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ORIGINAL ARTICLE

# Alcohol sensitivity, alcohol use and hypertension in an older Chinese population: the Guangzhou Biobank Cohort Study

Wei Sen Zhang[1], Chao Qiang Jiang[1], Kar Keung Cheng[2], Peymane Adab[2], G Neil Thomas[2], Bin Liu[1], Kin-Bong Hubert Lam[2], C Mary Schooling[3] and Tai Hing Lam[3]

**Although the J-shaped association between alcohol consumption and blood pressure (BP) is well known, the effect of alcohol sensitivity on this relationship is less clear. We studied the association of alcohol sensitivity and alcohol use with BP and hypertension. This cross-sectional analysis included 19 335 older participants from the Guangzhou Biobank Cohort Study recruited from 2003 to 2006, using clinically measured BP and self-reported alcohol use and alcohol sensitivity. Alcohol use was rare in women, in whom light-to-moderate drinkers (<140 g ethanol per week) without alcohol sensitivity had lower systolic and diastolic BPs (mean difference 5.3 (95% CI 3.8–6.9) mm Hg and 1.9 (1.1–2.7) mm Hg, respectively) and a reduced risk of hypertension (0.62 (0.53–0.72)) relative to never drinkers. Similarly, excessive drinkers (⩾140 g ethanol per week) without alcohol sensitivity had a significantly higher systolic and diastolic BP and risk of hypertension than did nondrinkers (mean difference 5.1 (2.8–7.4) mm Hg, 2.7 (1.5–4.0) mm Hg and 34% (8–66%), respectively, for men). These differences were even greater for men with alcohol sensitivity (mean differences 12.0 (8.9–15.2) mm Hg, 6.2 (4.5–7.9) mm Hg and 95% CI (46–159%), respectively). Alcohol sensitivity and alcohol use were both associated with elevated BP and risk of hypertension in an older Chinese population. Alcohol sensitivity may aggravate the effect of drinking on BP. Limiting alcohol use to two drinks per day for men and one drink a day for women may be suitable for East Asians. Reduction of alcohol consumption should be an important public health target.**
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**Keywords:** alcohol sensitivity; alcohol use; blood pressure; Chinese

## INTRODUCTION

Alcohol has been reported to be a common and modifiable risk factor for hypertension,[1] with a J-shaped relation. Light-to-moderate alcohol consumption may be beneficial for reducing the risk of hypertension, although residual confounding cannot be ruled out. In contrast, heavy drinking increases the risk of hypertension.[2–4] The metabolism of alcohol involves the enzymes, alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH), which break down ethanol into acetaldehyde and subsequently into acetic acid.[5,6] Sensitivity to alcohol is mainly because of functional differences in ALDH2 polymorphisms.[6] Alcohol sensitivity is low in people with ALDH2*1/*l, intermediate in those with ALDH2*1/*2 and high in those with ALDH2*2/*2.[7] Most of those who are sensitive to alcohol experience symptoms, such as facial flushing or palpitations, during alcohol consumption.[8] Alcohol sensitivity is more common in East Asian than in Western populations.[8–10] Alcohol sensitivity may modify the effect of alcohol on blood pressure (BP), possibly making alcohol less protective in East Asians, thus altering the appropriate definition of light-to-moderate drinking with corresponding public health implications. Earlier studies are few, small and inconsistent,[11–16] with no evidence from China or Chinese populations. Some studies showed that the effects of alcohol drinking on BP and incidence of hypertension were not different in individuals with high or low levels of ALDH2 activity.[11–14] However, two studies showed that there is a higher risk of hypertension associated with drinking large amounts of alcohol in those who are highly sensitive to alcohol.[15,16] In the Guangzhou Biobank Cohort Study (GBCS), data on drinking, alcohol sensitivity and BP have been collected in detail.[17] We used baseline data from the GBCS to investigate the association between alcohol use, alcohol sensitivity and BP in an older Chinese population.

[1]Guangzhou Number 12 Hospital, Guangzhou, Guangdong, China; [2]Unit of Public Health, Epidemiology and Biostatistics, University of Birmingham, Birmingham, UK and [3]School of Public Health, Li Ka Shing Faculty of Medicine, The University of Hong Kong, Hong Kong SAR, China
Correspondence: Professor K-K Cheng, Public Health, Epidemiology and Biostatistics, School of Health and Population Sciences, University of Birmingham, Birmingham B15 2TT, UK.
E-mail: k.k.cheng@bham.ac.uk
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## METHODS

### Sources of data

The GBCS is a collaborative project between the Guangzhou No. 12 Hospital, Guangzhou, China, the Universities of Hong Kong, Hong Kong, China and of Birmingham, UK, and has been described elsewhere in detail.[17] Briefly, participants underwent a half-day session with a detailed medical interview, from which data on lifestyle habits (such as drinking, smoking and physical activity), socioeconomic position, symptoms after drinking alcohol, disease history and reproductive history were obtained. They also had a full physical examination, including BP assessment. This analysis is based on participants from phases 1 and 2 of the recruitment examined in 2003–2004 and 2005–2006, respectively. The Guangzhou Medical Ethics Committee of the Chinese Medical Association approved the study and all participants gave their written, informed consent.

Of the 20 412 eligible participants, 19 335 (94.7%), who had complete data on all items of interest, were included in the present cross-sectional analysis. The exclusion of 1077 (5.7%) participants was largely because of missing physical activity data ($n$=765, 3.7%), as the International Physical Activity Questionnaire[18] was not introduced into the study until 1 month after the first phase of recruitment started.

