The sodium-potassium ratio: adherence to guidelines in the Multi-Ethnic Study of Atherosclerosis

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Abstract

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Introduction: Few Americans meet the guidelines for sodium and potassium consumption. Yet, sodium reduction remains a major public health aim due to its relationship with hypertension and cardiovascular disease (CVD), increasing potassium is not equally emphasized. The sodium-potassium ratio may better reflect dietary quality with respect to hypertension. Few studies have investigated guideline adherence to a sodium-potassium ratio in an ethnically diverse sample. This is a notable gap due to ethnic differences in CVD risk and cultural differences in food intake among varying groups.

Methods: Secondary analysis of data collected in a large, prospective cohort study, the Multi-Ethnic Study of Atherosclerosis, was performed using sodium and potassium estimates from food frequency questionnaires and spot urine measures. Dietary and urinary sodium-potassium ratios were compared to assess compliance to the guidelines. The association between the urinary sodium-potassium ratio and systolic blood pressure was assessed using linear regression and adjusting for covariates.

Results: With a mean dietary sodium-potassium ratio equal to 1.0, very few participants (0.3%) met the guidelines. The average urinary sodium-potassium ratio (1.3) was greater than dietary estimates. A 1.0 unit change in the urinary sodium-potassium ratio was associated with a 3.4 mmHG increase in systolic blood pressure (P<0.0001) even after adjusting for antihypertensive medication use. When modeled separately, the change in systolic blood pressure for 1 gm/dl of potassium (-2.5 mmHG, p<0.0001) was much greater than that for sodium (1.0 mmHG, P<0.001).

Conclusion: Current sodium and potassium guidelines are generally not achievable and require reassessment. Dietary potassium greatly impacts systolic blood pressure and should be considered with sodium in nutrition interventions for hypertension. Use of a sodium-potassium ratio will strengthen public health planning and clinical interventions.

Table of Contents

INTRODUCTION

Health professionals have long recognized that sodium and potassium intake impacts blood pressure; however, the current guidelines for sodium and potassium consumption are not being met at a population level and may not be feasibly achieved by individuals.[1,2] Optimizing dietary sodium and potassium can improve blood pressure, a major modifiable risk factor for cardiovascular disease.[3] Because cardiovascular disease is the leading cause of death in the United States, it is critical that clinicians intervene by working with clients to achieve recommended sodium and potassium intake guidelines.[4] By reducing sodium and increasing potassium intake, some individuals experience blood pressure lowering and thus reduce risk of cardiovascular outcomes.[5,6] Yet, improvement cannot be made if the current sodium and potassium guidelines are idealistic rather than achievable.

The purpose of this study is to investigate the population level feasibility of the sodium and potassium guidelines, assessed as a ratio of sodium to potassium, for older adults and to determine if the sodium-potassium ratio is associated with systolic blood pressure, a marker of CVD risk. Background information will be provided to introduce why sodium and potassium guideline feasibility is questionable and how the sodium-potassium ratio is beneficial to research. In the introduction, the reasoning behind the current sodium and potassium guidelines will be explained and the sodium-potassium ratio will be introduced as a useful tool for population level research.

In Part I of the introduction, the rationale for current guidelines of sodium and potassium intake will be described by investigating proposed mechanisms for why sodium/potassium impacts blood pressure, reviewing results from key clinical trials, and highlighting evidence from population level studies. Then, in Part II the concept of a sodium-potassium ratio will be introduced by arguing the importance of potassium in the hypertension pathway, describing how the ratio better represents individuals' diets and the food supply, and presenting current research supporting use of the ratio as a marker for CVD risk.

Part I. Understanding Current Sodium and Potassium Guidelines

At a public health level, many organizations and guideline generating groups encourage dietary sodium reduction.[3,7-9] Reducing dietary sodium consumption is a major public health issue in the United States because across nationally representative studies researchers consistently report excess sodium intake.[10,11] Adults over the age of 20 consume 3592mg/day sodium and 2793mg/day potassium on average, according to the National Health and Nutrition Examination Survey (NHANES) 2011-2012.[10] Though sodium guidelines differ by organization, most recommend limiting sodium intake to a maximum of 2300-2400mg, meaning most Americans exceed the sodium guidelines by about 1200mg (Table 1).[3,9,12,13] Dietary potassium is equally concerning because the average American needs to increase daily potassium intake by nearly 2000mg to meet the adequate intake level of 4700mg.[14] Overall, these trends suggest that Americans consume a diet filled with processed foods (high sodium) and few fresh fruits and vegetables (low potassium). Reported sodium and potassium intake in this study was consistent with levels reported five years prior, suggesting this trend is consistent over time and requires intervention.[11]

Even though most Americans need to reduce dietary sodium and increase potassium, the Dietary Guidelines for Americans 2010 recommend even greater sodium reductions to 1500mg for at-risk populations.[12] The at-risk population includes adults 51 years of age or older, children, African Americans, or individuals with hypertension, diabetes, or chronic kidney disease.[7] This recommendation is not universally accepted and is controversial due to recent publications questioning the feasibility and effectiveness of lowering sodium intake to 1500mg.[1,15] It is important for the more restrictive sodium guideline to be investigated further because clinicians use the guidelines on a daily basis to advise patients about making diet changes. Meanwhile, the potassium guidelines are far less contentious, but are supported by less research than sodium.

In order to understand the context of dietary sodium and potassium guidelines, it is important to review the mechanisms linking sodium and potassium intake with blood pressure, the clinical trials attempting blood pressure reduction through diet, and the key observational studies defining sodium and potassium trends at a population level.

A. *Proposed mechanisms of sodium and potassium in the hypertension pathway*

The mechanisms determining blood pressure in the body result from complex interactions between the sympathetic nervous system, cardiac system, renal system, and renin-angiotensin-aldosterone system.[16-19] Dietary sodium and potassium are both thought to affect these systems, thus impacting blood pressure. Though dietary sodium and potassium have been associated with primary hypertension, the mechanisms underlying this relationship are not well understood.

