Taurine Supplementation Improves the Utilization of Sulfur-Containing Amino Acids in Consecutive Alcohol Administration Rats

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Abstract

The proposed study is to evaluate the effectiveness of taurine supplementation on the utilization of sulfur-containing amino acid (SCAA) in consecutive alcohol administration rats. Ninety Sprague Dawley rats (male and female 45 each) were consecutively treated with 20% alcohol water solution and taurine (2 g/kg BW taurine) for 4weeks. Food and water were available *ad libitum*. In the beginning, ten animals (M5, F5) were sacrificed and the biological lesions (blood, brain, and liver) for basal level of SCAAs and other biochemical parameters. The other rats were then sacrificed every week for following four weeks. In results, there is no difference on alcohol-water solution consumption. During the experiment, the plasma alcohol concentration increased during the study, however, taurine-treated animals showed the lower plasma level in week2. Furthermore, homocysteine level of plasma and liver significant elevated in week 2. The plasma SAM/SAH ratio also decreased in week1. On the other hand, the key cofactor of transsulfuration, Vitamin B6, significantly declined in plasma after a week of ethanol intervention whereas and increase was observed in brain tissue. Under the taurine supplementation, some recoveries of SCAA were shown significantly by delaying taurine depletion, increasing SAM/SAH ratio, and elevating plasma and brain level of vitamin B6 in week 2. No biochemical differences between genders were found. In conclusion, taurine supplementation could recover the brain and blood abnormal utilization of SCAA under consecutive alcohol administration in rats.

Keywords：taurine, sulfur-containing amino acids, alcohol, SAM/SAH, pyridoxal 5'-phosphate, homocysteine

Material & Methods

Experimental design

four-week old Sprague Dawley rats were housed in stainless cages under a humidity and temperature with 12hr light-dark cycle. After a week of acclimation, five animals were randomly sacrificed as baseline (B). The rest animals were divided into two groups (A, alcohol, and AT; alcohol +taurine groups) and given drinking water which contained 30% alcohol with or without taurine supplement (2 g/Kg BW). In each group, every five animals were sacrificed for the first, the second, and the forth week. Brain, liver, and blood samples were collected for further biochemical analysis.

Results

Table 1.The changes of SCAA amounts in liver during the experiment.

		Methionine	SAM	SAH	Cysteine	Taurine	Hypotaurine
		nmol/g tissue				μmol/g tissue	
B	Taurine	130.5±28.1	85.1±12.5	16.9±1.8	32.3±11.7	7.03±0.73	1.95±0.64
	Control	117.8±13.5	79.8±8.5	18.7±1.8	30.5±8.02	8.24±1.13	2.01±0.58
Wk1	Taurine	98.8±21.4	116.3±51.7*	23.9±1.8*	31.2±10.3	6.52±0.82	1.87±1.28
	Control	73.8±21.4	122.7±35.1*	37.8±2.8**	55.4±11.4	4.98±0.32**	1.97±0.46
Wk2	Taurine	98.0±19.9	137.8.8±41.4*	21.5±1.2*	72.0±21.3	5.52±0.95*	1.72±0.13
	Control	68.9±17.3*	143.8±22.2*	43.8±1.3**	45.1±6.2**	4.73±1.01*	2.31±0.79
Wk4	Taurine	75.8±13.4*	109.2±25.5*	10.7±2.0	91.5±9.6*	6.31±1.67	1.05±0.51*
	Control	59.7±8.32**	175.8±34.9*	49.8±5.0**	42.1±5.8**	4.23±0.15**	2.35±0.13*

Table 2. The changes of SCAA amount in brain during the experiment.

		Methionine	SAM	SAH	Cysteine	Taurine	Hyptaurine
		nmol/g tissue				μmol/g tissue	
B	Taurine	103.4±28.5	20.1±3.6	15.3±1.4	106.9±29.7	6.11±0.41	0.64±0.09
	Control	102.8±16.7	20.8±4.2	14.5±1.8	96.9±9.6	6.61±0.81	0.62±0.16
Wk1	Taurine	88.7±6.42	24.2±2.4	19.1±0.8*	94.8±10.4	5.32±1.03	0.58±0.17
	Control	82.2±12.7	25.1±2.2	19.3±1.9*	82.1±11.7*	4.07±0.73**	0.46±0.13
Wk2	Taurine	70.5±10.2*	28.2±3.1*	20.3±1.2*	90.1±8.3	4.98±0.36*	0.59±0.25
	Control	66.5±11.8*	27.8±2.3*	32.4±0.9**	73.9±9.6**	4.01±0.63*	0.37±0.21**
Wk4	Taurine	67.8±10.2*	30.1±3.2*	17.3±2.1	110.3±18.5	5.38±0.06	0.49±0.21
	Control	41.9±3.6**	28.8±2.1*	37.8±2.2**	70.7±9.2**	3.46±1.24**	0.30±0.09**

Table 3. Time changes of plasma SCAA concentration via taurine intervention.

