzyme that catalyzes the formation of a biogenic amine from its precursor, L-histidine (Schayer, 1966). HDCs fall into two classes: those that use pyridoxal 5'-phosphate (PLP) as a cofactor and those that have a pyruvoyl coenzyme, is quite different. Many investigators demonstrated that HDC regulates the level of histamine in tissues or cells, and that modulation of HDC activity under various conditions markedly affects the level of the amine (Schneider et al., 1987; Mamune-Sato et al., 1990; Yatsunami et al., 1993). It is thus of great importance to clarify the mechanism underlying regulation of the HDC activity.

Recently, we reported the structure of the human HDC gene, and proposed the possibility that HDC activity is transcriptionally regulated (Yatsunami and Ohtsu et al., 1994). Accumulated evidence suggests that HDC activity is also regulated at the translational level. HDC cDNAs have been isolated, and characterized, from human basophilic leukemia cells (Yamauchi et al., 1990), human erythroleukemia cells (Zahnow et al., 1991), fetal rat liver (Joseph et al., 1990), and mouse mastocytoma P-815 cells (Yamamoto et al., 1990). The molecular masses of HDC enzymes (74 kDa), calculated from amino acid sequences derived from the respective cDNAs, are larger than those of the purified HDC subunits (53–54 kDa) by about 20–21 kDa. It is, therefore, conceivable that primary translated products are processed after translation to yield mature enzymes and that such post-translational processing of HDCs changes the activity or nature of the enzymes.

Until recently, however, it had not been clarified whether or not a primary translated 74-kDa HDC has activity, because it has never been isolated from any tissue or cells. Most recently, mouse recombinant 74-kDa HDC exhibiting enzyme activity was expressed in SF9 cells (Yamamoto et al., 1993). However, it was not clearly shown whether or not the enzyme activity was due to the recombinant 74-kDa HDC itself, because the mouse recombinant 74-kDa HDC was unstable and its purification was unsuccessful. In addition, it is totally unknown whether 74-kDa HDCs from other mammalian sources have a molecular nature similar to that of mouse 74-kDa HDC. Although the 54-kDa HDCs purified so far characteristically resemble each other, the nature of 74-kDa HDCs might differ among man, mouse, and rat in some respects, because their C-terminal regions are not so homologous (Mamune-Sato et al., 1992).

To clarify whether or not a 74-kDa HDC has HDC activity, and whether and how 74- and 54-kDa HDCs take part in histamine synthesis, we have expressed cDNAs for human 74- and 54-kDa HDCs using a baculovirus system. To our surprise, the human 74- and 54-kDa HDCs exhibited equivalent enzyme activity, and the latter existed as a monomer form.

MATERIALS AND METHODS

Construction of cDNAs for 74- and 54-kDa HDCs—A human HDC cDNA, pTN-2 (Yamauchi et al., 1990; Mamune-Sato et al., 1992), in the Okayama-Berg cDNA cloning/expression vector (Okayama and Berg, 1982) was kindly provided by Dr. Kohei Yamauchi (Tohoku University,
**FIG. 1. Structure and construction of recombinant transfer vectors pVLHDC74 and pVLHDC54.** Human HDC cDNA in the Okayama-Berg vector was digested with PstI and BamHI. A long 5'-untranslated region or the 3'-end corresponding to the C-terminal 20 kDa of HDC was deleted using the polymerase chain reaction protocol, as described under "Materials and Methods." The resulting HDC fragments (pHDC74 and pHDC54) were subcloned downstream of the polyhedrin promoter in pVL1392. The solid bars indicate the untranslated 5'- and 3'-ends of human HDC cDNA. A bold arrow indicates the polyhedrin promoter.

