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Acute reduction in inferior vena cava diameter following alcohol consumption: a substudy of a pilot cluster-randomized trial

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Article

Keywords: Alcohol consumption, inferior vena cava, intravascular volume, hemodynamics, ultrasonography, vital signs

Posted Date: November 20th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11609675/v1

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Additional Declarations: Competing interest reported. Hisashi Yoshimoto has received research funding from Asahi Breweries, Ltd., Kirin Breweries, Ltd., Sanwa Shurui Co., Ltd., Mizkan Holdings Co., Ltd., and CureApp, Inc. He has also received consulting fees from Otsuka Pharmaceutical Co., Ltd. and honoraria from Asahi Breweries, Ltd., Otsuka Pharmaceutical Co., Ltd., Kirin Breweries, Ltd., Sanwa Shurui Co., Ltd., and Sawai Pharmaceutical Co., Ltd.

Abstract

Background

Acute alcohol consumption induces cardiovascular and diuretic effects; however, its influence on intravascular volume distribution remains poorly understood. Inferior vena cava (IVC) ultrasonography is a non-invasive method for assessing intravascular volume status, but changes in IVC morphology following alcohol consumption have not previously been investigated. This study evaluated acute changes in IVC measurements and vital signs after alcohol consumption.

Methods

This prespecified physiological substudy analyzed 59 healthy adult social drinkers enrolled in a pilot cluster-randomized trial. Maximum IVC diameter and respiratory collapsibility were assessed immediately before and after a standardized 2-hour ad libitum drinking session. Heart rate, blood pressure, breath alcohol concentration, and alcohol intake were serially measured throughout the session. Exploratory univariable linear regression analyses were performed to identify factors associated with post-drinking IVC diameter and changes in IVC diameter.

Results

Maximum IVC diameter significantly decreased after alcohol consumption, from 14.4 ± 3.4 mm to 10.8 ± 3.0 mm (paired t test, $P < 0.001$). The proportion of participants with respiratory collapsibility ≥ 50% increased from 79.3% to 91.4%, although this difference was not statistically significant (McNemar's test, $P = 0.096$). During alcohol consumption, heart rate and shock index gradually increased, whereas systolic and diastolic blood pressure decreased. In exploratory analyses, pre-drinking maximum IVC diameter was the strongest predictor of post-drinking IVC measurements and was inversely associated with the change in IVC diameter from baseline (β = −0.653; 95% CI, − 0.862 to − 0.444; $P < 0.001$).

Conclusions

Acute alcohol consumption was associated with a significant reduction in maximum IVC diameter, suggesting alterations in intravascular volume distribution beyond previously recognized cardiovascular responses. The magnitude of this reduction was strongly associated with pre-drinking IVC diameter, highlighting the potential importance of baseline intravascular volume status in determining the physiological response to alcohol.

INTRODUCTION

Alcohol is one of the most widely consumed psychoactive substances worldwide.[1] Alcohol consumption has long been incorporated into social and cultural practices; however, it also produces a variety of physiological effects that may compromise cardiovascular stability and increase the risk of alcohol-related adverse events.[2] A better understanding of these physiological responses may contribute to the development of evidence-based strategies for safer alcohol consumption.

Among the acute physiological effects of alcohol, cardiovascular responses have been the subject of considerable investigation.[3] Shortly after alcohol consumption, heart rate increases whereas blood pressure transiently decreases.[4] Alcohol also stimulates catecholamine release, indicating activation of neurohumoral mechanisms involved in cardiovascular regulation.[5] Furthermore, ambulatory electrocardiographic studies have demonstrated that acute alcohol intake can precipitate cardiac arrhythmias,[6] further supporting its immediate influence on cardiovascular physiology.

Alcohol also alters body fluid homeostasis. Ethanol suppresses vasopressin secretion, resulting in transient diuresis and increased urine output.[7] Experimental studies have suggested that this diuretic effect becomes more pronounced as alcohol concentration increases.[8,9] However, alcohol-induced diuresis is attenuated under dehydrated conditions, suggesting that alcohol consumption does not inevitably produce dehydration.[10] Instead, changes in intravascular volume and body fluid distribution are likely to be more complex than can be explained by urine output alone.

Despite these findings, little is known about how acute alcohol consumption affects intravascular volume distribution. Previous studies have evaluated hydration indirectly using urine volume or laboratory measurements, without directly assessing circulating volume.[11,12] Ultrasonographic assessment of the inferior vena cava (IVC) is now widely used as a non-invasive bedside indicator of intravascular volume status.[13] However, to the best of our knowledge, no previous study has examined IVC morphology before and after alcohol consumption. Although some studies performed echocardiography during alcohol intake, these focused exclusively on cardiac function and did not evaluate hydration status or intravascular volume.[4,14]

