ORIGINAL ARTICLE

Relationship Between Alcohol Intake and Lipid Accumulation Product in Middle-aged Men

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Abstract — Aims: Lipid accumulation product (LAP), defined as a product of waist circumference and triglycerides, has recently been proposed as a predictor of cardiovascular disease and diabetes mellitus. The purpose of this study was to determine whether and how LAP is associated with alcohol drinking. Methods: Subjects were 21,378 men aged 35–60 years and they were divided by alcohol intake into non-, light (<22 g ethanol/day), heavy (≥22 and <44 g ethanol/day) and very heavy (≥44 g ethanol/day) drinkers. Relationships between alcohol intake and LAP were analyzed by using multivariate analyses with adjustment for age, smoking and habitual exercise. Results: Log-transformed LAP levels in light drinkers and very heavy drinkers were significantly (P < 0.01) lower and higher, respectively, than the level in non-drinkers, and the levels were comparable in non- and heavy drinkers (non-drinkers, 1.335 ± 0.005; light drinkers, 1.290 ± 0.009; heavy drinkers, 1.348 ± 0.005 and very heavy drinkers, 1.414 ± 0.006). The inverse association of alcohol intake with LAP was more prominent in smokers and subjects without regular exercise than in non-smokers and subjects with regular exercise, respectively, while the positive association of alcohol with LAP was more prominent in non-smokers than in smokers. Odds ratio for hyperglycemia of subjects with vs. subjects without high LAP was significantly higher than a reference level of 1.00, and this association was not different among the four alcohol groups. Conclusion: There is a J-shaped relationship between alcohol intake and LAP, which is confounded by smoking and habitual exercise.

INTRODUCTION

Light-to-moderate alcohol consumption is known to be associated with a lower risk of cardiovascular disease, particularly coronary artery disease, compared with the risk in non-drinkers (Corrao et al., 2000). This beneficial effect of alcohol is, in part, explained by the improvement in the blood lipid profile in drinkers. About half of the protection and an additional 18% of the protection against coronary artery disease afforded by moderate alcohol consumption have been reported to be mediated by an increase in HDL cholesterol and a decrease in LDL cholesterol, respectively (Langer et al., 1992). There is solid evidence of a positive association between alcohol intake and HDL cholesterol, while the results of previous studies with respect to the relationship between alcohol intake and LDL cholesterol were inconsistent: there are previous studies showing that LDL cholesterol was lower, higher or not different in drinkers compared with that in non-drinkers (Whitehead et al., 1996; Wu et al., 2001; Wakabayashi, 2008). On the other hand, heavy alcohol drinking is associated with an increase in triglycerides (TGs; Castelli et al., 1977; Van de Wiel, 2012), which is known to be a prevalent risk factor of cardiovascular disease (Hokanson and Austin, 1996; Labreuche et al., 2009). Excessive alcohol drinking is also a risk factor of hypertension (Klatsky, 1996), and this detrimental effect of alcohol, in part, explains the higher risk of hemorrhagic types of stroke, such as cerebral hemorrhage and subarachnoid hemorrhage, in heavy drinkers (Mazzaglia et al., 2011). Thus, alcohol has diverse actions on cardiovascular risk factors, which cause a J-shaped relationship between alcohol consumption and all-cause mortality (Di Castelnuovo et al., 2006).

Lipid accumulation product (LAP) has recently been proposed to be a continuous marker of lipid over-accumulation and is determined by TGs and waist circumference (WC) as follows: LAP = TGs (mmol/l) × (WC (cm) – 65) for men and TGs (mmol/l) × (WC (cm) – 58) for women (Kahn, 2005). LAP has been shown to be a better predictor than the body mass index for cardiovascular disease (Kahn, 2005; Wehr et al., 2011) and diabetes (Kahn, 2006; Bozorgmanesh et al., 2010). However, it is not known whether and how alcohol drinking influences LAP. The purpose of this study was, therefore, to determine the relationship between alcohol intake and LAP in middle-aged men and also whether the relation of LAP to hyperglycemia is modified by alcohol drinking.

