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Efficacy Verification of Four Hangover Cure Products for Reducing Blood Alcohol and Acetaldehyde Concentrations in Sprague-Dawley Rats

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Recently, many hangover cure products containing natural ingredients have been made available in the market that are effective for alcohol-related liver damage or for improved liver function. However, the cure for of liver damage or medication for improved liver function are different from hangover cure. Therefore, the efficacy hangover cure products needs to be verified. In this study, we investigated and compared the ameliorating effect of four commercially available hangover cure products on acute ethanol-induced hangover in Sprague-Dawley rats. The four samples were labeled as C, M, R, and S. The efficacy of the samples was evaluated based on the serum concentration and area under the curve (AUC) of blood ethanol and acetaldehyde concentrations to quantitatively assess the hangover cure effect. Ethanol administration to the rats significantly raised the serum alcohol and acetaldehyde levels. The Cmax reduction rates of ethanol for the samples C, M, R, and S were 5.9%, 3.1%, 8.4%, and 11.7%, and the AUC were 8.9%, 2.2%, 12.1%, and 19.6%, respectively, whereas the Cmax reduction rates of acetaldehyde were 14.2%, 15.2%, 28.2%, and 35.0%, and the AUC were 21.6%, 7.5%, 22.4%, and 29.9%, respectively. In conclusion, all samples showed a tendency to relieve hangover in the order of S, R, C, and M in terms of the ethanol concentration, but only sample S showed a statistically significant decrease in both Cmax and AUC for ethanol and acetaldehyde. These results suggest that an objective method for verifying the efficacy of hangover cure products is lacking.

Key words : Acetaldehyde, acute alcohol-induced hangover animal model, alcohol, hangover, hangover cure product

Introduction

A hangover refers to symptoms such as headache, diarrhea, poor appetite, nausea, vomiting, chills, and cold sweats that occur after alcohol drinking. Physical symptoms include decreased cognitive, exercise ability, and hemodynamic and hormonal changes [23]. The causes of hangover are known as dehydration, toxicity of alcohol and alcohol metabolites, acetaldehyde, and nutrient deficiency caused by malabsorption [16, 18, 19]. Ethanol itself can cause hangover symptoms by directly affecting urine formation, blood glucose level, sleep patterns, etc [19]. Excessive alcohol consumption can directly damage the gastrointestinal wall and small intestine by causing inflammation; it can cause fatty liver by changing the metabolic state that appears in the liver and other organs, and also may result in hypoglycemia due to inhibition of glucose production as a result of the accumulation of lactic acid [3, 17, 20, 22]. Acetaldehyde is the main causative agent of hangover produced as a primary metabolite in the process of ethanol decomposition. When acetaldehyde is slowly decomposed in the body, the time remaining in the body and the concentration increase, accumulating fatigue substances such as lactic acid, causing hangover symptoms such as headache, vomiting, decreased blood pressure, and allergic reactions [4].

Many studies are currently being conducted on the effect of alcohol metabolism. Alcohol is oxidized into acetaldehyde by alcohol dehydrogenase (ADH) in the liver, and acetaldehyde is metabolized to acetic acid by acetaldehyde dehydrogenase (ALDH) and finally decomposed into carbon dioxide and water [2, 6]. Social and economic problems caused by hangovers are also emerging as more people experience hangover symptoms [10, 13, 21].

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It has been reported that about 75% of alcohol drinkers experience hangover symptoms more than once a year, and 15% experience hangover symptoms every month [8]. A hangover cure refers to a product that contains effective ingredients for protecting and recovering the liver and detoxifying alcohol and acetaldehyde accumulated in the liver, which helps relieve hangover symptoms after drinking [11].

As many people have recently shown interest in hangover products, natural materials that have the effect of improving hangovers are being developed. *Hovenia dulcis* fruit extract has been recognized as a functional ingredient that protects the liver from alcohol damage. Three of the four hangover cure products selected in this study also contain *Hovenia dulcis* fruit extract. The other contains curcuminm, a functional substance of fermented *curcuma longa* that is recognized for its beneficial effects on liver health. In addition, other ingredients that are known for effective on liver health such as yeast extract and milk thistle extract [1, 14] were also contained in the hangover cure product with main ingredients.

