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Taurine supplementation improves the utilization of sulfur-containing amino acids in rats continually administrated alcohol

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Abstract

The main purpose of this study was to evaluate changes in brain sulfur-containing amino acid (SCAA) metabolism to determine whether taurine intervened under continuous alcohol intake. We fed 80 male Sprague–Dawley rats 30% alcohol-containing water for 4 weeks. Eighty animals were divided into two groups (with or without 2 g/kg body weight taurine supplementation), and five were killed every week in each group for monitoring SCAA changes in the brain, liver, kidneys and heart. Results indicated that the plasma alcohol concentration increased from Weeks 1–4; however, animals with taurine supplementation showed a lower plasma concentration of ethanol in Week 2. As to SCAA concentrations, cysteine and taurine were both lower after a week of alcohol ingestion in the brain and plasma; the same declining trend was shown in the liver in Week 2. In contrast, plasma and hepatic concentrations of homocysteine were elevated in Week 2, and the plasma *S*-adenosylmethionine (SAM)/*S*-adenosylhomocysteine (SAH) ratio also decreased in Week 1. Furthermore, the key cofactor of transsulfuration, pyridoxal-5′-phosphate, significantly declined in the plasma after a week of the ethanol intervention, whereas an increase was observed in brain tissue. Under taurine supplementation, some recoveries were shown by delaying taurine depletion to Week 2, increasing the SAM/SAH ratio and elevating plasma and brain levels of vitamin B_6 in Week 2. In conclusion, daily consumption of 30% alcohol interfered with SCAA metabolism, thus decreasing taurine's role in neurotransmission. The possible mechanism involved might be that ethanol interrupts the production of cysteine, which is the upstream SCAA of taurine, thus decreasing the homocysteine level. Additionally, taurine supplementation delayed this process.

Keywords: Taurine; Sulfur-containing amino acids; Alcohol; SAM/SAH ratio; Pyridoxal-5′-phosphate

1. Introduction

It has long been known that alcohol consumption interferes with the regulation of sulfur-containing amino acids (SCAAs). However, most studies focused on interactions of single amino acids and alcohol-induced hepatic disorders. Few studies have examined the overall process of SCAA metabolism in the central nervous system (CNS) under alcohol consumption; they generally focused on the liver [1–4]. With increasing incidences of alcohol abuse and related psychological nutrient metabolism, more studies on the alcoholic brain have been carried out.

With both acute and chronic consumption, ethanol interrupts the methionine and folate metabolic cycles [3], not to mention disrupting transmethylation affecting the *S*-adenosylmethionine (SAM)/*S*-adenosylhomocysteine (SAH) ratio [2,4], which is the key of DNA methylation. Research showed that DNA hypomethylation was involved in animal model of hepatic carcinogenesis [5,6]. Alcohol dehydrogenase localized not only in liver but also in kidney.

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Alcohol metabolism would also cause interferences in several enzymes which are related to SCAAs, such as cysteine conjugate β-lyase, and not to mention, alcoholic ketoacidosis caused kidney overloading [7]. Acute ethanol administration also increases cysteine utilization and taurine synthesis in hepatic tissues [8].Vitamin B_6, a coenzyme of cysteine transsulfuration, is also reduced under alcohol administration [9]. This information indicates that ethanol intake is highly correlated with SCAA metabolism in the liver and brain.

As an end product of SCAA metabolism, taurine plays a crucial role in brain neurotransmission, and vitamin B_6 is the cofactor of GABA transaminase and glutamate decarboxylase. It is of great interest to study SCAA metabolism and taurine in the brain under alcohol administration.

Although it was first labeled a nonessential, biologically inert amino acid, taurine and its analogues are now believed to be correlated with many psychological disorders, such as major depression, epilepsy, anxiety and alcoholism [10–12]. Research shows an increase in taurine transport and efflux under chronic alcohol exposure [13]. Moreover, acamprosate, a derivative of taurine, is now approved for use in alcohol withdrawal [14]. Since we previously reported that acute ethanol gavagement produced dramatic imbalances in the SCAA metabolism in various tissues, especially the brain, a significant correlation between changed plasma levels of pyridoxal-5′-phosphate (PLP) and taurine was reported [15]. The results indicated strong influences on taurine and transsulfuration under ethanol intake. Thus, further interest was aroused for evaluating the effectiveness of a taurine intervention on SCAA metabolism during chronic alcohol ingestion.

