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Original Article

Alcohol Consumption is Positively Associated with Handgrip Strength Among Japanese Community-dwelling Middle-aged and Elderly Persons

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SUMMARY

Background: Alcohol consumption is an important lifestyle factor for a variety of health problems, we investigated whether alcohol consumption is associated with handgrip strength (HGS), which is a useful indicator of sarcopenia, among Japanese community-dwelling persons.
Methods: The present study included 764 men aged 70 (69–70) years and 955 women aged 70 (69–70) years from a rural village. Daily alcohol consumption was measured using the Japanese liquor unit in which a unit corresponds to 22.9 g of ethanol, and the participants were classified into never drinkers, occasional drinkers, daily light drinkers (1–2 units/day), and daily moderate drinkers (2–3 units/day).
Results: HGS were significantly correlated with age in both men and women. HGS increased significantly with increased daily alcohol consumption in both genders, and in men HGS in daily moderate drinkers were significantly greater than those in never, occasional, and daily light drinkers. In women, HGS in daily light and moderate drinkers were significantly greater than those in never drinkers. In men, Multivariate-adjusted HGS were significantly greater in daily light {mean: 33.4 (95% confidence interval: 32.3–34.5) kg} and moderate drinkers {33.6 (32.8–34.0) kg} than in never drinkers {31.7 (30.8–32.7) kg}, and in women multivariate-adjusted HGS in occasional drinkers {21.5 (21.0–22.1) kg} was significantly greater in never drinkers {20.7 (20.5–21.0) kg}.
Conclusion: These results suggest that alcohol consumption may have a protective role in aging-associated decline in muscle strength in community-dwelling persons.

1. Introduction

Muscle weakness is consistently reported as an independent risk factor for high mortality in older adults,[1] and is an important public health problem. Thus, Handgrip strength (HGS) is an easily obtainable measure of physical health and muscle function, and is a reliable test to estimate cognitive performance, adverse outcomes (mortality, functional decline, institutionalization),[2] and mortality.[1]

Alcohol consumption is an important lifestyle factor for a variety of health problems, and excessive alcohol consumption increases morbidity and mortality because it is associated with an increase risk of conditions such as liver disease, several types of cancer, and sarcopenia (e.g., loss of muscle mass and strength).[3] Although Mild to moderate alcohol consumption has also been known to improve of inflammation[4] and insulin resistance,[5] and is able to cause decreased cardiovascular disease (CVD).[6] The results of meta-analysis to explore the relationship between alcohol consumption and sarcopenia in people aged ≥ 65 years old do not support alcohol consumption as a risk factor for sarcopenia.[7] Thus, there is a great deal of controversy surrounding this relationship, and relevant literature on the relationship between the influence of alcohol intake and HGS is sparse.[8] To our knowledge, there are few studies that demonstrate a relationship between alcohol consumption and HGS in Japanese population.

We hypothesized that alcohol consumption is associated with muscle strength. To confirm this hypothesis, we investigated whether alcohol consumption is associated with HGS, which is a useful indicator of sarcopenia, among Japanese community-dwelling persons.

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2. Methods

2.1. Subjects

The present study was designed as part of the Nomura study. The study population aged ≥40 years was selected through a community-based annual check-up process from the Nomura health and welfare center in a rural town located in Ehime prefecture, Japan in 2014. 1760 subjects were assessed for eligibility to participate in the study. The physical activity level of subjects (e.g., exercise habits), information on medical history, present conditions, and medications (e.g., antihypertensive, antidyslipidemic, antidiabetic, and uric acid lowering medication) were obtained by interview using a structured questionnaire. For all these individuals, overnight fasting plasma samples were made available. The final study sample included 1719 eligible persons without miss data. The study complies with the Declaration of Helsinki, and was approved by the ethics committee of Ehime University School of Medicine with written informed consent obtained from each subject (Institutional Review Board: 1402009).

