Dietary sodium intake and mortality: the National Health and Nutrition Examination Survey (NHANES I)

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Summary

Background Population-wide restriction of dietary sodium has been recommended. However, little evidence directly links sodium intake to morbidity and mortality. The aim of this study was to assess the relation of sodium intake to subsequent all-cause and cardiovascular-disease (CVD) mortality in a general population.

Methods The first National Health and Nutrition Examination Survey established baseline information during 1971–75 in a representative sample of 20 729 US adults (aged 25–75). 11 348 underwent medical examination and nutritional examination based on 24 h recall. Two had no data on sodium intake available. Vital status at June 30, 1992, was obtained for the 11 346 participants through interview, tracing, and searches of the national death index. Mortality was examined in sex-specific quartiles of sodium intake, calorie intake, and sodium/calorie ratio. Multiple regression analyses were done to assess the relations with mortality.

Findings There were 3923 deaths, of which 1970 were due to CVD. All-cause mortality (per 1000 person-years; adjusted for age and sex) was inversely associated with sex-specific quartiles of sodium intake (lowest to highest quartile 23·18 to 19·01, p<0·0001) and total calorie intake (25·03 to 18·40, p<0·0001) and showed a weak positive association with quartiles of sodium/calorie ratio (20·27 to 21·71, p=0·14). The pattern for CVD mortality was similar (sodium 11·80 to 9·60, p<0·0019; calories 12·80 to 8·94, p<0·0002; sodium/calorie ratio 9·73 to 11·35, p=0·017). In Cox multiple regression analysis, sodium intake was inversely associated with all-cause (p=0·0069) and CVD mortality (p=0·086) and sodium/calorie ratio was directly associated with all-cause (p=0·0004) and CVD mortality (p=0·0056). By contrast, calorie intake in the presence of the two measures of sodium intake was not independently associated with mortality (all-cause p=0·86; CVD p=0·74). Analysis restricted to participants with no history of CVD at baseline gave similar results.

Interpretation This observational study does not justify any particular dietary recommendation. Specifically, these results do not support current recommendations for routine reduction of sodium consumption, nor do they justify advice to increase salt intake or to decrease its concentration in the diet.

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Introduction

Ecological, observational, and experimental data largely, but not invariably, support the view that a restricted-sodium diet is associated with, and can induce, lower blood pressure.[1–12] Based on these and other associations[13] with intermediate physiological variables, some groups recommend restriction of daily sodium intake to 106 mmoles (approximately 2400 mg).[14] This recommendation is justified by the expectation that the beneficial changes in intermediate physiological variables outweigh any harmful effects, so that the net effect would be lower cardiovascular morbidity and mortality, or an overall improvement in the quality of life. We could find no empirical data to support these expectations directly.

Although favourable effects on intermediate variables translate into health benefits in many circumstances, this is not always the case.[15,16] In previous studies in hypertensive patients, a low-sodium diet was associated with adverse effects on quality of life[17] and an increase in cardiovascular morbidity and mortality.[18] To find out whether dietary sodium is associated with mortality in a general population, we examined the relation of sodium intake, measured in 1971–75, to all-cause and cardiovascular-disease (CVD) mortality, up to mid-1992, among participants in the first National Health and Nutrition Examination Survey (NHANES I).

Methods

Participants

There were 20 729 individuals aged 25–75 years at the time of the NHANES I survey from 1971 to 1975. From the total sample, 14 407 (70%) underwent medical examination, and 11 348 of these underwent a nutrition investigation based on a 24 h recall. Data on sodium intake were missing for two participants, who were therefore excluded. Vital status was obtained for the remaining 11 346 participants through interview, tracing, and searches of the national death index. Deaths and causes of death were confirmed from death certificates. Details on the plan and operation of NHANES I have been published previously.[19,20] Since information on mortality was incomplete after June, 1992, the cut-off date for this analysis was June 30, 1992. Participants not reported as dead before this date were presumed to be alive.

The length of follow-up for each individual, expressed as the number of person-years contributed, was calculated from the baseline to the date of death or to the cut-off date.

Additional analyses were done on a restricted sample of 9962 participants who, at baseline, reported no pre-existing cardiovascular disorders. Those excluded had a history of circulatory diseases (International Classification of Diseases, 9th Revision, codes 390–450), rheumatic heart disease, or heart operation (ICD-9 procedure codes 29–30).