However, hangover cure is different from liver health or liver protection. Hangover cure products activate alcohol-metabolizing enzymes with a single intake to lower blood alcohol and acetaldehyde levels, but functional ingredients for liver focuse on the liver protection effect when taken for more than 3 months. In addition, there are no individually recognized materials for hanover cure or hanover relieving from KFDA. Therefore, in this study, we aimed to objectively confirm whether these hangover cure products are actually effective on lowering blood ethanol and acetaldehyde concentration in acute alcohol-induced hangover animal model.

We didn't use positive control because there is no approved positive control in Korea due to hangover cure products are not accepted as health functional food. According to the standard test method of the Korea Food & Drug Administration (KFDA), the hangover relief effect is quantitatively assessed by blood alcohol and acetaldehyde levels over time [9].

Materials and Methods

Chemial and reagents

In this study, prethanol A was purchased from Duksan Chemical Co., Ltd., and carboxymethylcellulose sodium (CMC) was obtained from Twinbio. Ethanol and acetaldehyde concentrations were measured by purchasing ethanol UV method (Roche Diag-nostics, Darmstadt, Germany) acetaldehyde UV method (Roche Diag-nostics, Darmstadt, Germany) kit. The samples used in this study were commercially available four hangover cure products, and the component information of each sample is shown in Table 1.

Experimental samples

Four hangover cure products used in this study are commercially available, and the single intake amount of each product was 3 g, 100 ml, 12 g, and 3 g in the order of sample C, M, R, and S. In order to find out the hangover-relieving effect according to one serving amount of each product intake, the administration dose of the rat was converted from the one serving amount of human based on a body surface area coefficient of rats and humans. This calculation method using a coefficient based on body surface area is from U.S. FDA guidance [5]. For example, the dose conversion factor from human to rat is 0.16. Therefore, the calculation formula is as follows; single intake amount for rat (mg/kg) = single

Table 1. Detail information of hangover cure products

Sample name	Main ingredient	Formulation	Single intake amount for human	*Converted single intake amount for rat
Sample C	*Hovenia dulcis* fruit combined extract, *Salix alba* extraction powder, *Laurus nobilis* extraction powder, curcuma longa extraction powder, barley grass extraction powder	Pilule	3 g	312.5 mg/kg
Sample M	*Hovenia dulcis* fruit extract, Korean pear extract, milk thistle extract	Liquid	100 ml (14.7 g)	1,527.3 mg/kg
Sample R	Curcumin pigment, Mango concentrate	Chew	12 g	1,250.0 mg/kg
Sample S	Yeast extract, diverse plants concentrate (*Hovenia dulcis* fruit, atractylodes, *Crataegus pinnatifida* fruit, *Pueraria lobata*), *Citrus unshiu* peel powder	Pilule	3 g	312.5 mg/kg

*single intake amount for rat (mg/kg) = single intake amount for human (mg)/ 0.16/ 60 kg (average human body weight)

intake amount for human (mg) / 0.16 / 60 kg (average human body weight). The 100 ml of liquid sample M was lyophilized, and we obtained 14.7 g of sample M in powder form. Other solid samples were ground prior to mixing into 0.5% CMC, which is a dispersant. The rat single intake amount of each sample was calculated, dissolved in 0.5% CMC, and 1.5 ml of the solution was administered per rat.

Experimental animals

In this experiment, 250 to 300 g of eight weeks old SD rats (Sprague-Dawley, male) were obtained from Raonbio Co., Ltd. (Yongin, Korea). They were bred in an animal breeding room that maintained temperature (22.5±0.4℃), humidity (50±20%) and a 12-hr lighting cycle, and water and feed were supplied without restrictions during the experiment. After adapting to the breeding environment for a week, the experiment was conducted. The experimental animals in this study were bred with the approval of the Institutional Animal Care and Use Committee of Kyung Hee University (KHUASP(SE)-20-023).