METHODS

Subjects

The subjects were Japanese men aged 35–60 years (n = 21,378) who had received periodic health checkup examinations at workplaces in Yamagata Prefecture in Japan. This study was approved by the Ethics Committee of Yamagata University School of Medicine (No. 112 from April 2005 to March 2006, approved on March 13, 2006). Histories of alcohol consumption, cigarette smoking, regular exercise (almost every day with exercise for 30 min or longer per day) and illness were surveyed by questionnaires. One pack of cigarette generally contains 20 cigarettes, a number that is often used for dividing light and heavy smokers. The subjects were divided into three groups by average cigarette consumption (non-smokers; light smokers, <20 cigarettes per day; heavy smokers, 20 or more cigarettes per day). Those who had been receiving drug therapy for dyslipidemia (4.7%) were excluded from subjects of this study. Subjects showing WC ≤65 cm (0.86%) were also excluded because their log-transformed LAP could not be calculated.

Evaluation of alcohol consumption

Average alcohol consumption of each subject per week was reported on questionnaires. Frequency of habitual alcohol drinking was asked in the questionnaire as ‘How frequently
do you drink alcohol?’. Frequency of weekly alcohol drinking was categorized as ‘every day’ (regular drinkers), ‘sometimes’ (occasional drinkers) and ‘never’ (non-drinkers). Only regular drinkers who answered ‘every day’ were used as drinkers for the analysis in this study, since it was difficult to know the correct average alcohol consumption of occasional drinkers who answered ‘sometimes’. Occasional drinkers were, thus, excluded from subjects for analysis, and regular drinkers were compared with non-drinkers in this study. Usual weekly alcohol consumption was recorded in terms of the equivalent number of ‘go’, a traditional Japanese unit of amount of sake (rice wine). The amounts of other alcoholic beverages, including beer, wine, whiskey and shochu (traditional Japanese distilled spirit), were converted and expressed as units of ‘go’. One ‘go’ contains ~22 g of ethanol, and this amount was used to separate moderate-to-heavy drinkers from light drinkers, since it is generally accepted that alcohol intake should be reduced to <20–30 g ethanol per day from the viewpoint of prevention of hypertension (Mancia et al., 2007; Ogihara et al., 2009). Average daily alcohol intake (grams of ethanol per day) was then calculated. The subjects were divided into four groups according to ethanol consumption per day (non-drinkers; light drinkers: <22 g of ethanol per day; heavy drinkers: ≥22 and <44 g of ethanol per day; very heavy drinkers: ≥44 g ethanol per day). In general, alcohol intake of two to three drinks per day and alcohol intake of three drinks or more per day are considered as moderate drinking and heavy drinking, respectively. In the present study, heavy drinkers and very heavy drinkers were defined as those with alcohol intake of ≥22 g but <44 g of ethanol per day and ≥44 g ethanol per day, respectively, because ‘go’, a traditional Japanese unit of amount of sake, which corresponds to ~22 g of ethanol, was used in the questionnaire. Thus, the category of ‘moderate drinkers’ used in general was included in the category of ‘heavy drinkers’ in the present study.

Measurements

WC was measured at the navel level according to the recommendation of the definition of the Japanese Committee for the Diagnostic Criteria of Metabolic Syndrome (Anonymous, 2005). Fasted blood was sampled from each subject, and concentrations of serum TGs were measured by an enzymatic method using a commercial kit, pureauto S TG-N (Sekisui Medical Co., Ltd, Tokyo, Japan). The values of LAP were arranged in the ascending order, and the subjects were then divided into three groups of approximately equal sizes. Subjects in the highest tertile group of LAP were defined as subjects with high LAP, since the common cut-off value for LAP has not been confirmed. Blood hemoglobin A1c, which reflects the glucose tolerance status more correctly than does blood glucose, was used for the evaluation of hyperglycemia. Hemoglobin A1c was determined by the latex cohesion method using a commercial kit (Determiner HbA1c, Kyowa Medex, Tokyo, Japan). Coefficients of variation for reproducibility of each measurement were ≤3% for TGs and ≤5% for hemoglobin A1c. Hemoglobin A1c values were calibrated using a formula proposed by the Japan Diabetes Society as hemoglobin A1c (National Glycohemoglobin Standardization Program) (%) = hemoglobin A1c (Japan Diabetes Society) (%) + 0.4% (The Committee of Japan Diabetes Society on the diagnostic criteria of diabetes mellitus, 2010). Hyperglycemia including diabetes and pre-diabetes was defined as hemoglobin A1c ≥5.7% (The Committee of Japan Diabetes Society on the diagnostic criteria of diabetes mellitus, 2010). Subjects receiving drug therapy for diabetes were included in the hyperglycemia group.

