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Hepatic and Renal Cysteine Sulfinic Acid Decarboxylase Activities in Cats Fed Different Levels of Dietary Protein and Taurine

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Abstract

In order to evaluate the dietary regulation of cysteine sulfinic acid decarboxylase (EC 4.1.1.29) in cats, activity and protein content of CSAD were assessed in the liver and kidney of cats fed different levels of dietary protein, with and without taurine. Four groups of cats were fed one of the following diets for 5 weeks: 20% protein and taurine-free diet (LP0T); 20% protein and 0.15% taurine diet (LPNT); 60% protein and taurine-free diet (HP0T); and 60% protein and 0.15% taurine diet (HPNT). CSAD activity was determined in the liver and kidney of cats by measuring $^{14}CO_2$ released from [1-^{14}C]-L cysteine sulfinic acid. CSAD protein was quantified using an immunochemical method. CSAD activity was extremely low in cat tissues, among which kidney showed the highest activity which was 0.118± 0.050, and 0.377±0.056 nmol · min^{-1} · mg soluble protein^{-1} in animals fed LP0T and HP0T, respectively. Even though renal CSAD protein content was 18~55% of the hepatic CSAD protein content, renal CSAD activity was 1.3~6.5 times of the hepatic CSAD activity. Renal CSAD activities of cats fed 60% protein were about 1.6~3.2 times those of animals fed 20% protein, and hepatic CSAD activity was not significantly affected by the dietary level of protein. Taurine depletion significantly elevated both hepatic and renal CSAD activities above the values for cats having normal taurine status most probably as an adaptive response.

Key words: cysteine sulfinic acid decarboxylase, taurine, dietary protein, kidney, cat

INTRODUCTION

Taurine is biosynthesized from sulfur amino acids, methionine and cysteine in most mammals. Although the exact pathway of taurine biosynthesis is still controversial, the main route in many mammalian tissues involves the formation of cysteine sulfinic acid (CSA) from cysteine via cysteine dioxygenase, followed by the decarboxylation of CSA to hypotaurine and subsequent oxidation to taurine (CSA-dependent pathway). The enzyme, cysteine sulfinic acid decarboxylase (CSAD, EC 4.1.1.29) which catalizes the conversion of CSA to hypotauine has been purified for the first time from the dog (1) and rat (2) liver. It is a soluble, pyridoxal 5'-phosphate(PALP)-dependent enzyme containing SH-group as an essential part. The activity of CSA-dependent pathway in various organs show considerable species variation, and in general, it is found high in the liver and brain (3-5), and absent from the mammalian heart (6). CSA can possibly be transaminated with α-ketoglutarate (αKG) to its α-keto acid, β-sulfinyl pyruvate which is readily converted to pyruvate and sulfite (SO_3^{2-}). Griffith (7) studied the in vivo partitioning of CSA between decarboxylation and transamination, and suggested that in mice about 85% of administered CSA was decarboxylated to hypotaurine, whereas about 15% was transaminated to β-sulfinylpyruvate.

Most of the cysteine catabolic enzymes have been reported to be regulated by protein, methionine and cysteine levels in the diet (8-11) as well as by the developmental stage of an animal (12-14). The activity of rat liver cysteine dioxygenase was positively correlated with the dietary intakes of protein (9) and sulfur amino acids (9,10-12). High protein or high cysteine diets significantly increased the activity of cysteine desulfhydratase in chick (15) and rat livers (8). It has been found that CSAD in the rat liver also responds rapidly to the dietary level of protein, in a reversible manner, and feeding rats a high protein diet significantly decreased hepatic CSAD activity (16). On the other hand, taurine depletion significantly increased hepatic CSAD activity in kittens, presumably as part of an adaptive response (17).

