Selenocysteine lyase (SCL) (EC 4.4.1.16) is a pyridoxal 5′-phosphate-dependent enzyme that specifically catalyzes the decomposition of L-selenocysteine to L-alanine and elemental selenium. The enzyme was proposed to function as a selenium delivery protein to selenophosphate synthetase in selenoprotein biosynthesis (Lacourciere, G. M., and Stadtman, T. C. (1998) J. Biol. Chem. 273, 30921–30926). We purified SCL from pig liver and determined its partial amino acid sequences. Mouse cDNA clones encoding peptides resembling pig SCL were found in the expressed sequence tag database, and cDNA clones encoding peptides resembling pig SCL. We also determined the sequence of the N-terminal region of putative human SCL. These enzymes were shown to be distantly related in primary structure to NifS, which catalyzes the desulfurization of L-cysteine to provide sulfur for iron-sulfur clusters. The recombinant mSCL overproduced in Escherichia coli was a homodimer with the subunit $M_r$ of 47,000. The enzyme was pyridoxal phosphate-dependent and highly specific to L-selenocysteine (the $K_{cat}/K_m$ value for L-selenocysteine was about 4,200 times higher than that for L-cysteine). Reverse transcriptase-polymerase chain reaction and Western blot analyses revealed that mSCL is cytosolic and predominantly exists in the liver, kidney, and testis, where mouse selenophosphate synthetase is also abundant, supporting the view that mSCL functions in cooperation with selenophosphate synthetase in selenoprotein synthesis. This is the first report of the primary structure of mammalian SCL.

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The nucleotide sequence(s) reported in this paper has been submitted to the GenBank™/EBI Data Bank with accession number(s) AF175407 (mouse Scly) and AF175767 (a sequence of cDNA encoding putative human SCL).

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The abbreviations used are: SPS, selenophosphate synthetase; bp, base pair(s); EST, expressed sequence tag; kbp, kilobase pair(s); PAGE, polyacrylamide gel electrophoresis; RT-PCR, reverse transcriptase-polymerase chain reaction; PLP, pyridoxal 5′-phosphate; SCL, selenocysteine lyase; Tricine, N-(2-hydroxy-1,1-bis(hydroxy-methyl)ethyl)glycine.

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mined the sequence of the N-terminal region of putative human SCL. The primary structures of these enzymes revealed that mammalian SCLs are distantly related to NiFs proteins and have characteristic sequences that are not found in other NiF-like proteins hitherto known. This is the first report on the primary structure of mammalian SCL.

**EXPERIMENTAL PROCEDURES**

**Materials—** Selenocysteine was synthesized as described previously (13). Oligonucleotides were provided by Espeol Oligo Service (Tuskuba, Japan). Restriction and DNA modification enzymes were purchased from New England Biolabs (Beverly, MA) and Takara Shuzo (Kyoto, Japan). All chemicals were of analytical grade.