2.2. Evaluation of risk factors

Information on demographic characteristics and risk factors was collected using clinical files. Body mass index (BMI) was calculated by dividing weight (in kilograms) by the square of the height (in meters). Smoking status was defined as the number of cigarette packs per day multiplied by the number of years smoked (pack-year), and the participants were classified into never smokers, past smokers, light smokers (<20 pack-year) and heavy smokers (≥20 pack-year). Beverage-specific quantities of alcohol consumption were calculated according to data reported via interview using a structured questionnaire. The following ethanol concentrations were assumed: 22.9 g/180 ml of Japanese sake, 500 ml of beer, 60 ml of whiskey, 180 ml of wine, or 110 ml of shochu (white spirits), and the participants were classified into never drinkers, occasional drinkers (<1 unit/day), daily light drinkers (1–2 units/day), and daily moderate drinkers (2–3 units/day). In this study, there was no alcohol abuser (≥3 units/day). We measured systolic blood pressure (SBP) and diastolic blood pressure (DBP) in the right upper arm of participants in the sedentary position using an automatic oscillometric blood pressure recorder while the subjects were seated after having rested for at least 5 min. Triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), hemoglobin A1c (HbA1c), uric acid (UA), and creatinine (Cr) were measured during fasting. Estimated glomerular filtration ratio (eGFR) was calculated using CKD-EPI equations modified by a Japanese coefficient: Male, $Cr \leq 0.9$ mg/dl, $141 \times (Cr/0.9)^{-0.411} \times 0.993^{age} \times 0.813$; $Cr > 0.9$ mg/dl, $141 \times (Cr/0.9)^{-1.209} \times 0.993^{age} \times 0.813$; Female, $Cr \leq 0.7$ mg/dl, $144 \times (Cr/0.7)^{-0.329} \times 0.993^{age} \times 0.813$; $Cr > 0.7$ mg/dl, $144 \times (Cr/0.7)^{-1.209} \times 0.993^{age} \times 0.813$.[9] Moreover, ischemic stroke, ischemic heart disease, and peripheral vascular disease were defined as CVD.

2.3. HGS test

This test was used to measure the isometric strength of the hand and forearm muscles using Takei Digital Hand Grip. Some studies have focused on the reliability and validity of the Takei Digital Hand Grip.[10] The subject holds the dynamometer in the hand to be tested, with the arm at right angles and the elbow by the side of the body. The handle of the dynamometer is adjusted if required - the base should rest on the first metacarpal (heel of palm), while the handle should rest on the four middle fingers. When ready the subject squeezes the dynamometer with maximum isometric effort, which is maintained for about 5 s. No other body movement is allowed.[10] The mean of two right and left measurements was used for analysis.

2.4. Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics Version 20 (Statistical Package for Social Science Japan, Inc., Tokyo, Japan). All values are expressed as mean (95% confidential interval), unless otherwise specified. Data for TG, HbA1c, and HGS are skewed, presented as median (interquartile range) values, and log-transformed for analysis. Subjects were divided into four groups based on alcohol consumption according to gender, and differences among the groups were analyzed by ANOVA for the continuous variables or the χ^2 -test for the categorical variables. ANCOVA was performed using a general linear model approach to determine the association between the confounding factors and HGS. In these analyses, HGS was the dependent variable, the four categories of alcohol consumption were the fixed factors, and confounding factors were added as covariates. A p-value <0.05 was considered significant.

3. Results

3.1. Characteristics of subjects categorized by gender

Gender-specific characteristics of the subjects are illustrated in Table 1. The study included 764 men aged 70 (69–70) years and 955 women aged 70 (69–70) years. HGS was 34.0 (28.4–39.2) kg in men and 21.4 (18.7–24.1) kg in women. In men, BMI, smoking status, daily alcohol consumption, history of CVD, DBP, TG, prevalence of antidiabetic medication, serum UA, and HGS were significantly higher than in women, but HDL-C, LDL-C, prevalence of antidyslipidemic medication, and eGFR were significantly lower.

3.2. Characteristics of subjects categorized by gender and daily alcohol consumption

We thought that sex-specific analyses were also required as alcohol consumption and handgrip strength are higher in men than in women. Gender-specific characteristics of the subjects categorized by gender and alcohol consumption are illustrated in Table 2a. In men, smoking status, SBP, DBP, HDL-C, UA, and eGFR were significantly higher with increased daily alcohol consumption, but age, history of CVD, LDL-C, and prevalence of antidyslipidemic medication were significantly lower. In women, smoking status, HDL-C, UA, and eGFR were significantly higher, but age and SBP were significantly lower with increased daily alcohol consumption. Table 2b.

3.3. A relationship between age and HGS of subjects categorized by gender

As shown in Fig. 1, HGS were significantly correlated with age in both men ($r = -0.619$, $p < 0.001$) and women ($r = -0.478$, $p < 0.001$).