Experimental design

After acclimatizing the experimental animals for a week, 72 rats were divided into six groups (n=12) with the same weight average: a normal control group (Normal), ethanol administration group (EtOH), ethanol + sample C administration group (sample C), ethanol + sample M administration group (sample M), ethanol + sample R administration group (sample R), and ethanol + sample S administration group (sample S) (Table 2). All experimental groups were fasted for 18 hours before administration to prevent obstruction of ethanol absorption in the gastrointestinal tract due to feed intake, and then each sample was dissolved in 0.5% CMC for homogeneous absorption and ingestion. 0.5% CMC was administered independently for Noraml group and EtOH group. Edible alcohol (prethanol) was diluted to 50% concentration, and 3 g/kg body weight of alcohol was orally administered to all experimental groups except the Normal group. The amount of ethanol, 3 g/kg, is most commonly used dose in hangover studies [7, 12, 15, 24]. Alcohol administration was conducted 30 minutes after administering each sample. Blood was collected from tail vein at zero hour, one hour, three hours, five hours and eight hours after ethanol administration. Blood collected by time was placed in tubes and centrifuged at 2,259× g for 15 minutes at 4℃ to obtain serum.

Table 2. Experimental group design

Groups	Treatment
Normal	Saline + 0.5% CMC
EtOH	Ethanol + 0.5% CMC
Sample C	Ethanol + Sample C (312.5 mg/kg body weight)
Sample M	Ethanol + Sample M (1,527.3 mg/kg body weight)
Sample R	Ethanol + Sample R (1,250.0 mg/kg body weight)
Sample S	Ethanol + Sample S (312.5 mg/kg body weight)

Measurement of blood concentrations of ethanol and acetaldehyde

The ethanol and acetaldehyde concentration in serum collected over time was measured using a spectrophotometer (Jasco V-730, Jasco, Japan) according to instructions of assay kit (Roche Diag-nostics, Darmstadt, Germany). The ethanol and acetaldehyde concentration of each group was calculated using the formula presented in the kit.

Statistical analysis

The results of this experiment are shown by the mean ± standard deviation (the mean ± SD). For significance verification between each group, Statistical Packages for Social Science (SPSS) 25 software (SPSS Inc., Chicago, IL, USA) was used. Since the normalization was satisfied by the normalization distribution test, it was judged to be statistically significant when $p<0.05$ after analysis by the Turkey test using One way ANOVA.

Results and Discussion

Changes in blood ethanol concentration

This experiment was conducted to investigate the effects of commercially available four hangover cure products on blood ethanol concentration. Fig. 1. shows the result of measuring the blood ethanol concentration and the result of blood ethanol concentration-time AUC. The blood ethanol concentration of the EtOH group tended to decrease gradually after showing Cmax (6.19±0.52 g/l) at one hr after administration. The EtOH group showed a statistically significant difference compared to the Normal group at one hr, three hr, five hr, and eight hr (0.05±0.04 g/l, 0.11±0.08 g/l, 0.06±0.05 g/l, 0.05±0.03 g/l *v.s* 6.19±0.52 g/l, 6.12±0.45 g/l, 4.33±0.46 g/l, 2.79±0.81 g/l, $p<0.05$). The Cmax of each sample admin-

