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Data Article

# Data on alcohol consumption and coronary artery calcification among asymptomatic middle-aged men for the ERA-JUMP study

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*Abbreviations:* BMI, body mass index; CAC, coronary artery calcification; CHD, coronary heart disease; CRP, C-reactive protein; CVD, cardiovascular diseases; EBCT, electron beam computed tomography; the ERA JUMP Study, the EBCT risk factor assessment among Japanese and the United States (US) men in the post-World-War-II birth cohort; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol

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ABSTRACT

Data presented in this article are supplementary data to our primary article 'Association of Alcohol Consumption and Aortic Calcification in Healthy Men Aged 40–49 Years for the ERA JUMP Study' [1]. In this article, we have presented supplementary tables showing the independent association of alcohol consumption with coronary artery calcification using Tobit conditional regression and ordinal logistic regression.



**Specification Table**

| | |
|---|---|
| Subject area | Medicine |
| More specific subject area | Cardiology- subclinical atherosclerosis |
| Type of data | Tables |
| How data was acquired | Physical examination (weight, height, systolic and diastolic blood pressure etc.), a lifestyle questionnaire (smoking, alcohol consumption, physical activity, medications etc.), and a laboratory assessment (Serum lipids, glucose, C-reactive proteins, fibrinogen etc.), coronary artery calcification measured by Electron Beam Computed Tomography (a GE-Imatron C150 Electron Beam Tomography scanner, GE Medical Systems, South San Francisco, US) |
| Data format | Analyzed |
| Experimental factors | Association between alcohol consumption and coronary artery calcification using SAS version 9.4 (SAS Institute, Cary, North Carolina) and STATA version 14.0 (StataCorp LP, College Station, TX, US) |
| Experimental features | Population-based cross-sectional study |
| Data source location | Pittsburgh, PA, USA; Honolulu, Hawaii, USA; Kusatsu City, Shiga, Japan |
| Data accessibility | Data is with this article |

**Value of the data**

- Pathophysiological mechanisms underlying the J-shaped relationship between alcohol consumption and CHD are not completely understood.
- Data concerning alcohol consumption and atherosclerosis are scarce.
- In this cross-sectional study, the heavy alcohol consumption was positively associated with CAC.
- Mechanisms other than the reduced deposition of calcium in the atherosclerotic lesions may be responsible for the beneficial association of light to moderate alcohol consumption with CHD.
- This data may be useful for scientists interested in exploring the mechanisms underlying the association between alcohol and CHD.

## 1. Data

The tables presented in the current article are supplementary material to our primary article 'Association of Alcohol Consumption and Aortic Calcification in Healthy Men Aged 40–49 Years for the ERA JUMP Study' [1]. In an international population-based cross-sectional study, we have assessed the relationship between alcohol consumption and CAC among middle-aged asymptomatic men. Tables 1-1 and 2-1 (Tobit conditional regression models) and 1–2 and 2-2 (ordinal logistic regression models) present the overall as well as race/ethnicity-stratified association of alcohol consumption with CAC. The results showed that the heavy alcohol consumption was positively and significantly associated with CAC after adjusting for cardiovascular risk factors.

## 2. Experimental design, materials, and methods

### 2.1. Study population

An international study, the ERA JUMP, was initiated between 2002 to 2006 to assess the prevalence and risk factors associated with subclinical atherosclerosis among 300 Japanese men in Kusatsu, Japan, 300 US White and 100 Black men in Pittsburgh, US, and 300 Japanese American men in Honolulu, US. The ERA-JUMP study enrolled men aged 40–49 years old, free of clinical CVD or other severe diseases. In Japan, participants were randomly selected using basic residents' register. In Pittsburgh, White and Black study participants were randomly selected from the voter registration list. In Honolulu, study participants were randomly selected from the offspring of the members of the Honolulu Heart Program cohort [2].

### 2.2. Experimental design, materials, and methods

Following standardized protocols, information from study participants was obtained using a lifestyle questionnaire, physical examination, and a laboratory assessment. Data were collected on body weight, height, blood pressure, heart rate, smoking, use of medications (antihypertensive, antidiabetic, and lipid-lowering), meat intake, physical activity related to the current job, and alcohol consumption. Collected blood samples were stored at -70°C and shipped on dry ice from all the centers to the University of Pittsburgh. Total cholesterol, HDL-C, and triglycerides were determined using the protocol standardized by the Centers for Disease Control and Prevention [3]. The Friedewald equation was used to calculate LDL-C [4]. Serum sample were also used to measure glucose (a hexokinase-glucose-6-phosphate-dehydrogenase enzymatic assay), CRP (a calorimetric-competitive-enzyme-linked-immuno-sorbent assay), and fibrinogen (an automated-clot-rate assay). Using standardized protocol across all centers, CAC was evaluated by EBCT using a GE-Imatron C150 Electron Beam Tomography scanner (GE Medical Systems, South San Francisco, US) [2,5] and quantified using the Agatston method [6].

