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Red ginseng relieves the effects of alcohol consumption and hangover symptoms in healthy men: a randomized crossover study

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Heavy drinking causes hangover symptoms, because the action of alcohol dehydrogenase forms acetaldehyde, which is metabolized by acetaldehyde dehydrogenase into acetate. Red ginseng shows positive effects on alcohol metabolism in animal studies. We investigated the effects of red ginseng on relieving alcohol and hangover symptoms in 25 healthy men in a randomized crossover study. At each visit (0, 1, and 2 weeks), the subjects drank 100 mL whiskey (40% alcohol) and either 100 mL water or 100 mL of a 0.321 mg mL^{-1} red ginseng anti-hangover drink (RGD). We took blood samples periodically until 240 min after alcohol consumption, and we investigated the blood profiles, alcohol levels, and acetaldehyde levels. We also measured anthropometric parameters, expiratory air-alcohol levels, and hangover symptoms. The plasma alcohol concentrations within the RGD group were significantly lower than those within the placebo group after 30 min ($p = 0.002$), 45 min ($p = 0.016$), and 60 min ($p = 0.009$); the areas under the response curves revealed a positive effect of RGD ($p = 0.051$). Furthermore, the expiratory alcohol concentration was significantly lower after 30 min ($p = 0.005$) and 60 min ($p = 0.065$), and the areas under the response curves ($p = 0.058$) likewise revealed a positive effect of RGD. The plasma acetaldehyde level was significantly elevated at 120 min ($p = 0.020$), but the areas under the response curves showed a similar trend ($p = 0.054$). While the plasma acetaldehyde concentration slightly increased, the RGD showed positive effects on hangover symptoms. Considering the reduction of plasma alcohol levels, expiratory concentrations, and hangover severity, we conclude that red ginseng relieves the symptoms of alcohol hangover.

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Introduction

Recently, societal diversification and economic growth have caused people to drink more than in the past, increasing the incidences of alcoholism and hangovers.[1] Worldwide, persons more than 15 years of age consumed 6.13 liters of alcohol on average in 2005 (WHO, 2011). Drinking cost ranged between 1.3% and 3.3% of GDP in high-income countries and two middle-income countries in 2007.[2] Chronic, excessive alcohol consumption can cause many diseases, including cancer, diabetes mellitus, neuropsychiatric disorders, cardiovascular disease, cirrhosis of the liver, unintentional injuries, and others.[2] Acute heavy drinking can cause hangover symptoms because of acetaldehyde, formed by the action of alcohol dehydrogenase (ADH), which is metabolized by acetaldehyde dehydrogenase (ALDH) into acetate.[3] Acetaldehyde can bind to biologically important proteins; high levels can cause severe side effects, including fatigue, nausea, vomiting, dizziness, thirst, headache, muscle pain, increased systolic blood pressure, rapid heartbeat, tremor, sweating, and others.[3]

The increasing rates of alcohol consumption and severe hangover symptoms are sufficient reasons to make a product to relieve hangover symptoms. A few products, like coffee, tea, fluids, vitamin B6, and painkillers, are recommended for relieving hangover symptoms.[4] Even ingredients that have some positive effects on acute alcohol toxicity are limited in terms of their effects against hangover symptoms.[5,6] Red ginseng has long been used as a medicinal plant.[7,8] In Korea, red ginseng has been used as a traditional medicinal plant for thousands of years.[9,10] Red ginseng extract in particular has short-term effects on ethanol metabolism in rats[9] and helps to reduce blood ethanol concentration in intoxicated rats[9] and dogs.[10] Many studies of red ginseng have been conducted in animals, but few have been conducted in humans. Furthermore, there are few

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studies on prospective treatments for hangover symptoms. Therefore, we investigated the effects of red ginseng on improving alcohol metabolism and reducing hangover symptoms in healthy men.

Materials and methods

Study subjects

For this study, 25 subjects were recruited from among volunteers who responded to advertisements for a nutrition study conducted by the Clinical Nutrigenetics/Nutrigenomics Laboratory at Yonsei University in 2011. The trial participants were healthy men from 25 to 49 years of age with a body mass index (BMI) ranging from 18.5 to 29.9 kg m^{-2}. The exclusion criteria were a history of cardiovascular disease, diabetes mellitus, cancer, kidney, liver, or thyroid disease, allergy to red ginseng; or any other serious life-threatening illness requiring regular medical treatment.