2. Experimental design

2.1. Animals and treatment

Thirty-five 4-week-old Sprague–Dawley rats were housed in stainless cages in a humidity- and temperature-controlled environment with a 12-h light–dark cycle. After a week of acclimation, five animals were randomly sacrificed at the baseline (B). The remaining animals were divided into two groups [alcohol (A) and alcohol+taurine (AT) groups] and given drinking water which contained 30% alcohol with or without taurine supplementation [2 g/kg body weight (BW)]. In each group, five animals were sacrificed for in the first, second and fourth weeks. Brain, liver and blood samples were collected for further biochemical analysis. All prepared biological samples were used for SAM, SAH, PLP and SCAA analyses. The NADH/NAD ratio and alcohol level of plasma were also determined.

2.2. Biochemical analysis

2.2.1. Analysis of SAM and SAH

SAM and SAH were analyzed using method described by [16]. All procedures of sample preparations were carried out at 4°C. Two hundred μl of plasma or tissue homogenates was deproteinized with an equal volume of 0.4 M perchloric acid, and the mixture was centrifuged at 10,000*g* at 4°C for 20 min. The supernatants were filtered and then directly applied to a high-performance liquid chromatography (HPLC) analytical system. The equipment for the SAM/SAH analysis consisted of a model Hitachi L-7100 pump equipped with an L-7200 autosampler, an L-7455 Photo Diode Array detector, and an Insil ODS-2 column (5 μm, 4×250 mm, GL Sciences, Torrance, CA). Conditions for the SAM/SAH analysis were a 1-ml/min flow rate and detection at 254 nm. The mobile phase consisted of 40 mM $NH_4H_2PO_4$, 8 mM 1-heptanesulfonic acid sodium salt and 18% (by volume) methanol (pH 3.0, adjusted with hydrochloric acid).

2.2.2. Analysis of taurine and other SCAAs

Analyses of taurine and other SCAAs were modified from the description of Vasanits et al. [17,18]. Borate buffer was used to homogenize specimens (1/1, by volume) then the proper amount of *o*-phthaldialdehyde-3-mercaptopropionic acid was added for derivatization. The centrifuged supernatants were then applied to HPLC for analysis. The HPLC apparatus consisted of a model L-7100 Hitachi pump equipped with an L-6200 UV detector. A Hypersil amino acid column was used. The mobile phase consisted of isocratic eluent A (0.03 M sodium acetate with 0.25% tetrahydrofuran by volume) and eluent B (80% acetonitrile with 20% 0.1 M sodium acetate by volume) at a flow rate of 1 ml/min and detection at 254 nm.

2.2.3. Glutathione analysis

A commercial kit (BIOXYTECH GSH-400TM) was used to determine the glutathione (GSH) level of all samples. Metaphosphoric acid (33–37%) was used to resuspend the erythrocyte lysate (4:1, by volume). The centrifuged (3000*g* at 4°C for 10 min) supernatant was collected and reacted with 50 ml of reagent A (4-chloro-1-methyl-7-trifluromethyl-quinolinium methylsulfate) to produce thioesters. Chromophoric thione was produced by adding reagent B (30% NaOH), with detection at 400 nm.

2.2.4. PLP analysis

The analysis of PLP was carried out with minor modifications as described by Ref. [19]. Plasma and tissue homogenates were deproteinized with 1 N perchloric acid. The supernatants were then directly injected into the HPLC for the PLP analysis. The HPLC apparatus consisted of a model L-7100 Hitachi pump equipped with an L-6200 fluorescence detector. A C18 HypersilTM amino acid column was used with a mobile phase of 0.03 M sodium phosphate under a flow rate of 1 ml/min.