Measurements

Data on nutrient intake were available from a single 24 h dietary recall. Sodium intake was expressed as mg per day, and total calorie intake as calories per day. Qualitative data on use of table salt were also available from the dietary frequency questionnaire (responses never, sometimes, or always).

Characteristic*	Men				Women			
	Q1 (n=1120)	Q2 (n=1118)	Q3 (n=1120)	Q4 (n=1120)	Q1 (n=1717)	Q2 (n=1721)	Q3 (n=1713)	Q4 (n=1717)
Demographic characteristics								
Age (years)	56·9 (14·3)	54·4 (15·5)	51·7 (15·5)	48·6 (15·1)	49·8 (16·0)	49·2 (16·0)	47·8 (15·9)	43·9 (14·9)
Black race	24·4%	17·0%	13·3%	8·8%	26·0%	18·3%	15·4%	11·5%
History								
CVD	17·6%	15·4%	14·5%	11·3%	11·5%	12·0%	9·4%	9·6%
Hypertension	21·6%	18·1%	15·5%	14·9%	16·8%	15·0%	14·2%	12·6%
Anthropometric characteristics								
Body-mass index (kg/m^2)	25·7 (4·3)	25·4 (4·0)	25·2 (4·2)	25·5 (4·1)	26·6 (6·0)	25·6 (5·7)	25·3 (5·5)	24·6 (5·5)
Bodyweight (kg)	76·0 (14·5)	76·4 (13·4)	76·4 (14·2)	77·7 (13·7)	68·4 (16·3)	66·3 (15·2)	65·6 (14·3)	64·3 (14·9)
Blood pressure (mm Hg)								
Systolic	142·4 (24·9)	138·8 (33·2)	136·0 (22·3)	134·4 (20·6)	136·7 (26·8)	134·9 (26·1)	133·7 (26·2)	129·5 (24·5)
Diastolic	87·3 (14·0)	85·8 (13·0)	84·5 (12·1)	84·6 (11·8)	83·5 (13·7)	82·6 (13·8)	81·8 (13·1)	80·2 (12·8)
24 h dietary recall								
Sodium intake (mg)	1041 (322)	1832 (195)	2647 (282)	4538 (1489)	678 (229)	1232 (138)	1791 (196)	3105 (1002)
Calorie intake (kcal)	1473 (638)	1930 (708)	2297 (732)	2937 (1050)	989 (408)	1331 (434)	1589 (518)	1976 (682)
Sodium/calorie (mg/kcal)	0·80 (0·35)	1·07 (0·42)	1·27 (0·44)	1·67 (0·61)	0·76 (0·35)	1·02 (0·35)	1·25 (0·48)	1·70 (0·66)
Use of table salt								
Always	33·9%	39·2%	37·2%	45·9%	20·4%	21·5%	24·5%	30·0%
Never	46·2%	35·5%	36·4%	31·9%	57·4%	57·0%	52·5%	46·9%

*Data presented as mean (SD) or as % of participants.

Table 1: **Baseline characteristics by quartile (Q) of sodium intake from 24 h recall**

As part of the physical examination in NHANES I, one blood-pressure value was recorded. For 33% of participants this value was the mean of three readings, two with the participant seated and once with him or her supine. In the remaining 67% of participants, one reading was done with the participant seated. Of the total 11 346 participants, 16% had baseline blood-pressure readings in the hypertensive range (systolic ⩾160 mm Hg, diastolic ⩾95 mm Hg, or both). Both the actual systolic blood pressure and history of hypertension were included in multivariate analyses as independent variables.

Statistical analysis

Baseline characteristics, including blood pressure, were defined for male and female participants. All statistical tests for continuous (Student's *t* test) and categorical variables (χ^2) were two-tailed. Since distributions of sodium intake for men and women differed significantly, baseline characteristics were assessed according to sex-specific quartiles of 24 h dietary sodium and calorie intake. The differences between quartiles were tested for statistical significance by ANOVA. All-cause and CVD death rates (per 1000 person-years), adjusted for age and sex, were calculated by specific dietary quartiles separately for men and women, standardised by the direct method, with age and sex distribution of the whole cohort as reference. The differences in mortality rates between the lowest and highest quartiles were tested for statistical significance.