3.4. Mean HGS of subjects categorized by gender and daily alcohol consumption

As shown in Table 3, HGS increased significantly with increased daily alcohol consumption in both genders, and in men HGS in daily moderate drinkers were significantly greater than those in never, occasional, and daily light drinkers. In women, HGS in daily light and moderate drinkers were significantly greater than those in never drinkers. Moreover, in men, age and BMI, and multivariate-adjusted HGS were significantly greater in daily light and moderate drinkers than in never drinkers. In women, age, age and BMI,

Table 1
Characteristics of subjects by gender.

Characteristics N = 1719	Men	Women	*P* -value*
	N = 764	N = 955	
Age (years)	70 (69–70)	70 (69–70)	0.473
Body mass index (kg/m^2)	23.2 (23.0–23.4)	22.6 (22.4–22.8)	**<0.001**
Exercise habits (%)	36.1	38.2	0.394
Smoking status (%)	41.5/39.1/5.8/13.6	96.6/2.2/0.7/0.4	**<0.001**
Daily alcohol consumption (%)	24.3/22.1/16.2/37.3	71.1/22.3/4.4/2.2	**<0.001**
History of cardiovascular disease (%)	9.8	4.4	**<0.001**
Systolic blood pressure (mmHg)	136 (135–137)	136 (135–137)	0.716
Diastolic blood pressure (mmHg)	80 (79–81)	77 (76–77)	**<0.001**
Antihypertensive medication (%)	45.5	43.9	0.495
Triglycerides (mg/dl)	90 (68–132)	87 (65–117)	**<0.001**
HDL cholesterol (mg/dl)	62 (61–63)	69 (68–70)	**<0.001**
LDL cholesterol (mg/dl)	114 (112–116)	125 (123–127)	**<0.001**
Antidyslipidemic medication (%)	13.6	29.1	**<0.001**
Hemoglobin A 1c (%)	5.7 (5.4–6.0)	5.7 (5.5–5.9)	0.151
Antidiabetic medication (%)	12.8	5.3	**<0.001**
Serum uric acid (mg/dL)	6.0 (5.9–6.1)	4.7 (4.7–4.8)	**<0.001**
Estimated GFR (ml/min/1.73 m^2)	70.4 (69.5–71.2)	72.4 (71.7–73.1)	**<0.001**
Handgrip strength (kg)	34.0 (28.4–39.2)	21.4 (18.7–24.1)	**<0.001**

HDL, high-density lipoprotein; LDL, low-density lopoprotein; GFR, glomerularfiltration ratio. Data presented as mean (95% confidence interval) values. Data for triglycerides, hemoglobin A1c, and handgrip strength are skewed, presented as median (interquartile range) values, and log-transformed for analysis.
Significant values (P < 0.05) are presented in bold.

Table 2a
Characteristics of subjects categorized by gender and alcohol consumption.

Men	Daily alcohol consumption (unit/day)				*P* for trend*
	Never	Occasional	Daily light	Daily moderate	
	0	<1 unit/day	1–2 units/day	2–3 units/day	
Characteristics N = 764	N = 186	N = 169	N = 124	N = 285	
Age (years)	72 (71–74)	70 (68–71)	72 (70–73)	67 (65–68)	**<0.001**
Body mass index (kg/m^2)	23.2 (22.7–23.6)	23.1 (22.6–23.6)	23.2 (22.7–23.7)	23.3 (23.0–23.6)	0.905
Exercise habits (%)	38.2	35.5	33.1	36.5	0.829
Smoking status (%)	47.3/33.9/3.2/15.6	50.9/32.0/7.1/10.1	38.7/47.6/4.8/8.9	33.3/43.2/7.0/16.5	**0.002**
History of cardiovascular disease (%)	15.6	12.4	10.5	4.2	**<0.001**
Systolic blood pressure (mmHg)	131 (129–134)	137 (134–139)	137 (133–140)	138 (136–140)	**<0.001**
Diastolic blood pressure (mmHg)	76 (75–78)	80 (79–82)	79 (77–81)	82 (81–83)	**<0.001**
Antihypertensive medication (%)	42.5	42.0	57.3	44.6	**0.036**
Triglycerides (mg/dl)	90 (66–130)	92 (68–135)	86 (64–126)	93 (72–139)	0.202
HDL cholesterol (mg/dl)	56 (53–58)	59 (57–61)	63 (60–65)	67 (65–69)	**<0.001**
LDL cholesterol (mg/dl)	117 (112–121)	117 (112–121)	114 (109–119)	110 (107–113)	**0.039**
Antidyslipidemic medication (%)	22.0	13.6	12.1	8.8	**0.001**
Hemoglobin A 1c (%)	5.7 (5.4–6.1)	5.7 (5.4–6.0)	5.7 (5.4–6.0)	5.6 (5.4–5.9)	0.246
Antidiabetic medication (%)	15.1	13.6	11.3	11.6	0.665
Uric acid (mg/dL)	5.7 (5.6–5.9)	5.9 (5.8–6.1)	5.9 (5.6–6.1)	6.2 (6.1–6.4)	**0.001**
Estimated GFR (ml/min/1.73 m^2)	67.1 (65.1–69.0)	69.4 (67.4–71.4)	68.3 (66.4–70.2)	74.0 (72.7–75.3)	**<0.001**