**Purification of Pig SCL (pSCL)—** All steps were carried out at 4 °C unless otherwise stated. A potassium phosphate buffer (KPB) (pH 7.4) containing 20 μM PLP and 0.01% 2-mercaptoethanol was used as the standard buffer. A pig liver (1.5 kg) was minced with an ice-cold meat mincer and homogenized in a Waring Blender in 7.5 liters of a 50 mM standard buffer. The homogenate was centrifuged, and the supernatant was passed through a nylon mesh. The crude extract was fractionated with ammonium sulfate (25–45% saturation) and dialyzed with a 10 mM standard buffer. The enzyme solution was applied to a DEAE-Toyopearl column (16 by 1 cm) equilibrated with a 10 mM standard buffer and eluted with a 10-liter linear gradient (0–0.2 M) of KCl in the same buffer. The fractions containing the enzyme were concentrated by ammonium sulfate precipitation (50% saturation). The enzyme was applied to a Phenyl-Toyopearl column (5 by 15 cm) equilibrated with a 10 mM standard buffer containing 0.6 M ammonium sulfate and eluted with 1.5 liters of the same buffer. The enzyme fractions were concentrated by ultrafiltration with a UP-20 membrane (Advantec, Naha, Japan). The enzyme was dialyzed against a 10 mM standard buffer. The enzyme was loaded onto a hydroxyapatite column (2.5 by 14 cm) equilibrated with a 20 mM standard buffer and eluted with a 20 mM standard buffer containing 0.7 M ammonium sulfate, applied to a second Phenyl-Toyopearl column (5 by 14 cm) equilibrated with the same buffer, and eluted with 10 mM standard buffer containing 0.6 M ammonium sulfate. The active fractions were pooled, concentrated by ultrafiltration as above, and dialyzed against 20 mM standard buffer. The enzyme was washed twice with 150 mM standard buffer. The enzyme fractions were collected and dialyzed against 10 mM standard buffer. The enzyme solution was applied to a DEAE-Toyopearl column (16 by 30 cm) and fractionated with the 20 mM standard buffer. The enzyme was poured on a hydroxyapatite column (2.5 by 10 cm) equilibrated with 20 mM standard buffer and eluted with a 20 mM standard buffer. The enzyme was subjected to sequence analysis with an automated protein sequencer PPSQ-10 (Shimadzu, Kyoto, Japan). In order to determine the internal sequences of pSCL, in-gel digestion was performed with a 4% polyacrylamide gel, stained with 0.1% Ponceau S, excised as horizontal lines and subjected to sequence analysis.

**Isolation of cDNA Encoding mSCL—** BLAST analysis of the EST data bases using the pSCL sequences resulted in the identification of highly homologous mouse and human cDNA sequences (Fig. 1). Mp1 (5'-CGGAAAGTGGCTCCTCTTCAA-3') and Mp2 (5'-GTGAAACTGATCATCCTCTAGG-3') were used to amplify a 340-bp fragment of AA107712, and Mp3 (5'-CAGGATCGGTGCTCTGTATG-3') and Mp4 (5'-GGCTTGTTCAAAATGGATGTTCTCT-3') were used to amplify a 250-bp fragment of MUS94C09 (Fig. 2). The PCR products were gel-purified, labeled with digoxigenin, and used as probes to obtain a mouse cDNA clone from a mouse liver ZAP cDNA library constructed with ZAP-cDNA Synthesis Kit (Stratagene, La Jolla, CA). Immunodetection was performed with Anti-DIG, Fab fragment AP Conjugate (Roche Diagnostics, Basel, Switzerland), nitro blue tetrazolium, and 5-bromo-4-chloro-3-indolyolphosphate-p-toluidine salt. Positive clones were isolated, and their cDNA inserts were excised in the form of pBluescript plasmid and analyzed by sequencing. Sequence analysis of an isolated clone, Scly1, revealed that it is comprised of 2,137 bp including a poly(A)尾 tail in the 3' end region. However, the clone lacked 4 bp, TAGG, at position 165, which corresponds to position 196–199 of the EST clone AA107712, and no in-frame ATG codon was found near the 5' end of this sequence (Fig. 2). To determine the sequence of the 5' region of the transcript, 5'-rapid amplification of cDNA ends (14) and CapFinder (CLONTECH, Palo Alto, CA) techniques were employed. The 4 bp, TAGG, were found in the PCR products from the fresh preparation of a transcript. A possible initiation ATG codon was found in a position adjacent to the 5' end of Scly1 (Fig. 2). The initiation codon, ATG, and four bases, TAGG, were introduced into the incomplete cDNA clone Scly1 (Fig. 2) by two-step PCR. Primers Mp5 (5'-GGGAAATTCATGGACGACGGCGCCGAAATG-3') and Mp6 (5'-CTCGGTGAATTCAGGCTGATGCGAATG-3') were used.

