ORIGINAL ARTICLES

Sodium Intake and Mortality Follow-Up in the Third National Health and Nutrition Examination Survey (NHANES III)

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BACKGROUND: Sodium restriction is commonly recommended as a measure to lower blood pressure and thus reduce cardiovascular disease (CVD) and all-cause mortality. However, some studies have observed higher mortality associated with lower sodium intake.

OBJECTIVE: To test the hypothesis that lower sodium is associated with subsequent higher cardiovascular disease (CVD) and all cause mortality in the Third National Health and Nutrition Examination Survey (NHANES III).

DESIGN: Observational cohort study of mortality subsequent to a baseline survey.

PARTICIPANTS: Representative sample (n=8,699) of non-institutionalized US adults age ≥30, without history of CVD events, recruited between 1988–1994.

MEASUREMENTS AND MAIN RESULTS: Dietary sodium and calorie intakes estimated from a single baseline 24-h dietary recall. Vital status and cause of death were obtained from the National Death Index through the year 2000. Hazard ratio (HR) for CVD mortality of lowest to highest quartile of sodium, adjusted for calories and other CVD risk factors, in a Cox model, was 1.80 (95% CI 1.05, 3.08, p=0.03). Non-significant trends of an inverse association of continuous sodium (per 1,000 mg) intake with CVD and all-cause mortality were observed with a 99% CI of 0.73, 1.06 (p=0.07) and 0.86, 1.04 (p=0.11), respectively, while trends for a direct association were not observed.

CONCLUSION: Observed associations of lower sodium with higher mortality were modest and mostly not statistically significant. However, these findings also suggest that for the general US adult population, higher sodium is unlikely to be independently associated with higher CVD or all-cause mortality.

KEY WORDS: sodium intake; mortality; cardiovascular disease; Third National Health and Nutrition Examination Survey (NHANES III).
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INTRODUCTION

Government agencies, expert panels and health associations routinely recommend lower sodium intake to reduce blood pressure (BP) in order to reduce risk of cardiovascular disease (CVD).[1–3] Randomized clinical trials confirm that lower sodium intake lowers mean systolic/diastolic BP values in the range of 1/0.6 to 5/3 mmHg with substantial interpersonal variation.[4] By contrast, there are no randomized clinical trial data to define the effect of different sodium intakes on CVD events or mortality.

Instead, several observational studies have examined the association of baseline sodium intake with subsequent cardiovascular events and mortality with mixed results. Some find more events with higher sodium,[5–9] others no difference,[10,11] and some find fewer events associated with higher sodium intake.[5,12–14] While a pattern in these observational studies may be emerging,[15] the data do not support any general causal inference.

Examinations of the relationship of dietary sodium intake with mortality in NHANES I and II[13,14] suggested the hypothesis that lower sodium may be associated with increased CVD and all-cause mortality. We undertook to test this hypothesis using the third of the series of standardized national surveys, NHANES III, with the 12-year linked mortality data that recently became available. We also examined the evidence for the competing hypothesis that higher sodium is associated with increased mortality.

METHODS

Study Population

The NHANES III study participants were recruited between 1988–1994 as a representative sample of the non-institutionalized US population. Participants gave informed consent, and the study was conducted in accord with the principles of the Declaration of Helsinki. Detailed methods for the NHANES III baseline data collection have been described elsewhere.[16] The mortality follow-up was based on National Death Index[17] searches and was recently made available as a limited access dataset through the NCHS Research Data Center.[18] Of 13,065 civilian adults over the age of 30 who participated in the mobile examination (MEC) and provided baseline data, we excluded 73 who died within the first 6 months after baseline exam, 1,327 who reported a history of heart attack, stroke or congestive heart failure, 673 who reported daily calorie intake <500 or >5,000 calories and 117 others who reported daily sodium intake <500 or >10,000 mg (<21.74 or >434.78 mmol). Those who reported a low sodium diet at baseline, (n=2,176) for hypertension were excluded since their

sodium intake may have been directly related to a risk factor for CVD and thus might severely confound the results. There remained 8,699 for the study sample.