## 3. Statistical analysis

Tobit conditional regression and ordinal logistic regression were used to model the association of alcohol consumption and CAC after adjusting for potential confounders and intermediary variables. Alcohol consumption was categorized into four groups: 0 (non-drinkers), $\leq 1$ (light drinkers), $>1$ to $\leq 3$ (moderate drinkers) and $>3$ drinks per day (heavy drinkers) (1 drink = 12.5 g of ethanol). For Tobit regression, outcome variable was natural log of (CAC $+1$). For ordinal logistic regression, we used four CAC score categories: 0–9, 10–99, 100–299 and $\geq 300$. For Tobit regression and ordinal regression, Model I was adjusted for age, race/ethnicity, and the years of education; Model II was further adjusted for potential confounders (pack-years of smoking, BMI, diabetes, lipid-lowering medications, physical activity at job, meat intake, LDL-C, and CRP); Model III was additionally adjusted for intermediary variables (hypertension, HDL-C, triglycerides, and fibrinogen) in the relation

**Table 1-1**
Tobit regression describing the association between alcohol consumption and coronary artery calcification for the ERA-JUMP Study [Reference category= non-drinkers (never + former drinkers)].

| **Alcohol Categories All Participants (n = 1006)** | **Nondrinkers** | **Light Drinkers** | **Moderate Drinkers** | **Heavy Drinkers** | |
|---|---|---|---|---|---|
| n (%) | 258 (25.7) | 355 (35.3) | 236 (23.5) | 157 (15.6) | |
| Mean CAC score | 25.8 | 24.9 | 37.3 | 41.0 | |
| | **TR** | **TR (95% CI)** | **TR (95% CI)** | **TR (95% CI)** | ***p*-trend**[a] |
| Unadjusted | 1.00 | 0.77 (0.42, 1.40) | 0.48 (0.25, 0.95) | 1.07 (0.51, 2.24) | 0.82/0.03 |
| Model I | 1.00 | 0.99 (0.55, 1.77) | 0.72 (0.38, 1.37) | 2.22 (1.07, 4.58) | 0.07/0.02 |
| Model II | 1.00 | 1.18 (0.68, 2.07) | 0.98 (0.52, 1.82) | 2.75 (1.36, 5.56) | 0.01/0.07 |
| Model III | 1.00 | 1.16 (0.66, 2.05) | 0.99 (0.53, 1.87) | 2.37 (1.11, 5.08) | 0.03/0.12 |
| **US White (n = 301)** | | | | | |
| n (%) | 57 (18.9) | 162 (53.8) | 71(23.6) | 11(3.7) | |
| Mean CAC score | 35.7 | 23.1 | 17.5 | 26.7 | |
| | **TR** | **TR (95% CI)** | **TR (95% CI)** | **TR (95% CI)** | ***p*-trend**[a] |
| Model II | 1.00 | 1.06 (0.44, 2.57) | 0.68 (0.24, 1.94) | 0.79 (0.10, 6.04) | 0.68/0.96 |
| Model III | 1.00 | 0.95 (0.39, 2.34) | 0.65 (0.22, 1.89) | 0.68 (0.08, 5.60) | 0.63/0.95 |
| **Japanese in Japan (n = 310)** | | | | | |
| n (%) | 53(17.10) | 82(26.45) | 81(26.13) | 94(30.32) | |
| Mean CAC score | 10.8 | 5.3 | 1.87 | 25.2 | |
| | **TR** | **TR (95% CI)** | **TR (95% CI)** | **TR (95% CI)** | ***p*-trend**[a] |
| Model II | 1.00 | 0.76 (0.22, 2.56) | 0.39 (0.11, 1.39) | 2.05 (0.63, 6.61) | 0.44/0.02 |
| Model III | 1.00 | 0.62 (0.17, 2.18) | 0.38 (0.10, 1.39) | 1.71 (0.48, 6.09) | 0.57/0.02 |
| **Japanese American (n = 292)** | | | | | |
| n (%) | 113 (38.7) | 75 (25.7) | 59(20.2) | 45(15.4) | |
| Mean CAC score | 32.9 | 57.7 | 104.0 | 69.5 | |
| | **TR** | **TR (95% CI)** | **TR (95% CI)** | **TR (95% CI)** | ***p*-trend**[a] |
| Model II | 1.00 | 1.71 (0.55, 5.32) | 2.11 (0.63, 7.06) | 3.51 (0.95, 12.97) | 0.06/0.98 |
| Model III | 1.00 | 1.79 (0.58, 5.59) | 2.13 (0.63, 7.17) | 2.15 (0.51, 9.09) | 0.22/0.66 |