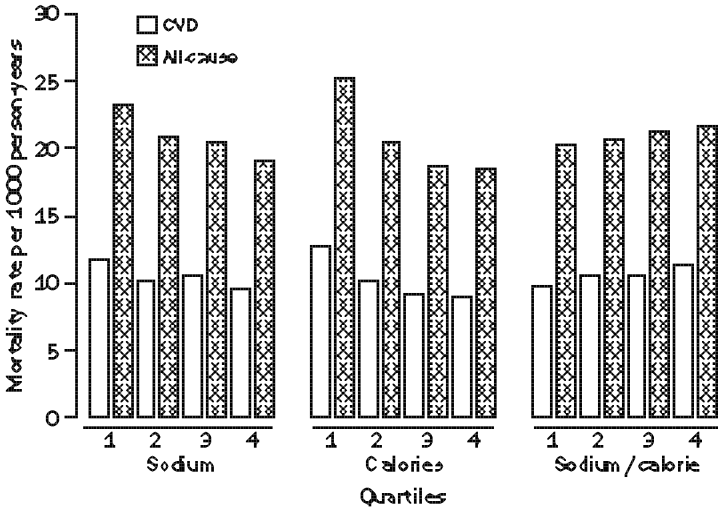
Because sodium intake and total calorie intake were highly correlated (r=0·65), a sodium/calorie ratio (expressed as sodium per calorie intake per day) was calculated for each participant to reflect the concentration of salt in the diet. Participants were stratified according to sex-specific quartiles, and all analyses described for sodium and total calorie intake were repeated for sodium/calorie ratio. Cox proportional-hazards regression models were constructed individually as well as in combination, to assess the association of the three dietary measures (sodium, total calories, and sodium per calorie) with all-cause and total CVD mortality, with simultaneous control for such available relevant variables as age at baseline, sex, race, body-mass index, history of CVD and hypertension, and systolic blood pressure. Stepwise backward regression analysis was done to estimate hazard ratios and 95% CI from a full model. To assess whether models with inclusion of other dietary measures were better than that with sodium intake as an individual measure, the change in the log-likelihood value was tested for significance as a χ^2 statistic with 1 degree of freedom.

All analyses were repeated on the restricted study sample of participants with no reported history of pre-existing CVD at baseline. All statistical analyses were done by means of SPSSWIN (version 7.0).

Characteristic*	Men				Women			
	Q1 (n=1118)	Q2 (n=1120)	Q3 (n=1120)	Q4 (n=1119)	Q1 (n=1716)	Q2 (n=1717)	Q3 (n=1717)	Q4 (n=1717)
Demographic characteristics								
Age (years)	50·3 (15·9)	52·9 (15·3)	54·0 (15·2)	54·5 (15·0)	46·1 (15·5)	48·1 (16·0)	48·7 (16·0)	47·9 (15·9)
Black race	20·1%	14·8%	17·0%	11·6%	23·2%	17·4%	16·8%	13·8%
History								
CVD	12·5%	14·6%	15·4%	16·0%	9·3%	10·4%	11·7%	11·1%
Hypertension	17·1%	15·6%	18·4%	19·0%	13·9%	14·2%	14·6%	16·0%
Anthropometric characteristics								
Body-mass index (kg/m^2)	25·7 (4·2)	25·4 (4·1)	25·2 (4·2)	25·4 (4·1)	25·6 (5·9)	25·5 (5·5)	25·4 (5·9)	25·6 (5·6)
Bodyweight (kg)	77·7 (14·3)	76·4 (13·7)	76·0 (14·1)	76·2 (13·7)	66·4 (15·9)	66·2 (14·9)	65·7 (15·5)	66·1 (14·7)
Blood pressure (mm Hg)								
Systolic	137·0 (22·8)	137·0 (22·9)	138·8 (22·6)	138·8 (23·7)	131·6 (25·5)	133·7 (25·8)	134·9 (26·4)	134·5 (26·4)
Diastolic	85·7 (12·8)	85·1 (12·8)	85·9 (12·8)	85·6 (12·9)	81·7 (13·7)	82·1 (13·3)	82·3 (13·1)	82·0 (13·4)
24 h dietary recall								
Sodium intake (mg)	1436 (730)	2134 (904)	2663 (1177)	3827 (1845)	920 (483)	1408 (579)	1815 (767)	2662 (1254)
Calorie intake (kcal)	2330 (1069)	2208 (916)	2098 (922)	2003 (898)	1520 (706)	1506 (609)	1467 (604)	1391 (604)
Sodium/calorie (mg/kcal)	0·62 (0·16)	0·97 (0·82)	1·27 (0·10)	1·95 (0·52)	0·60 (0·16)	0·94 (0·08)	1·24 (0·11)	1·96 (0·58)
Use of table salt								
Always	45·5%	37·8%	37·8%	35·3%	23·7%	25·4%	23·9%	23·5%
Never	35·5%	36·8%	37·8%	39·9%	53·9%	52·2%	53·5%	54·0%

