Dietary Sodium Content, Mortality, and Risk for Cardiovascular Events in Older Adults: The Health, Aging, and Body Composition (Health ABC) Study

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Original Investigation

Dietary Sodium Content, Mortality, and Risk for Cardiovascular Events in Older Adults
The Health, Aging, and Body Composition (Health ABC) Study

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IMPORTANCE Additional information is needed about the role of dietary sodium on health outcomes in older adults.

OBJECTIVE To examine the association between dietary sodium intake and mortality, incident cardiovascular disease (CVD), and incident heart failure (HF) in older adults.

DESIGN, SETTING, AND PARTICIPANTS We analyzed 10-year follow-up data from 2642 older adults (age range, 71-80 years) participating in a community-based, prospective cohort study (inception between April 1, 1997, and July 31, 1998).

EXPOSURES Dietary sodium intake at baseline was assessed by a food frequency questionnaire. We examined sodium intake as a continuous variable and as a categorical variable at the following levels: less than 1500 mg/d (291 participants [11.0%]), 1500 to 2300 mg/d (779 participants [29.5%]), and greater than 2300 mg/d (1572 participants [59.5%]).

MAIN OUTCOMES AND MEASURES Adjudicated death, incident CVD, and incident HF during 10 follow-up years. Analysis of incident CVD was restricted to 1981 participants without prevalent CVD at baseline.

RESULTS The mean (SD) age of participants was 73.6 (2.9) years, 51.2% were female, 61.7% were of white race, and 38.3% were black. After 10 years, 881 participants had died, 572 had developed CVD, and 398 had developed HF. In adjusted Cox proportional hazards regression models, sodium intake was not associated with mortality (hazard ratio [HR] per 1 g, 1.03; 95% CI, 0.98-1.09; P = .27). Ten-year mortality was nonsignificantly lower in the group receiving 1500 to 2300 mg/d (30.7%) than in the group receiving less than 1500 mg/d (33.8%) and the group receiving greater than 2300 mg/d (35.2%) (P = .07). Sodium intake of greater than 2300 mg/d was associated with nonsignificantly higher mortality in adjusted models (HR vs 1500-2300 mg/d, 1.15; 95% CI, 0.99-1.35; P = .07). Indexing sodium intake for caloric intake and body mass index did not materially affect the results. Adjusted HRs for mortality were 1.20 (95% CI, 0.93-1.54; P = .16) per milligram per kilocalorie and 1.11 (95% CI, 0.96-1.28; P = .17) per 100 mg/kg/m² of daily sodium intake. In adjusted models accounting for the competing risk for death, sodium intake was not associated with risk for CVD (subHR per 1 g, 1.03; 95% CI, 0.95-1.11; P = .47) or HF (subHR per 1 g, 1.00; 95% CI, 0.92-1.08; P = .92). No consistent interactions with sex, race, or hypertensive status were observed for any outcome.

CONCLUSIONS AND RELEVANCE In older adults, food frequency questionnaire–assessed sodium intake was not associated with 10-year mortality, incident CVD, or incident HF, and consuming greater than 2300 mg/d of sodium was associated with nonsignificantly higher mortality in adjusted models.


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Excess dietary sodium intake is associated with risk factors for cardiovascular disease (CVD) and heart failure (HF), most prominently with elevated blood pressure\(^1\)–\(^3\) but also with worse renal function,\(^4\)–\(^6\) left ventricular hypertrophy,\(^7\)–\(^9\) and increased arterial stiffness.\(^10\)–\(^12\) Therefore, limiting sodium intake might be an important intervention to reduce risk for CVD and HF.

Based on the effects of sodium reduction on blood pressure and the current levels of sodium consumption, investigators have projected substantial benefits on outcomes with stricter dietary sodium control.\(^13\)–\(^15\) These projections are based on extrapolation from investigations with high baseline sodium intake (>3000 mg/d) and assume neutral or beneficial effects on other risk factors.\(^16\) Also, except for a recent modeling study,\(^17\) the differential blood pressure–lowering effect of sodium restriction in prehypertensive and hypertensive vs normotensive populations was not taken into account. Strict dietary sodium control may unfavorably modulate insulin resistance,\(^17\)–\(^18\) serum lipid levels,\(^19\) and neurohormonal activity,\(^19\)–\(^20\) factors that predispose to CVD and HF. The uncertain net effect of these opposing forces is highlighted by findings in recent observational studies,\(^21\)–\(^24\) including a multinational cohort of more than 100 000 participants\(^24\) and a meta-analysis,\(^25\) all of which suggest a J-shaped or U-shaped association between dietary sodium intake and outcomes.

In addition to the concerns raised by these reports, inadequate caloric intake and medication interactions are additional concerns associated with very low sodium intake in older adults.\(^26\)–\(^28\) Data on sodium restriction are scarce in this population, especially for those with blood pressure at target. Also, achieving sodium intake of less than 1500 mg/d is difficult, particularly in older adults with long-held dietary habits.\(^29\) Therefore, the incremental benefit of restricting sodium intake to lower targets (<1500 mg/d) instead of the general recommendation of 2300 mg/d would need to be prospectively evaluated, as suggested by a recent Institute of Medicine report.\(^30\) In this direction, data from cohort studies can provide useful insights and facilitate the design of future prospective studies.

In this study, we investigated the association between dietary sodium intake, as assessed in year 2 with a food frequency questionnaire (FFQ), and risk for (1) all-cause mortality, (2) incident CVD, and (3) incident HF in the Health, Aging, and Body Composition (Health ABC) Study using subsequent 10-year follow-up data. In secondary analyses, we (1) evaluated for interactions with sex, race, and hypertensive status at baseline and (2) repeated analyses with sodium intake indexed for body mass index and total caloric intake.