### Exposure assessment

The participants were asked about the usual (on a typical occasion or during a typical day) frequency (days per week) and amount (ml) of drinking alcoholic beverages, including beer, grape wine, rice wine and whisky. Mean weekly alcohol consumption (g ethanol per week) was then calculated and categorized into three groups: nondrinkers, light-to-moderate drinkers (LMDs) and excessive drinkers (EDs). Nondrinkers were defined as those who explicitly reported no drinking currently and previously, or drinking less than six times per year. LMDs were those who drank less than once per week (occasional drinking) or who, on an average, drank $<$140 g ethanol per week. EDs were weekly drinkers consuming, on an average, 140 g or more ethanol per week. The latter category could only be used for men as there were very few women who fulfilled the criteria. Self-reported alcohol intake has been found to be reliable and valid in populations.[19]

Alcohol sensitivity was assessed by asking ever drinkers 'After drinking alcohol, do you usually experience facial flushing, palpitation and/or dizziness?' An affirmative response is compatible with having alcohol sensitivity.[15,20]

### Outcome specification of hypertension

Seated BP was measured three times, 1 min apart, after a 3-minute rest, using the Omron 705CP (HEM-705CP-E) sphygmomanometer (Omron Matusaka Co. Ltd., Masusaka, Japan). The average of the last two readings was used. Pulse pressure was defined as the difference between systolic BP and diastolic BP. Mean artery BP was defined as diastolic BP plus one-third of pulse pressure. Hypertension was defined as systolic and/or diastolic BPs $\geqslant$140/90 mm Hg, or being on treatment with medication for hypertension.[3]

### Statistical analysis

Means and 95% confidence intervals (95% CIs) are reported for BP. Number and percentage are reported for categorical variables. Analysis of covariance (ANCOVA) was used to calculate the age-adjusted means. Linear regression was used to assess the age-adjusted linear trend for alcohol sensitivity and amount of alcohol consumption. Logistic regression was used to estimate the risk of hypertension. The potential confounders considered were age, lifestyle habits (smoking and physical activity),[18] socioeconomic position (education and personal annual income) and other vascular risk factors (family history of hypertension, body mass index, waist circumference, total cholesterol, triglyceride, HDL-cholesterol and glucose).

Models were built to investigate the possible confounding effects of each confounder in turn. We present three models: model 1 adjusted for age; model 2 additionally adjusted for lifestyle and socioeconomic position; and model 3 additionally adjusted for other vascular risk factors.

Ex-drinkers are more likely to have developed hypertension or to be receiving antihypertensive medication. Furthermore, reducing excessive alcohol intake can produce a reduction in BP.[4,21] Therefore, ex-drinkers were excluded for analyzing the association between alcohol use, alcohol sensitivity and BP to avoid possible bias.

## RESULTS

There were more women (13 779) than men (5556), and the women were generally younger (mean age 61.5 years (s.d. $\pm$ 6.6)) than the men (mean age 64.4 years (s.d. $\pm$ 6.2)). Male and female drinkers with alcohol sensitivity were slightly younger than drinkers without sensitivity and nondrinkers (Table 1).

Ex-drinkers and current drinkers accounted for 1.0% (135 of 13 779) and 11.1% (1532 of 13 779) in women and 5.1% (286 of 5556) and 35.3% (1962 of 5556) in men, respectively. Among ever drinkers, the prevalence of alcohol sensitivity was 46.0% (1033 of 2248) in men, which was similar to that in women (47.2% (786 of 1667), $P$=0.48). However, the amount of alcohol consumed was greater in men than in women. Among ever drinkers, the proportion of ED in men (30.6%, 688 of 2248) was much higher than that among women (3.3%, 55 of 1667), $P<0.001$. The proportion of ED in those with alcohol sensitivity was lower than that among those without alcohol sensitivity (23.4 *vs.* 36.7% in men and 1.8 *vs.* 4.7% in women, respectively, Table 1).

In addition, the prevalence of smoking was higher in drinkers, with over 80% of male ever-drinkers being ever smokers. The prevalence of ever smoking was significantly higher among men than among women. Most of the participants had had primary to secondary education, with an annual personal income of 10 000–15 000 Yuan (8 Yuan=1 USD), and were physically active. Nondrinkers tended to more likely report a family history of hypertension. Women were more likely than men to have a large waist circumference and elevated total and low-density lipoprotein-cholesterol levels. There were also some minor differences in lipid profile among nondrinkers and ever drinkers, with and without alcohol sensitivity, but no difference in blood glucose levels.

### Alcohol consumption, alcohol sensitivity and BP

Table 2 shows that in men, EDs had higher mean systolic, diastolic, pulse and arterial BPs than did nondrinkers, regardless of alcohol sensitivity, adjusted for age. EDs with alcohol sensitivity had the highest systolic, diastolic, pulse and mean arterial BP. Furthermore, all these BP measures were significantly higher in EDs with alcohol sensitivity than in those without alcohol sensitivity. We additionally stratified EDs into two groups (140–279 and $\geqslant$280 g ethanol per week), and the observations were consistent in each group (Appendix 1). However, BP was almost the same in nondrinkers and in LMDs regardless of alcohol sensitivity. Light-to-moderate drinking was not associated with an elevated BP in men. A stratified analysis by medication for hypertension, the association between alcohol use, alcohol sensitivity and BP remained unchanged (data not shown).

Table 3 shows that, in women, LMDs with or without alcohol sensitivity had significantly lower adjusted mean systolic, diastolic, pulse and arterial BPs than did nondrinkers ($P$-values from 0.006 to $<0.001$). The interquartile ($P_{25}$, $P_{50}$, $P_{75}$) levels of alcohol intake (g ethanol per week) in the LMDs who drank once per week or more were 11, 27 and 54, respectively, in women, and 28, 54 and 84, respectively, in men. In stratified analysis by medication for hypertension, the results were similar (data not shown).

Table 4 shows that the results were similar when the prevalence and adjusted odds ratios (ORs) of hypertension were examined. The prevalence of hypertension was 46.7% (2593 of 5556) in men and 43.8% (6041 of 13 779) in women. Compared with nondrinkers, the prevalence of hypertension was lower in LMDs, both in men and women, and higher in male EDs. Male EDs, especially those with