Statistical analysis

Statistical analyses were performed using a computer software program (SPSS version 16.0 J for Windows, Chicago IL, USA). The mean levels of each variable were compared using the analysis of variance followed by Scheffé’s F-test in univariate analysis and analysis of covariance followed by Student’s t-test after the Bonferroni correction in multivariate analysis. Since TGs and LAP did not show a normal distribution, they were compared between groups non-parametrically by using the Kruskal–Wallis test followed by the Steel–Dwass test in univariate analysis or were used after log-transformation in multivariate analysis. Comparisons of the percentages of smokers, subjects doing regular exercise and subjects showing hyperglycemia were performed using the Chi-square test for independence. In logistic regression analysis, the odds ratios (ORs) for high LAP or hyperglycemia were calculated. Age, history of smoking and history of habitual exercise were used as other explanatory variables in multivariate analyses. In multivariate analyses, smoking and exercise were defined categorically as a history of smoking in three levels (non, light and heavy smokers) and a history of habitual exercise in two levels (with and without history), respectively. Age was used as a quantitative explanatory variable. Crude ORs were compared between different alcohol groups by using the Breslow–Day test. Probability (P) values <0.05 were defined as statistically significant.

RESULTS

Characteristics of subjects

Table 1 shows profiles of subjects in the four alcohol groups and overall subjects. Age was significantly older in each drinker group than in the non-drinker group and tended to be older with an increase in alcohol intake. The percentages of smokers and subjects doing regular exercise were significantly higher in each drinker group than in the non-drinker group and tended to be higher with an increase in alcohol intake. WC was significantly smaller in light and heavy drinkers than in non-drinkers. TGs and LAP were significantly lower in light drinkers and significantly higher in very heavy drinkers than in non-drinkers. Hemoglobin A1c and the percentage of subjects showing hyperglycemia were significantly lower in light, heavy and very heavy drinkers than in non-drinkers.

Comparison of log-transformed LAP, WC, log-transformed TGs and hemoglobin A1c levels in non-, light, heavy and very heavy drinkers in overall subjects

Log-transformed LAP, WC, log-transformed TGs and hemoglobin A1c levels calculated after adjustment for age, smoking and regular exercise are shown in Fig. 1. Log-transformed LAP and log-transformed TGs were significantly lower in
light drinkers and significantly higher in very heavy drinkers than in non-drinkers and were not significantly different in non-drinkers and heavy drinkers (Fig. 1a and c). WC was significantly smaller in light and heavy drinkers than in non-drinkers and was not different between non-drinkers and very heavy drinkers (Fig. 1b). Hemoglobin A1c was significantly lower in light, heavy and very heavy drinkers than in non-drinkers (Fig. 1d). When subjects showing very small WC (≤65 cm) were included in the analysis by treating all subjects showing WC of 66 cm or below as those with WC of 66 cm, a minimum WC by which log-transformed LAP is able to be calculated, the results of the associations between alcohol intake and LAP were similar to the results for subjects with WC of >65 cm (data not shown). In addition, the associations between alcohol intake and LAP were not altered by including subjects receiving therapy for dyslipidemia (data not shown).