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**Studies of Human Recombinant L-Histidine Decarboxylases**

J. apan. Recombinant transfer vectors for 74- and 54-kDa HDcs, pVLHDC74 and pVLHDC54, respectively, were constructed with pTN-2 and the transfer vector, pVL1392, as shown in Fig. 1. Briefly, human recombinant 74-kDa HDC cDNA (pHDC74) was obtained by deleting a long 5'-untranslated sequence in pTN-2 using a specific primer that contains an Xba I site at its 5'-end. The sense oligonucleotide used for the construction of pHDC74 was 5'GGTCTAGAAGTCTAGTGA-3', whereas human recombinant 54-kDa HDC cDNA (pHDC54) was constructed by deleting the 3'-end of pHDC74 using a specific primer, which contains a stop codon just upstream of the Gln-477 in the human HDC sequence, based on the results of Okamura and colleagues (1990), except that 80 mM NaCl was used to elute HDC from the phenyl-Sepharose HP column (mouse HDC was eluted from the column by 5 mM NaCl). The purified enzyme (34 kDa) was stored at -80°C.

**RESULTS**

**Subcellular Distribution of Recombinant 74- and 54-kDa HDcs—**Human recombinant 74- and 54-kDa HDcs, rHDC74 and rHDC54, respectively, were constructed in a baculovirus vector. Gln-477 was given as the C-terminal amino acid of rHDC54, because the C-terminal residue of purified fetal rat liver HDC was reported to be Gln-479, which corresponds to Gln-477 in the human HDC sequence, based on the results of Ohmori et al. (1990), except that 80 mM NaCl was used to elute HDC from the phenyl-Sepharose HP column (mouse HDC was eluted from the column by 5 mM NaCl). The purified enzyme (34 kDa) was stored at -80°C.

**SDS-Polyacrylamide Gel Electrophoresis and Molecular Mass Estimation—**Polyacrylamide gel electrophoresis was performed on slabs (4-20% gradient gels) according to Laemmli (1970). Proteins were stained with a silver stain kit (Daiichi Pure Chemicals Co., Ltd, Tokyo, Japan). A molecular mass standard, an electrophoresis calibration kit (Daiichi Pure Chemicals Co., Ltd, Tokyo, Japan) was used.

**Gel filtration on a Superdex-200 FPLC column was performed using a gel filtration calibration kit (Pharmacia) as standards: bovine serum albumin (66 kDa), ovalbumin (43 kDa), chymotrypsinogen A from bovine pancreas (25 kDa), and ribonuclease A from bovine pancreas (13.7 kDa).**

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**RESULTS**
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Mammalian HDCs have been characterized and purified from various types of cells and tissues (Ohmori et al., 1990; Taguchi et al., 1984; Martin and Bishop, 1986; Grzanna, 1984). These studies, however, focused only on a soluble 53–54-kDa HDC, although the primary translated HDCs are 74 kDa in size. The purpose of this work was to clarify whether and how 74- and 54-kDa HDCs take part in histamine synthesis. Resolution of this question will provide a clue as to the mystery that particulate and the supernatant fractions. As shown, a strong band with a molecular mass of 54 kDa was observed for the supernatant fraction, whereas for the particulate fraction, a weak but a distinct band corresponding to a molecular mass of 74 kDa was detected. Quantitative analysis by densitometry demonstrates that the ratio of HDC activity to immunoreactivity is similar for the two forms of the enzyme (Fig. 3C).

Characterization of the Purified Recombinant 54-kDa HDC—Fig. 4 shows a typical elution profile on Superdex 200 column chromatography. HDC activity was eluted in fractions where a globular protein with a molecular mass of 54 kDa would be. When the supernatant fraction from cultured KU-812-F cells was applied on a Superdex-200 gel filtration FPLC column, HDC activity was eluted at the same position as the purified rHDC54 (data not shown).

Fractions from the Superdex 200 column were subjected to SDS-polyacrylamide gel electrophoresis in the absence of 2-mercaptoethanol and the gel was silver-stained. As shown in Fig. 4 (inset), a single intense band corresponding to a molecular mass of about 54 kDa was exclusively obtained for the active fractions. Immunoblot analysis showed that the anti-HDC antibody only reacted with the single 54-kDa subunit protein (data not shown).