**Table 1 Sample characteristics by alcohol sensitivity for 19 335 older Chinese men and women in phases 1–2 of The Guangzhou Biobank Cohort Study**

| | *Men* | | | | *Women* | | | |
|---|---|---|---|---|---|---|---|---|
| | | *Alcohol sensitivity*[a] | | | | *Alcohol sensitivity*[a] | | |
| | *Nondrinkers* | *No* | *Yes* | P-*value* | *Nondrinkers* | *No* | *Yes* | P-*value* |
| *n* | 3308 | 1215 | 1033 | | 12112 | 881 | 786 | |
| Proportion, % | 59.5 | 21.9 | 18.6 | <0.001* | 87.9 | 6.4 | 5.7 | — |
| Age, years (s.d.) | 64.6±6.1 | 64.4±6.3 | 63.7±6.4 | <0.001 | 61.6±6.6 | 61.4±6.4 | 60.2±6.8 | <0.001 |
| *Alcohol use, %* | | | | | | | | |
| <140 g ethanol per week | — | 63.3 | 76.6 | <0.001 | — | 95.3 | 98.2 | 0.001 |
| ⩾140 g ethanol per week | — | 36.7 | 23.4 | | — | 4.7 | 1.8 | |
| *Smoking, %* | | | | | | | | |
| Ever smoked | 61.5 | 81.2 | 82.1 | <0.001 | 5.6 | 11.6 | 13.1 | <0.001 |
| *Physical activity* | | | | | | | | |
| Inactive | 5.0 | 5.8 | 4.0 | <0.001 | 4.8 | 3.2 | 3.1 | <0.001 |
| *(IPAQ), %* | | | | | | | | |
| Minimally active | 49.9 | 56.1 | 50.6 | | 48.2 | 48.2 | 56.9 | |
| HEPA[b] active | 45.1 | 38.1 | 45.1 | | 47.0 | 48.6 | 40.1 | |
| *Education, %* | | | | | | | | |
| <Primary | 2.1 | 4.7 | 2.5 | <0.001 | 14.3 | 11.9 | 11.5 | <0.001 |
| Primary | 25.5 | 31.9 | 28.9 | | 38.0 | 34.5 | 31.7 | |
| Secondary | 53.1 | 46.7 | 51.7 | | 42.1 | 47.1 | 50.3 | |
| Tertiary | 19.2 | 16.7 | 16.8 | | 5.6 | 6.5 | 6.6 | |
| *Income group, % (annual)* | | | | | | | | |
| <10 000 Yuan | 18.9 | 23.5 | 22.5 | <0.004 | 43.1 | 41.0 | 36.3 | <0.001 |
| 10 000–15 000 Yuan | 43.7 | 42.6 | 43.6 | | 40.9 | 42.8 | 50.3 | |
| ⩾15000 Yuan | 32.3 | 30.1 | 30.0 | | 10.8 | 12.4 | 10.7 | |
| Unknown | 5.1 | 3.7 | 4.0 | | 5.3 | 3.9 | 2.8 | |
| Family history of hypertension, % | 12.6 | 7.7 | 9.8 | <0.001 | 12.7 | 9.3 | 10.3 | 0.002 |
| *Body mass index (kg m$^{-2}$), %* | | | | | | | | |
| ⩽18.5 | 5.6 | 5.5 | 4.0 | 0.15 | 4.4 | 3.4 | 2.4 | 0.008 |
| 18.5–22.9 | 38.2 | 39.8 | 36.6 | | 37.3 | 36.1 | 34.6 | |
| 23–24.9 | 25.7 | 24.0 | 28.0 | | 24.5 | 22.7 | 26.2 | |
| ⩾25 | 30.5 | 30.6 | 31.5 | | 33.8 | 37.8 | 36.8 | |
| Waist circumference (Male⩾90 cm, Female⩾80 cm), % | 19.8 | 21.7 | 18.9 | 0.21 | 43.0 | 42.2 | 41.5 | 0.65 |
| Total cholesterol (⩾6.2 mmol l$^{-1}$), % | 25.3 | 26.3 | 27.9 | 0.24 | 42.2 | 43.9 | 47.6 | 0.008 |
| Triglyceride (⩾1.7 mmol l$^{-1}$), % | 30.0 | 28.4 | 31.6 | 0.26 | 34.4 | 31.0 | 30.4 | 0.011 |
| HDL-cholesterol (male: <0.9 mmol l$^{-1}$, female: <1.0 mmol l$^{-1}$), % | 1.9 | 2.6 | 3.4 | 0.018 | 1.3 | 2.7 | 0.8 | 0.001 |
| LDL-cholesterol (⩾3.36 mmol l$^{-1}$), % | 22.3 | 27.2 | 27.1 | <0.001 | 38.9 | 47.7 | 51.4 | <0.001 |
| Fasting glucose (⩾6.1 mmol l$^{-1}$), % | 21.4 | 20.9 | 20.9 | 0.91 | 21.4 | 21.6 | 19.5 | 0.43 |

Abbreviations: HDL, high-density lipoprotein; LDL, low-density lipoprotein.
[a]Among former and current drinkers.
[b]Health-enhancing physical activity, that is, vigorous activity at least 3 days a week achieving at least 1500 MET min per week or activity on 7 days of the week achieving at least 3000 MET min per week.
8 Yuan=1 USD.
**P*-value for difference by sex.

alcohol sensitivity, had a higher risk of hypertension, whereas female LMDs had a lower risk of hypertension, especially those without alcohol sensitivity. The association between alcohol use, alcohol sensitivity and hypertension remained consistent when the data were stratified by dividing EDs into two groups (140–279 and ⩾280 g ethanol per week) (Appendix 2).

**Table 2 Association between alcohol use, alcohol sensitivity and BP in older Chinese men (*n*=5270)[a]**

| | | | *Age-adjusted means (95% CI)* | | | |
|---|---|---|---|---|---|---|
| *Amount of drinking* | *Alcohol sensitivity* | n | *Systolic BP* | *Diastolic BP* | *Pulse pressure* | *Mean artery pressure* |
| Nondrinkers | — | 3308 | 133.2 (132.4–133.9) | 76.1 (75.7–76.5) | 57.0 (56.6–57.5) | 95.1 (94.7–95.6) |
| LMD, <140 g/week | No | 698 | 132.0 (130.4–133.6) | 75.7 (74.9–76.5) | 56.3 (55.3–57.3) | 94.5 (93.4–95.5) |
| LMD, <140 g/week | Yes | 719 | 133.2 (131.6–134.7) | 76.4 (75.6–77.2) | 56.8 (55.7–57.8) | 95.3 (94.3–96.3) |
| **ED, ⩾140 g/week** | **No** | **361** | **138.3 (136.1–140.4)***** | **78.8 (77.7–80.0)***** | **59.4 (58.0–60.8)**** | **98.6 (97.2–100.1)***** |
| **ED, ⩾140 g/week** | **Yes** | **184** | **145.2 (142.2–148.2)*****,### | **82.3 (80.7–83.9)*****,## | **62.9 (60.9–64.9)*****,## | **103.3 (101.3–105.3)*****,### |
| *P* for trend | | | <0.001 | <0.001 | <0.001 | <0.001 |

Abbreviations: BP, blood pressure; CI, confidence interval; ED, excessive drinker; LMD, light-to-moderate drinker.
[a]Excludes ex-drinkers.
ED, weekly drinkers drinking ⩾140 g ethanol per week; LMD, drinking <1 time per week or weekly drinking <140 g ethanol per week.
Comparing with nondrinkers: ***P*<0.01, ****P*<0.001; comparing with ED without alcohol sensitivity: ##*P*<0.01, ###*P*<0.001.
Significant *P*-values are in bold.