Feline species have a peculiar susceptibility to taurine deficiency because of their limited capacity to synthesize taurine (18,19) and high demand for dietary taurine for some biological functions, such as bile acid conjugation (20). Human adults also have limited ability to synthesize taurine similar to that of cats, but they rarely develop taurine deficiency. This is because of the small demand of taurine by mature tissues and the increased glycine conjugation of bile acid as a part of an adaptive response to reduced dietary intake of taurine. In the present study, the activity and protein content of CSAD, a rate limiting enzyme in taurine biosynthetic pathway in most mammalian tissues, were evaluated in the kidney and liver of cats. Strong adaptive responses of hepatic and renal CSAD activities to dietary levels of protein and taurine have been suggested in cats.

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MATERIALS AND METHODS

Animals and diets

Twenty four female cats (1773±60 g, 16~20 week old) from a specific pathogen-free colony at UC, Davis were randomly selected from a larger group maintained on a purified diet containing 435 g soy protein and 1.5 g taurine/kg diet. Half of them were prefed 400 g protein, 1.5 g taurine/kg diet for 1 week, and the other half were prefed a taurine-free diet containing 400 g protein/kg diet for 10 weeks ahead of time for depletion of body taurine. Cats maintained on 400 g protein, 1.5 g taurine/kg diet were switched to either 200 g protein, 1.5 g taurine/kg diet (LPNT) or 600 g protein, 1.5 g taurine/kg diet (HPNT), and those maintained on 400 g protein, 0 taurine/kg diet were switched to either 200 g protein, 0 taurine/kg diet (LP0T) or 600 g protein, 0 taurine/kg diet (HP0T) at the end of the prefeeding period. Each of the 4 groups had 6 cats. The compositions of experimental diets are shown in Table 1. All diets were fed in the form of a pellet.

Cats were maintained on these experimental diets for 5 weeks prior to being sacrificed for enzyme studies. All animals were housed in individual cages in a room having a light cycle from 06:00 to 22:00 daily and maintained at 24°C to 26°C. Food and water were provided ad libitum and changed each morning (09:00~10:00). Animals were weighed twice a week, and daily food intake was measured.

Sample collection

Animals were sacrificed between 9:00~11:00 without preceding fasting. Blood samples were collected from the jugular vein into heparinized 3 ml syringes right before the animals were sacrificed. Plasma was separated from the heparinized blood by centrifugation at 1,500×g for 10 min, and kept at -20°C until the taurine assay. The liver and kidney were removed from the animals under pentobarbital anesthesia, immediately freeze-clamped in liquid nitrogen, and stored at -80°C until the enzyme assay was performed.

Table 1. Composition of experimental diets

	LP0T	HP0T	LPNT	HPNT
	g/kg diet			
Soyprotein	100	300	100	300
Casein	100	300	100	300
Animal tallow	100	100	100	100
Sucrose	150	150	150	150
Starch	486	86	484	84
Mineral mix[1)]	50	50	50	50
Vitamin mix[2)]	10	10	10	10
70% choline · Cl	4.3	4.3	4.3	4.3
Taurine	-	-	1.5	1.5

LPNT, low protein/normal taurine diet; HPNT, high protein/normal taurine diet; LP0T, low protein/taurine-free diet; HP0T, high protein/taurine-free diet

[1)]The mineral mixture contained (g/100 g) $CaHPO_4$ 39.0; K_2HPO_4 9.0; $CaCO_3$ 11.0; $MgSO_4$ 4.5; KCl 10.0; $KHCO_3$ 10.0; $NaHCO_3$ 14.0; $MnSO_4 \cdot H_2O$ 0.384; $ZnSO_4 \cdot 7H_2O$ 0.445; $CuSO_4 \cdot 5H_2O$ 0.080; $FeC_6H_5O_7 \cdot 3H_2O$ 1.000; KI 0.003; $SnCl_2 \cdot 2H_2O$ 0.010; Na_2SeO_3 0.003; $(NH_4)_6Mo_7O_4 \cdot 4H_2O$ 0.004; $CrCl_3 \cdot 6H_2O$ 0.026; $NiCl_2 \cdot 6H_2O$ 0.030; NaF 0.014; $NH_4VO_3 \cdot 4H_2O$ 0.002; NaCl 0.499.