**DISCUSSION**

An anti-HDC peptide antibody, which recognizes only denatured HDC, was raised and used for the detection of human recombinant 74- and 54-kDa HDCs. The peptide sequence, Ser-218 to Lys-232, in the human HDC sequence is highly hydrophilic as well as antigenic and was selected to completely match those of the mouse (Ser-225 to Lys-239) (Yamamoto et al., 1990) and rat (Ser-221 to Lys-235) (Joseph et al., 1990) particulate and the supernatant fractions. As shown, a strong band with a molecular mass of 54 kDa was observed for the supernatant fraction, whereas for the particulate fraction, a weak but a distinct band corresponding to a molecular mass of 74 kDa was detected. Quantitative analysis by densitometry demonstrates that the ratio of HDC activity to immunoreactivity is similar for the two forms of the enzyme (Fig. 3C).

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HDCs. Immunoblot analysis using the antibody demonstrated that 87% of the total human rHDC74 expressed in Sf9 cells existed as an insoluble form with histamine-synthesizing activity. Although quantitative determination by immunoblot analysis has limitations, equal amounts of HDC activity of the two species apparently gave bands of equal intensity of rHDC74 and rHDC54, suggesting that the specific activities of the two HDCs are equivalent. It is unlikely that rHDC74 was cleaved to yield rHDC54 during the enzyme assay, and the latter exhibited HDC activity because no processing from rHDC74 to rHDC54 was observed during incubation of the former with histidine for 3.5 h at 37 °C (data not shown). When 74- and 54-kDa human HDC CDNsAs were subjected to in vitro transcription and translation, synthesis of the respective HDC proteins was observed. In addition, both the in vitro translated proteins exhibited HDC activity (data not shown). Overall, we concluded that human rHDC74 possesses as much histamine-synthesizing ability as rHDC54.

Fig. 3 suggests that the insoluble HDC is not an artifact in the present expression study, but that a fairly large amount of an insoluble 74-kDa HDC exists and functions. Approximately 29% of the total HDC activity was found to exist in the insoluble fraction from human leukemia cells, KU-812-F, which is the source of human HDC DNA. Possible contamination by soluble HDC of these fractions was ruled out because less than 3% of the lactate dehydrogenase activity was detected in either fraction. Immunoblot analysis using an extract of KU-812-F cells supports the existence of 74-kDa HDC in the cells, and the same ratio of activity to immunoreactivity for the 74-kDa and 54-kDa forms. Although the figure contains multiple bands corresponding to higher and lower molecular masses, there is no contradiction between the intensities of the two immunoreactive bands and the respective HDC activities. We have no evidence ruling out the possibility that these bands were derived from aggregation or processing of the respective enzymes, but we tentatively conclude that these bands appeared nonspecifically for the following reasons. First, the activity to immunoreactivity ratio of the two forms is not contradictory. Second, an anti-peptide antibody often gives nonspecific bands when samples are very crude and the amount of samples, applied to a gel, are very large, which was the case in this experiment.

If, as we have suggested, an insoluble 74-kDa HDC does contribute to histamine synthesis in physiological tissues and cells, how might soluble 54 and particulate 74-kDa HDCs participate in it? This is a difficult question because the molecular nature of the particulate HDC is totally unknown, and we could...
not determine its cellular location by means of immunohistoch-
chemistry using the anti-HDC peptide antibody we raised. We
assume that a particular 74-kDa HDC binds to cellular
membranes and promptly responds to signals from outside the cell.
In contrast to stored histamine, newly formed histamine is
believed to act soon after its synthesis at the site or in the cell
where it is formed (Kahlson and Rosengren, 1968). The newly
formed histamine, sometimes called induced histamine or nas-
cent histamine, plays an important role in cell growth (Kahlson
and Rosengren, 1968; Brandes and LaBella, 1993), platelet
aggregation (Saxena et al., 1989), and gastric acid secretion
(Kahlson and Rosengren, 1968; Glavin and Brandes, 1988). It
is possible that a particulate 74-kDa HDC synthesizes newly
formed histamine, whereas a soluble 54-kDa HDC produces
stored histamine. In any case, the hydrophobic C-terminal
20-kDa regions of mammalian 74-kDa HDCs probably partici-
brate in anchoring of the proteins to cellular membranes. These
assumption requires further investigation.