Study design

The study was conducted with a randomized crossover design over a 2-week period, and written informed consent was obtained from all subjects (Fig. 1). Volunteers were asked to participate in three visits (0, 1, and 2 weeks) and to abstain from drinking alcohol for 3 days and fast for 12 h prior to each visit. During testing, the 25 subjects were divided into placebo and test groups. At the first visit, the subjects consumed 100 mL blended scotch whiskey containing 40% alcohol (Passport, William Longmore Co., Ltd) and one piece of cheese. The subjects then drank either 100 mL water (placebo) or 100 mL red ginseng anti-hangover drink (RGD) as a test within 5 min of consuming the alcohol. The RGD was made by a water-extract method and contained 0.321 mg mL^{-1} red ginseng extract (Korean Ginseng Corporation, Korea). Blood samples were taken 0, 30, 45, 60, 90, 120, 180, and 240 min after the subjects consumed the alcohol and the RGD or placebo. At the second visit, the subjects followed the same protocol, and the placebo and RGD treatments were switched between the two groups. At the third visit, the subjects brought their survey papers, which they received during the first and second visits, and they completed the hangover symptom survey again while in a normal physiological state. The Institutional Review Board of Yonsei University (2012–21) approved our study protocol.

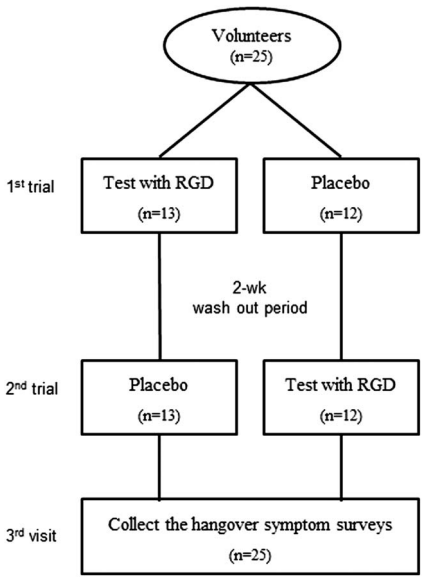

Fig. 1 The study protocol: a randomized crossover study was performed during a 2-week period. Twenty-five healthy volunteers were recruited.

Anthropometric parameters, blood pressure, and blood and urine collection

The body weight and height of the subjects without clothes and shoes were measured in the morning. The BMI was calculated as body weight in kilograms divided by height in square meters (kg m^{-2}). Body-fat percentages and lean body mass (LBM) were measured with a TBF-105 body-fat analyzer (Tanita, Tokyo, Japan). Waist circumference was measured at the umbilical level with the subjects standing after normal expiration; hip girth was measured at the widest part of the hip; and the waist-and-hip ratio (WHR) was calculated. After a 20 min rest, blood pressure (BP) was measured in the left arm of seated subjects using an automatic blood-pressure monitor (TM-2654, A&D, Tokyo, Japan). After a 12 h fasting period, the subjects consumed 100 mL whiskey and either water or the RGD. Venous blood specimens were collected 0, 30, 45, 60, 90, 120, 180, and 240 min after alcohol consumption in plain and EDTA-treated tubes and then centrifuged and stored at −70 °C until analysis. Urine was collected in polyethylene bottles containing 1% butylated hydroxytoluene after a 12 h fast. The tubes were immediately covered with aluminum foil and stored at −70 °C until analysis.

Serum lipid profile

Fasting serum concentrations of total cholesterol and triacylglycerol were measured using an Auto Chemistry Analyzer Express Plus (Chiron Diagnostics Co., MA, USA). After the precipitation of serum chylomicrons, we measured the low-density lipoprotein (LDL), very low-density lipoprotein, and high-density lipoprotein (HDL) cholesterols in the supernatants using an enzymatic method. LDL cholesterol was estimated directly for subjects with a serum triacylglycerol concentration greater than or equal to 400 mg dL^{-1} and indirectly using the Friedewald formula for subjects with a serum triacylglycerol concentration less than 400 mg dL^{-1}.

