



Interaction of current alcohol consumption and abdominal obesity on hypertension risk



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HIGHLIGHTS

- The first study to examine the combined effects of alcohol consumption and abdominal obesity
- Independent effects of current alcohol consumption on essential hypertension
- Independent effects of duration of alcohol consumption on essential hypertension

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ABSTRACT

The high prevalence of alcohol consumption and abdominal obesity and increased incidence of essential hypertension (EH) in China indicates that there may be an interaction between alcohol consumption and abdominal obesity on EH risk. Therefore, we aimed to examine the independent and combined effects of alcohol consumption and abdominal obesity on risk of EH in a Chinese cohort. We analyzed data from a population-based prospective cohort of 2778 participants aged 35–74 years from Jiangsu China who were free of hypertension, diabetes and CVD at enrollment and were followed for hypertension events. Cox proportional hazards regression model was used to calculate the hazard ratio (HR) of hypertension and corresponding 95% confidence interval (CI). Logistic regression model was used to examine the interaction between alcohol consumption and abdominal obesity on risk of hypertension. After adjusting for age, sex, smoking status, family history of hypertension, current alcohol consumption significantly increased EH risk. Compared with those without alcohol consumption, the hazard ratio of EH for participants with alcohol consumption was 1.65 (95% confidence interval 1.29–2.12). There was a statistically significant additive interaction between current alcohol consumption and abdominal obesity on EH risk. The risk of EH for current alcohol consumers with abdominal obesity was 4.49 times as high as the sum of risks in participants with a single condition alone. Both alcohol consumption and abdominal obesity are strong risk factors of EH in the Chinese population. Moreover, this study further demonstrates an additive interaction of alcohol consumption and abdominal obesity on EH risk.

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1. Introduction

Obesity is a worldwide phenomenon and indicates excessive deposition of fat in the body. In the Chinese middle-aged population, a significant increase in the prevalence of obesity originated in the early 19th to early 20th century [1]. Elevated morbidity and mortality rates in obese people are generally attributed to the increased incidence of obesity-related diseases, such as hypertension, which is associated with an increased risk of mortality and morbidity from

stroke, coronary heart disease, congestive heart failure, and end-stage renal disease [2,3]. Alcohol consumption also has an obvious effect on blood pressure (BP) and risk of hypertension. A large number of investigations in experimental, clinical, and epidemiological settings have focused on alcohol consumption and hypertension [4–8]. Some studies indicated a relationship between alcohol intake and incident hypertension, and some other studies suggested that light-to-moderate alcohol consumption decreased hypertension risk.

Alcohol consumption and obesity are two of the best known but most avoidable and controllable risk factors related to hypertension. Given the high prevalence of alcohol intake and obesity in China, the coexistence of these two conditions may impose a huge burden on public health. Gu et al. [9] provide evidence for the first time that body mass index (BMI) and alcohol intake might interactively

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influence systolic hypertension, especially for the overweight and obese patients in a cross-sectional epidemiologic study. Hence, in this study, we aimed to examine the independent and combined effects of alcohol consumption and obesity on incident hypertension in a population-based Chinese cohort, so as to find an effect modification of alcohol by obesity status and hypothesize that the association between alcohol drinking and hypertension may vary across populations.

2. Material and methods

2.1. Study cohort

The Prevention of MS and Multi-metabolic Disorders in Jiangsu Province of China Study (PMMJS) is an ongoing prospective cohort study aimed to estimate the prevalence of MS and the incidence of CVD and type 2 diabetes in Jiangsu Province of China. The detailed design of this study has been described elsewhere [10]. Briefly, the cohort was established between 2000 and 2004 in Jiangsu, China. Overall, 6400 participants aged 35–74 years were randomly selected based on a multi-stage sampling method. In the first survey, 5888 participants (92%) returned a completed questionnaire with information on diet, education, occupation, lifestyle factors, and medical history. The protocol was approved by the ethical committee of Soochow University.

In the second survey between 2006 and 2008, 4582 participants who have been followed for at least 5 years in this cohort were reached by actual re-contact. A total of 4083 participants completed the follow-up survey, with a follow-up rate of 89.1%. The characteristics of non-participants, such as age, sex, and metabolic variables, were similar to those who participated in the follow-up survey.

For this analysis, we excluded participants with hypertension ($n = 820$), diabetes ($n = 289$), CVD ($n = 36$), and missing data ($n = 133$) at baseline. We also excluded participants with $BMI < 18.5 \text{ kg/m}^2$ ($n = 27$), leaving 2778 eligible participants (1097 males and 1681 females) for final analysis. Each participant signed an informed consent form at the interview.