Primary hypertension may cause a number of abnormalities in sodium handling, including increased secretion of aldosterone and digitalis-like factor, which leads to greater renal reabsorption of sodium.[17] A diet in excess of sodium can further increase renal reabsorption of sodium due to increased extracellular fluid osmolality and subsequent production of antidiuretic hormone (ADH).[16] The increased total reabsorption of water and sodium in the collecting duct of the kidneys, related to increased aldosterone, digitalis-like factor, and/or ADH, results in increased vascular volume.[16] It is possible that long-term increased vascular volume, caused by excess reabsorption of fluid and sodium in the kidneys, leads to even greater cardiac output, constriction of the arteries, and arterial pressure.[17]

Excess dietary sodium and impaired mechanisms related to hypertension likely effect the smooth muscle cells as well. High serum osmolality decreases sodium pump activity on the smooth muscle cells, which causes decreased intracellular potassium and increased intracellular sodium and calcium.[17] Higher intracellular calcium leads to vasoconstriction of the vasculature.[17] Additionally, the increased serum sodium resulting from sodium reabsorption decreases synthesis of nitric oxide, a potent vasodilator.[17] Overall, excess sodium combined with impaired handling of hormones related to hypertension contributes to greater vasoconstriction and increased blood pressure.

If dietary sodium is reduced, blood pressure typically lowers due to increased excretion of sodium by the kidneys.[16] The kidneys do not immediately adapt to decreased dietary sodium intake, so they continue to excrete sodium at levels greater than overall intake, thus reducing extracellular volume and blood pressure.[16] However, some individuals do not experience blood pressure reduction with sodium reduction, which has led researchers to use the term salt sensitivity.[16] It appears that humans have a spectrum of individual salt sensitivity.[16] Specific racial groups, like African Americans, and age groups, like adults over 65 years, are typically more salt sensitive, meaning greater blood pressure reduction occurs with sodium restriction.[17] However, it is important to recognize that encouraging sodium reduction at a public health level may not benefit every individual through blood pressure reduction due to varying salt sensitivity.

At the same time, potassium impacts blood pressure primarily through mechanisms in the kidneys. Dietary potassium is almost entirely excreted in the urine.[16] To be excreted, sodium and potassium are exchanged at the level of the renal cortical collecting duct.[16] Sodium channels on epithelial cells of the renal cortex transport sodium into the cells, while potassium channels on the renal medulla transport potassium into the tubule lumen for excretion.[16] With a high potassium diet, more sodium needs to be secreted in the tubule fluid to create a favorable gradient allowing for greater potassium excretion. These mechanisms act to maintain an appropriate potassium level in the body.[16] Thiazide sensitive sodium-chloride cotransporters enhance secretion of sodium in the tubule lumen beyond activity of the epithelial cell sodium transporters, leading to greater excretion of both sodium and potassium. Thus, high dietary potassium likely decreases blood pressure by causing more sodium to be excreted by the kidneys.[16]

In addition, higher dietary potassium may impact the epithelial cells of arterial and arteriolar smooth muscle cells.[17] When serum potassium increases, endothelial cells become hyperpolarized, which leads to reduction of intracellular calcium levels.[17] These mechanisms promote vasodilation beyond the impact of high dietary potassium on the kidneys.[17] Finally, dietary potassium is thought to increase salt sensitivity in some individuals.[13]

By reviewing the mechanistic relationship between sodium, potassium, and hypertension, it is clear that these mechanisms are not fully understood. This is important when considering dietary guidelines because sodium reduction may not cause blood pressure reduction for all individuals.

B. *Clinical trials*

Apart from the research investigating proposed mechanisms of sodium and potassium in the hypertension pathway, the clinical effects of attempted sodium reduction and greater potassium intake on blood pressure in experimental studies should also be considered. In general, researchers agree that eating too much sodium contributes to increased blood pressure, especially for salt sensitive and hypertensive adults.[20-22] Several important clinical trials investigated sodium reduction, including the Dietary Approaches to Stop Hypertension (DASH) trial, the

Trial of Nonpharmacologic Interventions in the Elderly (TONE), and the Trials of Hypertension Prevention (TOHP). The major findings of these three studies are described below and compared to determine how decreased sodium or increased potassium intake may impact adults with and without hypertension.

The Dietary Approaches to Stop Hypertension Sodium trial is an example of a successful clinical trial where many hypertensive and non-hypertensive adults reduced blood pressure through sodium reduction and/or whole diet changes favoring more fruits, vegetables, whole grains, and lean protein sources.[23] Participants received all foods from the research centers and maintained or decreased sodium intake to three levels: 3450mg (high), 2300mg (intermediate), or 1150 (low) mg sodium.[23]

On the control diet, participants experienced systolic blood pressure reductions equal to 2.1mmHg when sodium intake was decreased from high to intermediate and an additional 4.6mmHg when sodium intake decreased from intermediate to low.[23] On the DASH diet, participants reduced systolic blood pressure by 1.3mmHg when sodium intake decreased from high to intermediate and an additional 1.7mmHg when sodium intake decreased from intermediate to low.[23] Participants with hypertension experienced more significant reductions in systolic blood pressure compared to non-hypertensive participants (-11.5 mmHg vs. -7.1mmHg).[23] The DASH researchers argued that blood pressure reduction seen with the DASH diet and sodium reduction was equal to blood pressure reductions seen with use of single hypertension medication.[23] As a result, the DASH trial results suggest that diet can be significantly effective in achieving blood pressure reduction for hypertensive and non-hypertensive adults.

However, the results from the DASH trials are stronger than other key clinical trials researching sodium reduction. While sodium reduction as a method for primary prevention of hypertension was successful in the Trial of Hypertension Prevention (TOHP) Phase II, participants achieved decreases in systolic blood pressure equal to 2.9mmHg, less than the highest reductions seen in DASH.[24] Participants saw greater reductions in blood pressure when paired with weight loss.[13,25,26] Sodium reduction was achieved through participant education, counseling, and extended follow-up with a goal of reducing sodium to 1840mg daily.[27] It appears that education alone may be less effective in achieving sodium reduction compared to providing all food to participants.