Table 1. Characteristics of subjects

<table>
<thead>
<tr>
<th></th>
<th>Overall subjects</th>
<th>Non-drinkers</th>
<th>Light drinkers</th>
<th>Heavy drinkers</th>
<th>Very heavy drinkers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>21,378</td>
<td>6332</td>
<td>2506</td>
<td>7688</td>
<td>4852</td>
</tr>
<tr>
<td>Age (years)</td>
<td>48.3 ± 7.5</td>
<td>47.0 ± 7.7</td>
<td>48.0 ± 7.6**</td>
<td>48.8 ± 7.4**</td>
<td>49.2 ± 7.2**</td>
</tr>
<tr>
<td>Smokers (%)</td>
<td>60.9</td>
<td>55.3</td>
<td>58.5**</td>
<td>62.9**</td>
<td>66.3**</td>
</tr>
<tr>
<td>Habitual exercise (%)</td>
<td>9.9</td>
<td>9.0</td>
<td>10.1</td>
<td>10.2*</td>
<td>10.4*</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>83.4 ± 8.5</td>
<td>83.8 ± 9.7</td>
<td>82.6 ± 7.9***</td>
<td>83.2 ± 7.9***</td>
<td>83.8 ± 8.1</td>
</tr>
<tr>
<td>Triglycerides (mmol/l)</td>
<td>1.34 (0.89, 2.10)</td>
<td>1.32 (0.90, 2.02)</td>
<td>1.24 (0.84, 1.85)**</td>
<td>1.33 (0.88, 2.08)</td>
<td>1.49 (0.91, 2.40)**</td>
</tr>
<tr>
<td>LAP</td>
<td>23.9 (12.2, 44.4)</td>
<td>23.7 (11.5, 44.3)</td>
<td>21.2 (10.8, 38.2)**</td>
<td>23.5 (12.2, 43.3)</td>
<td>26.6 (13.8, 50.4)**</td>
</tr>
<tr>
<td>Hemoglobin A1c (%)</td>
<td>5.50 ± 0.73</td>
<td>5.61 ± 0.87</td>
<td>5.48 ± 0.69**</td>
<td>5.45 ± 0.61**</td>
<td>5.46 ± 0.70**</td>
</tr>
<tr>
<td>Hyperglycemia (%)</td>
<td>20.5</td>
<td>25.6</td>
<td>19.1**</td>
<td>18.4**</td>
<td>17.9**</td>
</tr>
</tbody>
</table>

Numbers, means with standard deviations, medians with 25 and 75 percentile values in the parentheses, and percentages of each variable are shown. Asterisks denote significant differences from non-drinkers (*P < 0.05; **P < 0.01).
Comparison of log-transformed LAP, WC, log-transformed TGs and hemoglobin A1c levels in the alcohol groups of non-smokers, light smokers or heavy smokers

The relationship between alcohol intake and LAP was different among non-, light and heavy smokers (Fig. 2a): log-transformed LAP in non-smokers was significantly higher in heavy and very heavy drinkers than in non-drinkers, log-transformed LAP in light smokers was significantly lower in light drinkers but significantly higher in very heavy drinkers when compared with non-drinkers (inverted J-shaped relationship), and log-transformed LAP in heavy smokers was significantly lower in light and heavy drinkers than in non-drinkers (U-shaped relationship). These differences by the smoking status mainly reflect differences in the relationship between alcohol intake and WC (Fig. 2b): WC in non-smokers was significantly higher in very heavy drinkers than in non-drinkers, WC in light smokers was significantly lower in light and heavy drinkers than in non-drinkers, and WC in heavy smokers was significantly lower in light, heavy and very heavy drinkers than in non-drinkers. On the other hand, the relationship between alcohol intake and TGs was similar among non-, light and heavy smokers: there was an inverted J-shaped relationship between alcohol intake and log-transformed TGs (Fig. 2c). The inverse association between alcohol intake and hemoglobin A1c was also not modified by the smoking status: hemoglobin A1c levels in non-, light and heavy smokers were significantly lower in light, heavy and very heavy drinkers than in non-drinkers (Fig. 2d).

Comparison of log-transformed LAP, WC, log-transformed TGs and hemoglobin A1c levels in the alcohol groups of subjects with or without a habit of regular exercise

Both in the subject groups with and without regular exercise, log-transformed LAP and log-transformed TGs were significantly higher in very heavy drinkers than in non-drinkers, while significantly lower log-transformed LAP and log-transformed TGs compared with those in non-drinkers were
found only in light drinkers of the group without regular exercise (Fig. 3a and c). WC was, compared with non-drinkers, significantly smaller only in light and heavy drinkers of the group without regular exercise (Fig. 3b). On the other hand, the inverse association between alcohol intake and hemoglobin A1c was not modified by the status of exercise: hemoglobin A1c levels in both groups with and without regular exercise were significantly lower in light, heavy and very heavy drinkers than in non-drinkers (Fig. 3d).