Baseline Measures

Calorie, sodium and potassium intake were calculated from the 24-h dietary recall obtained by trained interviewers. Use of added salt was classified by three responses: does not add; adds some or adds a lot. Other baseline variables that were examined as potential confounders for the association of sodium with mortality included sex, age (years), serum cholesterol, race (coded as Black, White, and Hispanic/Other), treatment for hypertension, BP, smoking (current vs. else), alcohol consumption (dichotomized as ≥1 drink per week or else), weight, body-mass index (BMI), history of diabetes and education (dichotomized as <high school graduate or else). The coded metabolic equivalents (METS) for work and leisure activities for the previous month were summed and categorized by tertiles with the lower tertile considered inactive and the highest tertile very active. Less than 0.25% had missing values for BP, weight or BMI and less than 5% for cholesterol. These missing values were imputed using multivariable imputation.

Mortality Follow-up

Mortality status was based upon a National Center for Health Statistics (NCHS) search of the National Death Index (NDI) through the year 2000. The NDI provided underlying cause of death coded according to the ICD-9[19] through 1998, and the remainder according to ICD-10.[20] Only matches with a high degree of certainty were accepted. CVD was defined as ICD-9 codes 390–459 or ICD-10 codes I00-I99. Coronary heart disease (CHD) was defined as ICD-9 410–414 or ICD-10 codes I20-I25. Cerebrovascular accidents (CVA) were defined as 430–438 (ICD-9) or I60-I69 (ICD-10). All-cause mortality included all specified causes as well as unknown cause. Those without any indication of death were assumed alive and censored as of the end of 2000, the last available year of death in the NDI database at the time the matching was performed. Follow-up time was calculated as the time from the baseline exam to either a mortal event or the censoring date.

Statistical Analysis

Sodium, sodium/calorie ratio (sodium divided by calories) and residuals adjusted sodium (calculated according to method reported by Willett and Stampfer[21]) were analyzed as continuous variables and also categorized by quartiles. Sodium intake was also dichotomized at the median. Baseline characteristics were compared across quartiles of sodium by analysis of variance and chi-square for continuous and categorical variables, respectively. Age, sex and calorie-adjusted hazard ratios for the quartiles were estimated with Cox proportional hazards models with quartile 4 as reference.

Cox models were also constructed to estimate hazard ratios for the different measures of sodium while simultaneously adjusting for age, sex, race/ethnicity, education, smoking, history of diabetes, history of cancer, hypertension treatment, systolic blood pressure, serum cholesterol, alcohol use, weight, physical activity, added table salt and dietary potassium intake. Adjustment for total energy (calories) was done in three separate ways: calories added as a covariate, dividing sodium by calories (so-called energy density method) and using residuals derived from regressing sodium on calories. For the latter method, calories were also included as a separate covariate in multivariable models, as suggested by Willett.[22]

Interaction product terms of dichotomized sodium with each of the above listed covariates were created and tested along with the main effects terms in the fully adjusted models. To further test for heterogeneity, models stratified by each of the covariates were also constructed.

P values were calculated as two-tailed with an alpha of <0.05 designating statistical significance. Statistical analyses of baseline data were performed with STATA SE 9.2 (StataCorp, College Station, TX) and Cox models were created with SUDAAN 9 software (RTI International, Research Triangle Park, NC), in both cases taking into account the NHANES III complex sampling design. Power calculations were carried out with NCSS-PASS (Number Cruncher Statistical Systems, Kaysville, UT).

A subgroup was analyzed to determine whether findings from a recent study could be replicated in this sample.

In addition to the standard 95% confidence intervals (CIs), 99% CIs were constructed in order to estimate with greater confidence the likely upper limit of the true population value.

RESULTS

The 8,699 participants included represented over 99 million non-institutionalized US adults ≥30 years of age at entry exam during the 1988–1994 survey period. Mean (± SD) sodium intake for the study sample was 3,207±1,608 mg (139.43±69.91 mmol) with a median of 2,922 mg (127.04 mmol) and interquartile range of 2,060 to 4,048 mg (89.57 to 176.00 mmol). Mean energy intake was 2,007±847 calories with a median of 1,872 and interquartile range of 1,390 to 2,498 calories.

Baseline characteristics presented by sodium quartiles in Table 1 are expressed as mean (standard error), or percent, and were calculated using the complex sample weights. Given the large sample and the standard errors reduced by the complex sampling, all characteristics were significantly, or nearly so, different by quartile except for hypertensive status and history of diabetes. The lower sodium quartile group was more likely to be older, with less education and higher systolic BP. The higher quartile sodium group was more likely to be male, weigh more, smoke and have higher diastolic BP.