We recently conducted a cluster-randomized controlled trial involving 59 participants to determine whether pre-drinking education modified drinking behavior during alcohol consumption. During the trial, serial vital signs were recorded throughout alcohol intake, and ultrasonographic assessment of the IVC was performed before and after drinking.[13] These prospectively collected physiological data provided a unique opportunity to characterize the acute cardiovascular effects of alcohol beyond conventional vital signs. In addition, participants were genotyped for alcohol-metabolizing enzymes, allowing us to explore whether acute physiological responses differed according to genetic background. Previous studies have suggested that polymorphisms in alcohol-metabolizing enzymes, particularly ALDH2, influence cardiovascular responses to alcohol consumption.[15–19]

Therefore, the present study aimed to comprehensively evaluate acute alcohol-induced hemodynamic changes by combining serial vital sign measurements with ultrasonographic assessment of the IVC. By directly assessing changes in intravascular volume distribution, we sought to provide new physiological insights into the acute effects of alcohol and generate evidence that may ultimately contribute to the development of safer and more evidence-based drinking practices.

METHODS

This study was conducted as a prespecified physiological substudy of a single-center pilot cluster randomized trial performed in Japan.[20] The protocol was approved by the Institutional Ethics Committee for Human Genome and Genetic Research, University of Tsukuba (approval numbers G316 and G316-1), and all procedures were conducted in accordance with the Declaration of Helsinki. The parent trial was prospectively registered with the University Hospital Medical Information Network Clinical Trials Registry (UMIN-CTR; UMIN000060685; registered 17 February 2026). All participants provided written informed consent before participation.

The present analysis was conducted using data collected from participants enrolled in the parent pilot cluster randomized trial. The study enrolled healthy adult social drinkers aged 20 years or older who met predefined eligibility criteria, including previous experience with heavy alcohol consumption and absence of alcohol flushing. Participants attended a standardized 2-hour ad libitum drinking session under continuous medical supervision. During the session, participants were free to consume alcoholic and non-alcoholic beverages of their choice together with buffet-style snacks, including pizza, fried foods, and salad. Following the 2-hour drinking session, participants entered a 1-hour recovery session during which consumption of alcohol and food was not permitted, whereas non-alcoholic beverages could be consumed freely. Although the parent trial was designed to evaluate the effect of brief pre-drinking guidance on drinking behavior (including “drinking slowly during the first 30 min” and “eating while drinking”), the objective of the present substudy was to investigate physiological responses to alcohol consumption irrespective of the behavioral intervention. Therefore, participants from both trial arms were analyzed together, and intervention allocation was not considered in the present analyses. Details of participant eligibility, randomization procedures, intervention procedures, and safety monitoring have been reported in the primary trial manuscript.[20]

Measurements

Measurement time points are summarized in Fig. 1. IVC ultrasonography was performed immediately before the drinking session and immediately after completion of the 2-hour drinking period. The maximum IVC diameter (mm) was measured, and respiratory collapsibility was classified dichotomously according to whether the IVC diameter decreased by ≥ 50% during inspiration.[21]

Vital signs, including heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP), were measured immediately before drinking and every 30 minutes thereafter until 1 hour after

completion of drinking session. The shock index, calculated as heart rate (beats/min [bpm]) divided by systolic blood pressure (mmHg), was derived as a simple surrogate marker of hemodynamic status, with values > 1 generally considered indicative of hemodynamic instability.[22] Breath alcohol concentration was measured every 30 minutes from 30 minutes after the start of drinking until 1 hour after completion of the drinking session and was not measured before alcohol consumption.

Alcoholic and non-alcoholic beverage remnants, including partially consumed drinks, were collected at the end of each 30-minute interval during the drinking session. The volume consumed during each interval was used to calculate cumulative alcohol intake in grams of pure alcohol and cumulative fluid intake, assuming that 1 g of beverage corresponded to 1 mL.

Saliva samples were collected before the drinking session for genotyping of alcohol dehydrogenase 1B (ADH1B) and aldehyde dehydrogenase 2 (ALDH2).[16–18] Genotyping procedures were identical to those used in the parent trial.

Statistical analyses

The primary analyses evaluated changes in IVC measurements before and after alcohol consumption (0 min vs 120 min). Changes in maximum IVC diameter were analyzed using paired t-tests, whereas changes in the proportion of participants with respiratory collapsibility ≥ 50% were evaluated using McNemar's test.

Because this was an exploratory physiological study intended for hypothesis generation rather than hypothesis confirmation, no formal sample size calculation was performed for these analyses. Accordingly, all statistical tests should be interpreted as descriptive, and *P* values are presented as measures of compatibility with the observed data rather than confirmatory evidence. For descriptive purposes, $P < 0.05$ was used as a nominal reference threshold, without adjustment for multiple comparisons. This exploratory interpretation applies to the primary as well as all other analyses.

As secondary analyses, univariable linear regression models were fitted to identify factors associated with post-drinking maximum IVC diameter and with the change in maximum IVC diameter from baseline. Candidate explanatory variables included demographic characteristics, vital signs, breath alcohol concentration, alcohol intake, fluid intake, pre-drinking maximum IVC diameter, and genetic polymorphisms.