**Fig. 1.** Comparison of the amino acid sequences of pSCL fragments with those encoded by the EST clones, AA107712, MUS94C09, N56305, and R13893. The nucleotide sequences of the EST clones were found in the EST database by homology search using the BLAST program with the pSCL sequences.

**Fig. 2.** Schematic drawing of cDNA encoding mSCL. Differences in the coding region are shown. Numbers represent the positions in the nucleotide sequence of each cDNA. Scly1 was isolated by screening of the mouse cDNA library. The ATG codon and TAGG were introduced into Scly1 to construct Scly2. Scly encodes the functional enzyme. The horizontal lines indicate the positions of the sequences of the mouse EST clones, AA107712 and MUS94C09.
The entire coding sequence of mSCL cDNA (Fig. 2) was used for amplification of the DNA fragment encoding the N-terminal part of mSCL. Another set of primers, Mp7 (5′-AGGCGAAGCCACCTGGAGCTGGCC-3′) and Mp8 (5′-CCCCAAGCCTTGCCGACCCTGACGAGG-3′), was used to amplify the DNA fragment coding for the C-terminal fragment of mSCL. The initiation codon to be introduced is double underlined, and the underlined nucleotides indicate the 4 bases to be inserted. The restriction enzyme sites and N-terminal part of mSCL. Another set of primers, Mp7 (5′-AGGCGAAGCCACCTGGAGCTGGCC-3′) and Mp8 (5′-CCCCAAGCCTTGCCGACCCTGACGAGG-3′), was used to amplify the DNA fragment coding for the C-terminal fragment of mSCL. The initiation codon to be introduced is double underlined, and the underlined nucleotides indicate the 4 bases to be inserted. The restriction enzyme sites

**FIG. 3. Nucleotide and deduced amino acid sequences of mSCL cDNA.** Two possible overlapping polyadenylation signals are underlined. Numbers of nucleotides and amino acid residues are shown.

GGCGG-3′) and Mp6 (5′-CACCTGGTTGCTCTACGTGACATAGGC-3′) were used for amplification of the DNA fragment encoding the N-terminal part of mSCL. Another set of primers, Mp7 (5′-CAGGTCGQAAGGCGAAGCCACCTGGAGCTGGCC-3′) and Mp8 (5′-CCCCAAGCCTTGCCGACCCTGACGAGG-3′), was used to amplify the DNA fragment coding for the C-terminal fragment of mSCL. The initiation codon to be introduced is double underlined, and the underlined nucleotides indicate the 4 bases to be inserted. The restriction enzyme sites

**TABLE I**

| Step | Total protein | Activity<sup>a</sup> | Specific activity | Purity | Yield |
|------|--------------|---------------------|------------------|--------|-------|
|      | mg | units | units/mg | fold | % |
| Crude extract | 440 | 2600 | 5.9 | 1 | 100 |
| Ammonium sulfate precipitation | 550 | 3600 | 6.5 | 1.1 | 130 |
| Butyl-Toyopearl | 120 | 1600 | 13 | 2.2 | 62 |
| Q-Sepharose | 56 | 1600 | 29 | 4.9 | 62 |

<sup>a</sup> Measured with 5 mM L-selenocysteine.

**FIG. 4. Purification of recombinant mSCL.** SDS-PAGE was performed with the different preparations obtained during purification of mSCL. Lane 1, marker proteins; the molecular masses are shown in kDa; lane 2, crude extract; lane 3, ammonium sulfate precipitation; lane 4, Butyl-Toyopearl; lane 5, Q-Sepharose. The proteins were stained with Coomassie Brilliant Blue R-250.