**Methods**

**Study Population**

The institutional review boards at the University of Pittsburgh, the University of Tennessee, Knoxville, and the University of California, San Francisco, approved the Health ABC protocol. All participants provided written informed consent. The Health ABC Study enrolled 3075 well-functioning, community-dwelling individuals 70 to 79 years old between April 1, 1997, and July 31, 1998. Participants were identified from a random sample of Medicare beneficiaries of white race and all age-eligible black community residents in designated zip code areas surrounding Pittsburgh, Pennsylvania, and Memphis, Tennessee. Exclusion criteria included difficulties with walking, stair-climbing, or activities of daily living, as well as obvious cognitive impairment and inability to communicate. At the year 2 visit, our study baseline participants were asked to complete an FFQ. Data on sodium intake were available for 2713 of 2732 participants (99.3%) who were alive and attended the year 2 visit. This analysis includes data on 2642 participants (age range, 71–80 years); we excluded 63 participants with known HF at year 2 (assuming these participants received education on a low sodium diet) and 8 participants with implausibly low sodium intake values (<300 mg/d). Complete adjudicated 10-year follow-up data were available for 2628 of 2642 participants (99.5%). For incident CVD analyses, we considered only the subset of 1981 participants without prevalent CVD at baseline.

**Food Frequency Questionnaire**

Food intake was recorded at year 2 (at the first annual follow-up visit) with a 108-item FFQ designed specifically for the Health ABC Study by Block Dietary Data Systems (Berkeley, California) and based on reported intakes of non-Hispanic white and black residents of the Northeast and South 65 years or older in the Third National Health and Nutrition Examination Survey. The reference period was the preceding year. A trained interviewer administered the FFQ, and interviews were periodical monitored to assure quality and consistency. Woodblocks, real food models, and flash cards were used to help participants estimate portion sizes. Nutrient and food group intakes were determined by Block Dietary Data Systems. The Block Dietary Data Systems family of FFQs has undergone extensive validation, including early investigations in middle-aged and older adults\(^31\) and, more recently, validation of caloric and sodium components against 24-hour recalls in later versions for special populations.\(^32\)

**Study Definitions**

Race was self-defined. Hypertensive status was defined as self-reported history of hypertension, accompanied by the use of antihypertensive medications. Diabetes mellitus was based on self-report or the use of antidiabetic medications. Smoking was classified as current, past (≥100 lifetime cigarettes), or never. Exercise and lifestyle activities were summarized in kilocalories per week using a standardized questionnaire designed by the Health ABC Study. Major electrocardiogram abnormalities included (1) conduction defects, (2) irregular rhythm, (3) left ventricular hypertrophy, and (4) Q-wave or major T-wave and ST-segment abnormalities. Minor abnormalities included minor ST-segment or T-wave abnormalities. Prevalent CVD was defined as (1) coronary heart disease (history of myocardial infarction, angina treated with medications, or coronary revascularization), (2) cerebrovascular disease (history of stroke, transient ischemic attack, or carotid endarterectomy), or (3) peripheral vascular disease (history of intermittent claudication or vascular bypass or angioplasty) at...
year 2. These definitions follow those used in previous Health ABC Study publications,33,34

**Study Outcomes**
Surveillance was conducted by in-person examination alternating with a telephone interview every 6 months. The Health ABC Study Diagnosis and Disease Ascertainment Committee reviewed all hospital records, death certificates, informant interviews, and autopsy data to adjudicate immediate and underlying causes of death. A panel of clinicians verified diagnoses based on hospital records, interviews, and death certificates. Medical records for overnight hospitalizations were reviewed at each site by local adjudicators. Cardiovascular disease events were identified and adjudicated using the surveillance and adjudication process described above. Incident CVD was defined as new (1) coronary heart disease (myocardial infarction, angina, or coronary revascularization), (2) cerebrovascular disease (stroke, transient ischemic attack, or symptomatic carotid artery disease), (3) peripheral arterial disease, or (4) death owing to cardiovascular causes. Incident HF was defined as a first admission with overnight stay confirmed to be related to HF based on symptoms, signs, chest radiographs, and echocardiographic findings using criteria similar to those of the Cardiovascular Health Study.35-36

**Statistical Analysis**
We examined sodium intake as a continuous variable and as a categorical variable using the recommendation-level cutoff points (ie, <1500, 1500-2300, and >2300 mg/d).30 We examined for nonlinear associations using restricted cubic splines.37 For baseline characteristics, we used year 2 values whenever available; smoking status, physical activity, electrocardiogram findings, and creatinine and albumin levels were carried over from year 1. To examine the association between dietary sodium intake and 10-year mortality, we used Cox proportional hazards regression models. We adjusted for demographics and factors previously associated with mortality in the Health ABC Study,34 including age, sex, race, baseline hypertensive status, body mass index, smoking status, physical activity, prevalent CVD, pulmonary disease, diabetes mellitus, depression, blood pressure, heart rate, electrocardiogram abnormalities, and glucose, albumin, creatinine, and cholesterol levels. Covariate values were complete in 98.3% of participants. Confidence intervals were calculated with bootstrapping (normal-based with 1000 replications). Proportionality of hazards was evaluated with Schoenfeld residuals. The power to detect a 20% increase in mortality risk per 1 g of sodium intake (assuming a linear association) was 80.5% at 2-sided a = .05. For CVD, we used the competing risks model by Fine and Gray38 in the subset of participants without prevalent CVD at baseline to account for competing noncardiovascular death. We adjusted for the risk factors described above, which include risk factors for CVD previously identified in the Health ABC Study.34-39 We followed the same approach for HF risk (with death as a competing risk) and adjusted for the same risk factors, which include previously identified HF risk predictors in the Health ABC Study.40 Proportional hazards in competing risks models were evaluated using interaction terms with time. We repeated analyses, entering continuous sodium intake indexed for (1) body mass index (indexed sodium intake) and (2) total caloric intake (sodium density). In secondary analyses, we evaluated (1) the association between dietary sodium intake and self-reported appetite grade (on a 5-point Likert-type scale ranging from very good to very poor) and (2) self-reported adoption of a low-salt diet at the year 2 visit, as well as potential confounding effects from these characteristics. In exploratory analyses, we examined the association between dietary sodium intake and outcomes using alternative binary definitions for high intake (3000 and 4000 mg/d). Analyses were performed with statistical software (STATA 12.1; StataCorp LP).