2.2.5. Sample preparation for the NAD/NADH analysis

The protocol was modified according to the method developed by [20]. Plasma was preweighed to 0.2 ml or 0.2 g of tissue was ground in 0.5 ml phenol buffer (0.64 M phenol and 0.07 M phosphate-buffer saline; pH 7.8), and 0.5 ml of chloroform was added in the dark then centrifuged at

Table 1
Average alcohol and taurine intake during the experiment (n=5 for each time interval)

Group	30% Alcohol (A)	30% (AT)	
	Alcohol intake (ml)	Alcohol intake (ml)	Taurine intake (g)
Week 0	15.3±2.3	15.1±2.1	0.39±0.07
Week 1	19.2±2.6	19.3±1.7	0.49±0.08
Week 2	17.5±2.1	17.1±2.3	0.43±0.11
Week 4	18.7±1.7	19.5±2.4	0.49±0.09

10,000 rpm at 4°C. The supernatant was washed with 1 ml diethyl ether three times. The cleaned lower layer was filtered through a Multiscreen system (Millipore Multiscreen, lot no. F4PN82001) then injected into HPLC for analysis. The HPLC analysis was used an isocratic buffer system in buffer A [0.1 M KH_2PO_4, 5 mM tetrabutylammonium hydrogen sulfate (TBS) and 2.5% acetonitrile (pH 6.0)] and buffer B [0.1 M KH_2PO_4, 5 mM TBS and 25% acetonitrile] with a time-dependent gradient. The flow rate was 0.8 ml/min, and a photodiode array detector was used for detection. The analytical column was a C18 reversed-phase column (25×4.6 mm, 5 μm; Ascentis C18 581325-U, Supelco, St. Louis, MO), and the system consisted of a Hitachi L-7100 pump equipped with an L-7200 autosampler, an L-7455 Photo Diode Array detector and an Insil ODS-2 column (5 μm, 4×250 mm, GL Sciences).

2.3. Statistical analysis

Values are presented as the mean±S.D. All data were analyzed using the SAS system. Fisher's least significant difference test was performed to analyze differences among different ethanol consumption exposure durations. The acceptable level of significance was established at P<.05 except when otherwise indicated.

3. Results

3.1. Intake of alcohol and taurine

Each animal drank about 4.62±0.41 g of alcohol (15.4±1.37 g/kg BW) everyday, and in the AT group, the average taurine intake was 0.53±0.05 g each day (1.42±0.16 g/kg BW). According to Table 1, there were no differences in water intake among the two groups. Besides, there were no significant differences among time points during the experiment. Furthermore, there were also no significant changes of food intake between the two groups during the experimental period (25.35±6.42 g/day in AT group and 26.33±7.17g/day in A group, P=.301).

3.2. Plasma alcohol concentration and the hepatic NADH/NAD ratio

In the experiment, the plasma alcohol concentration continually increased remained inclined after 1 month of alcohol ingestion when compared to the baseline (Fig. 1A). Interestingly, taurine supplementation (in the AT group) suppressed the increasing of trend plasma alcohol resulting in a lower alcohol level than in the group which only consumed alcohol in Week 4. In hepatic tissue, the NADH/NAD ratio increased after alcohol treatment for 2 weeks in both groups (Fig. 1B). In the alcohol-only group (A), animals had a higher NADH/NAD ratio in Week 2 and an even higher one in week 4; it was also higher than that of taurine-supplemented group (AT) in week 4. The increasing trend remained in both groups, whereas taurine supplement suppressed the ratio in Week 4.

3.3. SCAAs attenuated in brain, liver and plasma

The increasing SAM/SAH ratios in brain and hepatic tissues after 1 month of alcohol treatment are shown in Fig. 2. The results showed that the SAM/SAH ratio began to decrease in brain and liver in week 2. However, taurine