Additional exploratory analyses were performed using baseline-adjusted linear regression models, in which post-drinking IVC diameter was regressed on each candidate variable after adjustment for pre-drinking maximum IVC diameter.

Statistical analyses were performed using R version 4.4.0 (R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

Of the 63 participants who were randomized in the parent pilot cluster-randomized trial, four withdrew before study procedures, leaving 59 participants included in this sub analysis. Participant recruitment, eligibility assessment, randomization, and study procedures have been described in detail in the primary trial report.[20] The mean age was 28.6 years (standard deviation [SD] 11.8 years; median 23.0 years [interquartile range (IQR), 21.0–30.5]), and 62.7% of participants were male (37/59). Additional participant characteristics are summarized in Table 1. Detailed participant-level data, anonymized to prevent identification of individual participants, are provided in the **Supplementary Material 1**.

Table 1
Characteristics of participants (n = 59)

Characteristics	Value
Age, year	
Mean ± SD	28.6 ± 11.8
Median [IQR]	23.0 [21.0–30.5]
Male, n (%)	37 (62.7)
Body weight, kg	
Mean ± SD	64.7 ± 14.1
Median [IQR]	63.1 [55.4–70.4]
Usual drinking frequency, n (%)	
1–3 days per month	20 (33.9)
1–2 days per week	23 (39.0)
3–4 days per week	9 (15.3)
5–7 days per week	4 (6.8)
Every day	3 (5.1)
Usual amount of alcohol consumed per drinking occasion, n (%)	
< 20 g	2 (3.4)
20–40 g	19 (32.2)
40–60 g	9 (15.3)
60–80 g	13 (22.0)
80–100 g	7 (11.9)
100–120 g	5 (8.5)
120–140 g	4 (6.8)
ADH1B genotype, n (%)	
*2/*2	31 (52.5)
*1/*2	24 (40.7)
*1/*1	4 (6.8)

ADH1B, alcohol dehydrogenase 1B; ALDH2, aldehyde dehydrogenase 2; CI, confidence interval; IQR, interquartile range; IVC, inferior vena cava; SD, standard deviation.

Characteristics	Value
ALDH2 genotype, n (%)	
*1/*1	47 (79.7)
*1/*2	12 (20.3)

ADH1B, alcohol dehydrogenase 1B; ALDH2, aldehyde dehydrogenase 2; CI, confidence interval; IQR, interquartile range; IVC, inferior vena cava; SD, standard deviation.

Figure 2 summarizes the mean time courses of vital signs, breath alcohol concentration, and body weight-adjusted alcohol and fluid intake during the study. Pulse rate increased progressively after the start of drinking, from 81.1 bpm (95% confidence interval [CI], 77.6–84.6 bpm) at baseline to a peak of 95.5 bpm (95% CI, 91.3–99.8 bpm) at the end of the 2-hour drinking session (120 min), followed by a gradual decline during the recovery period. Shock index showed a similar pattern, increasing from 0.66 (95% CI, 0.63–0.69) at baseline to 0.82 (95% CI, 0.77–0.86) at 120 min. Breath alcohol concentration also increased throughout the drinking session, reaching a maximum of 0.25 mg/L (95% CI, 0.21–0.28 mg/L) at 120 min. In contrast, both systolic and diastolic blood pressure gradually decreased during and after alcohol consumption. Mean systolic blood pressure declined from 124.3 mmHg (95% CI, 120.6–128.1 mmHg) at baseline to 118.3 mmHg (95% CI, 114.7–122.0 mmHg) at 120 min and remained slightly reduced at 150 min (117.7 mmHg; 95% CI, 114.6–120.9 mmHg) and 180 min (118.8 mmHg; 95% CI, 115.7–121.9 mmHg). Similarly, mean diastolic blood pressure decreased from 81.4 mmHg (95% CI, 78.2–84.6 mmHg) at baseline to 71.4 mmHg (95% CI, 68.1–74.7 mmHg) at 120 min and remained relatively low at 150 min (67.9mmHg; 95% CI, 64.8–71.1 mmHg) and 180 min (71.0 mmHg; 67.9–74.1 mmHg). Body weight-adjusted alcohol intake was highest during the first 30-minute interval (0.30 g/kg; 95% CI, 0.27–0.33 g/kg) and decreased thereafter. Body weight-adjusted fluid intake showed a broadly similar pattern, being highest during the first 30-minute interval (7.53 mL/kg; 95% CI, 6.91–8.16 mL/kg) and decreasing during the subsequent two intervals (second interval, 4.76 mL/kg; 95% CI, 4.13–5.39 mL/kg; third interval, 4.53 mL/kg; 95% CI, 3.87–5.19 mL/kg), followed by a marked increase during the final 30-minute interval (6.65 mL/kg; 95% CI, 5.86–7.45 mL/kg). Individual trajectories of vital signs, breath alcohol concentration, and alcohol intake are presented in **Supplementary Materials 2–5**.