**Table 3 Association between alcohol use, alcohol sensitivity and BP in older Chinese women (*n*=13 610)[a]**

| | | | *Age-adjusted means (95% CI)* | | | |
|---|---|---|---|---|---|---|
| *Amount of drinking* | *Alcohol sensitivity* | n | *Systolic BP* | *Diastolic BP* | *Pulse pressure* | *Mean artery pressure* |
| Nondrinkers | — | 12 112 | 130.9 (130.5–131.3) | 73.1 (72.9–73.3) | 57.8 (57.5–58.0) | 92.4 (92.1–92.6) |
| **LMD, <140 g per week** | **No** | **782** | **125.5 (124.0–127.0)***** | **71.2 (70.4–71.9)***** | **54.4 (53.4–55.4)***** | **89.3 (88.3–90.3)***** |
| **LMD, <140 g per week** | **Yes** | **716** | **127.8 (126.2–129.3)*****,# | **71.9 (71.1–72.7)**** | **55.9 (54.8–56.9)****,# | **90.5 (89.5–91.5)**** |
| *P*-value | | | <0.001 | <0.001 | <0.001 | <0.001 |

Abbreviations: BP, blood pressure; CI, confidence interval; LMD, light-to-moderate drinker.
[a]Excludes ex-drinkers and currently excessive drinkers, because of limitation of fewer excessive drinker cases in women.
LMD, drinking <1 time per week or weekly drinking <140 g ethanol per week.
Comparing with nondrinkers: ***P*<0.01, ****P*<0.001; comparing with those who were LMD without alcohol sensitivity: #*P*<0.05.
Significant *P*-values are in bold.

**Table 4 Prevalence and adjusted OR of hypertension for alcohol use and alcohol sensitivity in older Chinese men (*n*=5556) and women (*n*=13 779)**

| | | | | *OR (95% CI)*[a] | | |
|---|---|---|---|---|---|---|
| *Amount of drinking* | *Alcohol sensitivity* | n | *Hypertension (%)* | *Model 1* | *Model 2* | *Model 3* |
| *Men* | | | | | | |
| Nondrinkers | — | 3308 | 46.6 | 1.00 | 1.00 | 1.00 |
| LMD(<140 g per week) | No | 769 | 43.6 | 0.90 (0.76–1.05) | 0.93 (0.79–1.10) | 0.96 (0.81–1.14) |
| LMD(<140 g per week) | Yes | 791 | 43.1 | 0.92 (0.78–1.07) | 0.97 (0.82–1.13) | 0.95 (0.81–1.13) |
| **ED(⩾140 g per week)** | **No** | **446** | **51.1** | **1.21 (0.99–1.47)** | **1.30 (1.07–1.59)** | **1.34 (1.08–1.66)** |
| **ED(⩾140 g per week)** | **Yes** | **242** | **61.2** | **1.83 (1.40–2.40)*** | **2.01 (1.53–2.64)**** | **1.95 (1.46–2.59)*** |
| *P*-value | | | <0.001 | <0.001 | <0.001 | <0.001 |
| *Women*[b] | | | | | | |
| Nondrinkers | — | 12112 | 45.2 | 1.00 | 1.00 | 1.00 |
| LMD(<140 g per week) | No | 840 | 33.7 | 0.62 (0.53–0.72) | 0.63 (0.54–0.73) | 0.62 (0.53–0.72) |
| LMD(<140 g per week) | Yes | 772 | 34.7 | 0.71 (0.60–0.83) | 0.72 (0.62–0.85) | 0.69 (0.59–0.82) |
| *P*-value | | | <0.001 | <0.001 | <0.001 | <0.001 |

Abbreviations: CI, confidence interval; ED, excessive drinker; LMD, light-to-moderate drinker; OR, odds ratio.
[a]OR (95% CI)-odds ratio and its 95% CI. Model 1 adjusted for age; model 2 additionally adjusted for education, personal income, smoking status and physical activity; and model 3 additionally adjusted for family history of hypertension, BMI, waist circumference, total cholesterol, triglyceride, HDL-cholesterol and fasting blood glucose.
[b]Excludes ED because of limitation of fewer cases in women.
ED, weekly drinkers drinking ⩾140 g ethanol per week; LMD, drinking <1 time per week or weekly drinking <140 g ethanol per week.
Comparing with those who were ED without alcohol sensitivity, the final adjusted OR of hypertension was 1.45, 95% CI 1.04–2.04 (**P*<0.05, ***P*<0.01).
Significant *P*-values are in bold.

## DISCUSSION

In a large sample from an understudied Chinese population, with adjustment for many confounders, including lifestyle habits, socio-economic status and other vascular risk factors, we found that alcohol sensitivity was associated with higher BP in light-to-medium drinkers compared with that in similar drinkers without alcohol sensitivity, in both men and women. Excessive drinking men with alcohol sensitivity also had higher BP and risk of hypertension than did equivalent EDs without alcohol sensitivity.

Although alcohol-induced flushing is thought to be a deterrent factor to heavy consumption of alcohol,[20,22,23] the frequency of drinking of alcoholic beverages does not always differ between flushers

and nonflushers.[24] Social, psychological and cultural influences may be a better explanation for alcohol use among East Asians than alcohol sensitivity.[25] There is a lack of data describing the association between alcohol sensitivity and hypertension for Chinese or other East Asians, except the Japanese. Studies from Japan have had inconsistent results.[11–16] For example, a study including 4000 people from the general population showed that the ALDH2 genotype does not affect sensitivity to the pressor effects of alcohol in either men or women.[13] However, studies on male workers showed that heavy drinkers with sensitivity to alcohol (slight and visible face and/or skin flushing after drinking) had a higher risk of hypertension.[15,16] Our study also suggests that sensitivity to alcohol may aggravate the effect of excessive alcohol drinking on hypertension. Chronic alcohol intake by alcohol flushers increases blood acetaldehyde levels and thereby raises BP.[15] In women with hypertension, LMDs with alcohol sensitivity had a significantly higher BP (compared with LMDs without alcohol sensitivity) than did women without hypertension. In both men and women, the study showed that light-to-moderate drinking may not reduce BP in those who are sensitive to alcohol.