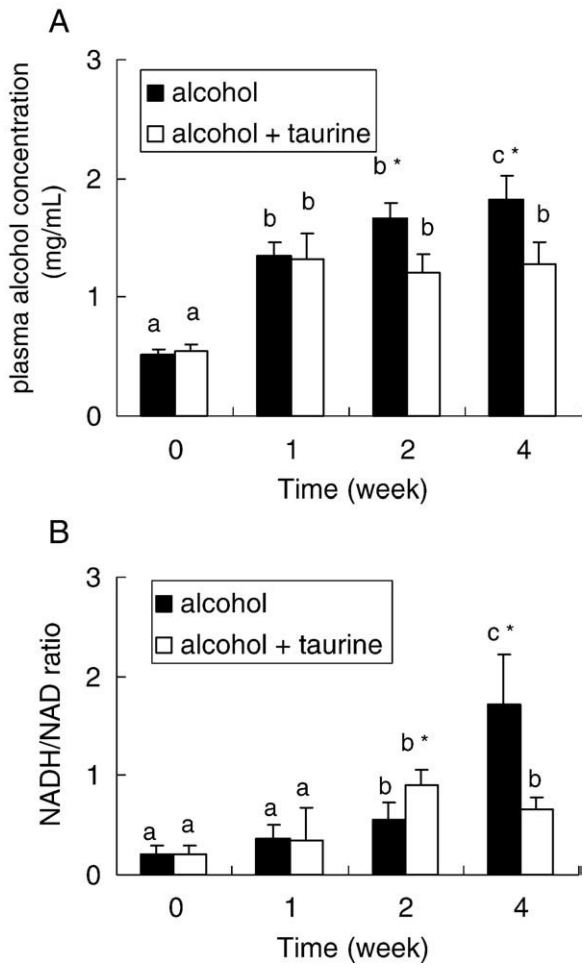

Fig. 1. The effects of taurine on plasma alcohol concentrations (A) and the hepatic NADH/NAD ratio (B) under alcohol administration. An asterisk represents statistical difference from another group at the same time point, and different letters indicate differences from time points within the same group, P<.05.

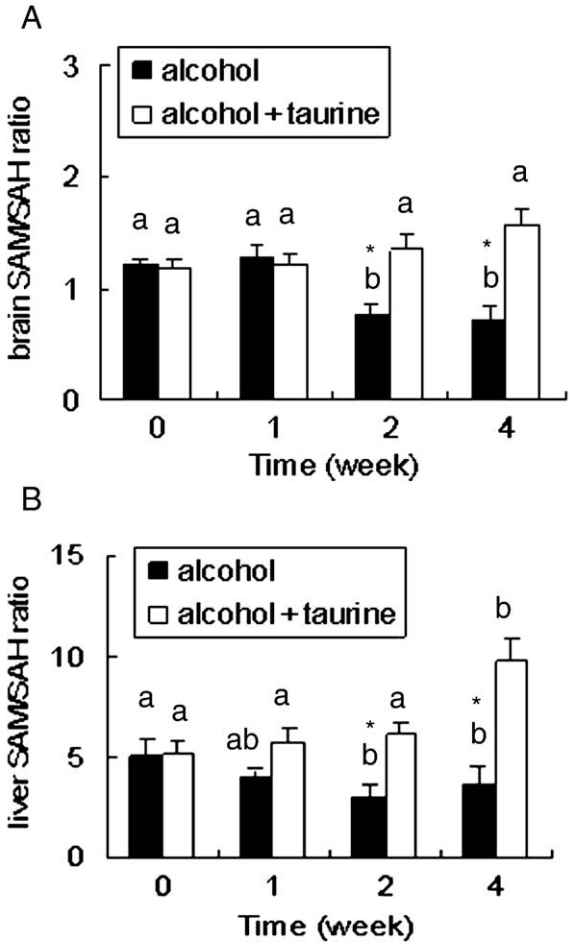

Fig. 2. Effects of SAM/SAH in the brain (A) and liver (B) after 4 weeks of alcohol ingestion with or without taurine supplementation. An asterisk indicates a statistical difference from another group at the same time point, and different letters indicate difference from time points within the same group at $P<.05$.

supplementation significantly reduced the decreasing effect on the SAM/SAH ratio by ethanol and showed an increasing trend in the liver in Week 4. Furthermore, the ratio showed a significant difference between the two groups in Week 2 which remained until Week 4.