[2)]The vitamin mixture contained (g/kg) cobalamine 0.005; riboflavin 1,002; nicotinic acid 10.022; calcium d-pantothenic acid 2.004; menadione sodium bisulfite complex 1.353; folic acid 1.002; pyridoxine·HCl 1.000; thiamin mononitrate 2.436; myo-inositol 20.042; d-biotin 0.100; ascorbic acid 40.084; retinyl acetate 2,004,182 I.U./kg; cholecalciferol 200,418 I.U./kg; DL-alpha-tocopheryl acetate 16,034 I.U./kg.

Taurine assay

Thawed plasma samples were deproteinized by using 10% sulfosalicylic acid, and analyzed for taurine concentration by an automatic amino acid analyzer based on ion-exchange chromatography (Beckman model 121-MB, USA).

CSAD activity

Forty percent homogenates (w/v) of frozen liver and kidney tissue were prepared using a polytron homogenizer in ice cold 50 mM phosphate buffer, pH 6.8 containing 0.3% Triton X-100, and centrifuged at 20,000×g for 30 minutes at 4°C. The supernatants were assayed for CSAD activity using a modification of the method described by De La Rosa and Stipanuk (21). The reaction was initiated with the addition of L-CSA containing 0.01 μCi of L-[1-^{14}C]CSA to each assay tube. The final assay volume of 800 μl contained 1mM DTT, 2 mM pyridoxal-5'-phosphate, 250 mM potassium phosphate (pH 7.1) and 30 mM L-CSA. $^{14}CO_2$ was trapped on KOH saturated filter paper in center wells suspended from septum stoppers. After incubation at 37°C in a shaking water bath, the reaction was terminated with 10% TCA, and $^{14}CO_2$ was collected for an additional hour. Filter papers were combined with 10 ml of scintillation fluid, and radioactivity was determined by a scintillation counter. Heat inactivated samples served as assay blanks. Product accumulation was linear as a function of incubation time and amount of tissue extract added. Protein concentration was measured in liver and kidney supernatants by the Bradford method (22) using bovine gamma globulin as the standard.

Measurement of CSAD protein content

CSAD was purified >1000-fold from rat liver following the method described by Weinstein and Griffith (23). Rabbits were injected with 200 μg and 100 μg of antigen for the initial and booster injections, respectively. Blood used to prepare preimmune serum was obtained prior to the initial injection. The presence of antibodies against CSAD was assessed by an ELISA. Rabbit IgG fractions were isolated from serum by protein A Sepharose chromatography. Immune IgG were further purified by affinity chromatography.

Samples of liver (60 μg) and kidney (30~40 μg) supernatant protein and incremental amounts of purified CSAD were subjected to SDS-PAGE. The separated proteins were then electroeluted onto nitrocellulose filters. Blots were probed with rabbit anti-CSAD IgG diluted in phosphate-buffered saline (pH 7.4) containing 0.05% Tween-20. Antigen-antibody complexes were detected as described by Jerkins and Steele (24) by activity staining using goat anti-rabbit IgG conjugated to alkaline phosphatase and a reactive mixture containing 0.1 M Tris (pH 9.5), 0.1 M NaCl, 9 mM $MgCl_2$, 1% dimethylformamide, 0.11 mM 5-

bromo-4-chloro-3-indolylphosphate and 0.12 mM nitroblue tetrazolium for color development. Blots were scanned with a Bio-Rad model 620 videodensitometer (Bio-Rad Laboratories, USA), and peak areas were integrated using a microcomputer.

Statistical analysis

Values in the tables and figures represent the mean±SEM of 6 cats. Signifiance of the effect of dietary protein or taurine on plasma taurine concentration or CSAD activity was tested by 2×2 factorial analysis of variance at $p<0.05$, $p<0.01$ or $p<0.001$ using the computer program, Statistical Analysis System (SAS/STAT version 6, SAS Institute Inc. Cary, NC).