Circulating levels of white blood cells, red blood cells, hemoglobin, hematocrit, and platelets

The hematocrit, hemoglobin, and numbers of white blood cells, red blood cells, and platelets were measured using an automated hematology analyzer (COULTER LH 750, Beckman Coulter, USA). The lytic reagent used for the complete blood-count parameters was also used for the whole-blood and leukocyte counts.

Creatinine, BUN, AST, and ALT concentrations

Serum creatinine was measured by the Jaffe reaction method using a Hitachi 7600-110 analyzer (Hitachi, Japan), and blood urea nitrogen was measured using the urease glutamate dehydrogenase method. Serum glutamic-oxaloacetic transaminase (GOT) and glutamic pyruvic transaminase (GPT) levels were measured by a kinetic-UV method based on the recommendations of the International Federation of Clinical Chemistry using commercially available kits and a Hitachi 7180 Auto analyzer (Hitachi Ltd., Tokyo, Japan).

Daily food intake and total energy expenditure

Dietary intake was assessed with a 24 h recall method and semi-quantitative food-frequency questionnaire. Dietary energy values and nutrient content were calculated using the Computer Aided Nutritional analysis program (CAN-pro 2.0, Korean Nutrition Society, Seoul, Korea). Total energy expenditure (kcal d^{-1}) was calculated from activity patterns, including basal metabolic rate and physical activity, over 24 h. The basal metabolic rate for each subject was calculated using the Harris–Benedict equation.

Plasma alcohol, acetaldehyde concentrations, and alcohol levels of expiratory air

We used the collected plasma for alcohol and acetaldehyde concentration measurements. We measured alcohol levels using an enzymatic assay with the COBAS Integra ethanol gen.2 test (Roche, USA) and a COBAS Integra 800 analyzer (Roche, USA). Acetaldehyde was detected by a UV method with an acetaldehyde kit (Boehringer Mannheim/R-biopharm, Germany) and a U-3010 UV-V spectrophotometer (Hitachi, Japan) according to the manufacturer's protocols. Expiratory air was checked 0, 30, 60, 90, 120, 150, 180, 210, and 240 min after alcohol consumption, with alcohol concentrations measured by an SD-400 expiratory alcohol measuring machine (Korea Ginseng Corporation, Korea).

Survey of hangover symptoms

We used adapted questionnaires from previous hangover-assessment tools.[11,12] The survey tool consisted of 15 questions addressing headache, nausea, vomiting, fatigue, concentration, thirst or dehydration, light sensitivity, sleeping difficulty, excessive sweating, anxiety, feelings of depression, trembling or shaking, dizziness, stomachache, and memory loss. The scores ranged from 0 (symptoms absent) to 4 (extremely severe symptoms) and covered 5 stages. The sum score for each of the symptoms was considered to be a symptom index. The survey was administered to participants during their third visit. At the first and second visit, we explained the hangover symptom survey papers and let the volunteers complete them as homework 24 h after drinking the alcohol and RGD or placebo. At the third visit, the subjects turned in the survey papers (RGD and placebo) and completed a survey of the hangover symptoms that they experienced under normal conditions (*i.e.*, after a typical instance of alcohol consumption).

Statistical methods

Statistical analyses were performed with SPSS version 18 for Windows (Statistical Package for the Social Science, SPSS Inc., Chicago, IL, USA). Each variable was examined for a normal distribution, and variables that were significantly skewed underwent a log transformation. Paired *t*-tests and Wilcoxon signed-rank tests were used to evaluate the effects of the RGD and the placebo on daily food intake, total energy expenditure, plasma alcohol levels, acetaldehyde concentrations, and expiratory alcohol concentrations. A one-way analysis of variance with least-significant differences was used to evaluate the differences between the groups. Areas under the curve (AUC) for plasma alcohol, expiratory alcohol, and plasma acetaldehyde levels were calculated after subtraction of the baseline value from each subsequent measurement using the trapezoidal method.[13] The results were expressed as mean ± standard error of the mean (SEM), and $p < 0.05$ was considered statistically significant.