**ORs for high LAP of each drinker group vs. the non-drinker group in overall subjects**

Table 2 shows crude and adjusted ORs for high LAP of drinkers vs. non-drinkers in overall subjects. Age, smoking and regular exercise were adjusted in multivariate analysis. Subjects with high LAP were defined as subjects in the highest tertile group of LAP. Crude and adjusted ORs for high LAP in light drinkers vs. non-drinkers were significantly lower than a reference level of 1.00, while the ORs in very

Fig. 3. Log-transformed LAP, WC, log-transformed TG and hemoglobin A1c levels in non-, light, heavy and very heavy drinkers of the subject groups with or without a history of regular exercise in multivariate analysis. Age and history of smoking were adjusted for calculating mean levels of each variable. Data are shown as means ± standard errors. Asterisks denote significant differences from non-drinkers (*P < 0.05; **P < 0.01).

<table>
<thead>
<tr>
<th>Non-drinkers</th>
<th>Light drinkers</th>
<th>Heavy drinkers</th>
<th>Very heavy drinkers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crude OR</td>
<td>1.00</td>
<td>0.76 (0.69–0.84)**</td>
<td>0.94 (0.87–1.01)</td>
</tr>
<tr>
<td>Adjusted OR</td>
<td>1.00</td>
<td>0.76 (0.68–0.84)**</td>
<td>0.93 (0.87–1.00)</td>
</tr>
</tbody>
</table>

Crude and adjusted ORs with their 95% confidence intervals in the parentheses are shown. The values of LAP were arranged in the ascending order, and the subjects were then divided into three groups of approximately equal sizes. Subjects in the highest tertile group of LAP were defined as subjects with high LAP. Adjusted ORs for high LAP were calculated using age, smoking and habitual exercise as other explanatory variables. Asterisks denote significantly higher or lower ORs compared with a reference level of 1.00 (**P < 0.01).
heavy drinkers vs. non-drinkers were significantly higher than the reference level. Crude and adjusted ORs for high LAP in heavy drinkers vs. non-drinkers were not significantly different from the reference level.

**ORS for high LAP of each drinker group vs. the non-drinker group in smokers or non-smokers**

Crude and adjusted ORs vs. non-drinkers for high LAP were significantly lower than the reference level of 1.00 in light drinkers of the non-, light and heavy smoker groups and in heavy drinkers of the light and heavy smoker groups, while the ORs were significantly higher than the reference level only in very heavy drinkers of the non-smoker group (Table 3).

**ORS for high LAP of each drinker group vs. the non-drinker group in subjects with or without a habit of regular exercise**

Crude and adjusted ORs vs. non-drinkers for high LAP were significantly higher than the reference level of 1.00 in very heavy drinkers of both groups with and without regular exercise, while the ORs were significantly lower than the reference level only in light drinkers of the group without regular exercise (Table 4).

**ORS for hyperglycemia of subjects with vs. subjects without high LAP in overall subjects**

Table 5 shows crude and adjusted ORs for hyperglycemia of subjects with vs. subjects without high LAP in each drinker group and overall subjects. Age, smoking and regular exercise were adjusted in multivariate analysis. Alcohol intake was also adjusted for the calculation of ORs in overall subjects. Crude and adjusted ORs for hyperglycemia in all of the drinker groups and the overall subjects were significantly higher than a reference level of 1.00, and there were no significant differences in the crude ORs of non-, light, heavy and very heavy drinkers in analysis using the Breslow–Day test. ORs of the interaction term consisting of drinker category (each drinker group vs. the non-drinker group) and LAP (high vs. not high) for hyperglycemia were not significantly different from the reference level.

**DISCUSSION**

In overall subjects, there was a J-shaped relationship between alcohol intake and LAP. This finding accords with the known relationship between alcohol drinking and cardiovascular risk (Corrao et al., 2000). Positive and inverse associations of alcohol intake with LAP were more prominent in non-smokers and smokers, respectively. The inverse relationship of alcohol intake with LAP was found in light drinkers without regular exercise but not in those with regular exercise. Therefore, smoking and exercise influence the association between alcohol intake and LAP. This study is the first study showing the relationship between alcohol drinking and LAP, a new marker of lipid over-accumulation.