The HDC activity of rHDC54 significantly decreased on di-
alysis for 16 h against buffer without PLP (Table I). The HDC
activity was not completely abolished on the dialysis, probably
because 16 h was not long enough to completely remove PLP
from the sample. The addition of PLP to the dialysate readily
increased the enzyme activity to the original level. These re-
results strongly suggest that the human HDC is PLP-dependent.

Human rHDC54 was purified by the same procedure as that
used for the purification of HDC from mouse mastocytoma cells
(Ohmori et al., 1990). In addition, the specific activity of the
purified human rHDC54 (1.12 μmol/mg/min) was proved to be
equivalent to that of the mouse and rat HDCs (800 and 260
nmol/mg/min, respectively), suggesting that human rHDC54 is
functionally similar to native HDCs from mammalian tissues
or cells. This is reasonable because the homology of the 54-kDa
HDC molecules is considerably high among three mammals
(Mamune-Sato et al., 1992). However, the activity of 74-kDa
HDCs might differ among them because the homology in their
C-terminal 20-kDa is not so striking. Assuming that the C-
terminale regions of HDC molecules affect their folding and
thereby influence the respective HDC activities, the activity of
74-kDa HDC in mouse or rat might not be similar to that of the
respective 54-kDa HDC.

Purified human rHDC54 was shown to be eluted as a mon-
omer form with an apparent molecular mass of 54 kDa on gel
filtration on a Superdex-200 column. Only a single band of
54-kDa HDC was detected on SDS-polyacrylamide gel electro-
phoresis of the eluted protein, even in the absence of 2-mercap-
tetoethanol. Until recently, HDC has been purified to homogene-
ity only from rat liver (Taguchi et al., 1984) and mouse
mastocytoma cells (Ohmori et al., 1990). In both cases, active
HDCs were reported to exist as a dimer form. A monomer and
active HDC was neither detected nor isolated. Human HDC is
the first example of an HDC existing as a monomer form. It is
unlikely that the active monomer HDC, found in the present
study, was an artifact produced during the process of purifica-
tion of the recombinant HDC, because HDC activity derived
from cultured KU-812-F cells was also eluted at the same
position as the purified rHDC54 was on a Superdex-200 gel
filtration column. The specific activity of the purified human
monomer, rHDC54, was equivalent to that of the dimer HDCs
from mouse and rat. However, we cannot rule out the possibil-
ity that the C terminus of 54-kDa human HDC is not identical
to those of other HDCs, which could contribute to their dimer-
ization. Further studies are needed to clarify this point.

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REFERENCES

Ausubel, F. M., Brent, R., Kingston, R. E., Moore, D. D., Seidman, J. G., Smith,
J. A., and Struhl, K. (1987) Current Protocols in Molecular Biology, pp.
16.9.1–16.10, Greene Publishing Associates/Wiley-Interscience, New York
Beaven, M. A. (1978) in Monographs in Allergy (Dukor, P., Kallos, P., Trunz,
Z., Waksman, B. H., and de Weck, A. L., eds) Vol. 13, pp. 1–113, Thusr S. Karger AG,
Basel, Switzerland

Brandes, L. J., and LaBella, F. S. (1993) Adv. Biosci. 89, 31–42

Glavin, G. B., and Brandes, L. J. (1988) Can. J. Physiol. Pharmacol. 66, 1139–1143

Grzanna, R. (1984) Neurochem. Res. 9, 1053–1059

Hopp, T. P., and Woods, K. R. (1988) Proc. Natl. Acad. Sci. U. S. A. 87, 3824–3828