From the aspect of SCAA metabolism, the results showed that alcohol depleted methionine with increased SAM and SAH synthesis in the brain and liver (Tables 2 and 3). In the liver, levels of SAM and SAH increased immediately after 1 week of ethanol administration. There was no significant difference in SAM levels between the 2 groups during the entire period, whereas SAH levels in the alcohol group were significantly higher than those of the taurine-supplemented group from Weeks 1–4. In brain tissue, the level of SAM was elevated in Week 2, and SAH was elevated in Week 1 in both groups. By comparison of the two groups, significant differences were revealed in Week 2. In the taurine-supplemented group, recovery of the SAH concentration was shown in Week 4 in both brain and liver tissues.

Alcohol administration decreased cysteine formation from Weeks 2–4 in the brain and liver; however, hypotaurine increased in Week 4 and was attributed to decreases in taurine in the liver (Week 4) and brain (Week 2). Surprisingly, taurine supplementation increased cysteine production in both tissues and decreased hypotaurine in the liver where recovering from taurine depletion. In terms of plasma SCAA utilization (Table 4), decreased methionine and increased homocysteine levels were observed in Weeks 2 and 4, respectively. Taurine supplementation delayed the process of transmethylation of these amino acids. Cysteine and taurine levels were significantly lower in Weeks 2 and 4, respectively. Alcohol with added taurine caused an increase in cysteine and postponed taurine depletion in the plasma. GSH, a competitive product of taurine, showed a significant decrease that begin in Week 1 and lasted to Week 4, and taurine supplement prevented it from being depleted.

3.4. Changes in PLP in tissues

From the point of view of the PLP-involved mechanism (Fig. 3), the result showed similar declines in brain and plasma after a week of alcohol ingestion; however, the liver level of PLP dropped in Week 4 (Fig. 3B, C). In addition, plasma PLP continued to drop in Week 2 and ceased being

Table 2
Changes in SCAAs amounts in the liver during the experiment

		Methionine	SAM	SAH	Cysteine	Taurine	Hypotaurine
		nmol/g Tissue				μmol/g Tissue	
B	Taurine	100.5±26.1	85.1±12.80	16.9±1.8	62.3±11.7	7.03±0.73	1.99±0.84
	Control	107.8±13.5	79.6±08.50	16.7±1.8	60.5±8.0	6.24±1.13	2.01±0.54
Week 1	Taurine	98.6±21.4	116.3±51.7[a]	23.9±1.6[a]	61.2±10.3	6.52±0.82	1.87±1.24
	Control	73.6±21.4	122.7±35.1[a]	37.8±2.8[a,b]	55.4±11.4	4.98±0.32[a,b]	1.97±0.46
Week 2	Taurine	98.0±19.9	137.6.6±41.4[a]	21.5±1.2[a]	72.0±21.2	5.52±0.55[a]	1.72±0.10
	Control	68.9±17.3[a]	143.8±22.2[a]	43.6±1.3[a,b]	45.1±6.2[a,b]	4.73±1.01[a]	2.31±0.78
Week 4	Taurine	75.6±13.4[a]	189.2±25.9[a]	18.7±2.0	91.8±9.8[a]	6.31±1.67	1.08±0.51[a]
	Control	58.7±6.32[a,b]	175.8±34.9[a]	49.6±3.0[a,b]	42.1±5.8[a,b]	4.23±0.15[a,b]	2.33±0.13[a]

B indicates Week 0. Values are presented as the mean±S.D. Animal numbers were eight at each time interval. Values in the same column and row with different superscripts significantly differ at $P<.05$ by one-way analysis of variance (ANOVA) with least significant difference.

[a] Significant difference compared to the control group.

[b] Difference between groups at the same time point.