The third key clinical trial, TONE, assessed effectiveness of sodium reduction for reducing blood pressure in older adults between 60-80 years.[28] All participants were hypertensive and taking one or two hypertension medications. Sodium reduction and weight loss were used to wean participants off of a hypertension medication by providing education and follow-up with a registered dietitian.[28] Participants decreased overall sodium intake by 920mg on average and experienced reductions in systolic blood pressure equal to 4.3mmHg on average.[28] Again, participants achieved less sodium reduction in TONE by education and counseling than was achieved by DASH researchers.

Overall, sodium reduction tends to be more effective in hypertensive adults than non-hypertensive adults. In a meta-analysis of randomized control trials, including both hypertensive and non-hypertensive participants, hypertensive participants reached greater blood pressure reduction by reducing sodium compared to non-hypertensive individuals.[19] The greatest reductions in blood pressure result from sodium reduction paired with other lifestyle changes, such as holistic diet changes or weight loss. It is also worth noting the absence of an emphasis on dietary potassium in this research.

The level of evidence linking potassium to blood pressure reduction is far less robust than that of sodium. The potassium recommendation, equal to 4700mg, was set by the IOM in a publication on Dietary Reference Intakes for electrolytes.[13] Because no dose-response studies have been done to assess the most appropriate level of potassium consumption, the panel based their recommendations on a few studies where intake of potassium between 3100-4700mg decreased risk of kidney stones in adults and decreased blood pressure in salt sensitive African Americans.[13] Meanwhile, the World Health Organization recommends that all adults consume at least 3510mg of potassium per day based on results of a meta-analysis, where consuming 3510mg of potassium was associated with the greatest blood pressure reductions.[29]

Although researchers have attempted to increase potassium intake through the use of supplements, a meta-analysis of randomized control trials supplementing adults with potassium found that potassium supplementation does not significantly impact blood pressure.[30] It is thought that the potassium from food is more effective in lowering blood pressure than supplemental forms of potassium.[13] The DASH trials suggest that a diet including a variety of fruits and vegetables, as a proxy for potassium, is effective in lowering blood pressure, though the exact amount of potassium included in the DASH diet was not defined.[23] In a meta-analysis of randomized control trials, increased dietary potassium was associated with a reduction of systolic blood pressure by 5.93mmHg. Thus, it is possible that increasing potassium in the diet may lower blood pressure.

Both dietary sodium and potassium impact blood pressure reduction in clinical trials; however, there is a lack of evidence defining the ideal amount of potassium needed to decrease blood pressure and the effectiveness of sodium reduction in clinical trials is dependent on the researcher methodology of achieving sodium reduction.

C. *Observational studies*

At a population level, the associations between dietary sodium, potassium, blood pressure, and CVD outcomes have been well researched. The Intersalt Cooperative Research group conducted a key study linking urinary sodium and potassium with blood pressure.[20] They collected urinary sodium and potassium from over 10,000 adults in 52 international centers.[20] They demonstrated that systolic blood pressure increased 4.3mmHg sodium per 2300mg urinary sodium excreted.[20] Meanwhile, urinary potassium was negatively associated with blood pressure, though significance was only reached after adjusting for confounders.[20] Since this early study, meta-analyses and systematic reviews assessing both observational and experimental research have strongly concluded that sodium is positively associated with blood pressure and moderately concluded that potassium is inversely associated with blood pressure.[13,19,31]

It has been more challenging to determine the relationship between dietary sodium, potassium, and CVD outcomes. A meta-analysis linked higher potassium diets with reduced risk of incident stroke (risk ratio = 0.76, CI = 0.66 to 0.89).[18] But, others have found no relationship between potassium intake and CVD or mortality outcomes.[3,32] A large, observational study using data collected from the Prospective Urban Rural Epidemiology (PURE) study investigated the association between global sodium consumption and risk of CVD events and mortality.[15] They found certain low levels of sodium consumption were associated with increased risk of events.[15] O'Donnell and colleagues concluded that the lowest risk of cardiovascular events and mortality occurred with sodium intake between 3000-6000mg daily.[15] With sodium intake below 3000 or above 6000mg, CVD events and/or mortality risk increased. The PURE study attempted to reduce the possibility of reverse causality by excluding participants with prior history of cardiovascular disease or an event within the past two years of enrollment.[15]

The PURE study findings conflict with dietary guidelines for sodium intake, which suggest disease risk is reduced with consumption between 1500-2400mg. Given the results of the PURE study, general intake in the US would be considered within range of reduced risk because current levels of intake in the US are near 3600mg.[10] Compared to the PURE findings, the guidelines set in the US would be in a range associated with increased risk of cardiovascular events or mortality.

At a population level, it is generally accepted that sodium is positively associated with blood pressure and potassium is inversely associated with blood pressure. However, it is difficult to conclusively state how dietary sodium and potassium impact cardiovascular events and/or mortality.