*Data presented as mean (SD) or as % of participants.

Table 2: **Baseline characteristics by quartile (Q) of sodium per calorie intake from 24 h recall**



All-cause and CVD mortality rates per 1000 person-years according to quartile of daily sodium intake, total calorie intake, and sodium per calorie intake (adjusted for age and sex)

Results

The 4478 (39·5%) men differed significantly (p<0·05) from the 6868 women in mean age (52·9 *vs* 46·7 years), weight (76·6 *vs* 66·1 kg), and blood pressure (138/86 *vs* 134/82 mm Hg) and frequency of reported history of CVD (15 *vs* 11%) and hypertension (18 *vs* 15%). Mean daily sodium intake (2515 *vs* 1701 mg) and calorie intake (2159 *vs* 1471 kcal [1 kcal=0·0042 MJ]) were also significantly higher for men than for women. In response to the question on use of table salt more men than women replied "always" (39 *vs* 24%) and fewer replied "never" (38 *vs* 53%).

Baseline characteristics of participants by quartile of sodium intake, total calorie intake, and sodium/calorie ratio were analysed for men and women separately. For men and for women, there were significant differences (ANOVA, p<0·05) across the four quartiles of sodium intake in mean age, blood pressure, bodyweight, and body-mass index (women only), use of table salt, and the proportions of black people and individuals with a history of CVD or hypertension (table 1). Mean calorie intake increased from the lowest to the highest sodium-intake quartile, but surprisingly, body-mass index was similar across the quartiles of sodium intake for men. Sodium and calorie intakes were closely correlated (r=0·65). The characteristics of the four quartiles of total calorie intake (not shown) were very similar to those of the quartiles of sodium intake.

Baseline characteristics generally differed (ANOVA, p<0·05), for men and for women, across quartiles of sodium/calorie ratio, with the exceptions of systolic blood pressure in men; weight, body-mass index, and use of table salt always in women; and diastolic blood pressure, history of hypertension, history of CVD, and use of table salt "never" in both sexes (table 2).

Dietary intake and mortality

Of the 11 346 participants, 7423 were presumed alive and 3923 had died (1970 from CVD) by June 30, 1992. All-cause mortality rates adjusted for age and sex (figure) were inversely and significantly related to sodium intake per day (lowest to highest quartile 23·18 to 19·01 per 1000 person-years; p<0·0001) and total calorie intake per day (25·03 to 18·40 per 1000 person-years; p<0·0001). A similar inverse relation was seen for CVD mortality rates from lowest to highest quartile of sodium intake (11·80 to 9·60 per 1000 person-years; p<0·0019) and calorie intake (12·80 to 8·94 per 1000 person-years; p<0·0002). The results were similar for the analysis limited to participants with no reported history of CVD at baseline (not shown).

For sodium/calorie ratio, there was a weak direct relation to all-cause mortality (lowest to highest quartile 20·27 to 21·71 per 1000 person-years; p=0·14) and a significant direct relation to CVD mortality (9·73 to 11·35 per 1000 person-years; p=0·017). When the analysis was restricted to participants with no reported history of CVD at baseline (not shown), both all-cause and CVD mortality were directly related to sodium/calorie ratio, but the relation did not achieve statistical significance.