During 8.7±2.3 years follow-up, there were 1,150 deaths recorded, with 436 CVD, 236 CHD and 82 CVA deaths. Table 2 provides age and sex adjusted hazard ratios of CVD and all-cause mortality for quartiles of sodium (also adjusted for calories), sodium per calorie ratio, and residual adjusted sodium. There was a statistically significant inverse association of sodium intake with CVD mortality (p=0.02) comparing the lowest to highest sodium intake quartile. The association of sodium intake with all-cause mortality and sodium/calorie ratio and residuals adjusted sodium, respectively, with both CVD and all-cause mortality, also revealed non-significant trends towards an inverse association. After full adjustment (Table 3), comparisons of sodium intake in quartile 1 to quartile 4 consistently showed a trend towards an inverse association, but this was not consistently the case for quartiles 2 and 3. However, no statistically

Table 1. Baseline Characteristics by Sodium Intake Quartile*†

Variable	Quartile 1	Quartile 2	Quartile 3	Quartile 4	p
	<2,060 mg	2,060–2,921	2,922–4,047	4,048–9,946	
	n=2,174	n=2,175	n=2,175	n=2,175	
Age (years)	51 (0.66)	49 (0.58)	48 (0.51)	44 (0.51)	<0.001
Male sex (%)	23.8	37.9	49.8	68.1	<0.001
Race:					0.004
White (%)	75.7	79.6	80.4	80.4	
Black (%)	11.1	8.8	8.8	8.4	
Hispanic (%)	7.6	8.9	8.3	7.0	
Other (%)	5.7	2.7	2.5	4.3	
Dietary Na intake‡ (mg/day)	1,501 (13.5)	2,483 (7.4)	3,441 (12.5)	5,497 (43.0)	<0.001
Dietary cal (kcal)	1,282 (14.0)	1,762 (16.7)	2,152 (16.6)	2,938 (26.8)	<0.001
Dietary Na/ calories (mg‡/kcal)	1.26 (0.01)	1.53 (0.01)	1.72 (0.01)	1.97 (0.02)	<0.001
Dietary K (mg/day)	1,979 (30.6)	2,463 (30.3)	2,911 (35.6)	3817 (45.9)	<0.001
Adds salt: (%)					<0.001
Rarely	69.0	64.9	59.2	54.4	
Sometimes	15.4	19.0	22.1	22.3	
Often	15.6	16.1	18.7	23.4	
BMI (kg/m^2) (BMI (kg/m^2)	25.8 (0.19)	26.4 (0.20)	26.3 (0.14)	26.6 (0.21)	0.006
BMI (%)					0.02
<25 kg/m^2	50.3	44.8	44.5	41.7	
25–29.9 kg/m^2	30.7	35.1	33.6	38.5	
≥30 kg/m^2	19.0	20.1	21.9	19.8	
Weight (kg)	69.2 (0.54)	73.8 (0.65)	75.5 (0.41)	79.3 (0.67)	<0.001
Education < high school (%)	26.0	20.9	20.5	17.8	<0.001
Smoker (%)	27.4	24.8	29.6	32.7	0.003
BP >140/90 mmHg (%)	16.8	15.4	13.9	13.5	0.17
Treatment for hypertension (%)	12.9	10.3	10.2	9.9	0.11
Systolic BP (mmHg)	122.6 (0.69)	121.5 (0.58)	120.8 (0.51)	120.8 (0.51)	0.08
Diastolic BP (mmHg)	73.7 (0.29)	74.0 (0.32)	74.6 (0.30)	75.8 (0.31)	<0.001
Cholesterol (mmol/l)	5.46 (0.037)	5.38 (0.042)	5.36 (0.037)	5.33 (0.037)	0.07
History of diabetes (%)	4.7	4.3	4.3	3.7	0.76
History of cancer (%)	8.6	9.0	9.2	5.1	<0.001
Physical activity (%)					0.05
Lowest tertile	20.8	17.5	15.5	15.5	
Middle tertile	49.0	52.3	52.4	53.9	
Highest tertile	30.2	30.2	32.1	30.6	

*Results for continuous variables are reported as mean values (with standard errors) with p values calculated by analysis of variance between the sodium categories. Categorical variables are reported as percentages with p values calculated by chi-square. Because of rounding, not all percentages total 100. All results take into account the complex sampling design of NHANES III

†To convert sodium values from mg to mmol, divide by 23

‡Abbreviations: Na = sodium; Cal = calories; K = potassium; HS = high school; Txt = treatment; BMI = body mass index

significant direct association of higher sodium with higher mortality was observed in any comparison.