LAP consists of levels of WC and TGs, which were influenced differently by alcohol drinking. The relationship between alcohol intake and TGs was similar to the relationship between alcohol intake and LAP. Therefore, the relationship between alcohol and LAP mainly reflects the

| Non-smokers | Crude OR | 1.00 | 0.84 (0.72–0.99)* | 1.11 (0.99–1.24) | 1.50 (1.32–1.71)** |
| Light smokers | Crude OR | 1.00 | 0.71 (0.61–0.83)** | 0.85 (0.76–0.95)** | 1.03 (0.90–1.17) |
| | Adjusted OR | 0.83 (0.71–0.98)* | 1.09 (0.97–1.22) | 1.48 (1.30–1.69)** |
| Heavy smokers | Crude OR | 1.00 | 0.77 (0.59–1.00)* | 0.80 (0.69–0.94)** | 1.00 (0.86–1.16) |
| | Adjusted OR | 0.77 (0.59–1.00)* | 0.81 (0.70–0.95)* | 1.02 (0.87–1.19) |

| Non-drinkers | Light drinkers | Heavy drinkers | Very heavy drinkers |
| Non-smokers | Crude OR | 0.75 (0.68–0.84)** | 0.91 (0.85–0.98) | 1.19 (1.10–1.30)** |
| Adjusted OR | 0.75 (0.67–0.83)** | 0.91 (0.84–0.98) | 1.19 (1.10–1.30)** |
| Light drinkers | Crude OR | 0.88 (0.63–1.23) | 1.23 (0.97–1.56) | 1.61 (1.24–2.08)** |
| Adjusted OR | 0.84 (0.60–1.19) | 1.16 (0.91–1.48) | 1.49 (1.14–1.93)** |

Crude and adjusted ORs with their 95% confidence intervals in the parentheses are shown. The values of LAP were arranged in the ascending order, and the subjects were then divided into three groups of approximately equal sizes. Subjects in the highest tertile group of LAP were defined as subjects with high LAP. Adjusted ORs for high LAP were calculated using age and smoking as other explanatory variables. Asterisks denote significantly higher or lower ORs compared with a reference level of 1.00 (*P < 0.05; **P < 0.01).
relationship between alcohol and TGs. The difference in the association between alcohol and LAP between smokers and non-smokers may be due to smaller WC in the drinker groups of smokers but not in those of non-smokers, compared with the corresponding non-drinker groups. There was an inverse relationship between alcohol intake and LAP in light drinkers without regular exercise but not in those with regular exercise, and this difference may result from smaller WC and lower TGs in light drinkers than in non-drinkers, which were found in subjects without exercise but not in those with exercise.

LAP has been shown to be associated with risk of diabetes (Kahn, 2006; Bozorgmanesh et al., 2010). This agrees with the finding in this study that there is a positive association between LAP and hyperglycemia (Table 5). Moderate alcohol consumption has been shown to be associated with a decreased incidence of diabetes (Howard et al., 2004; Baliunas et al., 2009), and this also agrees with the findings of the inverse associations of alcohol intake with the hemoglobin A1c level and the prevalence of hyperglycemia in the present study. In an analysis using data for subjects divided by age into four quartile groups, the inverse association between alcohol intake and hemoglobin A1c was not confounded by age (data not shown). The positive association of LAP with hyperglycemia was found independent of the status of alcohol drinking. Moreover, there was no interaction of alcohol and LAP for hyperglycemia in the analysis using the interaction term consisting of alcohol and LAP. Therefore, the association between LAP and glycemic status was not modified by alcohol drinking. This suggests that LAP can be used as a predictor of diabetes in drinkers as well as in non-drinkers. Moreover, in another analysis, LAP was positively correlated with the serum \( \gamma \)-glutamyl transpeptidase level both in the non-drinker and drinker groups (data not shown), and this supports the finding of a recent study that LAP is a good marker of liver steatosis (Bedogni et al., 2010).