		Homocystein	Methionine	Cysteine	Glutathinone	Taurine	Hypotaurine
		(nmol/L)			(μmol/L)		
B	Taurine	1.65±0.58	17.8±2.1	13.5±2.8	58.3±11.8	19.8±5.2	17.8±3.9
	Control	1.89±1.51	21.3±3.1	14.8±2.1	67.2±12.3	21.3±5.9	18.3±1.1
Wk1	Taurine	1.78±0.83	15.7±2.5	9.2±3.5*	43.2±11.5	20.8±5.2	20.7±9.2
	Control	1.71±0.73	15.8±3.6	10.4±1.37	45.2±9.2*	16.8±2.7*	19.5±3.7
Wk2	Taurine	2.17±0.53	18.8±1.3	11.5±2.1	65.7±15.1	18.5±5.5	18.2±3.0
	Control	2.51±1.07*	12.2±1.4**	10.9±1.3*	46.5±5.4	14.8±3.91*	18.5±2.8
Wk4	Taurine	2.79±0.81*	18.4±2.8	15.5±3.7	63.5±4.4*	15.1±1.92	19.1±2.1
	Control	3.85±0.81*	9.9±1.5**	7.2±1.5**	31.2±8.2**	9.1±1.13**	17.8±4.7

Figure 1.

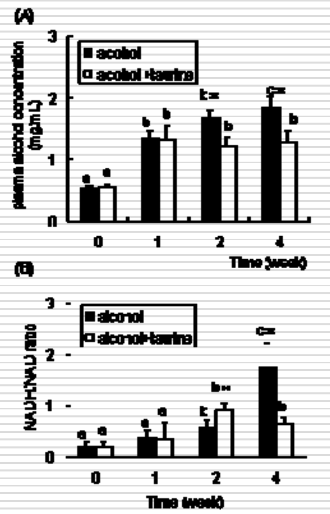

Figure 2.

Figure 3.

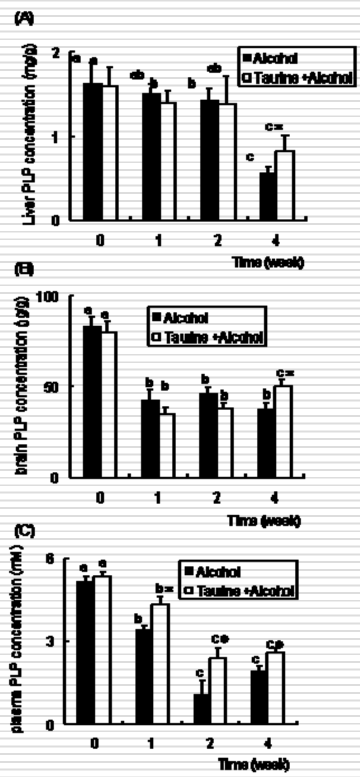

Figure 1.Effects of taurine on (A) plasma alcohol concentration and (B) hepatic NADH/ NAD ratio under alcohol exposure. The symbol "＊" represented statistically different from another group at the same time points, and different alphabetical letters indicated differences from time points within the group, $p < 0.05$.

Figure 2. Brain (A) and liver (B) SAM/SAH ratio in animals after four weeks of alcohol ingestion with or without taurine supplementation. The symbol "＊" represented statistically different from another group at the same time points, and different alphabetical letters indicated differences from time points within the group at $p < 0.05$.

Figure 3. The changes of pyridoxal - 5' phosphate in brain (A), liver (B), and plasma (C) during the experiment. The symbol "＊" represented statistically different from another group at the same time points, and different alphabetical letters indicated differences from time points within the group at $p < 0.05$.

Conclusion

The results provided some information for speculating how alcohol interrupted SCAA metabolism (that high alcohol drinking could lower methionine level thud interrupted transmethylation (Figure 4.). In other hand, decreased vitamin B6 level represented decreased transsulfuration by ethanol. Both of the results caused homocysteine retention. All the interferences might result the decrease of taurine. Furthermore, taurine supplementation could possibly recover the imbalance of transmethylation and transsulfuration thus replenish the level of taurine in tissues.

In conclusion, subchronic high level of ethanol consumption interrupted transmethylation and transsulfuration thus imbalanced SCAA metabolism. An extra supplementation of taurine could possibly replenish the damage brought by alcoholism. The study also verifies the preventive and protective role for development of functional nutrient on subchronic alcohol consumption. Further study is needed to clarify the actual mechanism and actions of taurine for evaluating the possible utilization on alcoholic abstinence.

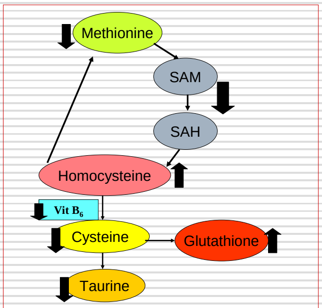

Figure 4. A speculated mechanism of alcohol drinking on SCAA metabolism.

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