TR: Tobit ratio; CI: confidence interval; CAC: coronary artery calcification;
Model I: Alcohol consumption, age, race/ethnicity, and years of education;
Model II: Model I + pack-years of smoking, BMI, diabetes, anti-lipid medication, job physical activity, meat intake, LDL-C, and CRP;
Model III: Model II + HDL-C, triglycerides, hypertension, and fibrinogen;

[a] *p-trend* shows *p*-value for linear and quadratic trend across the alcohol consumption categories calculated using contrast.

**Table 1–2**
Ordinal logistic regression describing the association between alcohol consumption and coronary artery calcification for the ERA-JUMP Study [Reference category= non-drinkers (never + former drinkers)].

| **Alcohol Categories All Participants (n = 1006)** | **Non-drinkers** | **Light Drinkers** | **Moderate Drinkers** | **Heavy Drinkers** | |
|---|---|---|---|---|---|
| n (%) | 258 (25.6) | 355 (35.3) | 236 (23.5) | 157 (15.6) | |
| Mean CAC score | 25.8 | 24.8 | 37.3 | 41.0 | |
| | **OR** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | ***p*-trend**[a] |
| Unadjusted | 1.00 | 0.93 (0.64, 1.36) | 0.85 (0.56, 1.31) | 1.28 (0.82, 2.01) | 0.32/0.60 |
| Model I | 1.00 | 1.15 (0.76, 1.73) | 1.13 (0.72, 1.77) | 2.14 (1.31, 3.50) | 0.74/0.03 |
| Model II | 1.00 | 1.31 (0.86, 2.00) | 1.34 (0.84, 2.14) | 2.39 (1.43, 4.00) | 0.75/0.01 |
| Model III | 1.00 | 1.30 (0.85, 2.00) | 1.36 (0.85, 2.19) | 2.25 (1.29, 3.93) | 0.69/0.02 |
| **Race/Ethnicity-Stratified Analyses** | | | | | |
| **US White (n = 301)** | | | | | |
| n (%) | 57 (18.9) | 162 (53.8) | 71(23.6) | 11(3.6) | |
| Mean CAC score | 35.7 | 23.1 | 17.5 | 26.7 | |
| | **OR** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | ***p*-trend**[a] |
| Model II | 1.00 | 1.10 (0.53, 2.28) | 1.09 (0.46, 2.57) | 0.80 (0.15, 4.41) | 0.67/0.80 |
| Model III | 1.00 | 1.08 (0.52, 2.25) | 1.16 (0.48, 2.81) | 0.95 (0.17, 5.42) | 0.76/0.98 |
| **Japanese in Japan (n = 310)** | | | | | |
| n (%) | 53 (17.1) | 82 (26.5) | 81 (26.1) | 94 (30.3) | |
| Mean CAC score | 10.7 | 5.3 | 1.9 | 25.2 | |
| | **OR** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | ***p*-trend**[a] |
| Model II | 1.00 | 0.75 (0.23, 2.42) | 0.26 (0.06, 1.15) | 1.84 (0.64, 5.30) | 0.14/0.61 |
| Model III | 1.00 | 0.70 (0.21, 2.40) | 0.22 (0.05, 1.05) | 1.73 (0.54, 5.50) | 0.12/0.52 |
| **Japanese American (n = 292)** | | | | | |
| n (%) | 113 (38.7) | 75 (25.7) | 59 (20.2) | 45 (15.4) | |
| Mean CAC score | 32.9 | 57.7 | 104.0 | 69.5 | |
| | **OR** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | ***p*-trend**[a] |
| Model II | 1.00 | 1.93 (0.98, 3.79) | 2.31 (1.14, 4.68) | 2.69 (1.26, 5.72) | 0.11/0.03 |
| Model III | 1.00 | 1.94 (0.97, 3.87) | 2.32 (1.12, 4.78) | 2.21 (0.97, 5.07) | 0.08/0.09 |

OR: odds ratio; CI: confidence interval; CAC: coronary artery calcification;
Model I: Alcohol consumption, age, race/ethnicity, and years of education
Model II: Model I + pack-years of smoking, BMI, diabetes, anti-lipid medication, job physical activity, meat intake, LDL-C, and CRP
Model III: Model II + HDL-C, triglycerides, hypertension, and fibrinogen

[a] *p- trend* shows *p*-value for linear and quadratic trend across the alcohol consumption categories calculated using contrast.