There are other blood lipids-related predictors for cardiovascular disease. The LDL cholesterol-to-HDL cholesterol ratio (LDL-C/HDL-C ratio) is a classical atherogenic index (Kannel, 1985; Fernandez and Webb, 2008) and tends to be lower as alcohol intake increases. Similarly, non-HDL cholesterol, a good predictor for cardiovascular disease (Cui et al., 2001; Packard and Saito, 2004), has been shown to be inversely correlated with alcohol intake (Wakabayashi and Groschner, 2009). The TGs-to-HDL cholesterol ratio (TG/HDL-C ratio) has been shown to be better for predicting coronary heart disease than the LDL-C/HDL-C ratio (Gaziano et al., 1997; Jeppesen et al., 1997) and to be lower in light, heavy and very heavy drinkers than in non-drinkers and lowest in light drinkers (Wakabayashi, 2012). Thus, the LDL-C/HDL-C ratio, non-HDL cholesterol and TG/HDL-C ratio are all inversely associated with alcohol intake. The significance of the above three lipids-related predictors of cardiovascular disease in very heavy drinkers is different from the significance of LAP in very heavy drinkers. In the present study, a J-shaped relationship was found between alcohol intake and LAP in the overall subjects, suggesting a detrimental effect of very heavy drinking on the risk of cardiovascular disease and agreeing with the known epidemiological finding of a J-shaped relationship between alcohol consumption and risk of cardiovascular disease (Corraro et al., 2000). Further studies are needed to evaluate the usefulness of the lipids-related parameters for the prevention of cardiovascular events.

There are limitations of this study. Although age, smoking and habitual exercise were adjusted in multivariate analyses, there are other possible confounding factors, including diet, nutrition and socioeconomic factors, for the relationship between alcohol drinking and LAP, and information on these factors was not available in the database used in this study. In addition, information on the kind of alcohol beverage was not available in this study. The subjects of this study were middle-aged Japanese men. Ethnicity-related differences have been shown in mortality from cardiovascular diseases, such as coronary heart disease, stroke and chronic kidney disease, and in the associated cardiovascular risk factors including lipid indicators (Cappuccio, 1997). Therefore, prediction of cardiovascular mortality by lipid indicators varies not only with the indicator used but also with age, gender, and, of especial relevance for the present study, ethnicity. Regarding gender, most observational studies showed a positive association or no association between beer intake and WC or waist-to-hip ratio in men, whereas results of the relationship between beer intake and abdominal obesity in women were inconsistent (Bendsen et al., 2013). This study is cross-sectional in its design and further prospective studies are needed to clarify causality of the relationship between alcohol drinking and LAP.

In conclusion, there is a J-shaped relationship between alcohol drinking and LAP, which is modified by smoking and exercise, while alcohol drinking may not modify the association between LAP and hyperglycemia.

The TGs-to-HDL cholesterol ratio (TG/HDL-C ratio) has been shown to be better for predicting coronary heart disease than the LDL-C/HDL-C ratio (Gaziano et al., 1997; Jeppesen et al., 1997) and to be lower in light, heavy and very heavy drinkers than in non-drinkers and lowest in light drinkers (Wakabayashi, 2012). Thus, the LDL-C/HDL-C ratio, non-HDL cholesterol and TG/HDL-C ratio are all inversely associated with alcohol intake. The significance of the above three lipids-related predictors of cardiovascular disease in very heavy drinkers is different from the significance of LAP in very heavy drinkers. In the present study, a J-shaped relationship was found between alcohol intake and LAP in the overall subjects, suggesting a detrimental effect of very heavy drinking on the risk of cardiovascular disease and agreeing with the known epidemiological finding of a J-shaped relationship between alcohol consumption and risk of cardiovascular disease (Corraro et al., 2000). Further studies are needed to evaluate the usefulness of the lipids-related parameters for the prevention of cardiovascular events.

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In conclusion, there is a J-shaped relationship between alcohol drinking and LAP, which is modified by smoking and exercise, while alcohol drinking may not modify the association between LAP and hyperglycemia.
Conflict of interest statement. None declared.

REFERENCES