2.2. Exposure assessment

We defined currently alcohol consumption as more than 1 drink of any type per month or not currently drinking as less than 1 drink of any type per month [11]; The cutoff values of waist circumference for abdominal obesity were 90 cm for male and 80 cm for female, which were in agreement with modification for Asian populations [12]; And abnormal BMI (overweight or overall obesity) was defined as $BMI \geq 25 \text{ kg/m}^2$ [13].

2.3. End point ascertainment

For this study, hypertension was defined as systolic blood pressures (SBP) $\geq 140 \text{ mm Hg}$ and/or diastolic blood pressures (DBP) $\geq 90 \text{ mm Hg}$ and/or the use of antihypertensive medication, as reported in the questionnaires [14].

2.4. Covariate measurement

Data on demographic characteristics, lifestyle risk factors, personal medical history and family history of hypertension for all participants were obtained using a standard questionnaire administered by trained staff. Three sitting blood pressure (BP) measurements were taken at 30-second intervals by trained observers using a standard mercury sphygmomanometer after the subjects had been resting for 5 min according to a standard protocol. The first and fifth Korotkoff sounds were recorded as the SBP and DBP, respectively. The mean of the three BP measurements was used in the analysis.

Body weight and height were measured using standard methods, and the BMI was calculated as the weight in kilograms divided by the square of the height in meters. The WC was measured two times at 1 cm above the umbilicus at minimal respiration by trained observers; the mean of the two WC measurements was utilized in the analysis.

Blood samples were collected in the morning after at least 8 h of fasting. All plasma and serum samples were frozen at $-80 \text{ }^\circ\text{C}$ until laboratory testing was performed. Plasma glucose was measured using an oxidase enzymatic method. The concentrations of HDL cholesterol and triglycerides were assessed enzymatically using an automatic biochemistry analyzer (Hitachi Inc, Tokyo, Japan) and commercial reagents. All analyses were performed by the same lab.

All of the participants signed the informed consent form. The study was approved by the Soochow University ethics committee.

2.5. Statistical analysis

Baseline continuous variables of participants were calculated as mean with standard deviation or median with interquartile range according to their distribution, and the categorical variables were presented as percentage. Baseline characteristics were compared according to smoking status using chi-square test for categorical variables and ANOVA test for continuous variables. We calculated person-years of follow-up for each participant from the date of return of the questionnaire to the date of hypertension diagnosis, the date of death, or August 31, 2008. Cox proportional hazards regression model was used to calculate the hazard ratio (HR) of hypertension and corresponding 95% confidence interval (CI). The multivariate HRs were further adjusted for sex, age, family history of hypertension, and smoking status. We next examined the interaction between alcohol consumption and obesity (defined by BMI) or abdominal obesity (defined by WC) on risk of hypertension, by dividing the participants into 4 groups according to alcohol consumption and obesity status. We then tested an interaction on an additive scale by calculating the relative excess risk due to interaction (RERI), the attributable proportion due to interaction (AP), and the synergy index (SI) based on the methods proposed by Andersson et al. [15]. $RERI > 0$, $AP > 0$, and $SI > 1$ indicate biological interaction. Both the point estimation and the 95% CI of RERI, AP, and SI were assessed using a method accounting for the asymmetric distribution of confidence limits for risk ratio [16]. All statistical analyses were performed using the SPSS statistical software system for Windows version 16.0 (SPSS Inc. Chicago, USA).

3. Results

A total of 2778 participants (1097 males and 1681 females) were studied; this number included 660 subjects with incident hypertension (254 males and 406 females) who developed hypertension by the follow-up investigation. Compared to subjects with normal BMI or WC, TC, TG, LDL-c, SBP, DBP were higher and HDL-c was lower in obese and abdominal obese subjects. The baseline characteristics of the 2778 study participants separated by abdominal obesity status and BMI are shown in Table 1.

Table 2 shows the association of BMI, WC, current alcohol consumption and duration of alcohol consumption and incident hypertension. After adjustment for age, sex, smoking status, family history of hypertension, HRs of hypertension were 1.76 (1.44–2.14) and 2.12 (1.46–3.10) in overweight and obese subjects respectively, compared with non-obese subjects. HRs of hypertension was 1.64 (1.32–2.03) in abdominal obese subjects, compared with non-abdominal obese subjects, and was 1.65 (1.29–2.12) in current alcohol consumption subjects, compared with non alcohol consumption subjects. Subjects with alcohol consumption for moderate years (1–15 years) and in subjects with alcohol consumption for more than 15 years

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