We found that in men, regular excessive drinking was associated with elevated BP and risk of hypertension, which is consistent with earlier reports.[26–29] However, we could not find a beneficial effect from light-to-moderate drinking. We used the same drinking definitions in women as in an earlier study,[30] but which are slightly higher than the recommendation of limit to one drink ($\sim$12.3 g ethanol) per day proposed by the US Department of Health and Human Services and of Agriculture.[31] We found that light-to-moderate drinking was associated with lower BP and risk of hypertension in women, especially in those without alcohol sensitivity. However, most of the woman alcohol users would have been occasional users, hence it is difficult to ascertain whether this was a real effect of occasional alcohol use or was because of residual confounding from other characteristics of the fairly uncommon woman drinkers (such as more health consciousness or self-restraint). Certainly, levels of alcohol intake in the light-to-moderate female drinkers who drank once per week or more were approximately half of those observed in the men, which may contribute to the observed differences. A prospective cohort study showed a strong positive association between higher alcohol consumption and an increased risk of developing hypertension in men, with no evidence of benefit in light-to-moderate drinking.[3] The risk of hypertension was much higher in men when total alcohol consumption exceeded five drinks per week. However, in women, light-to-moderate alcohol consumption contributed to a modestly lower hypertension risk.[3] In another prospective study, alcohol intake of up to 20 g alcohol per day was not associated with an increased risk of hypertension in women.[32] A population-based study including 5448 US adults showed that alcohol intake of up to two drinks per day had no effect on BP. There was a sex-specific effect of alcohol intake in excess of two drinks per day on BP, with increased BP in men but not in women.[33] Excessive alcohol intake has been shown to be associated with increased arterial stiffness in men.[34] In contrast, higher alcohol use was inversely related to arterial stiffness in women.[35]

A chronic low ethanol intake for LMDs may confer benefits mainly through higher antioxidant capacity and lower advanced glycation end products.[36] There was no beneficial effect of light-to-moderate drinking on BP in men in our study. This could be because most of the male drinkers also smoked, whereas female drinkers did not. In our study, we found that the prevalence of ever smoking in current drinkers was 81.6% in men and 11.3% in women. There is some evidence that the association between alcohol drinking and higher BP is stronger in smokers than in nonsmokers. A Japanese study found that BP was significantly higher in heavy drinkers than in nondrinkers, and these differences tended to be greater in light and heavy smokers than in nonsmokers.[37] Furthermore, the relatively greater prevalence of drinking and the larger amount of alcohol consumption in men (*vs.* women) in China may also, in part, explain the discrepancy in the effect of alcohol intake on BP in men and women.

Alcohol consumption is an established cause of cancer of the mouth, pharynx, larynx, esophagus, liver, colon, rectum and breast.[38] For each of these cancers, risk increases substantially with an intake of more than 50 g of alcohol per day.[38] A regular consumption of one or more drinks per day has been associated with an increased risk of breast cancer in women.[39] Given the evidence from this study and others, it is suggested that people should not drink, but for those who do, alcohol intake should be limited to no more than two drinks per day ($\sim$20–25 g ethanol per day) for men and one drink a day ($\sim$10–15 g ethanol per day) for women.[31] This may be suitable for Chinese adults and other Asians, in whom alcohol sensitivity is common. For the prevention of cancer and many other alcohol-related health problems, ceasing or reducing alcohol consumption should be an urgent public health target.

This is a large study in older Chinese including data on alcohol sensitivity, alcohol consumption, BP and medication for hypertension. The use of a standardized, detailed questionnaire and the measurement of fasting biochemical vascular risk factors enabled the adjustment of many potential confounders. These have allowed us to examine the independent association of alcohol sensitivity and alcohol consumption with BP and hypertension. To the best of our knowledge, this is the first large study on alcohol sensitivity, alcohol use and BP in a Chinese population, which may give evidence for alcohol control, especially for the many developing populations during a period of transition with massive promotion and a consequent rapid increase in alcohol consumption. Despite the strengths, there are some limitations. First, our findings would be biased if people with specific patterns of alcohol use and hypertension risk were systematically excluded, most likely heavy drinkers. However, heavy drinking is rare among Chinese. Second, our study is based on data from older people for whom the social and cultural setting means heavy alcohol use is uncommon, and thus precluded a full exploration of the effects of very high alcohol use, particularly in women. Third, our study from a setting in which rice wine and beer are the main alcoholic beverages means we cannot examine whether grape wine specifically is protective against hypertension; however, this does not detract from the relevance to China and other Asian populations. Fourth, we did not adjust for potential confounding by diet, especially by dietary sodium, which is an independent factor on BP. However, Cantonese are not heavy salt users, so confounding by dietary sodium is likely to be minimal. Moderate alcohol use can be associated with a healthier diet;[40] however, that pattern may be culturally specific. We cannot rule out the possibility that Chinese moderate alcohol users have a particularly unhealthy diet, which may mask a protective effect of moderate alcohol use on BP. Finally, this is a cross-sectional study, with all the inherent limitations, including the inability to determine causality.

In conclusion, excessive drinking with alcohol sensitivity may aggravate the detrimental effects of excessive drinking on BP and hypertension. Light-to-moderate drinking may be associated with reduced BP and risk of hypertension only in women, especially in women who are not sensitive to alcohol. Limiting alcohol consumption for those who drink to two drinks per day for men and one drink a day for women may also be appropriate for East Asians in whom alcohol sensitivity is common. Ceasing or reducing alcohol consumption should be an important public health target.