The primary analyses evaluated IVC measurements immediately before and immediately after the 2-hour drinking session (120 min). The mean maximum IVC diameter decreased from 14.4 mm before drinking (SD, 3.4 mm; median, 15.0 mm [IQR, 13.0–16.5 mm]) to 10.8 mm after drinking (SD, 3.0 mm; median, 11.0 mm [IQR, 9.0–13.0 mm]), representing a significant reduction in maximum IVC diameter (paired t test, $P < 0.001$; Fig. 3). One participant was excluded from the paired analysis of respiratory collapsibility because of missing data. Among the remaining 58 participants, the proportion with respiratory collapsibility ≥ 50% increased from 79.3% (46/58) before drinking to 91.4% (53/58) after drinking; however, this change was not statistically significant (McNemar's test, $P = 0.096$). Detailed individual changes in maximum IVC diameter before and after alcohol consumption are presented in **Supplemental Material 6**.

Univariable linear regression analyses were performed to identify factors associated with the maximum IVC diameter immediately after alcohol consumption and with changes in maximum IVC diameter following alcohol consumption (**Supplemental Materials 7 and 8**). Among all candidate exploratory variables, pre-drinking maximum IVC diameter showed the strongest association with both post-drinking maximum IVC diameter and the change in IVC diameter from baseline. The regression coefficient between pre- and post-drinking maximum IVC diameter was 0.347 (95% CI, 0.138 to 0.556; $P = 0.002$). The regression slope was substantially less than 1, consistent with the overall reduction in maximum IVC diameter observed after alcohol consumption. Pre-drinking maximum IVC diameter was inversely associated with the change in IVC diameter from baseline (regression coefficient $\beta = -0.653$; 95% CI, −0.862 to − 0.444; $P < 0.001$), indicating that participants with larger baseline IVC diameters tended to experience greater reductions after alcohol consumption. Conversely, participants with the smallest pre-drinking maximum IVC diameters showed little or no reduction in IVC diameter after alcohol consumption. These regression relationships are illustrated in Fig. 4. Apart from pre-drinking maximum IVC diameter, baseline pulse rate was positively associated with the change in maximum IVC diameter ($\beta = 0.085$, 95% CI, 0.021–0.149; $P = 0.012$), whereas baseline and 90-minute systolic blood pressure showed weak inverse associations with post-drinking maximum IVC diameter ($\beta = -0.053$, $P = 0.050$ and $\beta = -0.064$, $P = 0.028$, respectively). No clear associations were observed for age, sex, body weight, alcohol intake, breath alcohol concentration, or ADH1B and ALDH2 genotypes.

To account for the strong association between pre- and post-drinking maximum IVC diameters, additional baseline-adjusted linear regression models were fitted, in which post-drinking maximum IVC diameter was regressed on each candidate variable after adjustment for the pre-drinking maximum IVC diameter. After adjustment, none of the candidate variables remained clearly associated with post-drinking maximum IVC diameter (**Supplemental Material 9**).

DISCUSSION

In this prespecified physiological substudy of a pilot cluster-randomized trial, we evaluated acute hemodynamic responses to alcohol consumption in 59 healthy social drinkers. The principal finding was a significant reduction in the maximum IVC diameter immediately after a 2-hour drinking session. Furthermore, pre-drinking maximum IVC diameter was the strongest determinant of both post-drinking maximum IVC diameter and the change in IVC diameter following alcohol consumption. Participants with larger pre-drinking maximum IVC diameters tended to show greater reductions after alcohol consumption, whereas those with smaller baseline diameters showed little or no reduction. These findings suggest that the acute reduction in IVC diameter following alcohol consumption may depend not only on the physiological effects of alcohol itself but also on an individual's baseline intravascular volume status.

Previous studies investigating alcohol-induced changes in body fluid homeostasis have primarily evaluated urine output[10,23] or laboratory findings,[5,23–25] rather than directly assessing intravascular volume. Although alcohol is known to promote diuresis through suppression of vasopressin secretion

and other neurohumoral mechanisms,[23–25] changes in urine volume do not necessarily reflect alterations in circulating blood volume or fluid distribution. To our knowledge, this is the first study to evaluate IVC morphology before and after alcohol consumption.[13] Our findings suggest that acute alcohol intake may induce measurable changes in intravascular volume distribution beyond those inferred from conventional assessments of hydration.[9,11,12,26] This observation provides a novel physiological perspective on the acute effects of alcohol.