Data presented was mean (95% confidence interval). Data for triglycerides, hemoglobin A1c, and handgrip strength are skewed, presented as median (interquartile range) values.
Significant values (P < 0.05) are presented in bold.

and multivariate-adjusted HGS in occasional drinkers was significantly greater than in never drinkers.

4. Discussion

This study demonstrated that daily alcohol consumption was positively associated with HGS in Japanese community-dwelling persons aged 40–90 years. These results suggest that alcohol consumption may have a protective role in aging-associated decline in muscle strength, independent of confounding factors. To our knowledge, few epidemiology studies have quantified the link between alcohol consumption and HGS in both men and women.

Several prospective and cross-sectional studies have found that the link between alcohol consumption and HGS has been reported. The detrimental effects of acute and chronic excessive alcohol ingestion on human physiology have been well documented as affecting many aspects of metabolism, neural function, cardiovascular physiology, thermoregulation and skeletal muscle myopathy.[11] However, there are few studies that demonstrate a relationship between appropriate alcohol consumption and HGS. In a cohort of 890 men aged ≥50 years, Szulc et al.[12] demonstrated that moderate alcohol intake was associated with better physical performance (e.g., handgrip strength). In 5962 men aged ≥65 years, Cawthon et al.[13] showed that the association between alcohol intake and self-reported physical limitation was U-shaped, with the highest odds of physical limitation, which was evaluated by HGS. The findings of Bai et al.,[14] however, are in contrast to these. From 415 participants aged 60–99 years, they also reported that alcohol consumption ($\beta = -1.32$, $P < 0.001$) and smoking ($\beta = -1.47$, $P < 0.001$) were associated with low HGS in men, but were not in women. These conflicting findings are partly related to methodological differences and to participant characteristics. In addition, as alcohol consumption revels relate with increasing numbers of special metabolic risk factors, the effect of alcohol consumption on muscle strength might become negligible. It is very interesting to note a J-shaped association of alcohol consumption with CVD events and all-cause

Table 2b
Continued.

Women	Daily alcohol consumption (unit/day)				*P* for trend*
	Never	Occasional	Daily light	Daily moderate	
Characteristics N = 955	N = 679	N = 213	N = 42	N = 21	
Age (years)	71 (70–71)	69 (68–70)	68 (66–71)	61 (57–65)	**<0.001**
Body mass index (kg/m^2)	22.6 (22.4–22.9)	22.5 (22.1–23.0)	22.2 (21.2–23.2)	22.3 (21.2–23.4)	0.778
Exercise habits (%)	39.0	35.2	35.7	47.6	0.593
Smoking status (%)	97.8/1.5/0.4/0.3	95.8/2.8/0.9/0.5	92.9/4.8/2.4/0	76.2/14.3/4.8/4.8	**<0.001**
History of cardiovascular disease (%)	4.6	4.2	4.8	0	0.792
Systolic blood pressure (mmHg)	137 (136–139)	135 (132–137)	131 (125–137)	129 (120–138)	**0.009**
Diastolic blood pressure (mmHg)	77 (76–78)	76 (75–78)	76 (73–79)	73 (68–78)	0.227
Antihypertensive medication (%)	45.4	40.4	45.2	28.6	0.296
Triglycerides (mg/dl)	87 (67–121)	83 (61–111)	82 (58–103)	79 (57–130)	0.065
HDL cholesterol (mg/dl)	67 (65–68)	72 (69–74)	78 (71–85)	84 (75–93)	**<0.001**
LDL cholesterol (mg/dl)	125 (123–128)	125 (121–129)	121 (112–130)	115 (102–127)	0.324
Antidyslipidemic medication (%)	29.2	30.0	26.2	23.8	0.908
Hemoglobin A 1c (%)	5.7 (5.5–5.9)	5.7 (5.4–5.9)	5.8 (5.4–6.1)	5.5 (5.3–5.7)	0.093
Antidiabetic medication (%)	5.9	3.3	4.8	9.5	0.401
Uric acid (mg/dL)	4.7 (4.6–4.7)	4.9 (4.7–5.0)	4.9 (4.5–5.4)	5.4 (4.9–6.0)	**0.002**
Estimated GFR (ml/min/1.73 m^2)	71.6 (70.8–72.5)	73.6 (72.3–75.0)	72.7 (68.8–76.7)	82.1 (78.4–85.8)	**<0.001**