Results

General characteristics, daily total energy expenditure, and food intake of the participants

Table 1 shows the general characteristics of the 25 subjects. The mean age, height, weight, BMI, percent body fat, WHR, LBM, systolic BP, diastolic BP, triglyceride, total cholesterol, LDL cholesterol, and HDL cholesterol were all in the normal range. The hematologic examination revealed that the levels of white blood cells, red blood cells, hemoglobin, hematocrit, platelets, creatinine, blood urea nitrogen, GOT, and GPT were also within

Table 1 Characteristics of the study participants[a]

Identity	Score ($n = 25$)
Age (years)	28.1 ± 0.60
Height (cm)	175.0 ± 0.97
Weight (kg)	77.5 ± 2.66
Body mass index (kg m^{-2})	25.2 ± 0.75
Body fat (%)	22.0 ± 0.91
Waist hip ratio	0.86 ± 0.01
Lean body mass (kg)	59.9 ± 1.43
Blood pressure	
Systolic BP (mmHg)	118.2 ± 2.37
Diastolic BP (mmHg)	70.4 ± 2.60
Triglyceride (mg dL^{-1})	123.5 ± 14.2
Total-cholesterol (mg dL^{-1})	180.9 ± 6.79
LDL-cholesterol (mg dL^{-1})	106.6 ± 6.35
HDL-cholesterol (mg dL^{-1})	49.6 ± 1.88
White blood cell (10^3 μL^{-1})	6.24 ± 0.32
Red blood cell (10^3 μL^{-1})	4.80 ± 0.06
Hemoglobin (g dL^{-1})	14.7 ± 0.19
Hematocrit (%)	45.8 ± 0.62
Platelet ($\times 10^3$ mm^{-3})	224.9 ± 8.85
Creatinine (mg dL^{-1})	1.11 ± 0.03
Blood urea nitrogen (mg dL^{-1})	13.8 ± 0.65
Glutamic-oxaloacetic transaminase (IU L^{-1})	24.8 ± 1.22
Glutamic pyruvic transaminase (IU L^{-1})	28.9 ± 3.82

[a] Data are means ± SEM.

Table 2 Daily total energy expenditure and food intake[a]

	Baseline ($n = 25$)	Follow-up ($n = 25$)	p-value
Total energy expenditure (kcal)	2812.9 ± 74.0	2791.6 ± 65.0	0.376
Total energy intake (kcal d^{-1})	2626.7 ± 50.0	2639.1 ± 51.4	0.459
Carbohydrate (%)	62.5 ± 0.21	62.3 ± 0.21	0.721
Protein (%)	16.6 ± 0.20	16.3 ± 0.14	0.375
Fat (%)	22.3 ± 0.17	22.4 ± 0.14	0.706
Cholesterol (mg)	263.7 ± 17.5	218.9 ± 18.9	0.109

[a] Data are means ± SEM. p-values are from nonparametric paired t-tests.

the normal range (Table 1). The total energy expenditure, total energy intake, and percentages of carbohydrate, protein, fat, and cholesterol intake were measured in the first (baseline) and second (follow-up) visits, and the mean values were not significantly different between the two visits (Table 2).

Plasma alcohol concentration

The plasma alcohol concentrations within the RGD group were significantly lower than those within the placebo group 30 min (41.8 ± 2.98 mg dL^{-1} and 55.4 ± 3.50 mg dL^{-1}, respectively; $p = 0.002$), 45 min (42.3 ± 2.67 mg dL^{-1} and 50.3 ± 2.81 mg dL^{-1}, respectively; $p = 0.019$), and 60 min (39.7 ± 2.78 mg dL^{-1} and 46.8 ± 2.86 mg dL^{-1}, respectively; $p = 0.009$) after alcohol consumption. There were no significant differences between the two groups 0 min ($p = 0.166$), 90 min ($p = 0.106$), 120 min ($p = 0.252$), 180 min ($p = 0.287$), and 240 min ($p = 0.607$) after alcohol consumption. The AUC for plasma alcohol was smaller for the RGD group (105.1 ± 8.03 mg dL^{-1} × h) than for the placebo group (121.7 ± 7.84 mg dL^{-1} × h), although the difference was only marginally significant ($p = 0.051$) (Fig. 2A).

Expiratory alcohol concentration

The expiratory alcohol concentrations were significantly lower within the RGD group than within the placebo group 30 min (0.04 ± 0.00 mg dL^{-1} and 0.05 ± 0.00 mg dL^{-1}, respectively; $p = 0.005$) and 60 min (0.03 ± 0.00 mg dL^{-1} and 0.04 ± 0.00 mg dL^{-1}, respectively; $p = 0.065$) after alcohol consumption. There were no significant differences between the two groups 0 min ($p = 1.000$), 90 min ($p = 0.353$), 120 min ($p = 0.198$), 150 min ($p = 0.321$), 180 min ($p = 0.573$), 210 min ($p = 0.688$), and 240 min ($p = 0.285$) after alcohol consumption. The AUC for expiratory alcohol was smaller for the RGD group (0.07 ± 0.01% × h) than for the placebo group (0.08 ± 0.01% × h) ($p = 0.058$; Fig. 2B).