The present findings help bridge the gap between previous studies reporting alcohol-induced diuresis under normal hydration conditions and studies suggesting that low-alcohol beverages may facilitate rehydration under dehydrated conditions.[9–12,26] By directly measuring IVC diameter before and after alcohol consumption, our study suggests that the hemodynamic response to alcohol varies according to baseline hydration status. Previous experimental studies have shown that alcohol-induced diuresis is attenuated in dehydrated individuals, indicating that homeostatic mechanisms may limit further fluid loss under conditions of reduced body water.[10] Our findings provide complementary evidence from the perspective of intravascular volume distribution, showing that participants with smaller baseline IVC diameters exhibited little or no further reduction after alcohol consumption. In patients with chronic kidney disease and chronic heart failure, a larger baseline IVC diameter was associated with a greater subsequent reduction in body weight during treatment with tolvaptan, a vasopressin V2 receptor antagonist that promotes water diuresis.[27] Although the clinical settings and interventions differ substantially from those in the present study, this finding may be physiologically relevant because both tolvaptan and acute alcohol consumption promote water diuresis through modulation of the vasopressin pathway, with tolvaptan blocking renal V2 receptors and alcohol suppressing vasopressin release.[7] This raises the possibility that a similar baseline-dependent homeostatic response may contribute to the present findings.

Although the applicability of this concept to markedly dehydrated individuals remains speculative, our findings may also help explain previous reports suggesting that low concentrations of alcohol may be as effective as water in facilitating recovery from dehydration under certain conditions.[9,11,12,26] Specifically, if the relationship observed in Fig. 4B extends to individuals with substantially lower baseline IVC diameters, the regression line raises the possibility that alcohol-induced changes in IVC diameter may not only become negligible but could even reverse direction. This hypothesis should be interpreted cautiously because it is based on extrapolation beyond the observed range and requires confirmation in studies including dehydrated individuals. Nevertheless, it provides a potential unifying hypothesis that may reconcile previous apparently conflicting findings regarding the effects of alcohol on hydration status.

The observed changes in vital signs were generally consistent with previous interventional studies. Pulse rate progressively increased during alcohol consumption, whereas both systolic and diastolic blood pressure gradually decreased. Consequently, the shock index also showed a gradual increase throughout the drinking session. These findings are consistent with sympathetic activation and peripheral

vasodilation following acute alcohol intake.[14,17] The concurrent reduction in IVC diameter may indicate a decrease in central blood volume, suggesting that relative intravascular volume depletion may have played a role in the observed increase in pulse rate, possibly through compensatory sympathetic activation. Because a shock index > 1 is generally considered to indicate hemodynamic compromise, the frequent occurrence of values exceeding this threshold in our study may have potential clinical implications.[22] Although these transient physiological changes were well tolerated in the healthy participants included in this study, they could be of greater concern in individuals with impaired cardiovascular reserve or other comorbidities, warranting further investigation.[6,8]

As an exploratory analysis, we investigated factors associated with post-drinking IVC diameter and changes in IVC diameter. Although alcohol consumption was associated with a significant reduction in maximum IVC diameter in the primary analysis, few candidate variables were clearly associated with post-drinking IVC diameter or its change. Apart from the strong association with pre-drinking maximum IVC diameter, baseline pulse rate was positively associated with the change in maximum IVC diameter, whereas baseline and 90-minute systolic blood pressure showed weak inverse associations with post-drinking IVC diameter. However, these associations disappeared after adjustment for pre-drinking maximum IVC diameter, suggesting that they may primarily reflect baseline intravascular volume status rather than independently influencing the response to alcohol. Similarly, neither alcohol intake, fluid intake, breath alcohol concentration, nor polymorphisms in the alcohol-metabolizing enzymes ADH1B and ALDH2 showed clear associations with post-drinking IVC diameter or its change.

These findings should be interpreted cautiously because the relatively small sample size and substantial variability in alcohol intake under the ad libitum drinking setting may have limited our ability to identify weaker predictors. Nevertheless, the results suggest that alcohol-induced changes in intravascular volume are largely determined by baseline physiological status and are not readily explained by conventional vital signs, alcohol intake, fluid intake or alcohol-metabolizing genotypes alone. Acute alcohol consumption induces multiple physiological responses, including catecholamine release, vasodilation, and diuresis, and the balance among these mechanisms is likely to vary considerably between individuals.

This study has several limitations. First, although IVC ultrasonography provides a non-invasive estimate of intravascular volume status, we did not measure urine output, body weight changes, or overall fluid balance. Therefore, we could not evaluate whole-body fluid balance or determine whether the observed IVC changes reflected true volume loss or redistribution of body fluids. Second, participants consumed alcohol and food ad libitum, and dietary intake was not standardized. Third, because this analysis combined participants from both arms of the parent trial, the potential influence of the behavioral intervention was not considered. Fourth, the sample size was relatively small, limiting statistical power for exploratory analyses. Finally, IVC measurements were obtained only immediately before and immediately after the drinking session; therefore, the duration and recovery of these hemodynamic changes remain unknown.

In conclusion, acute alcohol consumption was associated with a significant reduction in maximum IVC diameter in healthy social drinkers. The observed relationship between baseline and post-drinking IVC measurements further suggests that the intravascular volume response to alcohol depends on baseline physiological status. Future studies including individuals with varying hydration status are needed to confirm this hypothesis.