RESULTS AND DISCUSSION

Body weight gain

There was no significant difference in average daily food intake among groups over the experimental period. At the end of a 5 week period, kittens fed HP0T and HPNT gained 383±53 g and 463±43 g of body weight, while those fed LP0T and LPNT lost 29±78 and 79±59 g of body weight from their initial weights, respectively. The low protein diets used in the present study did not support the growth of kittens. Protein reqirement for growing kittens is known to be 18~20% when the diets exceed all the essential amino acid requirements (25). Apparently, a diet containing 10% casein plus 10% soyprotein used in the present study does not provide an adequate level of all the essential amino acids for growth.

Plasma taurine concentrations

Changes in plasma taurine concentration over the experimental period is shown in Table 2. For the purpose of prolonged taurine depletion, cats fed LP0P and HP0T had been fed a taurine-free diet for 10 weeks prior to the experiment. Prefeeding the taurine-free diet for 10 weeks reduced the plasma taurine concentration to less than 2 μM even before starting the high and low protein experimental diets. At the end of the 5 week feeding period, plasma taurine concentration of cats fed LP0T and HP0T was reduced further to about 1 μM. Taurine concentrations in the plasma collected on the last week of the experiment were statistically analyzed by 2×2 factorial analysis of variance, and the results indicated that plasma taurine concentrations were significantly influenced by dietary levels of protein as well as taurine ($p<0.001$). Cats fed the HPNT had a plasma taurine concentration 47% lower than the value for animals fed the LPNT. It has been repeatedly observed in our laboratory that high protein diets, especially high soybean protein diets significantly reduced plasma taurine concentration (26). Further investigations are needed to provide an explanation for this phenomenon.

Table 2. Plasma taurine concentrations in cats fed LP0T, HP0T, LPNT and HPNT

	Weeks					
	0	1	2	3	4	5
	μmol/L					
LP0T	1.83 ±0.2	0.90 ±0.08	1.22 ±0.2	0.90 ±0.06	0.78 ±0.07	1.02* ±0.19
HP0T	1.59 ±0.14	0.94 ±0.15	1.61 ±0.19	1.21 ±0.18	0.87 ±0.10	1.21* ±0.16
LPNT	74.5 ±6.7	142 ±19.2	150 ±17.1	138 ±4.3	151 ±18.3	137* ±4.5
HPNT	69.7 ±10.1	70.1 ±4.32	71.8 ±8.8	49.3 ±6.2	53.2 ±1.8	71.8* ±5.2

Each value represents mean±SEM of 6 animals.
LPNT, low protein/normal taurine diet; HPNT, high protein/normal taurine diet; LP0T, low protein/taurine-free diet; HP0T, high protein/taurine-free diet
*Plasma taurine concentrations on week 5 were significantly affected by dietary levels of protein and taurine ($p<0.001$), when analyzed by 2×2 factorial analysis of variance.

Activity and protein content of CSAD

Activities and protein contents of CSAD in the liver and kidney of cats fed LP0T or HP0T are presented in Fig. 1 and Fig. 2. Hepatic CSAD activity of cats found in the present study (0.094~0.154 nmol $CO_2 \cdot min^{-1} \cdot mg\ protein^{-1}$) is comparable to the values in adult cat liver (about 0.075 nmol $CO_2 \cdot min^{-1} \cdot mg\ protein^{-1}$) reported previously by Sturman and Hayes (27), but is much lower than the values reported in rat liver (1.25~6.09 nmol $CO_2 \cdot min^{-1} \cdot mg\ protein^{-1}$, depending on the dietary level of protein) (16). In parallel with CSAD activity, CSAD protein contents in cat liver (37~42 μg CSAD protein · g $liver^{-1}$) were also much lower than those reported in rat liver (310~1506 μg CSAD protein · g $liver^{-1}$) (28). Animals vary in their ability to synthesize taurine from its precursors cysteine and methionine. Since the main pathway for taurine biosynthesis is limited in humans and cats, both species appear to have a requirement for taurine in the diet. Independent of the CSA-dependent pathway, cysteine undergoes desulfhydration, a process that is largely achieved by the direct desulfhydration which is catalyzed by cysteine desulfhydrase (8,15) and the transamination of cysteine via cysteine aminotransferase in