Hazard ratios and 95% CIs for the associations of sodium as a continuous variable (per 1,000 mg of sodium) for both CVD and all-cause mortality outcomes with the three different calorie adjustments revealed inverse associations that approached statistical significance for CVD mortality with sodium and residuals adjusted sodium (both p=0.07) (Table 4). The 99% CI for the HR per 1,000 mg (43.48 mmol) of both continuous sodium and residuals-adjusted sodium with CVD mortality was 0.73, 1.06. Similarly, the 99% CI for these HRs for all-cause mortality was (0.86, 1.04). In other models, there were non-significant trends towards an inverse association, with the exception of sodium per calorie ratio, which showed no trend for all-cause mortality. In none of these models was there a trend towards a direct association of any sodium measure with any mortality outcome. Using CHD and CVA mortality outcomes had results consistent with those for CVD, but with fewer outcomes, the robustness of these models was less.

Since it was not possible to quantify estimates of added sodium (at the table or in cooking), added sodium was included as qualitative dummy variables in the fully adjusted models. In sensitivity analyses, the subset (n=5,560) of those who reported no added sodium gave results similar to those of the full sample. Sensitivity analyses including individuals who reported a low salt diet for hypertension, had results in the same direction.

When lower was compared to higher sodium (dichotomized at the median 2,922 mg (127.04 mmol), there was a non-significant, inverse trend for an association with CVD mortality, with an adjusted hazard ratio (95% CI) of 1.39 (0.96, 2.02),

Table 2. Age-sex Adjusted Mortality Hazard Ratios and Number of Events by Sodium Quartiles

Mortality outcome	Quartile 1	Quartile 2	Quartile 3	Quartile 4
Sodium intake quartiles*				
CVD	1.87 (1.13, 3.12) p=0.02 (n=159)	1.86 (1.29, 2.70) p<0.001 (n=126)	1.44 (0.80, 2.59) p=0.21 (n=103)	1.00 (n=48)
All-cause	1.30 (0.94, 1.80) p=0.11 (n=389)	1.27 (0.93, 1.74) p=0.13 (n=321)	1.07 (0.81, 1.43) p=0.62 (n=256)	1.00 (n=184)
Sodium per calorie quartiles*				
CVD	1.22 (0.81, 1.84) p=0.33 (n=111)	0.87 (0.62, 1.23) p=0.43 (n=108)	0.91 (0.62, 1.32) p=0.60 (n=100)	1.00 (n=117)
All-cause	1.14 (0.90, 1.45) p=0.27 (n=286)	0.80 (0.61, 1.04) p=0.10 (n=271)	0.97 (0.76, 1.25) p=0.84 (n=279)	1.00 (n=314)
Residual adjusted sodium intake quartiles*				
CVD	1.39 (0.92, 2.08) p=0.11 (n=102)	1.06 (0.92, 2.08) p=0.69 (n=122)	1.24 (0.83, 1.86) p=0.28 (n=126)	1.00 (n=86)
All-cause	1.21 (0.95, 1.53) p=0.12 (n=268)	0.92 (0.74, 1.14) p=0.45 (n=304)	1.15 (0.94, 1.41) p=0.18 (n=319)	1.00 (259)

*Also adjusted for calories

†Age-sex adjusted rate ratios with quartile 4 as reference (95% confidence intervals) and p values. (n=number of events)

Table 3. Fully Adjusted Mortality Hazard Ratios by Sodium Quartiles*

Mortality outcome	Quartile 1	Quartile 2	Quartile 3	Quartile 4	p for Q 1 vs. Q4
Sodium intake quartiles†					
CVD	1.80 (1.05, 3.08)	1.94 (1.32, 2.85)	1.48 (0.82, 2.67)	1.0 (reference)	0.03
All-cause	1.24 (0.91, 1.70)	1.30 (0.96, 1.76)	1.06 (0.81, 1.40)	1.0 (reference)	0.17
Sodium/calorie intake quartiles					
CVD	1.25 (0.84, 1.86)	0.90 (0.64, 1.26)	0.90 (0.61, 1.32)	1.0 (reference)	0.26
All-cause	1.15 (0.92, 1.45)	0.81 (0.62, 1.06)	0.95 (0.74, 1.22)	1.0 (reference)	0.21
Residual adjusted sodium intake quartiles					
CVD	1.37 (0.90, 2.09)	0.95 (0.68, 1.33)	1.15 (0.76, 1.75)	1.0 (reference)	0.14
All-cause	1.21 (0.97, 1.53)	0.89 (0.70, 1.13)	1.09 (0.87, 1.38)	1.0 (reference)	0.09