Declarations

Conflict of interest

Hisashi Yoshimoto has received research funding from Asahi Breweries, Ltd., Kirin Breweries, Ltd., Sanwa Shurui Co., Ltd., Mizkan Holdings Co., Ltd., and CureApp, Inc. He has also received consulting fees from Otsuka Pharmaceutical Co., Ltd. and honoraria from Asahi Breweries, Ltd., Otsuka Pharmaceutical Co., Ltd., Kirin Breweries, Ltd., Sanwa Shurui Co., Ltd., and Sawai Pharmaceutical Co., Ltd.

Yuko Kusano and Takayo Kawabata are employees of Kirin Brewery Company, Limited. Takafumi Fukuda, Yuta Komano, Kentaro Umeda, and Masamori Iwase are employees of Kirin Holdings Company Limited.

The other authors declare no additional competing interests.

Funding

This study was funded by Kirin Brewery Company, Limited.

Acknowledgments

We thank the Center for Innovative Medicine and Engineering (CIME), University of Tsukuba Hospital, for institutional support and permission to conduct the study. This research was supported by AMED under Grant Number JP256f0137008.

Data sharing statement

De-identified participant-level data and analytic code are not publicly posted because the dataset includes small social-group sessions and genotype information. Data and code are available from the corresponding author on reasonable request, subject to ethics approval, applicable institutional requirements, and a data-sharing agreement.

Use of generative AI in manuscript preparation

During preparation of this manuscript, the authors used ChatGPT (OpenAI) to assist with Japanese-to-English translation and English-language editing. All outputs were reviewed and edited by the authors, who take full responsibility for the content of the manuscript.

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Figures

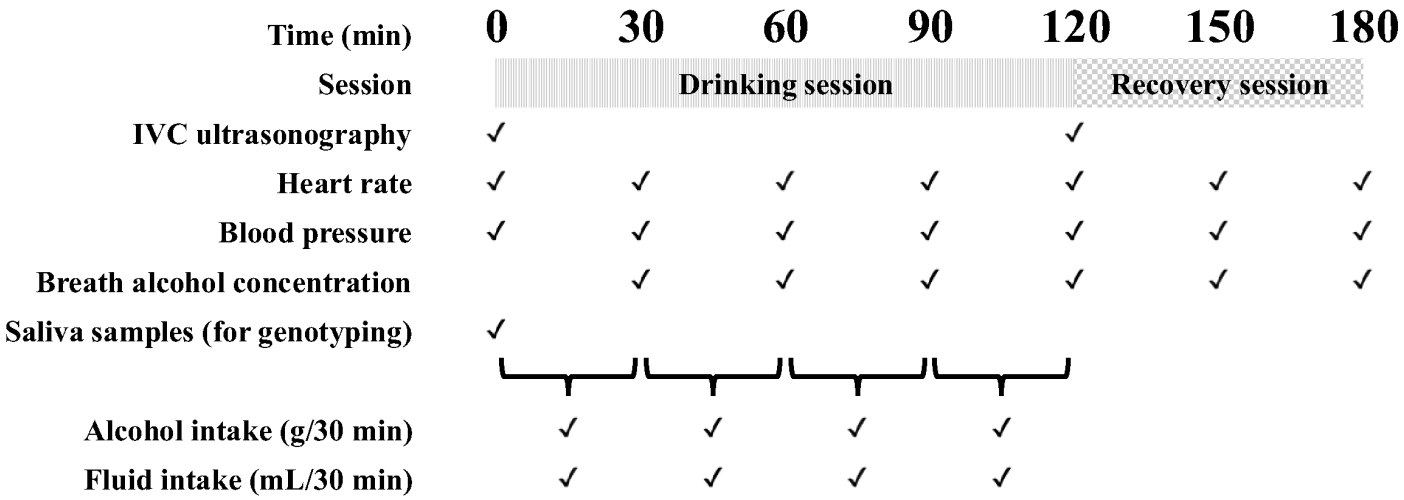


Figure 1

Schedule of study measurements.

IVC, inferior vena cava.