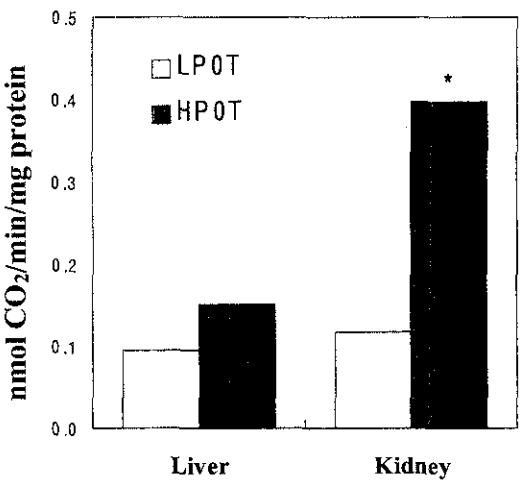

Fig. 1. Specific activity of cysteine sulfinic acid decarboxylase in cats fed LP0T and HP0T. LP0T, low protein/taurine-free diet; HP0T, high protein/taurine-free diet. *Significantly different from the value for LP0T within each tissue by Student's t-test at $^*p<0.01$.

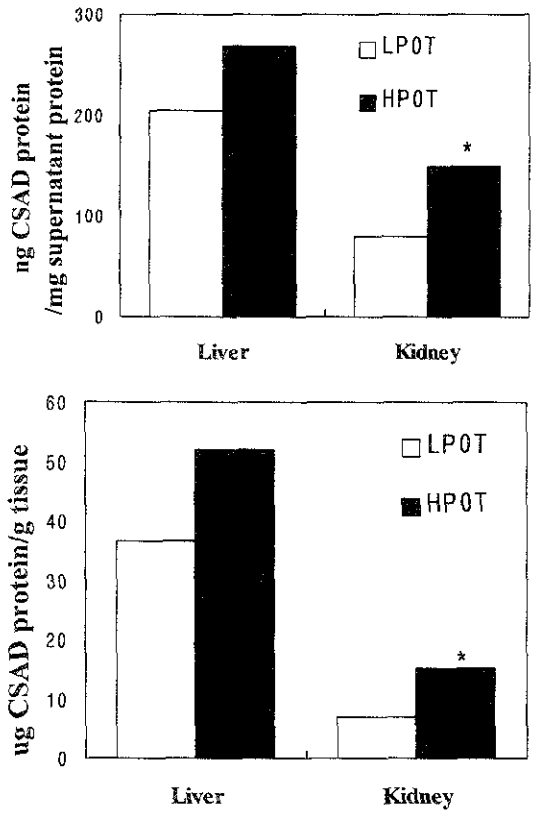

Fig. 2. Quantity of cysteine sulfinic acid decarboxylase protein in cats fed LPOT and HPOT. LPOT, low protein/taurine-free diet; HPOT, high protein/taurine-free diet. *Significantly different from the value for LPOT within each tissue by Student's t-test at *$p<0.05$.

conjunction with the reaction catalyzed by β-mercaptopyruvate sulfurtransferase (29,30). In both pathways, the end products are pyruvate and hydrogen sulfide (H_2S). Based on our findings of extremely low activity and protein content of CSAD in cat tissues, it is suggested that the main cysteine catabolic pathway results in pyruvate via desulfhydration pathway in cat tissues.

Renal CSAD activities were 126% and 258% of hepatic activities in cats fed LPOT and HPOT, respectively. However, CSAD protein contents in the liver were 1.8~2.6 times higher than those in the kidney of cats. These results suggest that, in cats, renal CSAD has a higher affinity for the substrate than hepatic CSAD. Within each tissue, CSAD protein content was closely correlated with CSAD activity ($r=0.91$, $p<0.001$). Tissue distributions of CSAD activity and CSAD protein content appear to be species-dependent since, in rats, renal CSAD activities were about 1/10 of hepatic activities (31), and renal CSAD protein contents were about 1/4~1/20 of hepatic CSAD protein contents (24).