Plasma acetaldehyde concentration

There were no significant differences in plasma acetaldehyde concentrations between the placebo group and the RGD group 0, 30, 45, 60, 90, and 240 min after alcohol consumption. The plasma acetaldehyde concentrations were significantly higher in the RGD group than in the placebo group (0.15 ± 0.02 mg dL^{-1} and 0.11 ± 0.02 mg dL^{-1}, respectively; $p = 0.020$) 120 min after alcohol consumption. These concentrations were similar to those 180 min after alcohol consumption (0.15 ± 0.02 mg dL^{-1} for the RGD group and 0.11 ± 0.02 mg dL^{-1} for the placebo group; $p = 0.065$). The AUC for plasma acetaldehyde was larger for the RGD group than for the placebo group (0.54 ± 0.07 mg dL^{-1} and 0.43 ± 0.06 mg dL^{-1}, respectively; $p = 0.054$) (Fig. 2C).

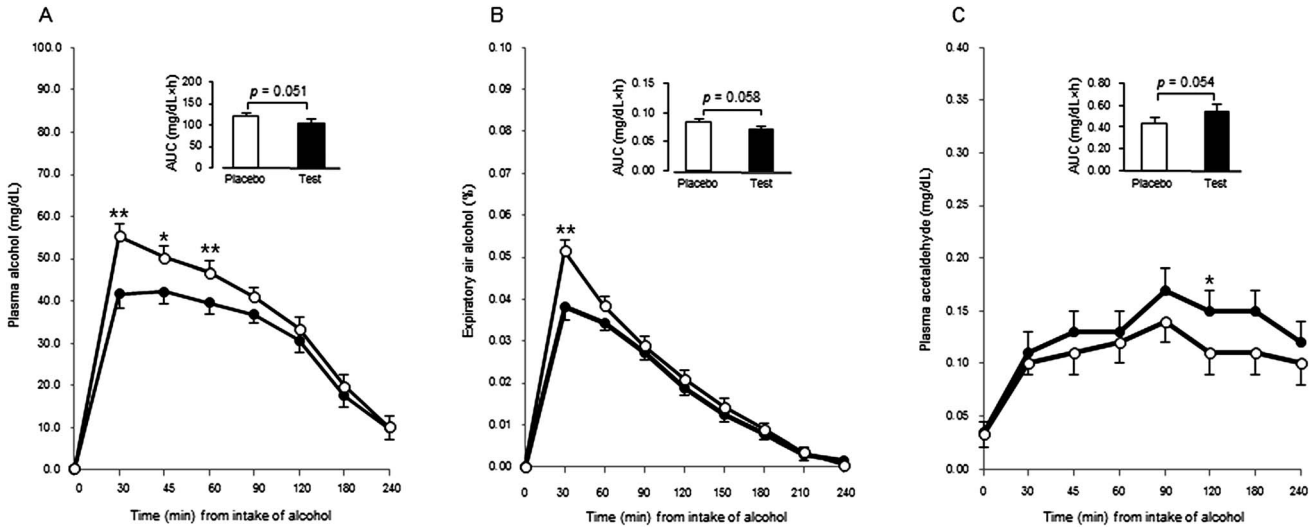

Fig. 2 Alcohol and acetaldehyde levels over 240 min following alcohol intake in placebo (○) and test (●) with red ginseng groups. (A) Alcohol levels in plasma; (B) alcohol levels in expiratory air; (C) acetaldehyde levels in plasma. Values are expressed as means ± SEM. p-values are from nonparametric paired t-tests. * $p < 0.05$, ** $p < 0.01$.