**Table 2-1**
Tobit regression describing the association between alcohol consumption and coronary artery calcification score for the ERA-JUMP Study [Reference category= never drinkers].

| **Alcohol Categories All Participants (n = 914)** | **Never-drinkers** | **Light Drinkers** | **Moderate Drinkers** | **Heavy Drinkers** | |
|---|---|---|---|---|---|
| n (%) | 166 (18.2) | 355 (38.8) | 236 (25.8) | 157 (17.2) | – |
| Mean CAC score | 24.2 | 24.8 | 37.3 | 41.0 | – |
| | **TR** | **TR (95% CI)** | **TR (95% CI)** | **TR (95% CI)** | ***p*-trend**[a] |
| Unadjusted | 1.00 | 0.87 (0.43, 1.74) | 0.54 (0.25, 1.16) | 1.21 (0.53, 2.76) | 0.94/0.07 |
| Model I | 1.00 | 1.01 (0.51, 2.00) | 0.72 (0.35, 1.49) | 2.27 (1.03, 5.03) | 0.09/0.03 |
| Model II | 1.00 | 1.25 (0.66, 2.38) | 1.05 (0.52, 2.12) | 3.02 (1.40, 6.52) | 0.01/0.10 |
| Model III | 1.00 | 1.23 (0.64, 2.37) | 1.06 (0.52, 2.17) | 2.60 (1.14, 5.96) | 0.03/0.17 |
| **Race/Ethnicity-Stratified Analyses** | | | | | |
| **US White (n = 276)** | | | | | |
| n (%) | 32 (11.6) | 162 (58.7) | 71 (25.7) | 11 (4.0) | – |
| Mean CAC score | 56.5 | 23.1 | 17.5 | 26.7 | – |
| | **TR** | **TR (95% CI)** | **TR (95% CI)** | **TR (95% CI)** | ***p*-trend**[a] |
| Model II | 1.00 | 0.77 (0.26, 2.32) | 0.50 (0.14, 1.73) | 0.55 (0.06, 4.71) | 0.47/0.79 |
| Model III | 1.00 | 0.65 (0.21, 2.07) | 0.45 (0.12, 1.69) | 0.48 (0.08, 4.54) | 0.45/0.72 |
| **Japanese in Japan (n = 305)** | | | | | |
| n (%) | 48 (15.74) | 82 (26.9) | 81 (26.6) | 94 (30.8) | |
| Mean CAC score | 11.0 | 5.3 | 1.9 | 25.2 | |
| | **TR** | **TR (95% CI)** | **TR (95% CI)** | **TR (95% CI)** | ***p*-trend**[a] |
| Model II | 1.00 | 0.92 (0.26, 3.32) | 0.47 (0.13, 1.80) | 2.51 (0.73, 8.63) | 0.30/0.04 |
| Model III | 1.00 | 0.77 (0.20, 2.91) | 0.45 (0.12, 1.76) | 1.99 (0.53, 7.48) | 0.45/0.04 |
| **Japanese American (n = 250)** | | | | | |
| n (%) | 71 (28.4) | 75 (30.0) | 59 (23.6) | 45 (18.0) | – |
| Mean CAC score | 22.9 | 57.7 | 104.0 | 69.5 | – |
| | **TR** | **TR (95% CI)** | **TR (95% CI)** | **TR (95% CI)** | ***p*-trend**[a] |
| Model II | 1.00 | 1.96 (0.56, 6.86) | 2.61 (0.69, 9.89) | 4.74 (1.13, 19.97) | 0.03/0.96 |
| Model III | 1.00 | 1.99 (0.57, 6.96) | 2.48 (0.65, 9.45) | 2.98 (0.61, 14.51) | 0.13/0.70 |

TR: Tobit ratio; CI: confidence interval; CAC: coronary artery calcification;
Model I: Alcohol consumption, age, race/ethnicity, and years of education;
Model II: Model I + pack-years of smoking, BMI, diabetes, anti-lipid medication, job physical activity, meat intake, LDL-C, and CRP;
Model III: Model II + HDL-C, triglycerides, hypertension, and fibrinogen;

[a] *p trend* shows *p*-value for linear and quadratic trend across the alcohol consumption categories calculated using contrast.