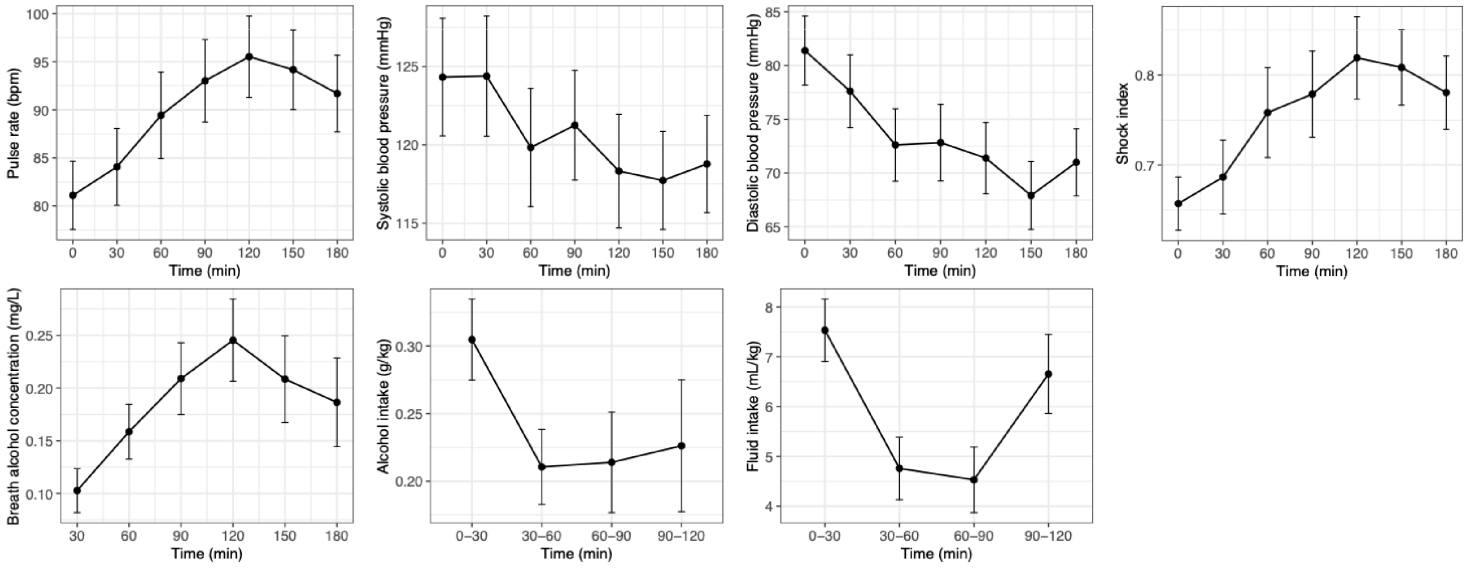


Figure 2

Time courses of vital signs during and after alcohol consumption.

Values are presented as means with 95% confidence intervals.