Table 3 Survey of hangover symptoms[a]

	Control ($n = 25$)	Placebo ($n = 25$)	RGD ($n = 25$)
Headache	1.32 ± 0.24	1.08 ± 0.22	1.04 ± 0.20
Nausea	1.00 ± 0.16	0.88 ± 0.18	0.56 ± 0.17
Vomiting	0.76 ± 0.21	0.68 ± 0.17	0.36 ± 0.18
Fatigue	1.88 ± 0.23[a]	1.68 ± 0.22[ab]	1.28 ± 0.17[b]
Concentration	1.48 ± 0.23[ab]	1.56 ± 0.22[a]	0.96 ± 0.18[b]
Thirst or dehydration	2.12 ± 0.20[a]	1.92 ± 0.22[a]	1.32 ± 0.16[b]
Light sensitivity	0.48 ± 0.15	0.52 ± 0.17	0.40 ± 0.14
Sleeping difficulty	0.16 ± 0.07	0.28 ± 0.12	0.04 ± 0.04
Excessive sweating	0.48 ± 0.20	0.52 ± 0.18	0.44 ± 0.19
Anxiety	0.08 ± 0.06	0.16 ± 0.09	0.16 ± 0.09
Feelings of depression	0.00 ± 0.00	0.16 ± 0.09	0.08 ± 0.08
Trembling or shaking	0.44 ± 0.14	0.28 ± 0.11	0.28 ± 0.12
Dizziness	1.20 ± 0.00	1.12 ± 0.20	0.76 ± 0.18
Stomachache	1.12 ± 0.27[a]	0.64 ± 0.18[ab]	0.40 ± 0.17[b]
Memory loss	0.48 ± 0.16[ab]	0.64 ± 0.19[a]	0.08 ± 0.06[b]
Sum of symptoms	13.8 ± 1.75[a]	12.7 ± 1.64[ab]	8.40 ± 1.27[b]

[a] Score range: 0 (symptoms absent), 1 (mild symptoms), 2 (moderate symptoms), 3 (severe symptoms), 4 (Extremely severe symptoms). Data are means ± SEM. Tested by one-way analysis of variance. Differences are significant if $p < 0.05$.

Survey of hangover symptoms

The severity of hangover symptoms under normal conditions (control) and within the placebo and RGD groups during the study is summarized in Table 3. Regarding the 15 hangover symptoms, the RGD group reported feeling less tired than usual, having fewer stomachaches, and feeling less thirsty or dehydrated than under normal conditions or compared with the placebo group. The RGD group also experienced less difficulty concentrating and less memory loss than the placebo group. Red ginseng did not significantly improve the other 10 symptoms; however, none of the symptoms, except for feelings of anxiety and depression, were worse for the RGD group than they were under normal conditions or compared with the placebo group. Overall, the symptom index for the RGD group (8.40 ± 1.27) was significantly lower than those for the control conditions (13.8 ± 1.75) and the placebo group (12.7 ± 1.64).

Discussion

This randomized crossover, placebo-controlled study demonstrates that, compared with subjects who consumed alcohol and placebo, subjects who drank alcohol and RGD had significantly lower plasma-alcohol concentrations, especially 30 min, 45 min, and 60 min after drinking. In a similar study of mice that were administered red ginseng and ethanol, the red ginseng significantly reduced the plasma alcohol concentration in the early stages of a 280 min test period,[9] and it also significantly reduced the expiratory alcohol concentration after 30 min. Even if there were no significant differences between the placebo and test groups in terms of the other parameters, all of the RGD groups showed lower amounts of expiratory alcohol than the placebo groups. Shin previously found that red ginseng treatment decreased the blood alcohol concentration in mice and increased the liver ADH activity in rats.[10] In addition, the results of the previous studies show that red ginseng increases ADH activity in the early stages of alcohol metabolism, indicating that acetaldehyde is produced rapidly in the early stages following alcohol consumption.[9,10] In our study, alcohol turned into acetaldehyde effectively, especially after 120 min in the RGD group, but the AUC for acetaldehyde was not significantly different between placebo and test groups. The acetaldehyde detection range was too small, and an extreme value of one subject can affect estimates of whole acetaldehyde concentration because of the small number of participants. In a similar clinical study comparing plant extracts and water, the experimental group showed significantly elevated blood acetaldehyde levels 1 h after drinking and experienced a relieving effect of the plant extracts on hangover severity.[14] In our study, even if the acute acetaldehyde concentration was slightly higher in the RGD group, the subjects had substantially reduced hangover symptoms. They experienced fewer stomachaches, less thirst and dehydration, less difficulty concentrating, and less memory loss than the normal hangover conditions of the placebo. These symptoms are generally observed during an alcohol hangover,[11,12] and the sum of the scores for all the questions was significantly lower ($p = 0.040$) in the RGD group, indicating that the RGD had a positive, long-term effect rather than a short-term effect on acetaldehyde metabolism. Some studies report a significant correlation between blood acetaldehyde concentration and hangover severity, but most convincing is the significant relationship between immune factors and hangover severity.[15] That means that the level of acetaldehyde is not a unique factor for hangover symptoms, and we can think about the antioxidant effects of red ginseng. Red ginseng is well known as an antioxidant, suggesting that it could help remove liver-harming oxidative substances caused by alcohol consumption[16] and that its antioxidant effects stimulate ADH and ALDH.[17] We hypothesize that the antioxidant effects of red ginseng improve alcohol metabolism and relieve hangover symptoms in the long term.