D. *Updates & Summary*

The Institute of Medicine recently published a review of evidence in which they could not support sodium intake guidelines under 2300mg for specialized populations to decrease risk of CVD outcomes given the lack of consistent, high-quality evidence supporting lower levels of consumption.[31] When they examined the evidence, they concluded that there is insufficient research supporting the guideline and that some evidence even indicated there might be increased mortality risk with sodium reduction to that level.[31]

The IOM report was received with much criticism.[33] The American Heart Association CEO spoke out against the report stating that, "we disagree with key conclusions… The report is missing a critical component – a comprehensive review of well-established evidence which links too much sodium to high blood pressure and heart disease." [34] The lifestyle guidelines set by the

American Heart Association endorse reducing sodium to 1500mg daily or reducing sodium by at least 1000mg, even if the guidelines are not met, due to small reductions in blood pressure that occur with reductions of 1000mg.[3] Because many of the studies used to assess outcome data for sodium consumption involved participants with CVD at the start of the study or other comorbidities, commentators argue that the IOM report made an inappropriate conclusion.[33,34]

The Centers for Disease Control commented on the report, suggesting that the studies used to assess the guideline were not appropriate to support their conclusions.[33] It is challenging to appropriately assess the relationship between sodium intake and cardiovascular outcomes. Some research suggests a positive relationship between sodium intake and CVD risk, while other evidence suggests an inverse relationship or no relationship at all.[13] Sodium intake varies from day-to-day and common measures either assess intake over 24-hours or rely on self-reported data to inform about usual intake, which introduces risk for misclassification of true sodium consumption.[35] Additionally, some outcome data from cohort studies are at risk of reverse causality.[35]

In summary, the evidence presented regarding the mechanistic relationship between dietary sodium, and hypertension, the clinical trials attempting blood pressure reduction through dietary changes, and the population level research questioning associations between diet and CVD outcomes has important implications for the dietary sodium and potassium guidelines. Seminal work in the field, including Intersalt and DASH, suggests dietary sodium and potassium are related to blood pressure and can be manipulated to decrease blood pressure, yet we have not conclusively determined what level of sodium and potassium intake is needed to reduce risk of cardiovascular disease. Likewise, the actual mechanisms underlying sodium, potassium, and hypertension are unclear. Despite the many uncertainties of sodium and potassium research, we have set guidelines that are not being achieved. Given current statistics regarding sodium and potassium consumption, it will be challenging to reach global compliance of sodium and potassium dietary guidelines.

Part II. Introducing the sodium-potassium ratio

The sodium and potassium guidelines critically require attention; as a result, we suggest that sodium and potassium could be considered as a ratio in research rather than individual nutrients. Use of the ratio would strengthen research because sodium and potassium both impact blood pressure at a mechanistic level, the sodium-potassium ratio better represents individuals' diets and the food supply, and the sodium-potassium ratio is likely a marker for CVD risk.

Even though the exact mechanisms describing how dietary sodium and potassium may lead to increased blood pressure, it is clear that dietary sodium and potassium do impact blood pressure.[17] At the cellular level, sodium and potassium are exchanged across membranes and an increase in one is likely to impact handling of the other.[16] As described previously, when more potassium is consumed in the diet, the kidneys will excrete more sodium to compensate.[16] While higher dietary sodium likely contributes to a more hypertensive environment in the vasculature through a number of mechanisms, higher dietary potassium favors more vasodilation and less arterial pressure by hyperpolarizing endothelial cells and excreting more sodium.[16] In clinical trials, increasing dietary potassium counteracts the effect of higher dietary sodium and it is thought that increased dietary potassium may increase salt sensitivity in some individuals.[13,23] Biologically, the joint activity of sodium and potassium impacts blood pressure.

Not only are activities of sodium and potassium clearly interrelated, but also the sodium-potassium ratio is more representative of the entire diet and better aligns with the food supply.

Researchers have evaluated the feasibility of the sodium and potassium guidelines being met either individually or as a ratio within the context of a healthy diet, which is important because the guidelines given to patients should be achievable.[2] Maillot and colleagues modeled the possibility of adults in the NHANES 2001-2002 meeting various dietary constraints, including meeting the sodium and potassium guidelines and 24 additional dietary reference intakes by age and sex groups.[2] Individual sodium and potassium guidelines could not be met in the model and maintain the additional dietary constraints, even when the researchers reduced sodium content in the food supply by 10%.[2] When they used a sodium-potassium ratio of 0.49 instead of individual guidelines in modeling, the diet constraints could be met with sodium intake between 1500-2500mg and potassium intake of 3000-5000mg.[2] The models could only hypothetically fulfill the 1500mg sodium guideline for men over 50 with significant changes from their current diet patterns.[2] From this research, it is evident that meeting both individual guidelines is unlikely given current food habits of the population and additional diet recommendations. Using a sodium-potassium ratio allows for a targeted range of sodium and potassium intake to be achieved as well as other goals for a healthy diet.

Part of the reason why the sodium-potassium ratio better allows individuals to meet goal ranges at a population level is because it inherently accounts for energy differences between individuals' diets. The current guidelines are not energy dependent and thus can be challenging to meet based only on caloric need. But, with a ratio, a targeted range of intake is appropriate for populations composed of adults with varying energy needs. Thus, the sodium-potassium ratio is more applicable to the needs of each individual within a population.

As a predictive tool, the sodium-potassium ratio is a better predictor of blood pressure than sodium or potassium intake alone and has been significantly associated with blood pressure.[18,20,26] In a study of adults who participated in the NHANES between 2005-2010, systolic blood pressure increased by 1.05mmHg for each 0.5 increase in the ratio.[36] The ratio has also been studied in the context of cardiovascular outcomes. As the sodium-potassium ratio increases, risk of cardiovascular disease also increases.[25,36] Using urinary sodium and potassium data, an assessment of the TOHP Phase II revealed that the sodium-potassium ratio was significantly associated with CVD outcomes.[25] Modeling the ratio provided the best fit compared to models of sodium or potassium alone.[25] Relative risk of CVD outcomes in the analysis was 1.24 per unit of the ratio.[25] However, other researchers have found no associations between the sodium-potassium ratio and CVD or mortality.[32] Overall, the available evidence suggests that the sodium-potassium ratio is valid predictor of blood pressure and may be associated with cardiovascular risk.