Multivariate analysis

Because sodium intake, total calorie intake, and sodium/calorie ratio were all associated with all-cause and CVD mortality, we undertook stepwise Cox proportional-hazards regression analysis with all three dietary measures in the model (table 3). Models were constructed separately for all-cause and CVD mortality as dependent variables. These models had significantly better likelihood values (p<0·001) than models limited to one dietary measure. In these single-measure analyses, sodium intake had a significant inverse association with both all-cause

Variable*	All-cause mortality			CVD mortality		
	β	p	Hazard ratio (95% CI)†	β	p	Hazard ratio (95% CI)†
Male	0·6286	<0·0001	1·88 (1·75–2·01)	0·6361	<0·0001	1·89 (1·71–2·09)
Black race	0·1585	0·0001	1·17 (1·08–1·27)	0·0465	0·4347	1·05 (0·93–1·18)
History of CVD	0·4033	<0·0001	1·50 (1·39–1·62)	0·4859	<0·0001	1·63 (1·46–1·80)
History of hypertension	0·1000	0·0241	1·11 (1·01–1·21)	0·0834	0·1668	1·09 (0·97–1·22)
Age (years)	0·0810	<0·0001	3·62 (3·44–3·82)	0·0922	<0·0001	4·33 (3·98–4·71)
Body-mass index (kg/m^2)	−0·0047	0·1932	0·98 (0·94–1·01)	0·0081	0·1000	1·04 (0·99–1·10)
Systolic blood pressure (mm Hg)	0·0057	<0·0001	1·15 (1·11–1·20)	0·0103	<0·0001	1·29 (1·23–1·36)
Sodium (mg)	−0·0001	0·0069	0·88 (0·80–0·96)	−0·00009	0·0864	0·89 (0·77–1·02)
Calories (kcal)	−0·00001	0·8562	0·99 (0·91–1·08)	−0·00002	0·7394	0·98 (0·87–1·11)
Sodium/calories (mg/kcal)	0·1955	0·0004	1·12 (1·05–1·19)	0·2159	0·0056	1·13 (1·04–1·24)
Table salt use (always)	0·0741	0·1201	1·08 (0·98–1·18)	−0·0130	0·8510	0·99 (0·86–1·13)
Table salt use (never)	0·0057	0·8889	1·01 (0·93–1·09)	−0·0012	0·9825	1·00 (0·89–1·12)

*For categorical variables, yes=1. For table salt use variables, reference=sometimes.

†For continuous variables, hazard ratios are for 1 SD change. SDs: age=15·9 years, body-mass index=5·15 kg/m^2, systolic blood pressure=24·98 mm Hg, sodium=1313 mg, calories=849 kcal, sodium/calorie=0·5787 mg/kcal.

Table 3: **Variables associated with risk of all-cause and CVD mortality in Cox proportional-hazards regression (full model)**

and CVD mortality. Sodium/calorie ratio showed a significant positive relation to both all-cause and CVD mortality. However, in the model with both measures of sodium intake, total calorie intake no longer had an independent relation to mortality. The relation of sodium intake to all-cause mortality, although significant ($p=0·0069$), was small. For example, an increase in dietary sodium of 1000 mg was associated with a 10% reduction in mortality. When potassium intake, alcohol use, family income, and education of head of household were each added to the models, the results for sodium intake and sodium/calorie ratio did not change substantially. Both potassium intake and alcohol use were removed from the all-cause and CVD models during backwards stepwise elimination. The results were similar when these analyses were restricted to the participants with no history of CVD at baseline.

All analyses were repeated with stratification for age (<65 and ⩾65 years). For the older age-group, the relation of sodium intake, sodium/calorie ratio, and calorie intake to all-cause and CVD mortality were similar to those observed for the whole study population and were statistically significant. Trends for the younger group were similar, but did not achieve significance.

Discussion

Our main findings are that dietary sodium intake is inversely associated with all-cause and CVD mortality, and that dietary sodium/calorie ratio is directly associated with both mortality rates. These associations, although small, are significant and independent, both of each other and of other factors known to influence mortality.

The inverse association of salt intake with mortality is consistent with the findings of a similar observational study of 3000 participants in a systematic programme to control hypertension.[18] In both studies, baseline sodium intake was assessed and related (with control for known confounders) to subsequent morbidity or mortality. Different ways of estimating sodium intake—dietary recall and 24 h urinary measurement—produced similar results in the two studies. NHANES I baseline data, unlike the study in hypertensive patients, included information on other dietary factors. Since there was a strong correlation of sodium intake with total energy consumption, we explored the relation of calorie intake to mortality, both as an isolated variable and in relation to its sodium content. We found that both sodium alone and the sodium concentration in the diet helped to explain, in opposite directions, variations in all-cause and cardiovascular mortality.