**TABLE II**

| Substrate | K<sub>H</sub> | V<sub>max</sub> | k<sub>cat</sub> | k<sub>cat</sub>/K<sub>H</sub> |
|-----------|-----------|------------|----------|-----------------|
| L-Selenocysteine | 9.9 | 58 | 46 | 4.6 |
| L-Cysteine sulfinate | 8.6 | 0.45 | 0.35 | 0.041 |
| L-Cysteine | 5.2 | 0.0074 | 0.0058 | 0.0011 |

methylsulfonyl fluoride, 1 mM EDTA, and 1 μg/ml pepstatin A, and then desalted with a Sephadex G-25 column (2 × 24 cm) equilibrated with 50 mM KPB. The active fractions were collected and applied to a Q-Sepharose column (3 × 10 cm) equilibrated with the same buffer. After the column was washed with the same buffer, the enzyme was eluted with a 0.8-liter linear gradient of 0–0.2M NaCl in the buffer, and the active fractions were pooled and concentrated with ammonium sulfate as above. The enzyme collected by centrifugation was resuspended in 10 mM KPB containing 1 mM dithiothreitol, 0.5 mM EDTA, and 20 μM PLP and then dialyzed against the same buffer. The final preparation was stored at −80 °C until use.

**Preparation of Cell Extracts and Subcellular Fractions from Mouse Liver**—The liver from a BALB/c mouse (6 weeks, male) was homogenized in ice-cold 0.25 M sucrose solution containing 3 mM Tris-HCl (pH 7.4), and 0.1 mM EDTA, and centrifuged for 10 min at 700 × g to obtain crude mitochondrial pellets. The supernatant was centrifuged at 105,000 × g to obtain a microsomal fraction and a cytosolic fraction. A pure nuclear fraction was obtained using the method of Blobel and Potter (15).

**RT-PCR Analysis**—Eight tissues (brain, heart, lung, liver, stomach, spleen, kidney, and testis) were excised from a BALB/c mouse (6 weeks, male), and total RNA was isolated with Sepasol-RNA I (Nacalai Tesque, Kyoto, Japan). RT-PCR was performed with the primers specific to the mSCL transcript. Mp10, 5′-TGGGCAGTGGAGAAGCTGC-3′ and Mp11, 5′-GTGCCAGCAGATGGAAGTGTGATGATG-3′.

**Western Blot Analysis with Anti-mSCL2 Antibody**—Proteins in various cell homogenates and the subcellular fractions were separated by SDS-PAGE and transferred to polyvinylidene difluoride membranes. Western blot analysis was performed with the anti-mSCL2 antibodies, and the proteins were detected by chemiluminescence using CDP-Star (Roche Diagnostics, Basel, Switzerland).

**Enzyme Assay**—The enzyme was assayed in a 0.12 M Tricine-NaOH buffer at pH 9.0. The enzymatic activity toward L-selenocysteine was determined with fuchsin (16). Sulfite production from L-cysteine sulfinate was determined with lead acetate as described previously (6). Sulfite production from L-cysteine sulfinate was determined with lead acetate as described previously (6). Sulfite production from L-cysteine sulfinate was determined with lead acetate as described previously (6). Sulfite production from L-cysteine sulfinate was determined with lead acetate as described previously (6).
BL21(DE3) harboring pESL. Purified mSCL (Table I) provided enzyme, mSCL, was produced, and the product amounted to dialyzed enzyme restored 90% of the original activity. Thus, M1 electrophoresis. Lanes: products were subjected to 1% agarose gel RT-PCR distribution of mSCL mRNA.

Nome DNA as a template. with the same primers using a mouse gene marker; Gen

spleen; T lung; B

tissuеs of mouse (B, brain; H, heart; Lu, lung; St, stomach; Lv, liver; K, kidney; Sp, spleen; T, testis) were subjected to 12.5% SDS-PAGE, blotted onto a polyvinylidene difluoride membrane, and analyzed with polyclonal anti-mSCL2 antibody. mSCL indicates purified mSCL loaded as a control. An arrow indicates the position of mSCL. Each lane was loaded with 30 µg of protein. B, the immunoblot of mSCL in each subcellular fraction; H, cell homogenate; Nu, the nuclear fraction obtained by low-spin centrifugation of the cell homogenate; Mt, the mitochondrial fraction; Mc, the microsomal fraction; Cy, the cytosolic fraction; pNu, the pure nuclear fraction prepared by the method of Blobel and Potter (15). Each lane was loaded with 1 x 10^3 of each fraction prepared from 0.45 g of mouse liver. The position of mSCL is indicated by an arrow.