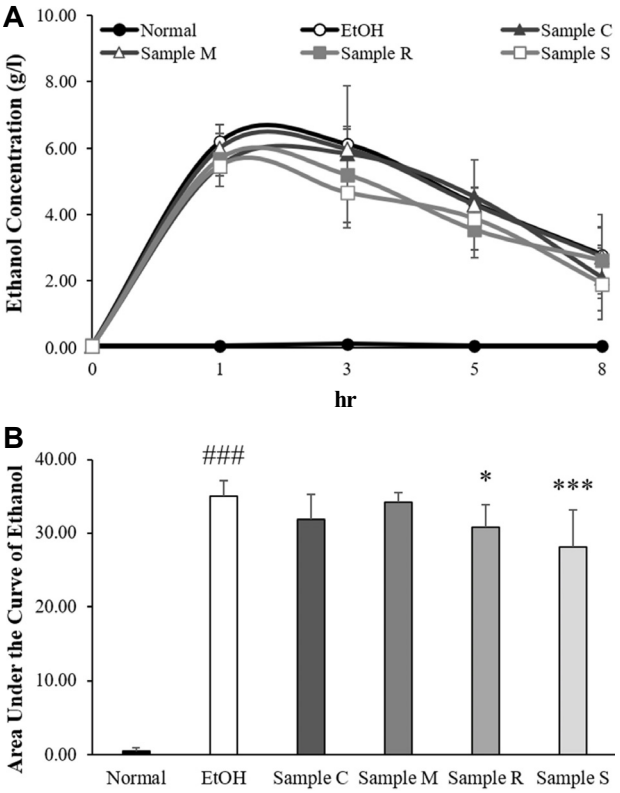

Fig. 1. Effect of four hangover cure products on ethanol level. (A) Etanol concentration. (B) Ethanol area under the curve. Normal (Saline + 0.5% CMC); EtOH (Ethanol + 0.5% CMC); Sample S (Ethanol + Sample S (312.5 mg/kg body weight)); Sample C (Ethanol + Sample C (312.5 mg/kg body weight)); Sample R (Ethanol + Sample R (1,250 mg/kg body weight)); Sample M (Ethanol + Sample M (1,527.3 mg/kg body weight)). All data were expressed as the mean ± SD (n=12). ### p<0.001 versus Normal, *p< 0.05 and ***p<0.001 versus EtOH.

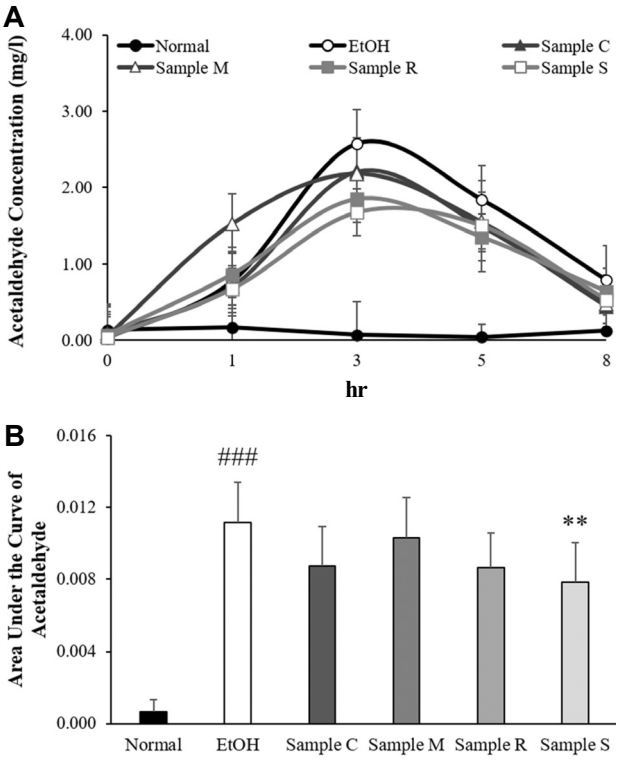

Fig. 2. Effect of four hangover cure products on acetaldehyde level. (A) Acetaldehyde concentration (B) Acetaldehyde area under the curve. Normal (Saline + 0.5% CMC); EtOH (Ethanol + 0.5% CMC); Sample S (Ethanol + Sample S (312.5 mg/kg body weight)); Sample C (Ethanol + Sample C (312.5 mg/kg body weight)); Sample R (Ethanol + Sample R (1,250 mg/kg body weight)); Sample M (Ethanol + Sample M (1,527.3 mg/kg body weight)). All data were expressed as the mean ± SD (n=12). ###p<0.001 versus Normal, **p<0.01 versus EtOH.