These findings suggest that, although there may be a specific relation of sodium to survival, it is not likely to be simple; at the very least, it must be considered in the total dietary context. Moreover, given the genetic, behavioural, environmental, and dietary heterogeneity in most industrialised societies, different individuals may have different optimum sodium intakes. Other research supports the concept that the diet as a whole may be a more important determinant of health outcome than an individual component.[21] The high degree of correlation between most dietary components and total calorie consumption suggests that there may be important interaction with other individual dietary components.

The available data do not provide an explanation for the observations. A favourable effect of sodium restriction on various intermediate physiological variables has been shown, particularly, but not exclusively, for blood pressure and other haemodynamic characteristics. In addition to the single outcome study suggesting increased morbidity in treated patients on a low-sodium diet,[18] there is convincing evidence of adverse effects of a low-sodium diet on important physiological characteristics, including the sympathetic system and the renin-angiotensin system in particular.[22–26] The ultimate health outcome of any intervention is the sum of all its biological effects. For a low-sodium diet, harm may outweigh benefit.

An important limitation of this study is the reliability of the dietary measures. Assessment of the exposure (dietary sodium and calories) was made only once, at baseline, and by recall rather than objective measurement. The dietary recall of sodium and calorie intake is probably an underestimation, as NHANES III, using new methods, suggests.[27] Memory can be faulty, estimates of portion size can be mistaken, and diet can change from day to day. However, to the extent that such variation was random, it would tend to mute the relation of exposure (sodium and sodium/calories) to outcome (death). Although a bias is possible, there is no inherent reason to suspect one. Also, we have no information about dietary patterns after the baseline examination. However, other studies have shown that middle-aged people are likely to have a stable nutrient intake over many years.[28,29] Nevertheless, no valid inferences can be made about the effect of usual, long-term dietary patterns. The substantial limitations of the dietary measures should be seen in the context that these data are the best available for a large, long-term population study in which mortality outcomes are also available. These data have been used for other studies linking nutrients to outcome.[30–32.]

The outcome data are more reliable, since all-cause mortality is an unequivocal endpoint; ascertainment was unbiased and complete. Confidence in less precise disease-specific mortality must be guarded.

All research, especially that with non-experimental designs, is prey to confounding. Ecological studies of sodium in populations are the most susceptible to confounding, but observational studies of individuals, such as ours, are not exempt. There can be control for some known confounders, but not for all. The problem arises when an unrecognised factor, or a factor for which insufficient data are available, confounds the observed associations by being associated with both exposure and outcome. Smokers, among whom we expect the mortality rate to be higher, might also have different dietary intake, but we did not have data to address this issue here. However, in a previous study, data on smoking were available, and smoking did not affect the inverse sodium to mortality relation.[18]

Another difficulty is that people with CVD, who are therefore at higher risk of mortality, might be more likely to adopt a low-sodium diet. To address this issue, we undertook stratified analysis that excluded participants with pre-existing CVD, as well as multiple regression analysis. In both cases, the relation of sodium to mortality persisted. Nevertheless, confounding by indication, despite efforts to control for it, cannot be entirely excluded as a possible contributor to these results.

Because of the controversy that surrounds the issue of appropriate sodium intake, these new data are valuable. Much research links dietary sodium and its variation to intermediate biological characteristics, but this study adds

to the smaller body of information relating sodium intake to ultimate morbidity and mortality. The information derives from a methodologically sound study carried out in a probability sample of the US population. During follow-up of more than two decades, 3923 deaths occurred.

Even if the association is valid, and confirmed elsewhere, it does not sustain any claim of causality. Even if the association is significant, dose-related, and independent, it may be a marker for some other dietary or non-dietary factor and therefore not causally related. In addition, the observational data here provide no insight into what might occur if consumption were artificially altered to modify absolute sodium intake or its dietary concentration. A manipulated sodium intake would not inevitably yield the outcomes that occur in individuals naturally consuming that amount of salt. In other words, these data provide no support for a recommendation to increase (or decrease) intake of salt or to decrease its concentration in the diet.

Contributors

All three investigators contributed to design, analysis, and the writing of the paper.

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