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1 Whitworth JA, World Health Organization and International Society of Hypertension Writing Group. 2003 World Health Organization (WHO)/International Society of Hypertension (ISH) statement on management of hypertension. *J Hypertens* 2003; **21**: 1983–1992.
2 Sesso HD, Cook NR, Buring JE, Manson JE, Gaziano JM. Alcohol consumption and the risk of hypertension in women and men. *Hypertension* 2008; **51**: 1080–1087.
3 Thadhani R, Camargo Jr CA, Stampfer MJ, Curhan GC, Willett WC, Rimm EB. Prospective study of moderate alcohol consumption and risk of hypertension in young women. *Arch Intern Med* 2002; **162**: 569–574.
4 Okubo Y, Suwazono Y, Kobayashi E, Nogawa K. Alcohol consumption and blood pressure change: 5-year follow-up study of the association in normotensive workers. *J Hum Hypertens* 2001; **15**: 367–372.
5 Asakage T, Yokoyama A, Haneda T, Yamazaki M, Muto M, Yokoyama T, Kato H, Igaki H, Tsujinaka T, Kumagai Y, Yokoyama M, Omori T, Watanabe H. Genetic polymorphisms of alcohol and aldehyde dehydrogenases, and drinking, smoking and diet in Japanese men with oral and pharyngeal squamous cell carcinoma. *Carcinogenesis* 2007; **28**: 865–874.
6 Harada S, Agarwal DP, Goedde HW. Aldehyde dehydrogenase deficiency as cause of facial flushing reaction to alcohol in Japanese. *Lancet* 1981; **2**: 982.
7 Takeshita T, Morimoto K, Mao X, Hashimoto T, Furuyama J. Characterization of the three genotypes of low Km aldehyde dehydrogenase in a Japanese population. *Hum Genet* 1994; **94**: 217–223.
8 Wolff PH. Ethnic differences in alcohol sensitivity. *Science* 1972; **175**: 449–450.
9 Chan AW. Racial differences in alcohol sensitivity. *Alcohol* 1986; **21**: 93–104.
10 Agarwal DP, Harada S, Goedde HW. Racial differences in biological sensitivity to ethanol: the role of alcohol dehydrogenase and aldehyde dehydrogenase isozymes. *Alcohol Clin Exp Res* 1981; **5**: 12–16.
11 Okayama A, Ueshima H, Yamakawa M, Kita Y. Low-Km aldehyde dehydrogenase deficiency does not influence the elevation of blood pressure by alcohol. *J Hum Hypertens* 1994; **8**: 205–208.
12 Tsuritani I, Ikai E, Date T, Suzuki Y, Ishizaki M, Yamada Y. Polymorphism in ALDH2-genotype in Japanese men and the alcohol–blood pressure relationship. *Am J Hypertens* 1995; **8**: 1053–1059.
13 Takagi S, Baba S, Iwai N, Fukuda M, Katsuya T, Higaki J, Mannami T, Ogata J, Goto Y, Ogihara T. The aldehyde dehydrogenase 2 gene is a risk factor for hypertension in Japanese but does not alter the sensitivity to pressor effects of alcohol: the Suita study. *Hypertens Res* 2001; **24**: 365–370.
14 Amamoto K, Okamura T, Tamaki S, Kita Y, Tsujita Y, Kadowaki T, Nakamura Y, Ueshima H. Epidemiologic study of the association of low-Km mitochondrial acetaldehyde dehydrogenase genotypes with blood pressure level and the prevalence of hypertension in a general population. *Hypertens Res* 2002; **25**: 857–864.
15 Itoh T, Matsumoto M, Nakamura M, Okada A, Shirahashi N, Hougaku H, Hashimoto H, Sakaguchi M, Handa N, Takeshita T, Morimoto K, Hori M. Effects of daily alcohol intake on the blood pressure differ depending on an individual's sensitivity to alcohol: oriental flushing as a sign to stop drinking for health reasons. *J Hypertens* 1997; **15**: 1211–1217.
16 Wakabayashi I. Sensitivity of circulatory response to alcohol influences the relationship between alcohol consumption and blood pressure in Orientals. *Blood Press* 2005; **14**: 238–444.
17 Jiang C, Thomas GN, Lam TH, Schooling CM, Zhang W, Lao X, Adab P, Liu B, Leung GM, Cheng KK. Cohort profile: the Guangzhou Biobank Cohort Study, a Guangzhou-Hong Kong-Birmingham collaboration. *Int J Epidemiol* 2006; **35**: 844–852.
18 Deng HB, Macfarlane DJ, Thomas GN, Lao XQ, Jiang CQ, Cheng KK, Lam TH. Reliability and validity of the IPAQ-Chinese: the Guangzhou Biobank Cohort study. *Med Sci Sports Exerc* 2008; **40**: 303–307.
19 Midanik LT. Validity of self-reported alcohol use: a literature review and assessment. *Br J Addict* 1988; **83**: 1019–1030.
20 Takeshita T, Morimoto K. Development of a questionnaire method to discriminate between typical and atypical genotypes of low Km aldehyde dehydrogenase in a Japanese population. *Alcoholism Clin Exp Res* 1998; **22**: 1409–1413.
21 Xin X, He J, Frontini MG, Ogden LG, Motsamai OI, Whelton PK. Effects of alcohol reduction on blood pressure: a meta-analysis of randomized controlled trials. *Hypertension* 2001; **38**: 1112–1117.
22 Harada S, Agarwal DP, Goedde HW, Tagaki S, Ishikawa B. Possible protective role against alcoholism for aldehyde dehydrogenase isozyme deficiency in Japan. *Lancet* 1982; **2**: 827.
23 Thomasson HR, Edenberg HJ, Crabb DW, Mai XL, Jerome RE, Li TK, Wang SP, Lin YT, Lu RB, Yin SJ. Alcohol and aldehyde dehydrogenase genotypes and alcoholism in Chinese men. *Am J Hum Genet* 1991; **48**: 677–681.
24 Yu PH, Fang CY, Dyck LE. Cutaneous vasomotor sensitivity to ethanol and acetaldehyde: subtypes of alcohol-flushing response among Chinese. *Alcohol Clin Exp Res* 1990; **14**: 932–936.
25 Johnson RC, Nagoshi CT. Asians, Asian–Americans and alcohol. *J Psychoactive Drugs* 1990; **22**: 45–52.
26 Wildman RP, Gu D, Muntner P, Huang G, Chen J, Duan X, He J. Alcohol intake and hypertension subtypes in Chinese men. *J Hypertens* 2005; **23**: 737–743.