Table 3
Changes in SCAAs amounts in the brain during the experiment

		Methionine	SAM	SAH	Cysteine	Taurine	Hyptaurine
		nmol/g Tissue				μmol/g Tissue	
B	Taurine	100.4±26.5	20.1±3.6	15.9±1.4	106.9±29.7	6.11±0.41	0.64±0.09
	Control	102.6±16.7	20.8±4.2	14.5±1.8	96.9±19.6	6.81±0.61	0.62±0.16
Week 1	Taurine	89.7±6.4	24.2±2.4	19.1±0.8 [a]	94.6±10.4	5.32±1.03	0.58±0.17
	Control	82.2±12.7	25.1±2.2	19.3±1.9 [a]	82.1±11.7 [b]	4.07±0.73 [a, b]	0.48±0.13
Week 2	Taurine	70.5±10.2 [a]	29.2±3.1 [a]	20.3±1.2 [a]	90.1±18.3	4.58±0.36 [a]	0.59±0.25
	Control	66.5±11.8 [a]	27.8±2.3 [a]	32.4±0.9 [a, b]	73.9±9.6 [a, b]	4.01±0.63 [a]	0.37±0.21 [a, b]
Week 4	Taurine	67.6±10.2 [a]	30.1±3.2 [a]	17.6±2.1	110.3±18.5	5.38±0.66	0.48±0.21
	Control	41.9±3.6 [a, b]	29.6±2.1 [a]	37.6±2.2 [a, b]	70.7±9.2 [a, b]	3.46±1.24 [a, b]	0.30±0.09 [a, b]

Values are presented as the mean±S.D. Animal numbers were eight at each time interval. Values in the same column and row with different superscripts significantly differ at $P<.05$ by one-way ANOVA with least significant difference.
[a] Significant difference compared to the control group.
[b] Difference between groups at the same time point.

depleted after Week 1 in the brain. With taurine supplementation, the brain PLP level showed a little, but insignificant, improvement in Week 4. In the plasma and liver, PLP levels might not have been improved with taurine supplementation, but they were still significantly higher than those of the control group (A) in Weeks 1 and 4. As to the dramatical declines in PLP levels in the plasma and liver with alcohol treatment, taurine supplementation did not alter PLP levels, and the low levels were maintained until the end of the study.

4. Discussion

In our study, the average amount of ingested ethanol was about 14 g/kg BW every day for each animal, which is an extremely high dosage among recent studies [21–24]. Chronic alcohol drinking might increase food intake but interrupt absorption of various nutrients [25]. Taurine supplementation at 1.45 g/kg BW could partially ameliorate the impaired SCAA metabolism induced by ethanol. The present study demonstrated that taurine supplementation can play a protective role in hepatic tissues and the brain with alcohol consumption. Acamprosate, a recently developed anticraving drug, is a derivative of taurine (calcium homotaurinate), and although it is the most easily tolerated anticraving drug, it still has side effects, for example, transient diarrhea [26]. In this study, we did not observe any adverse effects in the taurine-supplemented group. As to the high safety of taurine [27], its application for alcoholism could provide good nutrition support or be a functional food in the future.

It is evident that ethanol intake dysregulates methionine metabolism in various tissues [3,28]. However, DNA methylation might be one of the main reasons that ethanol impacts methionine metabolism [22]. In our study, 4 weeks of ethanol ingestion depleted brain and liver methionine levels and decreased SAM/SAH ratios. SAH is thought to be a potent inhibitor of SAM transmethylation so that the decreased ratio represented a decrease in SAM transmethylation. The results might also imply the activation of methionine adenosyltransferase and the interruption of glycine *N*-methyltransferase by ethanol [3]. The decreased SAM/SAH ratio in this study was consistent with previous studies and suggests the possibility of betaine or SAM supplementation with ethanol administration [10,24]. Although betaine and SAM supplementation can improve

Table 4
Time changes in plasma SCAAs concentration via taurine intervention.

		Homocysteine	Methionine	Cysteine	GSH	Taurine	Hypotaurine
		nmol/L	μmol/L				
B	Taurine	1.85±0.58	17.8±2.1	13.5±2.8	56.3±11.8	19.6±9.2	17.6±3.9
	Control	1.69±1.51	21.3±3.1	14.8±2.1	67.2±12.3	21.3±5.9	18.3±1.1
Week 1	Taurine	1.78±0.83	15.7±2.5	9.2±3.5 [a]	43.2±11.5	20.6±3.2	20.7±9.2
	Control	1.71±0.73	15.8±3.6	10.4±1.4	45.2±9.2 [a]	16.8±2.7 [b]	19.5±3.7
Week 2	Taurine	2.17±0.53	16.8±1.3	11.5±2.1	65.7±15.1	16.5±9.5	16.2±3.0
	Control	2.51±1.07 [a]	12.2±1.4 [a, b]	10.9±1.3 [a]	48.5±5.4	14.6±3.51 [b]	18.5±2.6
Week 4	Taurine	2.79±0.61 [a]	16.4±2.6	15.5±3.7	83.5±4.4 [a]	15.1±1.9	19.1±2.1
	Control	3.85±0.81 [a]	9.9±1.5 [a, b]	7.2±1.5 [a, b]	31.2±8.2 [a, b]	9.1±1.1 [a, b]	17.6±4.7