istration groups was reduced by 11.7%, 8.4%, 5.9%, and 3.1%, respectively in the order of sample S (5.47 ±0.62 g/l), sample R (5.67±0.31 g/l), sample C (5.83±0.69 g/l), and sample M (6.00±0.16 g/l) compared to the EtOH group (6.19±0.52 g/l). It was confirmed that only sample S (p<0.01) administration group showed statistically significant decrease compared to the EtOH group. Tmax was observed at one hour in sample M, R and S as in the EtOH group, and only sample C showed Tmax at three hours. The results of the blood ethanol Cmax and Tmax in each group are shown in Table 3. The AUC according to the blood ethanol concentration by time decreased by 19.6%, 12.1%, 8.9%, and 2.2% in the order of sample S, R, C, and M, respectively, and it was confirmed that sample S (35.03±2.16 v.s 28.15±5.07, p<0.001) and sample R (35.03±2.16 v.s 30.80±3.10, p<0.05) showed statistically significant decrease compared to the EtOH group. As a result of this experiment, when each

Table 3. Cmax and Tmax of etanol

	Normal	EtOH	Sample C	Sample M	Sample R	Sample S
Cmax (g/l)	0.11	6.19	5.83	6.00	5.67	5.47
Tmax (hr)	-	1	3	1	1	1

Normal (Saline + 0.5% CMC); EtOH (Ethanol + 0.5% CMC); Sample C (Ethanol + Sample C (312.5 mg/kg body weight)); Sample M (Ethanol + Sample M (1,527.3 mg/kg body weight)); Sample R (Ethanol + Sample R (1,250 mg/kg body weight)); Sample S (Ethanol + Sample S (312.5 mg/kg body weight)).

Table 4. Cmax and Tmax of acetaldehyde

	Normal	EtOH	Sample C	Sample M	Sample R	Sample S
Cmax (mg/l)	0.17	2.58	2.21	2.18	1.85	1.68
Tmax (hr)	-	3	3	3	3	3

Normal (Saline + 0.5% CMC); EtOH (Ethanol + 0.5% CMC); Sample C (Ethanol + Sample C (312.5 mg/kg body weight)); Sample M (Ethanol + Sample M (1,527.3 mg/kg body weight)); Sample R (Ethanol + Sample R (1,250 mg/kg body weight)); Sample S (Ethanol + Sample S (312.5 mg/kg body weight)).

hangover cure product was taken thirty minutes before ethanol intake, all samples lowered the blood ethanol Cmax and AUC, but only one sample out of the four samples showed a significant decrease in both Cmax and AUC.

Changes in blood acetaldehyde concentration

The results of measuring serum acetaldehyde concentrations, and the result of AUC according to the blood ethanol concentration by time are shown in Fig. 2. The serum acetaldehyde concentration in the EtOH group continuously increased up to three hours after alcohol administration and Cmax was 2.58±0.81 mg/l. The acetaldehyde concentration of EtOH group showed a statistically significant difference from the Normal group at one hour, three hours, five hours and eight hours (0.17±0.25 mg/l, 0.07±0.44 mg/l, 0.04±0.17 mg/l, 0.13±0.05 mg/l v.s 0.77±0.30 mg/l, 2.58±0.81 mg/l, 1.84±0.54 mg/l, 0.78±0.25 mg/l, $p<0.001$). The Cmax of each sample administration groups decreased by 35.0%, 28.2%, 15.2%, and 14.2%, respectively in the order of sample S (1.68 ±0.44 mg/l), sample R (1.85±0.46 mg/l), sample M (2.18±0.64 mg/l), and sample C (2.21±0.51 mg/l) compared to the EtOH group (2.58±0.81 mg/l). It was confirmed that sample S ($p<0.01$) and sample R ($p<0.05$) administration groups showed statistically significant decrease compared to the EtOH group. Tmax was observed at three hours in all samples the same as in the EtOH group. The results of the blood acetaldehyde Cmax and Tmax of each group are shown in Table 4. The AUC according to the blood acetaldehyde concentration by time decreased by 29.9%, 22.4%, 21.6%, and 7.5% in the order of sample S, R, C, and M, respectively, and it was confirmed that sample S (0.011±0.002 v.s 0.008± 0.002, $p<0.01$) administration groups showed statistically significant decrease compared to the EtOH group. Acetaldehyde is a toxic metabolite of alcohol and is known to cause headaches after drinking alcohol. As a result of this experiment, when each hangover cure product was taken thirty minutes before ethanol intake, all samples lowered the blood acetaldehyde Cmax but, as same as ethanol result, only sample S showed a significant decrease in both Cmax and AUC.