Significant values (P < 0.05) are presented in bold.

mortality,[15] implying that both lower and higher alcohol consumption lead to a higher risk. As long as we limit alcohol consumption to a moderate amount (roughly 2 drinks/day) there will be no negative side effects in the quest to gain muscle.

The mechanisms that lead to stronger HGS in individuals with daily light and moderate alcohol consumption are very complex and remain not to be clarified. A recent study has shown that oxidative protein damage is independently associated with low HGS among older persons, suggesting that oxidative stress might contribute to the loss of muscle strength and mass.[16] Moderate alcohol consumption has been known to be a neuroprotective antioxidant because of its free radical scavenger activity[17] and did not impair overload-induced muscle hypertrophy and protein synthesis.[18] In addition, alcohol decrease an effect in platelet activity, fibrinolysis and several other coagulation parameters.[19] While, ethanol is one of the few nutrients that is profoundly toxic, and alcohol causes both whole-body and tissue-specific changes in protein metabolism, and both chronically and acutely, alcohol causes reductions in skeletal muscle protein synthesis, as well as of skin, bone, and the small intestine. Animal studies also show chronically increased urinary nitrogen excretion and loss of skeletal muscle protein.[20] Several factors may be explained by these results: heavy alcohol consumption may deteriorate muscle, persons with poor health and low muscle strength intentionally limit alcohol intake, and moderate alcohol consumption is associated with better social participation and more active lifestyle. In our study there was not any heavy alcohol user (≥3 units/day).

We thought that sex-specific analyses were also required because at all ages, alcohol consumption and handgrip strength are higher in men than in women. We cannot explain the underlying mechanism that accounts for the gender difference from this study. A partial explanation for this result could be alcohol consumption,

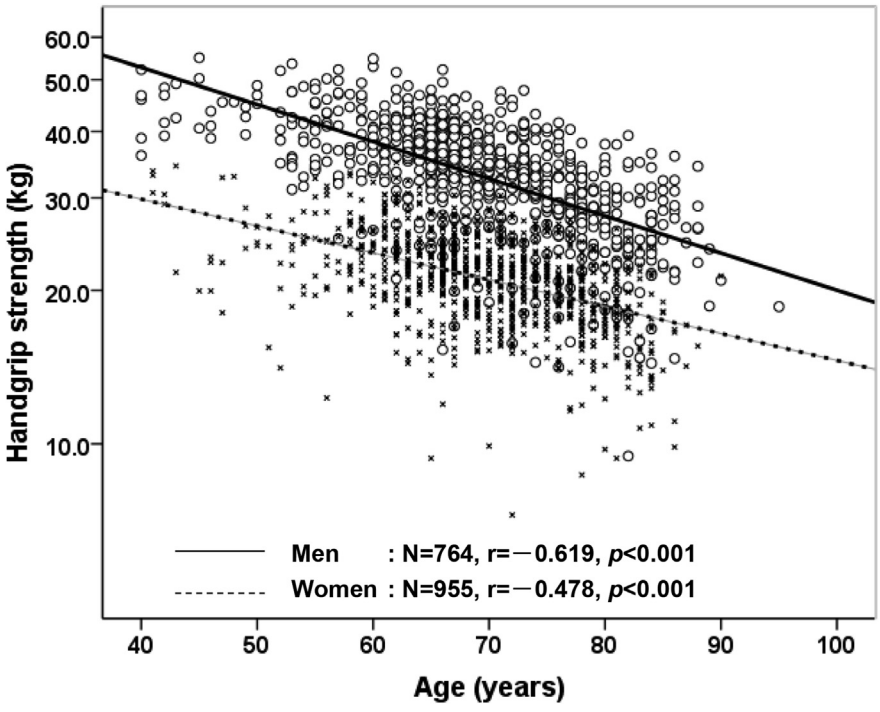

Fig. 1. Relationship between age and handgrip strength (HGS) by gender. Solid line, men; dashed line, women. HGS were significantly correlated with age in men (r = −0.619, $p < 0.001$), and in women (r = −0.478, $p < 0.001$). Data for handgrip strength is skewed and log-transformed for analysis.