Values are presented as the mean ± S.D. Animal numbers were eight at each time interval. Values in the same column and row with different superscripts significantly differ at $P<.05$ by one-way ANOVA with least- significant difference.
[a] Significant difference compared to the control group.
[b] Difference between groups at the same time point.

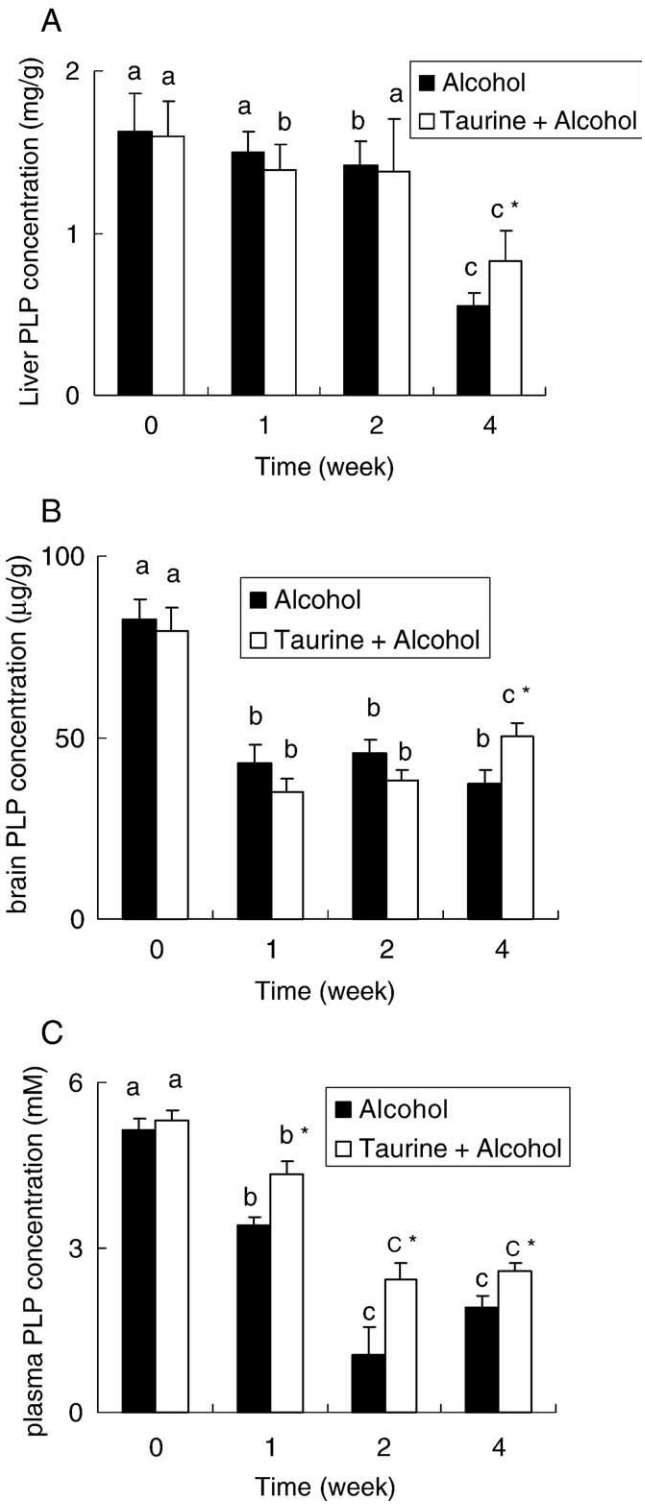

Fig. 3. Changes in PLP in the brain (A), liver (B) and plasma (C) during the experiment. An asterisk indicates a statistical difference from another group at the same time point, and different letters indicate a difference from different time points within the same group at $P<.05$.

the oxidative status and recover a few SCAA pools which are damaged by ethanol drinking, the results indicated that they cannot restore taurine levels of hepatocytes [29,30]. The inability to recover taurine depletion in the liver may cause an insufficient supply to various tissues (i.e., the brain), thus resulting in lower effectiveness. Consequently, direct supplementation with taurine may be a good choice for ameliorating alcohol-induced side effects.