Feeding HPOT for 5 weeks resulted in a significantly higher renal CSAD activity (3.4 times) than the value for animals fed LPOT ($p<0.01$). Renal CSAD protein contents, which are expressed per mg supernatant protein and per g tissue, were significantly higher in cats fed HPOT (1.9 times of the value for LPOT) than the value for animals fed LPOT ($p<0.05$). Hepatic activity and protein content of CSAD were also 64% and 31~42% higher, respectively, in cats fed HPOT compared to those fed LPOT, but a statistical significance was not found most probably due to large standard variations.

Effects of protein level and presence of taurine in diet on hepatic and renal CSAD activities are shown in Table 3. Hepatic CSAD activity expressed either per milligram protein or per g liver was not significantly affected by dietary level of protein. However, feeding a taurine-free diet for 15 weeks resulted in significantly higher hepatic CSAD activities which are 3.4~6.0 times those of cats fed diets with 0.15% taurine ($p<0.001$). Apart from the hepatic CSAD activity which did not respond significantly to the dietary level of protein, CSAD activities in the kidney of cats fed 60% protein were about 1.6~3.2 times those of cats fed 20% protein($p<0.001$). Renal CSAD activity was also significantly higher in cats fed HPOT compared to those fed HPNT ($p<0.05$ when CSAD activity was expressed per mg supernatant protein, and $p<0.01$ when expressed per g kidney).

We previously demonstrated that 60% protein diets significantly elevated the capacity of hepatic cysteine desulfhydration in cats compared to those fed 20% protein diets (32). Our results from cats showing high protein diets significantly in-

Table 3. Activities of cysteine sulfinic acid decarboxylase in the liver and kidney of cats fed different levels of dietary protein and taurine

	Diet				ANOVA		
	LPNT	HPNT	LPOT	HPOT	A	B	A×B
Hepatic Activity							
UA[1]/mg protein	0.028±0.008	0.030±0.014	0.094±0.025	0.154±0.035	***	N.S.	N.S.
UA/g liver	4.2±1.2	5.0±2.2	16.6±4.5	29.9±6.5	***	N.S.	N.S.
Renal Activity							
UA/mg protein	0.125±0.026	0.195±0.015	0.118±0.050	0.379±0.056	*	***	*
UA/g kidney	12.2±2.8	19.4±1.6	14.8±3.1	41.7±4.7	**	***	**

Each value is mean±SEM of 6 animals.
LPNT, low protein/normal taurine diet; HPNT, high protein/normal taurine diet; LPOT, low protein/taurine-free diet; HPOT, high protein/taurine-free diet; A, taurine effect; B, protein effect.
[1]UA is defined as nmol CO_2 produced/min.
*,**,***Significantly affected by the dietary modification when analyzed by 2×2 factorial analysis of variance at *$p<0.05$, **$p<0.01$, and ***$p<0.001$.

creased renal CSAD activity contradict the previous finding in rats. Rats fed a high-protein (60% casein) diet for 7~14 days showed hepatic CSAD activity which were 20~40% of the activity in liver of rats fed a moderate (18% casein) protein diet (16,33). Rats also differ from cats in that hepatic or renal CASD activities were decreased after feeding rats methionine (31,32) or cysteine (11) supplemented diets. Knopf et al. (34) reported that hepatic CSAD activity was the same in cats fed taurine-free diets as in cats fed diets containing 0.4% taurine. Our observation of 90%~410% increases in hepatic and renal CSAD activities in taurine-depleted cats is clearly not in agreement with their observation, but supports the findings of Rentschler et al. (17) that hepatic CSAD in taurine-deficient kittens was five-times the level in control kittens fed a diet with 0.1% taurine for 6 weeks. The physiological importance of CSAD regulation by dietary protein and sulfur amino acids is not fully understood, but may be related to the partitioning of CSA between sulfate- and taurine-synthesizing pathways. Increased hepatic and renal CSAD activities in cats fed taurine-free diets appear to be one of the adaptive responses of this species to conserve body taurine, although the significance of the increased capacity of taurine biosynthesis to taurine homeostasis is not yet known.

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