Joseph, D. R., Sullivan, P. M., Wang, Y.-M., Kazak, C., Federnmacher, D. A.,
Behrendsen, M. E., and Zahn, C. A. (1990) Proc. Natl. Acad. Sci. U. S. A. 87, 733–737

Kahlson, G., and Rosengren, E. (1968) Physiol. Rev. 48, 155–196

Laennec, U. K. (1970) Nature 237, 680–685

Mamune-Sato, R., Tanno, Y., Maeyama, K., Miura, Y., Takishima, T., Kishi, K.,
Fukuda, T., and Watanabe, T. (1990) Biochem. Pharmacol 40, 1125–1129

Mamune-Sato, R., Yamauchi, K., Tanno, Y., Ohkawara, Y., Ohtsu, H., Katayose,
D., Maeyama, K., Watanabe, T., Shibahara, S., and Takishima, T. (1992) Eur. J.
Biochem. 209, 533–539

Martin, S. A. M., and Bishop, J. O. (1986) Biochem. J. 234, 349–354

Merrifield, R. B. (1963) J. Am. Chem. Soc. 85, 2149–2154

Mitsuji, H. (1992) Osaka Univ. Igaku Zasshi (J. Japan) 44, 137–146

Ohmori, E., Fukui, T., Imanishi, N., Yatsunami, K., and Ichikawa, A. (1990)
J. Biochem. (Tokyo) 107, 834–839

Okayama, H., and Berg, P. (1982) Mol. Cell. Bid. 2, 161–170

Posnett, D. N., McGrath, H., and Tam, J. P. (1988) J. Biol. Chem. 263, 1719–1725

Raccii, P. A., and Sniids, E. E. (1984) Annu. Rev. Biochem. 53, 357–387

Sandanger, E., Hals, T. I., and Christen, P. (1994) Eur. J. Biochem. 221, 997–1002

Saxena, S. P., Brandes, L. J., Beeker, A. B., Simon, K. J., LaBella, F. S., and
Gerrard, J. M. (1989) Science 243, 1596–1599

Schayer, R. W. (1966) in Handbook of Experimental Pharmacology (Rocha e Silva,
M., ed.) Vol. 18, pp. 688–725, Springer, Berlin, Heidelberg, New York

Schneider, E., Pollard, H., Lepault, F., Guy-Grand, D., Minkowski, M., and Dy, M.
(1987) J. Immunol. 139, 3710–3717

Shore, P. A., Burkhalter, A., and Cohn, V. H., Jr. (1959) J. Pharmacol. Exp. Ther.
127, 182–186

Taguchi, Y., Watanabe, T., Kubota, H., Hayashi, H., and Wada, H. (1984) J. Biol.
Chem. 259, 5214–5221

Welling, G. W. (1985) FEBS Lett. 188, 215–218

Woolley, D. E. (1993) Adv. Biosci. 89, 1–29

Yamamoto, J., Yatsuuru, K., Ohmori, E., Sugimoto, Y., Fukui, T., Katayama, T.,
and Ichikawa, A. (1990) Biochem. Pharmacol. 325, 431–440

Yamachi, K., Sato, R., Tanno, Y., Ohkawara, Y., Maeyama, K., Watanabe, T.,
Satoh, K., Yoshizawa, M., Shibahara, S., and Takishima, T. (1990) Nucleic
Acids Res. 18, 5891

Yatsunami, K., Ohgoh, M., Yamamoto, J., Nakagawa, S., and Ichikawa, A. (1993)
Adv. Biosci. 89, 157–176

Yatsunami, K., Ohtsu, H., Tschikatka, M., Higuchi, T., Ishibashi, K., Shida, A.,
Shima, Y., Nakagawa, S., Ohmori, K., Yamamoto, M., Hayashi, N.,
Watanabe, T., and Ichikawa, A. (1994) J. Biol. Chem. 269, 1554–1559

Zahn, C. A., Yi, H.-F., McBride, O., and Joseph, D. R. (1991) DNA Seq. 1, 395–400.
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