Methionine plays a protective role in the brain; the possible mechanism is that methionine can be an antioxidant to protect brain cells from alcoholism-provoked oxidative stress [31–33]. Consequently, the decreased brain level of methionine might cause an increase in oxidative stress by subchronic ethanol consumption. Taurine may also be regarded as an antioxidative amino acid; therefore, it may function to delay methionine depletion and provide an explanation for methionine's recovery function [34,35].

It has mentioned that the primary effect of ethanol on the liver is its rapid conversion of NAD^+ into NADH [36]. The reason why ethanol alters the NADH/NAD^+ ratio is mainly based on its interaction with alcohol dehydrogenase [37]. The increased NADH/NAD^+ ratio is interpreted as a disturbance of carbohydrate metabolism under chronic alcoholism [38]. In other words, chronic ethanol ingestion might inhibit hepatic gluconeogenesis through stimulating NAD^+ conversion into NADH [39]. In the present study, taurine supplementation successfully reducing the ratio might be attributed to the following mechanisms. First, the antioxidative hepatoprotection of taurine by dietary supplementation can counteract oxidative stress in animal studies [40,41]. A prospective study even pointed out that NAD synthesis was positively correlated with the formation of taurine and relative SCAA pools [42]. In our study, a protective role of taurine on the liver was verified through significantly decreased NADH/NAD^+ ratios.

It is already known that abnormal transsulfuration and GSH metabolism exist in animal models of alcoholic liver disease [4]. In our study, the decreased levels of PLP, cysteine and taurine in the brain and liver can possibly be attributed to the damage from increased transsulfuration by ethanol (Figs. 2–5). Both chronic and acute alcohol consumption in our previous study produced transsulfuration dysregulation [15]. Ethanol interrupts vitamin B_6 absorption and circulation in nearly 51.5% of alcoholics [9]. Such a phenomenon might induce insufficient cysteine production and a further decline in taurine formation. The depletion of PLP might contribute to the activation of GABA-transmission through PLP-dependent GABA-transaminase in the CNS. Ethanol is thought to stimulate GABAergic activity to produce sedative effects under high-dose alcohol consumption [16].

Taurine, the end product of the transsulfuration of sulfur-containing amino acids, plays an important role in hepatoprotection and maintaining normal neurofunction [43]. Acute ethanol administration can increase taurine synthesis in the liver for further utilization [1,5]; however, chronic alcohol ingestion activates taurine catabolism [29,30]. In the CNS, taurine is not only the major osmolyte in astrocytes but also a neurotransmitter [4]; sometimes, it

serves as a neuroprotector and a free radical scavenger [35]; hence, the multifunctions of taurine lead to high utilization in the brain with alcoholic damage. Decreased taurine levels in the liver revealed a possible antioxidant function of taurine for preventing hepatocytes from oxidative stress with alcohol administration [4]. The results of our study showed a high correlation between alcohol ingestion and taurine metabolism. Taurine supplementation can replenish the decreased levels in the brain but not in liver, which might be explained by the liver being the commanding site for taurine, and the redistribution of taurine under ethanol ingestion contributes to its prior recovery in the CNS. In order to supplement the insufficient taurine levels in the brain, the hepatic levels remained lower to the end of the study.

Consequently, we concluded that subchronic high levels of ethanol consumption interrupted transmethylation and transsulfurations, thus imbalancing SCAA metabolism. An extra supplementation of taurine could possibly replenish the damage caused by alcoholism. The study also verifies the preventive and protective roles for development of functional nutrients with subchronic alcohol consumption. Further study is needed to clarify the actual mechanisms and actions of taurine to evaluate the possible utilization for alcoholic abstinence.

Acknowledgments

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