*Hazard ratios (95% confidence intervals) assessed with Cox proportional hazards models adjusting for age, sex, race, education, added table salt, exercise, alcohol use, current smoking, history of diabetes, history of cancer, systolic BP, cholesterol, dietary potassium, weight, treatment for hypertension

†Additionally adjusted for calories

p=0.08. To assess whether there was heterogeneity for this association, subgroup analyses were performed for each of the baseline characteristics with non-significant trends towards an inverse association for most subgroups. Formal tests of product terms in the Cox models showed no statistically significant interactions.

To compare our findings to those from the recently published observational follow-up of the TOHP study,[9] we created a subgroup of those who had BMI≥25 kg/m^2, aged 30–55 years and non-hypertensive, with systolic BP <140 mmHg and diastolic BP 80–89 mmHg. These criteria were met by less than 10% of our study sample and less than 4% of the entire US population represented by NHANES III. With only six CVD deaths (four in the low sodium and two in the high sodium), it was not possible to construct a robust model. For all-cause mortality (23 deaths), and after adjusting for age, sex, calories and added salt, there was no statistically significant association (p=0.28).

To assess the statistical power of this sample to detect an *increased* hazard ratio associated with each 1,000 mg (43.5 mmol) of sodium (i.e., a direct association), we found that we had >99% power to detect a true population HR≥1.20 and ≥1.15 for CVD and all-cause mortality, respectively.

DISCUSSION

These data are consistent with the hypothesis that lower sodium intake is associated with increased CVD and all-cause mortality. However, only a few of the observed associations reached statistical significance. In contrast, no analysis of the two mortality outcomes with three different adjustments for calorie intake, whether in the entire sample or in more than two dozen subgroups, generated any trend supporting the competing hypothesis that, within the range of sodium intakes in this sample, the highest sodium relative to the lowest is associated with increased mortality.

More than 100 clinical trials have identified a significant association of mean blood pressure with mean sodium intake, ranging from 1–5 mmHg systolic and 0.6–3 mmHg diastolic with sodium reductions of 75 to 100 mmols/day (1,725 to 2,300 mg/day).[4] There are, however, no randomized trial data linking sodium intake to CVD events or mortality. The several available observational studies linking baseline sodium with subsequent outcomes have yielded varied results.[5–14]

Most recently, a post-hoc intention to treat analysis (100% outcome ascertainment) of 10–15-year follow-up of two TOPH clinical trials reported no significant difference (p=0.34) in mortality.[9] In a post hoc subgroup analysis of 2,415 (77%) not protected by randomization, these investigators detected a statistically significant 25% lower risk of combined CVD events in those assigned to lower sodium intake. Although BP data for the follow-up period were not available, mean BP reduction of the TOHP I and II trials were modest (1.7/0.8 and 1.2/0.7 mmHg, respectively). Thus, this post-hoc analysis can best be seen as adding observational data for the subgroup studied. It should be noted, however, that individuals with characteristics approximating TOHP inclusion criteria regarding age, BP and weight/obesity constituted <10% of our study sample and <4% of the total US population.

In contrast, this study sample from this third in the NHANES series represents almost 100 million non-institutionalized US adults. Previously, we found an inverse association of sodium with CVD mortality, but not sodium/calorie ratio, in the NHANES I sample.[13] Another group, applying different methods, reported a direct association of sodium and sodium/calorie quartiles with CVD events in an overweight subgroup (28%) of NHANES I-but no association in the normal weight majority.[6] NHANES II data revealed a stronger, statistically significant inverse association