**Results**

**Baseline Characteristics**
The mean (SD) age of 2642 cohort participants was 73.6 (2.9) years, 51.2% were female, 61.7% were of white race, and 38.3% were black. The baseline characteristics according to sodium intake are summarized in Table 1. More sodium was consumed by men (median, 2850 mg/d; 25th to 75th percentile, 2140-3640 mg/d) than by women (median, 2320 mg/d; 25th to 75th percentile, 1760-2950 mg/d) (P < .001). Participants of white race and participants with diabetes mellitus consumed more sodium, whereas participants with hypertension had lower intake. Greater sodium intake was associated with higher albumin and creatinine levels but with lower cholesterol levels.

**Mortality**
After 10 years, 881 participants had died (Kaplan-Meier mortality, 33.7%). The association between dietary sodium intake and 10-year mortality was approximately linear; although the hazard ratios (HRs) for mortality per 1 g of sodium intake were 1.09 (95% CI, 1.04-1.16; P = .001) in the crude model and 1.03 (95% CI, 0.98-1.09; P = .27) in the adjusted model. The main confounder was sex, attenuating the unadjusted estimate by 48.4%. The crude association was stronger in women and in participants without hypertension (P = .07 and P = .10, respectively, for interaction), but these trends were attenuated in adjusted models (P = .48 and P = .11, respectively, for interaction). No evidence of differential association was observed across races. No association in subgroups retained significance in adjusted models.

Mortality rates were 33.8%, 30.7%, and 35.2% among participants consuming less than 1500, 1500 to 2300, and greater than 2300 mg/d of sodium, respectively (P = .07, log-rank test) (Figure 2). In crude models, sodium intake exceeding 2300 mg/d was associated with higher mortality than sodium intake of 1500 to 2300 mg/d, driven by the higher risk in women, blacks, and participants without hypertension (Table 2). However, none of these findings retained significance in adjusted models.
When sodium intake was indexed for caloric intake (sodium density), the results were not materially different: HRs per milligram per kilocalorie were 1.34 (95% CI, 1.06-1.70; P = .01) in the crude model and 1.20 (95% CI, 0.93-1.54; P = .16) in the adjusted model. No significant interaction with major subgroups of interest was observed. Indexing sodium intake for body mass index yielded similar results: HRs per 100 mg/kg/m² daily sodium were 1.36 (95% CI, 1.19-1.55; P < .001) in the crude model and 1.11 (95% CI, 0.96-1.28; P = .17) in the adjusted model, without significant interactions.

### Incident CVD
Among 1981 participants without CVD at baseline, 572 (28.9%) had developed CVD after 10 follow-up years. The lin-