PLP serves as a cofactor of the enzyme and probably binds to the ε-amino group of a lysine residue at the active site of the enzyme through an aldimine linkage, as in other PLP enzymes studied so far.

The specific activity of the purified enzyme (29 units/mg) for l-selenocysteine was comparable to that of pSCL (37 units/mg) (6) (Table I). The enzyme showed maximum reactivity at about pH 9 when measured in Tricine-NaOH (pH 7.0–9.5) and glycine-NaOH (pH 8.5–11) buffers. The optimum pH of mSCL activity is similar to that of pSCL activity (pH 9.0) (6). In the mSCL reaction, alanine and selenide were formed in a 1:1 stoichiometric ratio from l-selenocysteine in the presence of dithiothreitol (data not shown). However, the selenide product was shown to be produced by the nonenzymatic reduction of selenium by dithiothreitol as was shown in the previous studies on pSCL (20). This indicates that the product of the enzyme reaction is elemental selenium.

The substrate specificity and kinetic parameters for mSCL with l-selenocysteine, l-cysteine sulfinate, and l-cysteine are summarized in Table II. The enzyme exhibits an extremely high, although not absolute, specificity to l-selenocysteine. The kcat/Km value for l-selenocysteine is about 100 times and 4,200 times higher than that for l-cysteine sulfinate and l-cysteine, respectively, confirming that this enzyme is the mouse counterpart of pSCL. The production of alanine was not detected when any of the following amino acids were used as substrates (at 5 mM): L-aspartate, DL-kynurenine, L-selenocysteine, L-cysteine, D-selenocysteine, or D-selenocystine.
Tissue Distribution and Intracellular Localization of mSCL—In order to determine the tissue distribution of the Scly transcript, RT-PCR was performed using a set of primers (Mp10 and Mp11) specific to Scly and total RNAs prepared from various mouse tissues (brain, heart, lung, stomach, liver, kidney, spleen, and testis) as a template. As a control, PCR was performed using the same set of primers with the mouse genomic DNA as a template. As shown in Fig. 5, a 270-bp fragment was amplified in all examined tissues, indicating that the Scly gene is expressed ubiquitously. In the control reaction using genomic DNA, a fragment of about 4.5 kbp was amplified (Fig. 5). The results indicate that this region of the genomic DNA contains at least one intron and that the 270-bp fragments were derived from RNA and not from contaminating genomic DNA.

The tissue distribution of mSCL was examined by Western blotting. Polyclonal antibodies raised against mSCL2 were used to detect mSCL in various tissue homogenates of the mouse. An immunoreactive protein of 47 kDa was detected in all cell homogenates examined (Fig. 6A). Liver, kidney, and testis appeared to have the highest mSCL content.

We determined the intracellular localization of mSCL by Western blot analysis with anti-mSCL2 antibodies. Mouse liver was homogenized and fractionated into nuclear, mitochondrial, microsomal, and cytosolic fractions. The immunoreactive 47-kDa protein band was detected mainly in the cytosolic fraction (Fig. 6B).

DISCUSSION

In this study, we cloned cDNA encoding SCL from mouse liver. The properties of the recombinant mSCL are very similar to those of pSCL in molecular weight, amino acid sequence, optimum pH, subcellular localization, and high specificity toward L-selenocysteine.