Though the sodium-potassium ratio in research appears to be associated with risk, represents diets at a population level, and relates to the physiology of hypertension, few Americans actually meet the ideal sodium-to-potassium ratio, which is 0.49 for the general population and 0.32 for the at-risk populations described previously.[12] In an analysis of NHANES 2003-2008 data, 0.12% of adults over the age of 20 in the US categorized as the general population met the sodium-to-potassium ratio of 0.49.[1] Meanwhile, 0.015% of individuals categorized as the specific population requiring greater sodium reduction met the sodium-to-potassium ratio of 0.32.[1] The average sodium-potassium ratio in NHANES 2005-2010 was 1.41.[36] At no point from 1971 – 2006 has NHANES data revealed an average sodium-to-potassium ratio less than 0.83 among U.S. adults.[1] Thus, achievement of both the current sodium and potassium guidelines will be extremely difficult at a population level. It has been suggested by the World Health Organization that <1.0 would be a reasonable ratio to set as a guideline for the general population.[8]

Given low adherence to the dietary guidelines for sodium and potassium, researchers should continue to assess the diet patterns of the US population and consider the appropriateness of the current guidelines. Though preliminary data supports the relationship between the dietary sodium-to-potassium ratio and systolic blood pressure, no studies to our knowledge have investigated the relationship in an ethnically representative sample. This is a notable gap due to ethnic differences in CVD risk and cultural differences in food intake among varying ethnic groups.[37]

Specific Aims and Hypotheses

Our first aim is to describe the sodium-to-potassium ratio in an ethnically diverse sample by sociodemographic and cultural factors (race/ethnicity, age, gender). The Multi-Ethnic Study of Atherosclerosis (MESA) is a large prospective cohort study consisting of European American, African American, Chinese American, and Hispanic American participants carefully monitored for subclinical markers of cardiovascular diseases.[38] Participants completed food frequency questionnaires between April 2010 and December 2011 that will provide dietary estimates of sodium and potassium intake.

Next, we will contrast food frequency questionnaire estimates of sodium and potassium intake with spot urine estimates of excreted sodium and potassium. We anticipate that FFQ reports of sodium will be lower than excreted levels, while estimates of potassium from diet will be higher than urinary potassium levels, due to results from previous research.[39]

Finally, we will determine if the dietary sodium-to-potassium ratio is associated with systolic blood pressure, a marker of hypertension and CVD. Individual sodium and potassium levels will also be tested for associations with systolic blood pressure. We hypothesize that the sodium-to-potassium ratio will be associated with systolic blood pressure in a large ethnically diverse sample of adults. In addition, sodium will be associated with systolic blood pressure and potassium will be inversely associated with systolic blood pressure.

METHODS

Study Design

We performed secondary analysis of data collected in a large prospective cohort study, the Multi-Ethnic Study of Atherosclerosis (MESA). Participants were recruited at six field centers (New York, New York; Baltimore, Maryland; Chicago, Illinois; Los Angeles, California; Minneapolis-St. Paul, Minnesota; and Winston Salem, North Carolina). At enrollment, participants were age 45-84 and were selected based on having no clinical CVD events or CVD procedures. By design, four racial/ethnic groups were represented in selection of participants: European American, African American, Chinese American, and Hispanic American. The study includes five clinical exams to date, which were performed from July 2000 – January 2012. A more detailed description of the study design can be found elsewhere.[38]

Data Collection

Participants completed food frequency questionnaires (FFQs) at both Exam 1 and Exam 5, ten years apart. Exam 5, conducted between April 2010 and December 2011, has more recent data and thus is more relevant to current food consumption patterns and occurred under more recent guidelines. The data collected from Exam 5 FFQs was used to estimate dietary sodium, potassium, and sodium-potassium ratios. FFQs are frequently used in the field of nutrition as a useful estimate of usual dietary intake.[40] The MESA FFQ was adapted from the Insulin Resistance Atherosclerosis Study questionnaire to include culturally appropriate foods for all races/ethnicities represented in the study.[38] The Insulin Resistance Atherosclerosis Study questionnaire was validated for use in multi-ethnic populations.[41]

Additionally, stored spot urine measures of sodium and potassium excretion were made available for analysis by the MESA kidney team. MESA researchers collected spot urine samples from participants at the time of exam 1 (July 2000 – August 2002). Measures of sodium and potassium excretion were used as a short-term estimate of intake. The amount of sodium excreted in urine is nearly equivalent to intake of dietary sodium.[13] Researchers estimate that 77-90% of dietary potassium is excreted in the urine and urinary potassium is highly correlated with potassium intake.[13] Urine collection is subject to less measurement and recall error as compared to FFQs, but provides a measure of intake over a relatively short period of time. Meanwhile, FFQs estimate intake over a year and are a better estimate of long-term consumption patterns. Both urinary and FFQ measures were used in the analyses.

Statistical Analysis

MESA covariates of interest at Exam 5 were examined using descriptive statistics, including an evaluation of sodium, potassium, and sodium-potassium ratio means/proportions by covariates. The primary covariates of interest included age, sex, and race/ethnicity. Many common covariates were excluded as potential adjustment variables as they may be a consequence of the participant's diet (e.g. body mass index). We conducted sensitivity analyses to consider different outlier elimination strategies, comparing differences between removing the upper and lower 1% of the data and excluding participants who consumed fewer than 400 or greater than 6,000 kilocalories because those extremes are implausible estimates of participants' true energy intake. The individual sodium and potassium guidelines are not specific to varying levels of energy intake for adults, so we assessed sodium and potassium intake per kilocalorie to compare intake trends across groups of participant characteristics.

To assess compliance to an ideal sodium-potassium ratio, we created a new variable representing sodium/potassium consumption. The ideal ratios are 0.49 or less for the general population and 0.32 for at-risk populations, based on the Dietary Guidelines for Americans

2010.[12] It has been suggested elsewhere that a target ratio of 1.00 would be a reasonable public health goal, so this level was also assessed.[8] Using Pearson's correlation, we also compared FFQ measures of sodium and potassium intake at exams 1 and 5 to test the stability of these measures over time.

Because we wanted to assess the association between the sodium-potassium ratio and blood pressure independent of any potential initiated treatments, we tested for an association between reported hypertension medication usage (yes or no) and the sodium-potassium ratio at both exam 1 and exam 5 using logistic regression. This was done to ensure that dietary patterns, themselves, were not indicators for anti-hypertensive drug treatment. Additional covariates included in the model were race/ethnicity, sex, and age.