### Table 1. Baseline Participant Characteristics According to Baseline Dietary Sodium Intake

<table>
<thead>
<tr>
<th>Variable</th>
<th>&lt;1500 mg/d (n = 291)</th>
<th>1500-2300 mg/d (n = 779)</th>
<th>&gt;2300 mg/d (n = 1572)</th>
<th>P Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>74.4 (2.9)</td>
<td>74.6 (2.9)</td>
<td>74.6 (2.9)</td>
<td>.17</td>
</tr>
<tr>
<td>Male sex, No. (%)</td>
<td>88 (30.2)</td>
<td>315 (40.4)</td>
<td>887 (56.4)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Race, No. (%)</td>
<td></td>
<td></td>
<td></td>
<td>.03</td>
</tr>
<tr>
<td>Black</td>
<td>137 (47.1)</td>
<td>285 (36.6)</td>
<td>590 (37.5)</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>154 (52.9)</td>
<td>494 (63.4)</td>
<td>982 (62.5)</td>
<td></td>
</tr>
<tr>
<td>Body mass index, mean (SD)</td>
<td>27.7 (5.0)</td>
<td>27.2 (4.8)</td>
<td>27.1 (4.8)</td>
<td>.14</td>
</tr>
<tr>
<td>Indexed daily sodium, median (25th to 75th percentile), g/kg/m²</td>
<td>45 (36-52)</td>
<td>71 (62-83)</td>
<td>120 (99-148)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Smoking status, No. (%)</td>
<td></td>
<td></td>
<td></td>
<td>.91</td>
</tr>
<tr>
<td>Current smoker</td>
<td>32 (11.0)</td>
<td>65 (8.3)</td>
<td>149 (9.5)</td>
<td></td>
</tr>
<tr>
<td>Past smoker</td>
<td>131 (45.0)</td>
<td>359 (46.1)</td>
<td>728 (46.3)</td>
<td></td>
</tr>
<tr>
<td>Physical activity, median (25th to 75th percentile), kcal/kg/wk</td>
<td>65 (40-102)</td>
<td>68 (40-108)</td>
<td>66 (39-110)</td>
<td>.42</td>
</tr>
<tr>
<td>Total caloric intake, median (25th to 75th percentile), kcal/d</td>
<td>940 (790-1090)</td>
<td>1400 (1230-1600)</td>
<td>2130 (1800-2590)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Dietary sodium density, median (25th to 75th percentile), mg/kcal</td>
<td>1.24 (1.12-1.45)</td>
<td>1.37 (1.22-1.52)</td>
<td>1.50 (1.36-1.66)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Comorbidity, No. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>55 (18.9)</td>
<td>140 (18.0)</td>
<td>284 (18.1)</td>
<td>.81</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>27 (9.3)</td>
<td>61 (7.8)</td>
<td>103 (6.6)</td>
<td>.07</td>
</tr>
<tr>
<td>Peripheral vascular disease</td>
<td>17 (5.8)</td>
<td>35 (4.5)</td>
<td>78 (5.0)</td>
<td>.79</td>
</tr>
<tr>
<td>Any cardiovascular disease</td>
<td>74 (25.4)</td>
<td>203 (26.1)</td>
<td>384 (24.4)</td>
<td>.49</td>
</tr>
<tr>
<td>Pulmonary disease</td>
<td>39 (13.8)</td>
<td>81 (10.4)</td>
<td>180 (11.5)</td>
<td>.69</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>38 (13.1)</td>
<td>138 (17.7)</td>
<td>297 (18.9)</td>
<td>.03</td>
</tr>
<tr>
<td>Hypertension</td>
<td>167 (57.4)</td>
<td>427 (54.8)</td>
<td>803 (51.1)</td>
<td>.02</td>
</tr>
<tr>
<td>Uncontrolled hypertension</td>
<td>94 (32.3)</td>
<td>214 (27.5)</td>
<td>419 (26.7)</td>
<td>.08</td>
</tr>
<tr>
<td>Depression</td>
<td>31 (10.7)</td>
<td>75 (9.6)</td>
<td>163 (10.4)</td>
<td>.88</td>
</tr>
<tr>
<td>Blood pressure, mean (SD), mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>136 (22)</td>
<td>133 (20)</td>
<td>134 (21)</td>
<td>.36</td>
</tr>
<tr>
<td>Diastolic</td>
<td>71 (12)</td>
<td>70 (11)</td>
<td>70 (12)</td>
<td>.43</td>
</tr>
<tr>
<td>Heart rate, mean (SD), beats/min</td>
<td>64.7 (9.5)</td>
<td>64.7 (10.7)</td>
<td>64.9 (11.1)</td>
<td>.98</td>
</tr>
<tr>
<td>Electrocardiogram, No. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major abnormality</td>
<td>56 (19.2)</td>
<td>172 (22.1)</td>
<td>326 (20.7)</td>
<td>.96</td>
</tr>
<tr>
<td>Minor abnormality</td>
<td>57 (19.6)</td>
<td>129 (16.6)</td>
<td>260 (16.5)</td>
<td>.30</td>
</tr>
<tr>
<td>Laboratory test level, mean (SD)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucose, mg/dL</td>
<td>97.8 (23.8)</td>
<td>102.3 (31.3)</td>
<td>101.0 (29.6)</td>
<td>.02</td>
</tr>
<tr>
<td>Albumin, g/dL</td>
<td>3.96 (0.30)</td>
<td>3.98 (0.32)</td>
<td>4.00 (0.31)</td>
<td>.04</td>
</tr>
<tr>
<td>Creatinine, mg/dL</td>
<td>1.04 (0.37)</td>
<td>1.04 (0.40)</td>
<td>1.05 (0.37)</td>
<td>.007</td>
</tr>
<tr>
<td>Total cholesterol, mg/dL</td>
<td>212 (38)</td>
<td>208 (39)</td>
<td>204 (38)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Antihypertensive medication, No. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>β-Blocker</td>
<td>45 (15.5)</td>
<td>107 (13.7)</td>
<td>230 (14.6)</td>
<td>.99</td>
</tr>
<tr>
<td>ACE inhibitor or ARB</td>
<td>57 (19.6)</td>
<td>135 (17.3)</td>
<td>319 (20.3)</td>
<td>.31</td>
</tr>
<tr>
<td>Calcium channel inhibitor</td>
<td>70 (24.1)</td>
<td>209 (26.8)</td>
<td>333 (21.2)</td>
<td>.02</td>
</tr>
<tr>
<td>Thiazide diuretic</td>
<td>64 (22.0)</td>
<td>161 (20.7)</td>
<td>285 (18.1)</td>
<td>.06</td>
</tr>
<tr>
<td>Potassium-sparing diuretic</td>
<td>38 (13.1)</td>
<td>80 (10.3)</td>
<td>117 (7.4)</td>
<td>.001</td>
</tr>
<tr>
<td>Loop diuretic</td>
<td>19 (6.5)</td>
<td>36 (4.6)</td>
<td>91 (5.8)</td>
<td>.88</td>
</tr>
</tbody>
</table>

Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker. 
S to conversion factors: To convert glucose level to millimoles per liter, multiply by 0.0555; albumin level to grams per liter, multiply by 10; creatinine level to micromoles per liter, multiply by 88.4; and cholesterol level to millimeters per liter, multiply by 0.0259. 
*Nonparametric test for trend. 
1 Calculated as weight in kilograms divided by height in meters squared. 
2 Daily dietary sodium intake divided by body mass index (calculated as weight in kilograms divided by height in meters squared). 
3 Daily dietary sodium intake divided by daily caloric intake. 
4 Systolic blood pressure of 140 mm Hg or higher or diastolic blood pressure of 90 mm Hg or higher. 
5 Major Q-wave or QS-wave abnormality, major ST-segment or T-wave abnormality, left ventricular hypertrophy, or ventricular conduction defect. 
6 Minor Q-wave or QS-wave abnormality or ST-segment or T-wave abnormality.
ear form best represented the association between dietary sodium intake and CVD. Taking the competing risk for death into account, the subHR (sHR) for CVD per 1 g of sodium intake was 1.09 (95% CI, 1.01-1.16; \( P = .02 \)) in the crude model and 1.03 (95% CI, 0.95-1.11; \( P = .47 \)) in the adjusted model. The main confounder was sex, attenuating the unadjusted estimate by 61.0%. No differential association in subgroups was observed (Table 3).