Homology searches using the BLAST program against the nonredundant data base revealed that several human EST sequences show strong homology with mSCL. By assembling 11 independent EST sequences and a sequence of cDNA encoding putative human SCL isolated by the 5‘-rapid amplification of cDNA ends method,2 we determined an amino acid sequence encoding the N-terminal region of putative human SCL (hSCL) (Fig. 7). The peptide sequences of pSCL have striking similarity to mSCL and hSCL (Fig. 7), indicating that SCL is highly conserved in mammals. Overall sequence similarity (30%) is found between mammalian NifS homologs and SCLs. However, they are clearly classified into two distinct groups: one includes mSCL, hSCL, and pSCL, and the other includes a mouse NifS homolog (m-Nfs1) and a human NifS homolog (hNifS) (Fig. 7). In particular, the regions corresponding to Gln105-Gly121 and Asn205-Pro214 of mSCL are not found in mammalian NifSs.

We previously classified NifS family proteins into two groups

2 H. Mihara, T. Kurihara, and N. Esaki, unpublished results.
(21): group I includes m-Nfs1, hNiFs, A. vinelandii NiFs, E. coli IacS, etc., and group II includes E. coli CsdB, E. coli CSD, etc. Sequence alignment (Fig. 7) and the phylogenetic tree (Fig. 8) indicate that mammalian SCLs belong to neither of them. Although E. coli CsdB is similar to mSCL in its high specificity toward l-selenocysteine, it shows only 16% sequence identity with mSCL. Accordingly, mammalian SCLs define a new group of enzymes.

Recent studies on eukaryotic NiFs-like proteins from human (22), mouse (23), and yeast (23–25) showed that these proteins have mitochondrial sorting signals at their N-terminal regions. m-Nfs1 predominantly exists in the mitochondrial matrix (23). Yeast NiFs1 was shown to be sorted mainly to mitochondrial matrix (25). hNiFs is produced in two different forms because of the presence of two alternative initiation initiation codons in its gene transcript (22). The larger protein has a mitochondrial targeting signal and is transported into mitochondria. Many iron-sulfur proteins such as components of the TCA cycle and the respiratory chain are present in eukaryotic mitochondria. The subcellular localization of mammalian NiFsS is consistent with the hypothesis that they are directly involved in the de novo formation of iron-sulfur clusters (22).

In contrast, mSCL exists mainly in the cytosolic fraction. The subcellular localization of mSCL is consistent with the hypothesis that mSCL delivers an active form of selenium to the SPS reaction, which proceeds in cytosol. Recently, Lacourciere and Stadtman (9) found that the replacement of selenide by NifS in vitro resulted in an increased rate of formation of selenophosphate, indicating that selenium derived from l-selenocysteine by the action of NiFs serves as a better substrate than selenide for SPS. It is reasonable to assume that an enzyme specific toward l-selenocysteine, namely SCL, functions in a physiological selenium delivery system.

RT-PCR and immunoblot analysis showed that the mRNA of mSCL is distributed in brain, heart, lung, stomach, liver, kidney, spleen, and testis (Figs. 5 and 6A). This result is consistent with the wide distribution of this enzyme’s activity in various mammalian tissues described previously (6). mSCL was predominantly expressed in liver, kidney, and testis (Fig. 6A). Interestingly, SPS is also highly expressed in liver, kidney, and testis, where selenoproteins are produced (26). This supports the view that mSCL cooperates with SPS in the production of selenophosphate.

Free selenocysteine is formed by degradation of selenoproteins containing selenocysteine residues. It can also be produced from selenomethionine by cystathionine β-synthase and cystathionine γ-lyase in mammalian cells (27). Since a high concentration of free selenocysteine is toxic and its accumulation is lethal (28), excess selenocysteine has to be decomposed. SCL, which specifically decomposes selenocysteine into alanine and elemental selenium, may have the role of maintaining the normal concentration of selenocysteine. The result obtained in the present study that the SCL gene is expressed in various tissues is compatible with this hypothesis. The nucleotide sequence of cDNA encoding SCL determined in the present study enables us to examine the physiological role of SCL genetically.

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cDNA Cloning, Purification, and Characterization of Mouse Liver Selenocysteine Lyase: CANDIDATE FOR SELENIUM DELIVERY PROTEIN IN SELENOPROTEIN SYNTHESIS
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