Finally, to test for clinical outcomes as a result of dietary sodium and potassium intake, we estimated the association between the urinary sodium-potassium ratio and blood pressure. For this model, we used linear regression, while conditioning on plausible confounders, including race/ethnicity, sex, age, and hypertension medication usage. As before, many covariates were eliminated from consideration as potentially being partially a consequence of diet. The analysis was repeated using individual sodium and potassium estimates.

Statistical analysis was performed on Stata 11 (StataCorp LP). Results were considered to be statistically significant at the level of $p<0.05$.

RESULTS

Aim 1: Characterizing recent dietary sodium and potassium consumption by participant characteristics

At the time of exam five, participants were 53-94 years old, which is notable because the guidelines for sodium intake decrease to 1500mg for individuals who are ≥51 years old (Table 2). Mean age was equal to 69.9 years. More participants were female than male and 55% of participants reported using at least one medication for hypertension. Mean systolic blood pressure in the cohort was 124.1 mmHg and mean diastolic blood pressure was 68.2mmHg (Table 2).

Across all age groups, participants exceeded the sodium guideline by an average of 930mg (Table 3). For potassium, all age groups failed to meet the adequate intake level (4700mg) with a mean deficit of 2120mg potassium. 28% of the cohort met the sodium guideline and 6% of the cohort met the potassium guideline. Although it appeared that men consumed more sodium than women, this trend was eliminated when considering sodium intake as a ratio of total energy intake (Appendix I).

Sodium and potassium trends by race and ethnicity were inconsistent. When ordering racial/ethnic groups by level of sodium consumption from highest to lowest, Hispanic Americans consumed the most, followed by European Americans, African Americans, and Chinese Americans (Table 3). However, when considering sodium intake proportionate to total energy intake, Hispanic American participants consumed the most sodium followed by Chinese American, European American, and African American participants (Appendix I). Racial/ethnic group ordering was also inconsistent for potassium intake. From Table 3, ordering of racial/ethnic groups by potassium consumption was: 1. Hispanic American 2. European American 3. African American 4. Chinese American (Table 3). But, when energy intake was accounted for, Chinese-Americans consumed the most potassium, followed by Hispanic Americans, African Americans, and European Americans (Appendix I).

In the MESA cohort at exam 5, the average daily sodium-potassium ratio was 0.97 (Table 3). By age group, the ratio trended downward as age increased. The average sodium-potassium ratio for men was 1.04, which was higher than the average sodium-potassium ratio for women, equal to 0.93 (Table 3). Finally, ordering of the highest to lowest sodium-potassium ratio by racial /ethnic groups was: 1. African American 2. European American 3. Hispanic American 4. Chinese American (Table 3).

In the entire cohort, 0.32% of participants met the ideal sodium-potassium ratio (equal to 14/4388 participants; Table 4). If the sodium guideline were liberalized to 2300mg daily for adults older than 51 years, 4.1% of participants would meet the ideal ratio. Lastly, if the ratio were liberalized to 1.0, which has been suggested by the World Health Organization, then 58% of participants would meet the ideal ratio.[8]

Due to the presence of some implausible reported food consumption patterns, we performed a few sensitivity analyses by checking for outliers in the data. We removed the upper and lower one percent of the data. We also tested the effect of excluding participants who reported consuming fewer than 400 kcals/day or greater than 6,000 kcals/day because energy intake within those extremes likely does not represent the true energy intake of participants. Overall, we found that truncating the data produced no significant differences from the crude estimates of sodium and potassium intake, suggesting that outliers were not driving previous results (Appendix II).

Aim 2: Contrasting food frequency questionnaire measures and spot urine measures of sodium and potassium

Because stored urine samples were collected at Exam 1, cohort characteristics at Exam 1 are included in Table 5. At Exam 1, participant's usual mean consumption of sodium was 2147mg per day and mean consumption of potassium was 2628mg per day. The average sodium-potassium ratio was equal to 0.84, with little variation across age, gender, and racial/ethnic groups. Ranking of racial/ethnic groups by the sodium-potassium ratio was Hispanic American, European American, African American, and Chinese American, from highest to lowest sodium-potassium ratio. When comparing sodium and potassium consumption at Exams 1 and 5, it is evident that the sodium-potassium ratio has increased over time. There is a difference of 0.13 units between the sodium-potassium ratio at Exam 1 and Exam 5.

Meanwhile, the cohort excreted 2366mg/dL sodium and 2144mg/dL potassium according to spot urine measures (Table 7). The ratio calculated from urinary measures of sodium and potassium excretion was 1.30 on average, with consistency across age, gender, and racial/ethnic groups. When ranking racial/ethnic groups by the sodium-potassium ratio, European Americans and Chinese Americans had the highest ratio, followed by African Americans then Hispanic Americans. There was a difference of nearly 0.5 ratio units between the mean FFQ sodium-potassium ratio and the mean urinary sodium-potassium ratio (Table 9).

Aim 3: Estimating the association between the sodium-potassium ratio and systolic blood pressure

It is notable that at the time of exam 5, 55% of all participants reported taking at least one medication for hypertension, compared to only 37% at exam 1 (Table 10). At exam 1, the odds ratio for the sodium-potassium ratio increasing the likelihood of hypertension medication usage was close to 1.0 (Table 11). However, at exam 5, the odds ratio was 1.24 and reached significance, suggesting that a higher sodium-potassium ratio increased the likelihood of hypertension medication usage (p=0.04; Table 11). The channeling bias introduced by use of hypertension medications at exam 5 prevented us from assessing the association between the sodium-potassium ratio and systolic blood pressure at exam 5. As a result, we assessed systolic blood pressure and the urinary sodium-potassium ratio at exam 1 prior to greater treatment by hypertension medications. We chose to use the urinary sodium-potassium ratio versus the FFQ sodium-potassium ratio at exam 1 in the model because urinary measures are subject to less measurement error and are more accurate to recent sodium/potassium consumption at the time of exam 1.