The cumulative incidence rates of CVD during 10 years, accounting for competing mortality, were 28.5%, 28.2%, and 29.7% for less than 1500, 1500 to 2300, and greater than 2300 mg/d of sodium intake, respectively. Using the category of 1500 to 2300 mg/d as reference (lowest incidence), adjusted sHRs were 1.11 (95% CI, 0.77-1.61; \( P = .57 \)) for less than 1500 mg/d and 1.08 (95% CI, 0.86-1.36; \( P = .52 \)) for greater than 2300 mg/d intake.

In sodium density models, sHRs per milligram per kilocalorie of sodium for CVD were 1.15 (95% CI, 0.85-1.56; \( P = .36 \)) in the crude model and 1.00 (95% CI, 0.72-1.38; \( P = .98 \)) in the adjusted model. In body mass index sodium models, sHRs per 100 mg/kg/m^2 of sodium were 1.13 (95% CI, 0.94-1.35; \( P = .20 \)) in the crude model and 1.10 (95% CI, 0.90-1.34; \( P = .36 \)) in the adjusted model.

### Incident HF
Among 2642 participants (no participant had HF at baseline by design), 398 (15.1%) had developed HF after 10 follow-up years. The association between dietary sodium intake and HF was linear. The sHRs for HF per 1 g of sodium intake were 1.03 (95% CI, 0.95-1.12; \( P = .50 \)) in the crude model and 1.00 (95% CI, 0.92-1.08; \( P = .92 \)) in the adjusted model, accounting for competing mortality. No differential association in subgroups was observed (Table 3).

The cumulative incidence rates of HF during 10 years, accounting for competing mortality, were 15.7%, 14.3%, and 15.5% for less than 1500, 1500 to 2300, and greater than 2300 mg/d of sodium, respectively. Using the category of 1500 to 2300 mg/d as reference (lowest incidence), adjusted sHRs were 1.11 (95% CI, 0.77-1.61; \( P = .57 \)) for less than 1500 mg/d and 1.08 (95% CI, 0.86-1.36; \( P = .52 \)) for greater than 2300 mg/d intake.

In sodium density models, sHRs per milligram per kilocalorie of sodium for HF were 1.28 (95% CI, 0.87-1.89; \( P = .21 \)) in the crude model and 1.03 (95% CI, 0.70-1.51; \( P = .89 \)) in the adjusted model. In body mass index sodium models, sHRs per 100 mg/kg/m^2 of sodium were 0.94 (95% CI, 0.75-1.18; \( P = .60 \)) in the crude model and 1.00 (95% CI, 0.80-1.25; \( P = .99 \)) in the adjusted model.

### Self-reported Appetite and Self-reported Adoption of a Low-Salt Diet
At the year 2 visit, participants rated their appetite on a 5-point Likert-type scale (a higher grade represented a worse appetite). Among 2637 of 2642 responders (99.8%), only 3.5% rated their appetite as poor (3.0%) or very poor (0.5%). The correlation between sodium intake and appetite grade was significant but weak (Spearman rank correlation \( p = -0.05, P = .009 \)). Worse appetite was independently associated with higher mortality and HF but not with CVD risk. However, appetite grade did not confound or modify the association between dietary sodium intake and the outcomes of interest.

Based on year 2 questionnaire data, 538 of 2642 participants (20.4%) reported adoption of a low-salt diet. However, sodium intake among low-salt diet adopters (median, 2520 mg; 25th to 75th percentile, 1900-3370 mg) was similar to that among nonadopters (median, 2540 mg; 25th to 75th percentile, 1920-3330 mg) \( (P = .69) \). Self-reported low-salt diet was not associated with mortality risk \( (adjusted HR, 0.94; 95\% CI, 0.79-1.12; P = .50) \), CVD risk \( (adjusted sHR, 1.11; 95\% CI, 0.90-1.38; P = .34) \), or HF risk \( (adjusted sHR, 1.16; 95\% CI, 0.91-1.48; P = .22) \) and did not confound or modify the association between dietary sodium intake and these outcomes.

### Alternative Definitions of High Sodium Intake
In exploratory analyses, we examined 2 alternative binary definitions for high sodium intake (3000 and 4000 mg) \( (Table 4) \). Sodium intake exceeding 4000 mg was associated with more consistent estimates for mortality and incident CVD and HF risk in unadjusted analyses. However, the strength of the association was considerably dampened in adjusted analyses.

### Discussion
In our study, we observed no association between FFQ-determined dietary sodium intake and 10-year mortality or incident CVD and HF among older adults participating in the Health ABC Study. Compared with baseline sodium intake of 1500 to 2300 mg/d, no signal of benefit was observed with less than 1500 mg/d of sodium intake. However, a signal for potential harm was observed with greater than 2300 mg/d of sodium intake, driven mainly by women and black participants, but this finding needs further confirmation because of nonsignificance and multiple subgroup testing. Also, no signal was observed for an association between dietary sodium intake and incident CVD and HF in this older adult population.
Adults 65 years or older comprise 13% of the US population but account for most CVD and HF cases. By 2050, this population segment is projected to double, accompanied by almost doubling of new CVD cases and 50% more HF cases. Sodium intake of less than 1500 mg/d is recommended for adults 51 years or older as a means to prevent CVD, although this recommendation has been debated. In an analysis from the Third National Health and Nutrition Examination Survey, only 1.3% of adults 51 years or older were consuming less than 1500 mg/d of sodium; the mean consumption was 3100 mg/d. In a recent study among a Canadian cohort of older adults, sodium intake by FFQ was similar to that in our study. These findings highlight the difficulties in implementing stricter sodium intake, especially in older adults, and the tremendous efforts that would be required at the industry, community, interpersonal, and individual level to achieve this level of sodium intake.