Genetic variation in many of the alcohol-metabolizing genes contributes to differences in alcohol intake and hangover symptoms.[18] Alcohol is oxidized to acetaldehyde by the ADH enzymes, especially ADH1B, and acetaldehyde is oxidized further into acetate by the ALDH enzymes, mainly ALDH2.[19] Genetic polymorphisms for those enzymes affect alcohol metabolism and susceptibility to alcoholism.[20,21] Additionally, both ADH and ALDH must be analyzed to confirm such genetic variation. ADH1B*2, an ADH isoform, is common among Asians but rare among Europeans.[22] An ALDH2 heterotype is positively associated with hangover symptoms in Japanese persons, suggesting that acetaldehyde is linked to the development of hangover symptoms.[23] The presence of ALDH2*2 is associated with slower alcohol metabolism, whereas the presence of ADH2*2 and ALDH2*1 is correlated with slightly faster alcohol metabolism.[24] Acetaldehyde accumulates after alcohol consumption, because ALDH2 is inactivated in individuals with the ALDH2*2 allele, which is common in Asian populations.[18] Our results came from a crossover study, so they are limited by the absence of the consideration of genetic factors. For a better evaluation of alcohol metabolism, future studies should include a genetic analysis of the ADH and ALDH genes.

Despite our efforts, this paper has a few limitations. The subjects consumed water as a placebo, and even if the water and the RGD were served in the same dark bottle containing 100 mL aqueous solution, the subjects may have known what they drank, because the taste and smell of the RGD were different from those of the water. To reduce the placebo effect on the hangover symptom survey, we asked the subjects to finish their survey 24 h after drinking to obtain a relative check of the reduction between the RGD and placebo states. It is better, however, to check the hangover severity in a time-wise fashion, *e.g.*, before drinking, and 2 h, 4 h, and 14 h after drinking. Also, the method of checking should be changed from a self-administered survey to a survey conducted with a researcher on the phone to be sure that the survey is conducted at suitable times. Furthermore, we measured plasma and expiratory alcohol and acetaldehyde levels during 4 h without directly measuring ADH and ALDH activities and genotypes. For a better confirmation, it is necessary to measure levels of alcohol, acetaldehyde, ADH, and ALDH until they become undetectable. Moreover, the small sample size of our study may not be conducive to identifying weak associations because of low statistical power.

In conclusion, we confirmed that treatment with red ginseng significantly reduced plasma alcohol levels 30 min, 45 min, and 60 min after alcohol consumption. Red ginseng also reduced expiratory alcohol concentrations 30 min after alcohol consumption in the short term, and it relieved a variety of hangover symptoms in the long term. Our results suggest that red ginseng offers significant relief for individuals who are suffering from the effects of alcohol consumption.

Conflict of interest

There is no potential conflict of interest.

Declaration of interests

None of the authors have any conflicts of interest in relation to the materials presented in this paper.

Abbreviations

RGD	Red ginseng anti hangover drink
ADH	Alcohol dehydrogenase
ALDH	Acetaldehyde dehydrogenase
LBM	Lean body mass
WHR	Waist-and-hip ratio
BP	Blood pressure
LDL	Low-density lipoprotein
HDL	High-density lipoprotein
GOT	Glutamic-oxaloacetic transaminase
GPT	Glutamic pyruvic transaminase
AUC	Areas under the curve
SEM	Standard error of the mean

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