After adjusting for covariates, the sodium-potassium ratio was significantly associated with systolic blood pressure, where systolic blood pressure increased by 3.43mmHg per unit increase of the ratio. Meanwhile, individual measures of sodium and potassium excretion were significantly associated with systolic blood pressure, with an increase in systolic blood pressure of 1.18mmHg per 1000mg/dL sodium excretion and a decrease in systolic blood pressure of 2.43 for each 1000mg/dL increase in potassium excretion (Table 12).

DISCUSSION

In this evaluation of the controversial sodium and potassium guidelines, strengthened by the use of both dietary and urinary measures of intake/excretion, very few older, ethnically diverse adults met the sodium and potassium guidelines or an ideal sodium-potassium ratio. Because the mean urinary sodium-potassium ratio was much larger than the mean dietary sodium-potassium ratio, diets are likely to be worse than described in prior research from dietary estimates of sodium and potassium intake, thus further emphasizing the importance of sodium and potassium from a public health perspective. The current sodium and potassium guidelines are idealistic, especially at the 1500mg sodium level, based on the present evaluation where so few participants could meet them. By focusing on sodium reduction alone, individuals are not benefiting from the clinically significant blood pressure reductions associated with greater potassium consumption. Until better guidelines are formed, it may be beneficial to evaluate sodium and potassium consumption in the form of a ratio where ≤ 1.0 serves as an attainable initial goal for achieving balance of the two nutrients in the diet.

A. Public Health Implications

In order for nutrition guidelines to impact health at a population level, those guidelines should be attainable. In the MESA cohort, the sodium and potassium guidelines were not broadly attainable, where only 14 out of the 4288 participants, or 0.3%, met the ideal sodium-potassium ratio based on 1500mg sodium and 4700mg potassium guidelines. This is clinically significant because the MESA cohort is ideally positioned to assess the attainability of the controversial 1500mg sodium guideline. Multiple cohort characteristics, including age (over 51 years) and ethnic diversity, predisposed participants to a higher level of CVD risk associated with sodium intake. If only 0.3% of these participants were able to meet the ideal sodium-potassium ratio based on a sodium guideline of 1500mg, it is unlikely that population level compliance with the sodium and potassium guidelines will be achieved. In another nationally representative cohort, 0.015% of participants met the ideal sodium-potassium ratio, indicating that this trend is consistent across different cohorts.[1]

Considering dietary sodium and potassium as a ratio strengthens recommendations because the sodium-potassium ratio is strongly associated with blood pressure, a major risk factor for CVD. In the MESA cohort, the sodium-potassium ratio was associated with an increase in systolic blood pressure of nearly 3.5mmHg per unit increase of the ratio. Other researchers have reported more modest associations between the sodium potassium ratio and systolic blood pressure, ranging from 0.33-2.8mmHg per unit of the ratio.[20,36,42-44] Often, sodium reduction is emphasized strongly for reducing blood pressure and CVD risk, but increasing potassium is not considered. The relative absence public health messages emphasizing increasing dietary potassium for CVD risk reduction is shocking, considering the present analysis where potassium had almost twice the effect on systolic blood pressure compared to sodium. Potassium plays an important role in the hypertension pathway and should be emphasized with sodium in dietary pattern planning for reducing CVD risk.

An appropriate initial goal for the sodium-potassium ratio is ≤1.0. Because the World Health Organization endorses a sodium-potassium ratio ≤1.0 to improve population level health, ≤1.0 is also reasonable initial goal for US guidelines.[29] At this time, the ideal sodium-potassium ratio for older adults, derived from the Dietary Guidelines for Americans 2010, is 0.32; however it is based on a sodium guideline of 1500mg that is being questioned by researchers for its effectiveness, safety, and attainability.[1,15,31] In the MESA cohort, only 2.8% of participants were able to meet a urinary sodium-potassium ratio equal to 0.32. If the ideal sodium-potassium ratio were ≤1.0, 38.5% of MESA cohort participants would meet the goal, indicating that it is more widely achievable. While diet improvement would still need to be made to reach population level compliance with a goal of 1.0, it is more feasible compared to a goal of 0.32.

There are several applications for the sodium-potassium ratio to improve dietary planning at a public health level, including the USDA Food Patterns and the Healthy Eating Index. Historically, the USDA Food Patterns have exceeded the upper limit of 2300mg sodium for adults requiring the highest categories of total energy intake.[45] If the sodium-potassium ratio were used in planning of the USDA Food Patterns, guidelines could be met across every category of energy intake because a ratio inherently allows for different amounts of sodium and potassium to be consumed across a range of energy requirements. The Healthy Eating Index is another tool that would benefit from the inclusion of the sodium-potassium ratio as it is used in research to assess diet quality.

B. Clinical Applications

Because clinicians use evidence-based guidelines to set goals for patients, it is critical for those guidelines to not only be well researched, but also achievable. Clinicians may improve efforts at CVD risk reduction by emphasizing both potassium and sodium in dietary interventions, using the urinary sodium-potassium ratio as a screening tool, and considering diet as an integral tool to complement medication therapy.