A 2009 meta-analysis of 13 prospective studies reported that a 2000-mg-higher sodium intake was associated with a 14% higher risk for CVD. The estimates in our study were lower (approximately 6% higher risk per a 2000-mg-higher sodium intake) and did not reach significance. However, the Health ABC Study included only older adults 70 to 79 years old at the time of inception, and the mean sodium intake was much lower than that in the meta-analysis, emphasizing the importance of the population under investigation and the absolute levels of sodium intake for which the potential treatment effect is estimated. In a post hoc analysis of 28,880 individuals from 2 randomized trials, a significant association between sodium urinary excretion and CVD was observed when excretion exceeded 6500 mg/d, a much higher threshold than that recommended by current guidelines. This finding has been corroborated by a recent multinational cohort study. In our exploratory analysis, the signal toward risk appeared more con-

### Table 2. Association Between Baseline Dietary Sodium Intake and 10-Year Mortality

<table>
<thead>
<tr>
<th>Sodium Intake</th>
<th>No. of Events/No. of Participants</th>
<th>Unadjusted HR (95% CI)</th>
<th>P Value</th>
<th>Adjusted HR (95% CI)$\dagger$</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Participants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>881/2642</td>
<td>1.09 (1.04-1.16)</td>
<td>.001</td>
<td>1.03 (0.98-1.09)</td>
<td>.27</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>97/291</td>
<td>1.11 (0.88-1.40)</td>
<td>.37</td>
<td>1.12 (0.89-1.42)</td>
<td>.33</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>236/779</td>
<td>1 [Reference]</td>
<td>NA</td>
<td>1 [Reference]</td>
<td>NA</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>548/1572</td>
<td>1.19 (1.02-1.39)</td>
<td>.03</td>
<td>1.15 (0.99-1.35)</td>
<td>.07</td>
</tr>
<tr>
<td>Male Participants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>510/1290</td>
<td>1.01 (0.94-1.09)</td>
<td>.74</td>
<td>1.01 (0.94-1.09)</td>
<td>.76</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>39/88</td>
<td>1.11 (0.78-1.59)</td>
<td>.56</td>
<td>1.12 (0.78-1.60)</td>
<td>.54</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>127/315</td>
<td>1 [Reference]</td>
<td>NA</td>
<td>1 [Reference]</td>
<td>NA</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>344/887</td>
<td>0.95 (0.78-1.17)</td>
<td>.66</td>
<td>1.04 (0.84-1.29)</td>
<td>.72</td>
</tr>
<tr>
<td>Female Participants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>371/1352</td>
<td>1.13 (1.03-1.25)</td>
<td>.01</td>
<td>1.06 (0.96-1.17)</td>
<td>.27</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>58/203</td>
<td>1.25 (0.91-1.72)</td>
<td>.17</td>
<td>1.12 (0.80-1.55)</td>
<td>.51</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>109/464</td>
<td>1 [Reference]</td>
<td>NA</td>
<td>1 [Reference]</td>
<td>NA</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>204/685</td>
<td>1.33 (1.06-1.66)</td>
<td>.01</td>
<td>1.26 (0.99-1.59)</td>
<td>.06</td>
</tr>
<tr>
<td>Participants of White Race</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>498/1630</td>
<td>1.08 (1.01-1.17)</td>
<td>.03</td>
<td>1.03 (0.94-1.12)</td>
<td>.50</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>39/154</td>
<td>0.85 (0.60-1.20)</td>
<td>.36</td>
<td>0.84 (0.58-1.22)</td>
<td>.36</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>145/494</td>
<td>1 [Reference]</td>
<td>NA</td>
<td>1 [Reference]</td>
<td>NA</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>314/887</td>
<td>1.12 (0.93-1.35)</td>
<td>.24</td>
<td>1.06 (0.86-1.31)</td>
<td>.58</td>
</tr>
<tr>
<td>Participants of Black Race</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>383/1012</td>
<td>1.09 (1.01-1.17)</td>
<td>.02</td>
<td>1.03 (0.95-1.12)</td>
<td>.42</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>58/137</td>
<td>1.36 (0.98-1.89)</td>
<td>.07</td>
<td>1.41 (0.97-2.05)</td>
<td>.07</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>91/285</td>
<td>1 [Reference]</td>
<td>NA</td>
<td>1 [Reference]</td>
<td>NA</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>234/590</td>
<td>1.31 (1.03-1.67)</td>
<td>.03</td>
<td>1.29 (0.98-1.69)</td>
<td>.07</td>
</tr>
<tr>
<td>Participants With Hypertension</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>515/1397</td>
<td>1.05 (0.98-1.13)</td>
<td>.14</td>
<td>1.01 (0.93-1.09)</td>
<td>.84</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>69/167</td>
<td>1.29 (0.97-1.70)</td>
<td>.08</td>
<td>1.21 (0.89-1.66)</td>
<td>.22</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>144/427</td>
<td>1 [Reference]</td>
<td>NA</td>
<td>1 [Reference]</td>
<td>NA</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>302/803</td>
<td>1.14 (0.93-1.39)</td>
<td>.21</td>
<td>1.10 (0.90-1.34)</td>
<td>.36</td>
</tr>
<tr>
<td>Participants Without Hypertension</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>366/1245</td>
<td>1.15 (1.07-1.25)</td>
<td>&lt;.001</td>
<td>1.07 (0.98-1.17)</td>
<td>.13</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>28/124</td>
<td>0.83 (0.55-1.26)</td>
<td>.38</td>
<td>0.86 (0.56-1.31)</td>
<td>.48</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>92/352</td>
<td>1 [Reference]</td>
<td>NA</td>
<td>1 [Reference]</td>
<td>NA</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>246/769</td>
<td>1.30 (1.02-1.65)</td>
<td>.04</td>
<td>1.19 (0.91-1.57)</td>
<td>.21</td>
</tr>
</tbody>
</table>

Abbreviations: HR, hazard ratio; NA, not applicable.