Table 4. Adjusted Mortality Hazard Ratios for Continuous Sodium Measures

Mortality outcome	Sodium measure	Hazard ratio* (95% confidence interval)	p value
Cardiovascular disease	Sodium per 1,000 mg†	0.88 (0.77, 1.01)	0.07
	Sodium mg per calorie	0.91 (0.72, 1.14)	0.40
	Sodium residuals adjusted per 1,000 mg	0.88 (0.77, 1.01)	0.07
All-cause	Sodium per 1,000 mg	0.94 (0.88, 1.01)	0.11
	Sodium mg per calorie	1.00 (0.88, 1.12)	0.94
	Sodium residuals adjusted per 1,000 mg	0.95 (0.88, 1.02)	0.11

*Hazard ratios (95% confidence intervals) assessed with Cox proportional hazards models adjusting for age, sex, race, education, added table salt, exercise, alcohol use, current smoking, history of diabetes, history of cancer, systolic BP, cholesterol, dietary potassium, weight, treatment for hypertension

†1,000 mg sodium=43.48 mmol, additionally adjusted for calories

of CVD mortality with sodium, sodium/calorie ratio and residual adjusted sodium as well as sodium <2,300 mg/day (<100 mmol/day)-the threshold for current US dietary guidelines and close to the median intake in that study population.[14]

Our study has several important limitations. There was only a single measure of dietary intake at baseline with no follow-up measures to assess whether or to what extent sodium intake changed over time. Also, there were no urinary sodium excretions collected to validate the self-reported 24-h dietary intake or the impact of discretionary salt. As with all observational studies, the possibility of latent confounding persists, despite efforts to statistically adjust the models with appropriate available covariates, including alternate types of adjustment for calories. Mortality information was mostly provided by National Death Index searches, and specific cause of death from death certificate data is subject to misclassification. Finally, the trends toward inverse sodium to CVD associations observed here were, with only two exceptions, not statistically significant.

This study also has several important strengths. NHANES III was constructed to be a representative sample of the adult US population. Assessment of dietary intake benefited from the experience of the previous NHANES surveys, and NHANES III has been used extensively to estimate US adult dietary intakes. Although individual intake of discretionary salt was not quantified (estimated by others at about 5–10% of total intake[1]), analyses of sodium by quartiles and as a continuous variable lessens the importance of an absolute estimate.

The non-significant trends towards an inverse association of sodium intake with mortality observed here were remarkably consistent despite different mortality outcomes and different methods for calorie adjustment. In addition, the upper limits of the 99% confidence intervals for the HR for CVD and all-cause mortality of 1,000 mg (43.48 mmol) sodium (adjusted with calories as a covariate or with residuals adjustment) were 1.06 and 1.04, respectively, which means the probability of excess CVD >6% or all-cause mortality greater than 4% per 1,000 mg (43.48 mmol) sodium can be estimated as <0.01. In other words, while the probability estimated from these data of greater mortality being associated with lower sodium does not meet the conventional standard for statistical significance, the likelihood of higher mortality being associated with higher sodium is far less.

We have described elsewhere that when one looks at the results of the various observational studies conducted in populations with different mean values of sodium intake, the findings seem to follow a J-shape, with elevated risks associated with lower sodium among populations at the lower end of sodium intake (such as NHANES I and II), and with elevated risks associated with higher sodium among populations at the higher end of mean sodium intake (such as in Japan or in Finland) when compared to studies in populations with mean sodium intakes in the middle range.[15] The modestly higher mean sodium intake level observed in NHANES III compared to NHANES I and II was accompanied by an attenuated risk of mortality associated with lower sodium. Thus, it places the NHANES III cohort as a whole closer to the middle of this hypothesized "J." Of course, without clinical trial data, any causal inference regarding sodium and mortality in either direction remains speculative.

Sodium intake may actually be primarily a marker for other as yet unidentified individual characteristics in the diet or otherwise, or may have different contributory effects in different individuals, under different circumstances, and in different population subgroups. However, observational data available from other countries suggest that mean sodium intake greater than 4–5 g/day (174–217 mmol/day) may be harmful.[23] Given the evidence that US sodium and caloric intake are increasing and closely correlated, and because the burden of obesity is growing, efforts aimed at reducing caloric intake may be a useful strategy to halt the upward trajectory of both weight and sodium intake.

Sodium restriction is one of most widely publicized non-pharmacological recommendations for CVD prevention. These latest findings are consistent with, albeit not demonstrative of, the hypothesis that lower sodium may be associated with higher CVD and all-cause mortality risk. Conversely, these findings add to the evidence that for the broad general US population, higher sodium is unlikely to be independently associated with higher all-cause or CVD mortality.

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