**Table 2-2**
Ordinal logistic regression describing the association between alcohol consumption and coronary artery calcification for the ERA-JUMP Study [Reference category= never drinkers].

| **Alcohol categories All Participants (n=914)** | **Never-drinkers** | **Light Drinkers** | **Moderate Drinkers** | **Heavy Drinkers** | |
|---|---|---|---|---|---|
| n (%) | 166 (18.2) | 355 (38.8) | 236 (25.8) | 157 (17.2) | |
| Mean CAC score | 24.2 | 24.9 | 37.3 | 41.0 | |
| | **OR** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | ***p*-trend**[a] |
| Unadjusted | 1.00 | 1.01 (0.65, 1.57) | 0.92 (0.57, 1.50) | 1.38 (0.84, 2.29) | 0.59/0.47 |
| Model I | 1.00 | 1.20 (0.75, 1.93) | 1.17 (0.71, 1.95) | 2.20 (1.28, 3.80) | 0.91/0.03 |
| Model II | 1.00 | 1.38 (0.84, 2.25) | 1.42 (0.83, 2.41) | 2.53 (1.43, 4.49) | 0.07/0.01 |
| Model III | 1.00 | 1.40 (0.85, 2.31) | 1.47 (0.85, 2.53) | 2.44 (1.32, 4.51) | 0.55/0.02 |
| **Race/Ethnicity-Stratified Analyses** | | | | | |
| **US White (n = 276)** | | | | | |
| n (%) | 32 (11.6) | 162 (58.7) | 71 (25.7) | 11 (4.0) | – |
| Mean CAC score | 56.5 | 23.1 | 17.5 | 26.7 | – |
| | **OR** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | ***p*-trend**[a] |
| Model II | 1.00 | 0.91 (0.38, 2.20) | 0.92 (0.34, 2.47) | 0.66 (0.11, 3.88) | 0.98/0.67 |
| Model III | 1.00 | 0.96 (0.38, 2.41) | 1.05 (0.37, 3.01) | 0.89 (0.14, 5.63) | 0.99/0.98 |
| **Japanese in Japan (n = 305)** | | | | | |
| n (%) | 48 (15.7) | 82 (26.9) | 81 (26.6) | 94 (30.8) | – |
| Mean CAC score | 11.0 | 5.3 | 1.9 | 25.2 | – |
| | **OR** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | ***p*-trend**[a] |
| Model II | 1.00 | 0.77 (0.23, 2.62) | 0.28 (0.06, 1.29) | 1.93 (0.64, 5.83) | 0.18/0.69 |
| Model III | 1.00 | 0.73 (0.20, 2.63) | 0.23 (0.05, 1.15) | 1.76 (0.53, 5.89) | 0.16/0.56 |
| **Japanese American (n = 250)** | | | | | |
| n (%) | 71 (28.4) | 75 (30.0) | 59 (23.6) | 45 (18.0) | – |
| Mean CAC score | 22.9 | 57.7 | 104.0 | 69.5 | – |
| | **OR** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | ***p*-trend**[a] |
| Model II | 1.00 | 2.08 (0.99, 4.48) | 2.58 (1.15, 5.76) | 3.18 (1.35, 7.47) | 0.11/0.02 |
| Model III | 1.00 | 2.16 (0.98, 4.73) | 2.63 (1.16, 5.98) | 2.68 (1.07, 6.78) | 0.07/0.06 |

OR: odds ratio; CI: confidence interval; CAC: coronary artery calcification;
Model I: Alcohol consumption, age, race/ethnicity, and years of education;
Model II: Model I + pack-years of smoking, BMI, diabetes, anti-lipid medication, job physical activity, meat intake, LDL-C, and CRP;
Model III: Model II + HDL-C, triglycerides, hypertension, and fibrinogen;

[a] *p- trend* shows *p*-value for linear and quadratic trend across the alcohol consumption categories calculated using contrast.

between alcohol consumption and atherosclerosis/CHD. In model III, we tested the interaction between race/ethnicity and alcohol consumption on CAC. We also conducted the race/ethnicity-stratified analysis. All p-values were two-tailed and p-value $< 0.05$ was considered as significant.

## Transparency document. Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.dib.2018.02.032.

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