$\dagger$ Adjusted for age, sex, race, baseline hypertensive status, body mass index, smoking status, physical activity, prevalent cardiovascular disease, pulmonary disease, diabetes mellitus, depression, blood pressure, heart rate, electrocardiogram abnormalities, and serum glucose, albumin, creatinine, and cholesterol levels.
sistent with sodium intake exceeding 4000 mg/d, although this finding was not significant in adjusted analyses.

Previous individual prospective cohort studies have reported a positive association, no association, or an inverse relationship between sodium intake and mortality. Moreover, it is vexing that in some observational investigations the reductions in blood pressure achieved by dietary means did not translate into lower CVD rates. Discrepant findings of previous studies are likely due to differences in ranges of sodium intake, study populations, and methods of sodium assessment, as well as failure to explore nonlinear associations. The nonlinearity issue is highlighted by recent studies. In a meta-analysis that included 23 cohort studies and 2 follow-up clinical trials, risk for adverse outcomes was increased at intakes of less than 2600 and greater than 4900 mg/d of sodium. In a large multinational cohort of more than 100,000 participants 35 to 70 years old, risk for death and major cardiovascular events was increased when 24-hour urinary sodium excretion was 7000 mg/d or higher (6000 mg/d for participants with hypertension) and when excretion was below 3000 mg/d. Recently, long-term outcomes were reported from the control arms of the Trials of Hypertension Prevention, during which 24-hour urinary sodium excretion was repeatedly assessed. In the follow-up investigations, which started 10 and 5 years after the end of the Trials of Hypertension Prevention I and II, respectively, sodium excretion of less than 2300 mg/d was associated with lower CVD risk in middle-aged adults. No clear risk gradient was observed between the group consuming less than 2300 mg/d of sodium and the group consuming 2300 to 3600 mg/d of sodium, and linear trends were not significant. However, our study population consisted of older adults (71-80 years old), who are inherently at higher risk for CVD and HF, and the mean sodium intake was low (<3000 mg/d); therefore, the effect of high sodium intake might have been more difficult to ascertain.

Several trials, most of which were short-term studies, showed that a reduction in sodium excretion to levels consistent with current guidelines lowered blood pressure in participants with prehypertension or hypertension and in normotensive participants, although the effect was attenuated in the latter. However, investigators of younger participants with high-normal blood pressure reported no difference in CVD risk events on initial follow-up, and a statistically significant benefit during an extended observational follow-up period of 10 to 15 years was found only after multivariable adjustment. In line with these findings, a 2011 Cochrane review of randomized clinical trials evaluating reduced sodium intake detected no significant reduction in CVD risk or mortality. In another meta-analysis of 4 primary prevention trials (2 in hypertension and 2 in prehypertension), a marginally significant reduction in CVD events was reported among those randomized to reduced sodium intake. Therefore, these studies are suggestive but not conclusive of a benefit of sodium reduction to very low intake targets in a primary prevention population. Considering the special case of older adults, in whom comorbidities, inadequate caloric intake, and medication interactions are additional concerns with very low sodium intake, the effect of sodium restriction should probably be tested explicitly in this population before implementing a generalized recommendation for very low (<1500 mg/d) sodium intake target. In the interim, a more conservative approach to sodium restriction (eg, targeting <2300 mg/d) might be appropriate for older adults.

Our study has some limitations. First, although adequate from a broad epidemiological perspective, an FFQ is less accurate at the individual level than other measurement methods. Sodium intake estimates by FFQ are reasonably reproducible but are poorly correlated with 24-hour urinary excretion estimates, with Spearman rank correlation p values...
Table 3. Association Between Baseline Dietary Sodium Intake and 10-Year Incident Cardiovascular Disease and Heart Failure

<table>
<thead>
<tr>
<th>Sodium Intake</th>
<th>Incident Cardiovascular Disease</th>
<th>Incident Heart Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Events/No. of Participants</td>
<td>Adjusted sHR (95% CI)</td>
</tr>
<tr>
<td>All Participants</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>572/1981</td>
<td>1.03 (0.95-1.11)</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>63/237</td>
<td>1.05 (0.79-1.41)</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>161/576</td>
<td>1 (Reference)</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>348/1188</td>
<td>1.02 (0.84-1.24)</td>
</tr>
<tr>
<td>Male Participants</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>312/900</td>
<td>0.98 (0.89-1.08)</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>23/59</td>
<td>1.13 (0.72-1.77)</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>77/208</td>
<td>1 (Reference)</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>212/633</td>
<td>0.91 (0.69-1.18)</td>
</tr>
<tr>
<td>Female Participants</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>260/1081</td>
<td>1.12 (0.98-1.28)</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>40/158</td>
<td>1.04 (0.71-1.54)</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>84/368</td>
<td>1 (Reference)</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>136/555</td>
<td>1.12 (0.85-1.49)</td>
</tr>
<tr>
<td>Participants of White Race</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>353/1217</td>
<td>0.98 (0.88-1.09)</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>37/116</td>
<td>1.15 (0.80-1.67)</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>98/375</td>
<td>1 (Reference)</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>218/726</td>
<td>1.03 (0.80-1.33)</td>
</tr>
<tr>
<td>Participants of Black Race</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>219/764</td>
<td>1.08 (0.97-1.20)</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>26/101</td>
<td>0.82 (0.52-1.32)</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>63/201</td>
<td>1 (Reference)</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>130/462</td>
<td>1.12 (0.85-1.49)</td>
</tr>
<tr>
<td>Participants With Hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>302/951</td>
<td>1.02 (0.93-1.12)</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>33/113</td>
<td>0.87 (0.57-1.31)</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>90/286</td>
<td>1 (Reference)</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>179/552</td>
<td>1.02 (0.78-1.32)</td>
</tr>
<tr>
<td>Participants Without Hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per 1 g</td>
<td>270/1030</td>
<td>1.06 (0.93-1.20)</td>
</tr>
<tr>
<td>&lt;1500 mg/d</td>
<td>30/104</td>
<td>1.23 (0.82-1.84)</td>
</tr>
<tr>
<td>1500-2300 mg/d</td>
<td>71/290</td>
<td>1 (Reference)</td>
</tr>
<tr>
<td>&gt;2300 mg/d</td>
<td>169/636</td>
<td>1.02 (0.77-1.37)</td>
</tr>
</tbody>
</table>