27 Tsuruta M, Adachi H, Hirai Y, Fujiura Y, Imaizumi T. Association between alcohol intake and development of hypertension in Japanese normotensive men: 12-year follow-up study. *Am J Hypertens* 2000; **13**: 482–487.
28 Yoon YS, Oh SW, Baik HW, Park HS, Kim WY. Alcohol consumption and the metabolic syndrome in Korean adults: the 1998 Korean National Health and Nutrition Examination Survey. *Am J Clin Nutr* 2004; **80**: 217–224.
29 Puddey IB, Beilin LJ. Alcohol is bad for blood pressure. *Clin Exp Pharmacol Physiol* 2006; **33**: 847–852.
30 Schooling CM, Sun W, Ho SY, Chan WM, Tham MK, Ho KS, Leung GM, Lam TH. Moderate alcohol use and mortality from ischaemic heart disease: a prospective study in older Chinese people. *PLoS ONE* 2008; **3**: e2370.
31 Dietary Guidelines for Americans 2005. Available: http://www.health.gov/dietaryguidelines/dga2005/document/pdf/dga2005.pdf [Released on 12 January 2005].
32 Witteman JC, Willett WC, Stampfer MJ, Colditz GA, Kok FJ, Sacks FM, Speizer FE, Rosner B, Hennekens CH. Relation of moderate alcohol consumption and risk of systemic hypertension in women. *Am J Cardiol* 1990; **65**: 633–637.
33 McFarlane SI, von Gizycki H, Salifu M, Deshmukh M, Manieram M, Gebreegziabher Y, Gliwa AA, Bordia S, Shah S, Sowers JR. Alcohol consumption and blood pressure in the adult US population: assessment of gender-related effects. *J Hypertens* 2007; **25**: 965–970.
34 Kurihara T, Tomiyama H, Hashimoto H, Yamamoto Y, Yano E, Yamashina A. Excessive alcohol intake increases the risk of arterial stiffening in men with normal blood pressure. *Hypertens Res* 2004; **27**: 669–673.
35 Sierksma A, Lebrun CE, van der Schouw YT, Grobbee DE, Lamberts SW, Hendriks HF, Bots ML. Alcohol consumption in relation to aortic stiffness and aortic wave reflections: a cross-sectional study in healthy postmenopausal women. *Arterioscler Thromb Vasc Biol* 2004; **24**: 342–348.
36 Vasdev S, Gill V, Singal PK. Beneficial effect of low ethanol intake on the cardiovascular system: possible biochemical mechanisms. *Vasc Health Risk Manag* 2006; **2**: 263–276.
37 Wakabayashi I. Modification of the association of alcohol drinking with blood pressure by cigarette smoking. *Blood Press* 2008; **17**: 87–93.
38 Breast and colorectal cancers are associated with alcohol consumption, says IARC. Available: http://www.iarc.fr/en/media-centre/pr/2007/pr175.html [Released on 28 March 2007].
39 Hamajima N, Hirose K, Tajima K, Rohan T, Calle EE, Heath Jr CW, Coates RJ, Liff JM, Talamini R, Chantarakul N, Koetsawang S, Rachawat D, Morabia A, Schuman L, Stewart W, Szklo M, Bain C, Schofield F, Siskind V, Band P, Coldman AJ, Gallagher RP, Hislop TG, Yang P, Kolonel LM, Nomura AM, Hu J, Johnson KC, Mao Y, De Sanjosé S, Lee N, Marchbanks P, Ory HW, Peterson HB, Wilson HG, Wingo PA, Ebeling K, Kunde D, Nishan P, Hopper JL, Colditz G, Gajalanski V, Martin N, Pardthaisong T, Silpisornkosol S, Theetranont C, Boosiri B, Chutivongse S, Jimakorn P, Virutamasen P, Wongsrichanalai C, Ewertz M, Adami HO, Bergkvist L, Magnusson C, Persson I, Chang-Claude J, Paul C, Skegg DC, Spears GF, Boyle P, Evstifeeva T, Daling JR, Hutchinson WB, Malone K, Noonan EA, Stanford JL, Thomas DB, Weiss NS, White E, Andrieu N, Brêmond A, Clavel F, Gairard B, Lansac J, Piana L, Renaud R, Izquierdo A, Viladiu P, Cuevas HR, Ontiveros P, Palet A, Salazar SB, Aristizabel N, Cuadros A, Tryggvadottir L, Tulinius H, Bachelot A, Lê MG, Peto J, Franceschi S, Lubin F, Modan B, Ron E, Wax Y, Friedman GD, Hiatt RA, Levi F, Bishop T, Kosmelj K, Primic-Zakelj M, Ravnihar B, Stare J, Beeson WL, Fraser G, Bullbrook RD, Cuzick J, Duffy SW, Fentiman IS, Hayward JL, Wang DY, McMichael AJ, McPherson K, Hanson RL, Leske MC, Mahoney MC, Nasca PC, Varma AO, Weinstein AL, Moller TR, Olsson H, Ranstam J, Goldbohm RA, van den Brandt PA, Apelo RA, Baens J, de la Cruz JR, Javier B, Lacaya LB, Ngelangel CA, La Vecchia C, Negri E, Marubini E, Ferraroni M, Gerber M, Richardson S, Segala C, Gatei D, Kenya P, Kungu A, Mati JG, Brinton LA, Hoover R, Schairer C, Spirtas R, Lee HP, Rookus MA, van Leeuwen FE, Schoenberg JA, McCredie M, Gammon MD, Clarke EA, Jones L, Neil A, Vessey M, Yeates D, Appleby P, Banks E, Beral V, Bull D, Crossley B, Goodill A, Green J, Hermon C, Key T, Langston N, Lewis C, Reeves G, Collins R, Doll R, Peto R, Mabuchi K, Preston D, Hannaford P, Kay C, Rosero-Bixby L, Gao YT, Jin F, Yuan JM, Wei HY, Yun T, Zhiheng C, Berry G, Cooper Booth J, Jelihovsky T, MacLennan R, Shearman R, Wang QS, Baines CJ, Miller AB, Wall C, Lund E, Stalsberg H, Shu XO, Zheng W, Katsouyanni K, Trichopoulou A, Trichopoulos D, Dabancens A, Martinez L, Molina R, Salas O, Alexander FE, Anderson K, Folsom AR, Hulka BS, Bernstein L, Enger S, Haile RW, Paganini-Hill A, Pike MC, Ross RK, Ursin G, Yu MC, Longnecker MP, Newcomb P, Bergkvist L, Kalache A, Farley TM, Holck S, Meirik O, Collaborative Group on Hormonal Factors in Breast Cancer. Alcohol, tobacco and breast cancer—collaborative reanalysis of individual data from 53 epidemiological studies, including 58 515 women with breast cancer and 95 067 women without the disease. *Br J Cancer* 2002; **87**: 1234–1245.
40 Ruidavets JB, Bataille V, Dallongeville J, Simon C, Bingham A, Amouyel P, Arveiler D, Ducimetière P, Ferrières J. Alcohol intake and diet in France, the prominent role of lifestyle. *Eur Heart J* 2004; **25**: 1153–1162.