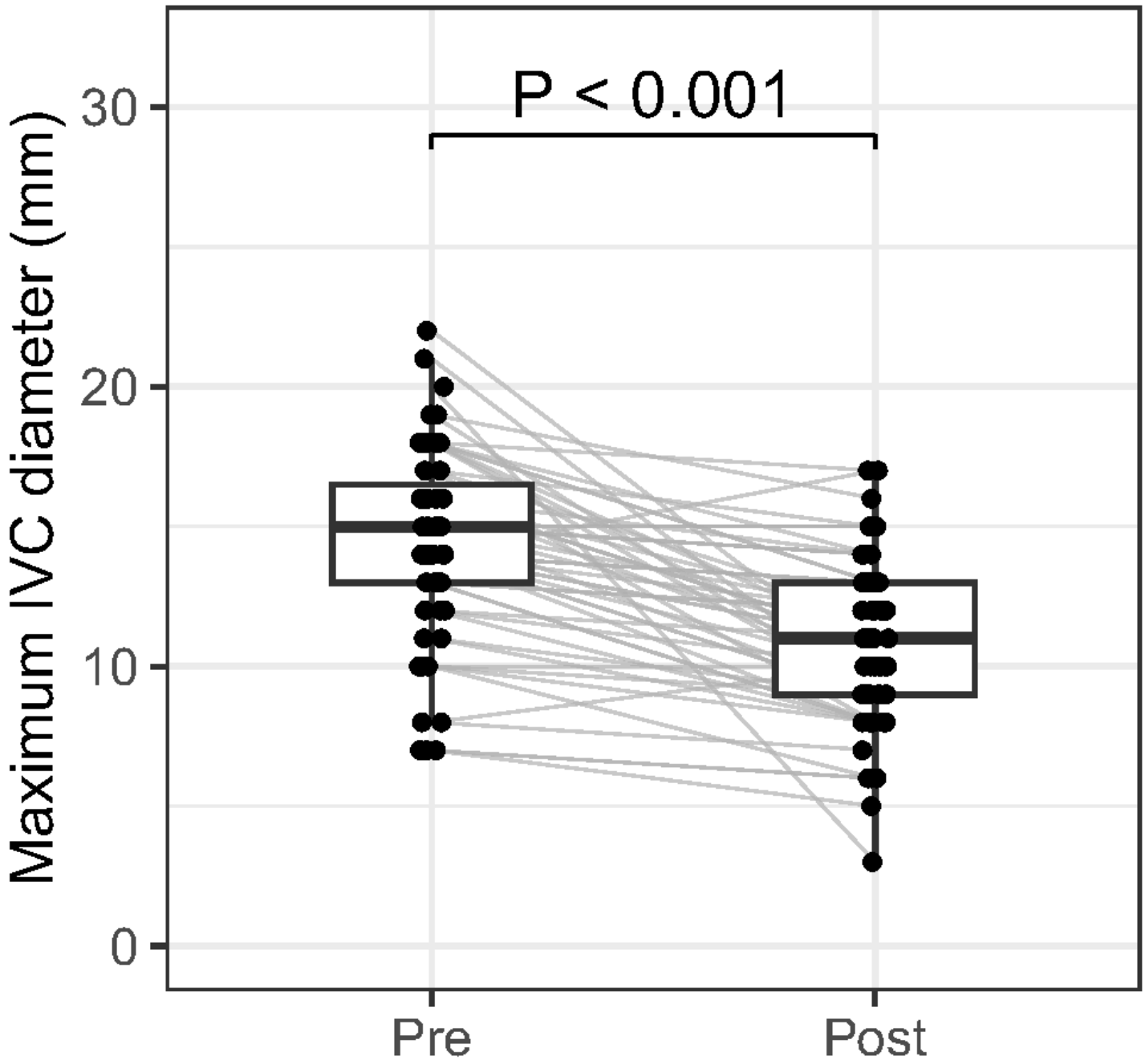


Figure 3

Maximum inferior vena cava diameter before and after alcohol consumption.

IVC, inferior vena cava.

The *P* value was calculated using a paired t test.

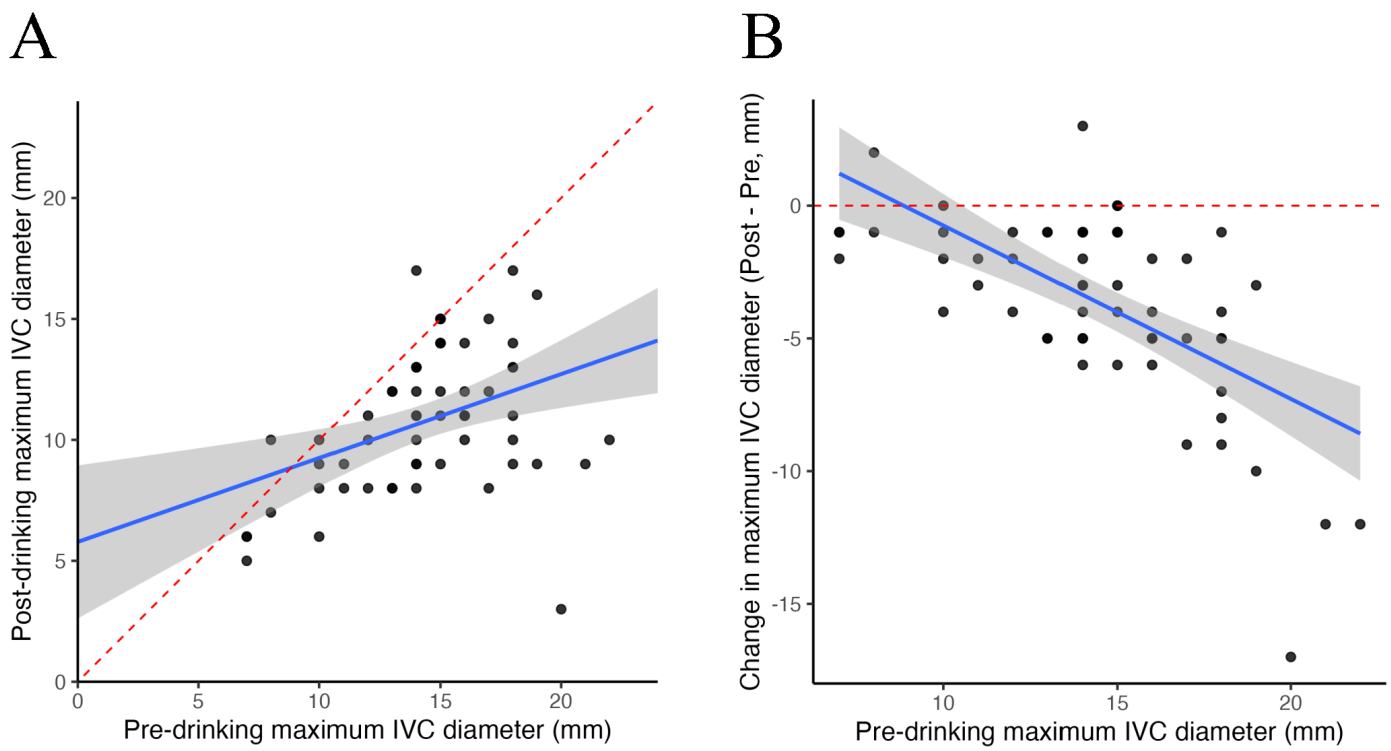


Figure 4

Relationships between pre-drinking maximum inferior vena cava (IVC) diameter and (A) post-drinking maximum IVC diameter or (B) changes in maximum IVC diameter.

IVC, inferior vena cava.

The blue lines represent fitted linear regression lines, and the shaded areas indicate 95% confidence intervals. The red dashed line in panel A represents the line of identity (y = x), whereas the red dashed line in panel B indicates no change (y = 0).

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