Korea's alcohol consumption is among the best in the world. Accordingly, a lot of hangover cure products are on the market, and the market is growing nowadays. However, there is no objective verification of its efficacy. In this study, we attempted to compare commercially available four hangover cure products by confirming the ability to reduce the blood ethanol concentration and the blood acetaldehyde concentration, which are the causative materials of hangover. As a result, we figured out all four samples showed a tendency to relieve hangover in the order of sample S > R > C > M. However, there was only one product had a statistically significant hangover-relief efficacy in both ethanol and acetaldehyde.

It is judged that the difference in efficacy of the products is due to the difference in the content of ingredients that promote alcohol metabolism. The difference in the efficacy of the three hangover cure products containing the *Hovenia dulcis* is also thought to be due to the different *Hovenia dulcis* extract content for each product and the additional effect of sub-ingredients. Although this experiment was conducted in an animal model rather than clinical trial, these experiment results suggested the need for objective efficacy verification of hangouver cure product.

The Conflict of Interest Statement

The authors declare that they have no conflicts of interest with the contents of this article.

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초록 : 국내 시판 숙취해소제 4종의 혈중 알코올 및 아세탈데히드 농도 감소 효능 비교

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최근 알코올성 간손상이나 간건강에 효과가 있는 천연물 원료를 포함하는 숙취해소제들이 시판되고 있다. 하지만 간손상이나 간건강에 대한 기능성은 숙취해소와는 다르기 때문에 숙취해소제들의 숙취해소 효능 검증이 필요하다. 본 연구는 국내의 4가지 숙취해소제의 효능을 알코올에 의한 숙취유도 흰쥐 모델에서 비교 조사하고자 하였다. 4가지 샘플들은 각각 C, M, R, S로 명명되었다. 동물실험에서 숙취 완화 효능은 혈중 알코올 및 아세탈데히드 농도로 평가되기 때문에 각 샘플들의 효과를 혈중 알코올과 아세탈데히드의 농도와 AUC (Area Under the Curve)로 평가하였다. 알코올 투여는 유의적으로 흰쥐의 혈중 알코올과 아세탈데히드 농도를 증가시켰다. 샘플 C, M, R, S순으로 에탄올의 Cmax와 AUC 감소율은 각각 5.9%, 3.1%, 8.4%, 11.7%와 8.9%, 2.2%, 12.1%, 19.6%였다. 아세탈데히드의 경우, 같은 샘플 순서로 Cmax는 14.2%, 15.2%, 28.2%, 35.0% 감소하였으며, AUC는 21.6%, 7.5%, 22.4%, 29.9%의 감소율을 보였다. 결론적으로, 샘플 S, R, C, M순으로 4개의 샘플 모두 EtOH군에 대비하여 숙취가 해소되는 경향을 보였으나, 샘플 S만이 에탄올과 아세탈데히드의 혈중 농도와 AUC 모두에서 통계적으로 유의적인 감소율을 보였다. 따라서 이러한 실험결과는 시중에 판매되는 숙취해소제의 객관적인 효능 검증이 부족하다는 것을 시사한다.