Table 3
Mean handgrip strength of subjects categorized by gender and alcohol consumption.

	Daily alcohol consumption (unit/day)				*P* for trend*
	Never	Occasional	Daily light	Daily moderate	
	0	<1 unit/day	1–2 units/day	2–3 units/day	
Men N = 764	N = 186	N = 169	N = 124	N = 285	
Mean handgrip strength (kg)					
Non-adjusted	30.3 (29.4–31.5)	32.1 (30.9–33.5)	32.5 (30.9–33.8)	35.2 (34.5–35.9)[d f g]	**<0.001**
Age & BMI-adjusted	31.7 (30.9–32.6)	32.3 (31.3–33.2)	33.6 (32.4–34.7)[a]	33.6 (32.8–34.3)[b e]	**0.007**
Multivariate 1-adjusted	31.8 (30.9–32.7)	32.3 (31.4–33.3)	33.6 (32.4–34.7)[a]	33.5 (32.7–34.3)[b]	**0.012**
Multivariate 2-adjusted	31.7 (30.8–32.7)	32.3 (31.3–33.2)	33.4 (32.3–34.5)[a]	33.6 (32.8–34.0)[b e]	**0.015**
Women N = 955	N = 679	N = 213	N = 42	N = 21	*P* for trend*
Mean handgrip strength (kg)					
Non-adjusted	20.5 (20.3–20.9)	21.8 (21.3–22.4)[c]	21.5 (20.3–23.1)	24.3 (23.1–25.5)[c]	**<0.001**
Age & BMI-adjusted	20.7 (20.5–21.0)	21.6 (21.1–22.1)[b]	21.3 (20.2–22.5)	21.9 (20.3–23.7)	**0.032**
Multivariate 1-adjusted	20.7 (20.5–21.0)	21.6 (21.1–22.1)[b]	21.3 (20.2–22.5)	22.1 (20.4–23.9)	**0.022**
Multivariate 2-adjusted	20.7 (20.5–21.0)	21.5 (21.0–22.1)[b]	21.3 (20.2–22.5)	22.2 (20.5–24.0)	**0.030**

BMI, body mass index. Data presented as mean (95% confidence interval) values. Multivariate 1-adjusted for age, BMI, exercise habit, smoking status, drinking status, history of cardiovascular disease. Multivariate 2-adjusted for Multivariate 1 model + systolic blood pressure, antihypertensive medication, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, antidyslipidemic medication, hemoglobin A1c, antidiabetic medication, uric acid, and estimated glomerular filtration ratio. [a] $P < 0.05$;[b] $P < 0.01$; [c] $P < 0.005$; [d] $P < 0.001$ versus never drinkers, [e] $P < 0.05$; [f] $P = 0.001$ versus occasional drinkers, and [g] $P < 0.01$ versus daily light drinkers by Bonferroni. Significant values ($P < 0.05$) are presented in bold.

which is more likely to be higher in men, and the influence of sex hormones by testosterone.[21] In our study, HGS were significantly greater in daily right and moderate drinkers in men and occasional drinkers in women.

Several limitations should be considered in this study. First, our cross-sectional study design does not eliminate potential causal relationships between alcohol consumption and muscle function. Second, alcohol consumption categories are based on a single assessment of interview, which may introduce a misclassification bias. Third, self-reported smoking status and alcohol consumption may be underreported due to recall and social desirability biases. Fourth, we could not eliminate the possible effect of medications for hypertension and dyslipidemia, and the possible effects of underlying diseases (e.g., under nutrition due to various illness and healthy diet and consequently lower HGS) on the present findings. Therefore the demographics and referral source may limit generalizability.

5. Conclusion

The present study showed that alcohol consumption is strongly associated with muscle function among Japanese community-dwelling persons. Thus, alcohol consumption might provide an important marker for the assessment of risk as well as a therapeutic target for the modification of sarcopenia.

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Conflicts of interest

Authors declare that there is no conflict of interest.

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