## APPENDIX 1

### Association between alcohol use, alcohol sensitivity and BP in older Chinese men ($n$=5270)[a]

| | | | *Age-adjusted means (95% CI)* | | | |
|---|---|---|---|---|---|---|
| *Amount of drinking* | *Alcohol sensitivity* | n | *Systolic BP* | *Diastolic BP* | *Pulse pressure* | *Mean artery pressure* |
| Nondrinkers | — | 3308 | 133.2 (132.4–133.9) | 76.1 (75.7–76.5) | 57.0 (56.6–57.5) | 95.1 (94.7–95.6) |
| LMD, <140 g per week | No | 698 | 132.0 (130.4–133.6) | 75.7 (74.9–76.5) | 56.3 (55.3–57.3) | 94.5 (93.4–95.5) |
| LMD, <140 g per week | Yes | 719 | 133.2 (131.6–134.7) | 76.4 (75.6–77.2) | 56.8 (55.7–57.8) | 95.3 (94.3–96.3) |
| **ED, 140–279 g per week** | **No** | **149** | **133.8 (130.4–137.1)** | **78.5 (76.6–80.3)*** | **55.3 (53.1–57.6)** | **96.9 (94.7–99.1)** |
| **ED, 140–279 g per week** | **Yes** | **75** | **142.5 (137.8–147.3)***,## | **80.9 (78.4–83.5)***** | **61.6 (58.5–64.8)**,## | **101.5 (98.3–104.6)***,# |
| **ED, ⩾280 g per week** | **No** | **212** | **141.4 (138.6–144.2)***** | **79.1 (77.6–80.7)***** | **62.3 (60.4–64.2)***** | **99.9 (98.0–101.7)***** |
| **ED, ⩾280 g per week** | **Yes** | **109** | **147.0 (143.1–151.0)***,§ | **83.2 (81.1–85.4)***,§ | **63.8 (61.2–66.4)***** | **104.5 (101.9–107.1)***,§§ |
| *P* for trend | | | <0.001 | <0.001 | <0.001 | <0.001 |

Abbreviations: BP, blood pressure; CI, confidence interval; ED, excessive drinker; LMD, light-to-moderate drinker.
[a]Excludes ex-drinkers.
ED, weekly drinkers drinking ⩾140 g ethanol per week; LMD, drinking <1 time per week or weekly drinking <140 g ethanol per week.
Comparing with nondrinkers: *$P$<0.05, **$P$<0.01, ***$P$<0.001; comparing with those who were ED (140–279 g per week) without alcohol sensitivity: #$P$<0.05, ##$P$<0.01; comparing with those who were ED (⩾280 g per week) without alcohol sensitivity: §$P$<0.05, §§$P$<0.01.
Significant *P*-values are in bold.

## APPENDIX 2

### Prevalence and adjusted OR of hypertension for alcohol use and alcohol sensitivity in older Chinese men ($n$=5556)

| | | | | *OR (95% CI)*[a] | | |
|---|---|---|---|---|---|---|
| *Amount of drinking* | *Alcohol sensitivity* | n | *Hypertension (%)* | *Model 1* | *Model 2* | *Model 3* |
| Nondrinkers | — | 3308 | 46.6 | 1.00 | 1.00 | 1.00 |
| LMD (<140 g per week) | No | 769 | 43.6 | 0.90 (0.76–1.05) | 0.93 (0.79–1.10) | 0.96 (0.81–1.14) |
| LMD (<140 g per week) | Yes | 791 | 43.1 | 0.92 (0.78–1.07) | 0.97 (0.82–1.13) | 0.95 (0.81–1.13) |
| **ED, 140–279 g per week** | **No** | **179** | **46.9** | **1.00 (0.74–1.36)** | **1.08 (0.80–1.47)** | **1.14 (0.82–1.58)** |
| **ED, 140–279 g per week** | **Yes** | **92** | **66.3** | **2.22 (1.43–3.45)**** | **2.39 (1.54–3.73)**** | **2.44 (1.54–3.86)**** |
| ED, ⩾280 g per week | No | 267 | 53.9 | 1.37 (1.06–1.76) | 1.47 (1.13–1.90) | 1.49 (1.14–1.96) |
| ED, ⩾280 g per week | Yes | 150 | 58.0 | 1.64 (1.17–2.29) | 1.81 (1.29–2.54) | 1.69 (1.18–2.42) |
| *P*-value | | | <0.001 | <0.001 | <0.001 | <0.001 |

Abbreviations: CI, confidence interval; ED, excessive drinker; LMD, light-to-moderate drinker; OR, odds ratio.
[a]OR (95% CI)-odds ratio and its 95% CI. Model 1 adjusted for age; model 2 additionally adjusted for education, personal income, smoking status and physical activity; and model 3 additionally adjusted for family history of hypertension, BMI, waist circumference, total cholesterol, triglyceride, HDL-cholesterol and fasting blood glucose.
ED, weekly drinkers drinking ⩾140 g ethanol per week; LMD, drinking <1 time per week or weekly drinking <140 g ethanol per week.
Comparing with those who were ED (140–279 g per week) without alcohol sensitivity, the final adjusted OR of hypertension was 2.14, 95% CI 1.24–3.71 (**$P$<0.01).
Significant *P*-values are in bold.