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## Association of soda consumption with subclinical cardiac remodeling in the Framingham heart study<sup>☆</sup>



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### ABSTRACT

**Objective.** Diet soda consumption increases cardiometabolic risk. The aim of this investigation was to assess the relations between self-reported soda consumption and subclinical cardiac remodeling.

**Methods.** We assessed the relations between self-reported soda consumption and left ventricular mass (LVM) and left atrial dimension (LAD) (both standardized within sex) in a sample of middle-aged attendees from the Framingham Heart Offspring cohort examination 5 and 6.

**Results.** The overall mean age was 55 years and 59% of the participants were women. Compared to non-consumers ( $n = 1010$ ), soda consumers ( $n = 3192$ ) had greater body weight (mean 86 vs. 82 kg among men, and 70 vs. 67 kg among women). Compared with non-consumers, age- and height-adjusted LAD was increased (standard deviation units) among soda consumers by 0.15 standard error 0.042, ( $p < 0.001$ ) for those drinking  $>0-7$  diet soda ( $n = 1023$ ),  $-0.010$  (0.043,  $p = 0.82$ ) for people drinking  $>0-7$  regular soda ( $n = 907$ ), 0.22 (0.057,  $p < 0.0001$ ) for individuals consuming  $>7$  diet soda ( $n = 372$ ), and 0.20 (0.092,  $p = 0.034$ ) for participants drinking  $>7$  regular soda ( $n = 116$ ) per week. LVM was increased among participants consuming diet soda ( $p < 0.05$ ), but not in regular soda consumers ( $p > 0.05$ ). Upon adjustment for weight, however, all aforementioned associations were attenuated.

**Conclusion.** The observed associations between soda consumption and LAD or LVM were likely related to the greater body weight of soda drinkers relative to non-drinkers.

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Abbreviations: BMI, Body mass index; CVD, Cardiovascular disease; LAD, Left atrial end-systolic dimension; LVM, Left ventricular mass.

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## 1. Introduction

Consumption of both sugar and aspartame-containing beverages has increased rapidly during the last couple of decades [1]. During the same time period, the prevalence and incidence of the metabolic syndrome, atrial fibrillation, and heart failure have increased as well [2]. The temporal increases in the prevalence and incidence of these conditions have been particularly pronounced among younger individuals and women [3–5], who often are the greatest consumers of soda. Soda consumption previously has been shown to increase the risk of developing the metabolic syndrome [4,6], but the relations between soda consumption and cardiovascular disease (CVD) have been less well investigated. Two recent epidemiological studies reported increased risks of stroke associated with diet soda, but not with regular soda consumption, whereas another study showed increased risk of coronary heart disease and adverse changes in metabolic risk factors among people consuming sugar-, but not artificially-sweetened soda beverages [7–9]. In order to explore one potential mechanism by which soda consumption might lead to CVD, we assessed the association between soda consumption and subclinical cardiac remodeling (using left ventricular mass [LVM] and left atrial [LA] size as endophenotypes for CVD and atrial fibrillation, respectively) in a middle-aged to older adult community-based sample of individuals free from overt CVD.

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## 2. Material and Methods

The Framingham Heart Study is a prospective community-based observational study aiming to investigate risk factors for CVD. The participants of the original cohort were enrolled in 1948. In 1971 the children of the original cohort and the spouses of the children were enrolled into the Offspring cohort [10]. For the present investigation, we used a pooled sample of participants from the 5th (1991–1995) and 6th (1995–1998) quadrennial examination cycles of the Offspring cohort when routine echocardiography was performed and information on soda consumption was collected ( $n = 7331$  person-observations). We excluded participants with prevalent CVD (including significant valve disease, prior acute myocardial infarction, clinical heart failure, atrial fibrillation, and/or cerebrovascular disease,  $n = 790$ ), inadequate echocardiograms ( $n = 1715$ ), incomplete data on soda consumption ( $n = 579$ ), or other important covariates ( $n = 45$ ), leaving 4202 person-observations for analyses. Of these 896 people contributed data from one examination cycle, and 1653 from two examination cycles.

Consumption of soda was based on self-reported questionnaires. Questions included how many soda drinks the participants consumed in a week and distinguished use of diet from regular soda. For the present purpose, weekly soda consumption was divided into six groups: “non-consumers” ( $n = 1010$ ), “consumption of >0–7” ( $n = 1023$  diet and  $n = 907$  regular consumers), or “>7” diet/regular soda drinks ( $n = 372$  diet and  $n = 116$  regular consumers) and a “mixed group” ( $n = 774$ ) including participants who consumed both diet and regular soda (see Table 1). All participants had fasting blood samples

drawn at the Framingham Heart Study clinic as well as standardized measures of anthropometric variables.

### 2.1. Echocardiograms

Experienced sonographers performed transthoracic echocardiographic examination of all participants using a standardized protocol. Measurements of left ventricular size and left atrial end-systolic dimensions (LAD) were performed from M-mode echocardiograms obtained from the parasternal long axis view. For calculation of left ventricular mass, we used the following formula:  $\text{LV mass (LVM)} = 0.8 (1.04 ([\text{LVIDD} + \text{PWTD} + \text{IVSTD}]^3 - [\text{LVIDD}]^3)) + 0.6 \text{ g}$ , where LVIDD is LV end-diastolic dimension, PWTD left ventricular posterior wall thickness, and IVSTD interventricular septum thickness.

### 2.2. Ethics

The study was approved by the Institutional Review Board of the Boston University Medical Center. All participants provided written informed consent before participation.

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## 3. Calculations

Multivariable linear regression models were used to estimate the associations between soda consumption category and different echocardiographic variables. LVM and LAD were included as dependent variables in analyses after being standardized within sex. All models were adjusted for age and height initially. In subsequent steps, we additionally adjusted for: 1) weight, and 2) systolic blood pressure, anti-hypertensive treatment, smoking, dyslipidemia, and diabetes. We repeated all analyses using generalized estimation equation models, which account for the dependence between observations and unless specified, similar results were observed. All analyses were performed in SAS version 9.3 (Cary, NC, USA). A two-sided  $p$ -value  $< 0.05$  was considered statistically significant for all analyses.

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## 4. Results

The overall mean age was 55 years and 59% of the participants were women. Twenty-four percent of the participants did not consume any soda, whereas 33% consumed diet soda only, 25% regular soda only, and 18% consumed both diet and regular soda. Soda consumers were younger, had a higher body mass index, and lower serum high-density lipoprotein concentrations than non-consumers (Table 1). Moreover, the total daily energy intake was higher for the regular and high diet soda consumption groups compared to the soda non-consumer group. The prevalence of diabetes was also higher in the high diet soda consumption group.

### 4.1. Association between soda consumption and cardiac remodeling

All of the soda consumption groups, except for the >0–7 regular soda intake per week group, were associated with

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