Abbreviations: NA, not applicable; sHR, subhazard ratio (hazard ratio conditional on the competing risk for death).

* Adjusted for age, sex, race, baseline hypertensive status, body mass index, smoking status, physical activity, prevalent cardiovascular disease (for heart failure events), pulmonary disease, diabetes mellitus, depression, blood pressure, heart rate, electrocardiogram abnormalities, and serum glucose, albumin, creatinine, and cholesterol levels.

of 0.20 or less, and underestimate sodium intake.56 As a consequence, the association between dietary sodium intake and outcomes is probably attenuated in our study. Also, participants at greatest risk may be more susceptible to underestimation of sodium intake. However, self-reported adoption of a low-salt diet was not associated with significantly higher risk for events in our study. Second, the Health ABC Study was not designed to specifically answer the question of appropriate dietary sodium intake. Secondary data analyses associating sodium intake with outcomes have several methodological drawbacks.57,58 Third, study participants were selected on the basis of voluntary participation and good functional capacity. Therefore, this sample may not fully reflect the general older adult population. However, individuals without mobility disability represent approximately 60% of the population 70 to 79 years old in the United States.59 Fourth, although we have comprehensively adjusted for risk factors previously identified in this cohort, we cannot exclude unobserved confounding. In addition, the lack of an association between dietary sodium intake and blood pressure or hypertensive status, one of the main mechanisms by which sodium intake leads to increased CVD and HF risk, might be an indication of reverse causality or, alternatively, of higher sodium intake thresholds for blood pressure effects in this population. Fifth, considering the 10-year horizon, we cannot exclude the possibility of regression dilution. Sixth, our approach probably has reduced power in the individual sodium intake groups (compared with a quantile-based approach) and may have led to unstable relative risk estimates. However, we have opted for recommendation-based cutoffs of sodium intake (<1500, 1500-2300, and
Table 4. Association Between Baseline Dietary Sodium Intake Exceeding 3000 mg/d and Exceeding 4000 mg/d and 10-Year Outcomes

<table>
<thead>
<tr>
<th>Variable Unadjusted sHR (95% CI)</th>
<th>P Value</th>
<th>Adjusted sHR (95% CI)*</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;3000 mg/d (n = 890) vs ≤3000 mg/d (n = 1752)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality</td>
<td>1.18 (1.03-1.36)</td>
<td>.02</td>
<td>1.06 (0.92-1.22)</td>
</tr>
<tr>
<td>Incident CVD</td>
<td>1.09 (0.92-1.30)</td>
<td>.31</td>
<td>0.97 (0.81-1.16)</td>
</tr>
<tr>
<td>Incident HF</td>
<td>0.90 (0.73-1.11)</td>
<td>.32</td>
<td>0.82 (0.65-1.02)</td>
</tr>
<tr>
<td>&gt;4000 mg/d (n = 336) vs ≤4000 mg/d (n = 2306)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality</td>
<td>1.25 (1.04-1.51)</td>
<td>.02</td>
<td>1.04 (0.85-1.25)</td>
</tr>
<tr>
<td>Incident CVD</td>
<td>1.33 (1.06-1.67)</td>
<td>.01</td>
<td>1.18 (0.93-1.49)</td>
</tr>
<tr>
<td>Incident HF</td>
<td>1.22 (0.93-1.61)</td>
<td>.14</td>
<td>1.12 (0.84-1.49)</td>
</tr>
</tbody>
</table>

Abbreviations: CVD, cardiovascular disease; HF, heart failure; sHR, subhazard ratio (hazard ratio conditional on the competing risk for death).

* Adjusted for age, sex, race, baseline hypertensive status, body mass index, smoking status, physical activity, prevalent CVD (for HF events), pulmonary disease, diabetes mellitus, depression, blood pressure, heart rate, electrocardiogram abnormalities, and serum glucose, albumin, creatinine, and cholesterol levels.

bn = 667 vs n = 1314.

* n = 248 vs n = 1733.

Conclusions

In conclusion, we observed that sodium intake estimated by FFQ was not associated with mortality or risk for CVD and HF in a cohort of adults 71 to 80 years old. These findings extended to sex-based and race-based subgroups and in participants with and without hypertension at baseline. Our data emphasize the need for stronger evidence, preferably from rigorous controlled trials testing additional thresholds for sodium intake, before applying a policy of further sodium restriction to older adults beyond the current recommendation for the general adult population (2300 mg/d).
Dietary Sodium, Mortality, and Cardiovascular Risk

Original Investigation Research