Both sodium and potassium should be emphasized in dietary interventions to reduce CVD risk. By focusing on sodium reduction alone, patients are not benefiting from the clinically significant effects of increasing potassium. Whereas a difference of 1000mg urinary sodium excretion was associated with 1.18mmHg systolic blood pressure in the MESA cohort, a difference of 1000mg urinary potassium excretion was associated with a decrease of 2.43mmHg systolic blood pressure. Potassium had twice the effect on systolic blood pressure as sodium, which suggests that potassium should have a greater role in dietary guidelines. Dietary potassium should be emphasized in all dietary interventions for hypertension and CVD risk reduction with a goal of achieving balance with sodium consumption. Notably, dietary potassium should be increased through greater consumption of potassium-rich foods rather than potassium supplements. Potassium supplements are not as effective at decreasing blood pressure as potassium from food.[13,30]

Achieving balance of sodium and potassium in dietary interventions is not only important because of the clinically significant effects of potassium, but also because patients typically cannot meet current sodium guidelines. When few adults with increased CVD risk are able to meet the more restrictive 1500mg sodium guideline, it is difficult to justify counseling patients to meet that guideline. In examples of clinical trials where participants successfully reduced sodium intake to a range between 1150 and 1850 mg, researchers either provided all food to participants or required extensive follow-up with a nutritionist over multiple months.[28,46] These conditions are typically not feasible in practice. Reaching sodium intake equal to 1500mg requires an extreme diet that places significant burden on patients and decreases quality of life. The evidence supporting a guideline for 1500mg sodium is inconsistent and controversial.[2,15,31] As a result, clinicians should not base recommendations on current guidelines but should instead consider working with patients to achieve balance of sodium and potassium consumption via the ratio.

In order to use the sodium-potassium ratio in clinical practice, clinicians can request spot urine collection for patients. Appropriate balance of sodium and potassium can be assessed using urinary sodium and potassium levels by calculating a ratio of sodium to potassium excretion. If the ratio exceeds 1, clinicians may consider referring a patient to a registered dietitian for further dietary evaluation and intervention. Assessing the urinary sodium-potassium ratio may be a way for clinicians to reduce the complications of hypertension by starting to work on improving the diet earlier in the disease progression.

Finally, clinicians should consider dietary intervention as a complement to medication therapy for hypertension. In the MESA cohort, use of hypertension medications increased within the cohort over time, as did the sodium-potassium ratio also increased over time (0.84 at exam 1; 0.97 at exam 5). The mean dietary sodium-potassium ratio at exam 5 is likely an underestimate of the true intake; yet, the mean ratio still exceeds current guidelines, suggesting little dietary change takes place even after medication is started for patients with hypertension. This is a critical opportunity for intervention by registered dietitians. Too often, clinicians neglect dietary intervention after medication has been initiated. Lifestyle changes, including decreased sodium and increased potassium consumption, can reduce the need for hypertension medications and/or increase the long-term efficacy of hypertension medication.[47,48] In an era where tight control of systolic blood pressure (<120mmHG) is thought to greatly decrease risk of CVD events and/or mortality, dietary intervention should be further emphasized to achieve greater blood pressure reduction.[49] Older adults should be encouraged by clinicians to work with registered dietitians for improvement of sodium and potassium intake even after hypertension medications have been started.

Strengths and Limitations

Several factors may have influenced our findings. First, the MESA cohort is relatively large and has been followed prospectively over time, thus increasing power and decreasing bias for our findings. However, participants in the MESA cohort have received a high level of medical follow-up, which may have predisposed them to more medical treatments compared to the adult US population overall. There is the possibility that reductions in blood pressure may have resulted from treatments other than sodium reduction, such as anti-hypertension medications. We attempted to mitigate this possibility in our model by considering hypertension medication usage as a covariate.

A possible limitation of our research is that we used FFQs to estimate participants' sodium and potassium intake because urine collection is thought to be a more objective measure of sodium and potassium consumption. However, our study is greatly strengthened by the use of spot urine samples to estimate participants' urinary sodium and potassium excretion. In fact, there were distinct differences between the urinary and FFQ estimated sodium-potassium ratios. This could be related to the fact that added sodium was not accounted for in the FFQ estimates of sodium consumption. Additionally, social desirability bias likely led to decreased reporting of sodium and increased reporting of potassium in FFQ estimates of intake. This trend is important for registered dietitians to consider because it will require in depth interviewing and detailed questions to best estimate sodium and potassium intake of patients.

Another possible limitation of our study relates to the comparison of self-reported food frequency questionnaires over a range of ten years. The sodium-potassium ratios at exam 1 and exam 5 were only moderately correlated (Appendix III). Over time, the ratio increased. Given the amount of time between exam 1 and 5, a moderate correlation between 0.44 and 0.46 indicates that there was some stability in the cohort diet over time, but some changes did occur.

The impact of hypertension medications in the MESA cohort was notable. While 37% of all participants reported taking at least one hypertension medication at the time of exam 1, this estimate increased to 55% by exam 5. The urinary sodium-potassium ratio was significantly associated with hypertension medication usage at exam 5 (p=0.04; Table 11). This trend was not apparent at exam 1, where the sodium-potassium ratio was not significantly associated with use of hypertension medications (p=0.76). It is evident that by exam 5 channeling bias impacted our findings, where participants with a higher sodium-potassium ratio were more likely to be medicated with hypertension medications. As a result, we could not use data from exam 5 to determine the association between systolic blood pressure and sodium intake because use of hypertension medications acted as a confounder in the causal pathway.

Future Directions

Public health professionals and clinicians should consider using the sodium-potassium ratio in dietary planning and as a screening tool at the level of 1.0. Attempting to meet this general guideline is an attainable way to improve dietary balance of sodium and potassium. Greater emphasis on improving potassium intake in making public health recommendations and counseling patients will likely improve attempts at blood pressure reduction. In the future, validation studies should be done to assess use of the sodium-potassium ratio as a marker of CVD outcomes and use of ≤1.0 as a sodium-potassium ratio guideline. Population level guidelines for sodium and potassium consumption would greatly benefit from additional research identifying levels of sodium and potassium consumption associated with CVD outcomes, especially for individuals at higher risk of CVD. Research in this area is necessary to identify appropriate and ideal sodium and potassium guidelines.

CONCLUSION

In a cohort of ethnically diverse, older adults, very few participants were able to meet the goal sodium-potassium ratio based on current sodium and potassium guidelines. Our findings contribute to a larger body of evidence emphasizing the need for updated sodium and potassium recommendations. The sodium-potassium ratio was highly correlated with systolic blood pressure, a major risk factor for cardiovascular disease. Including the sodium-potassium ratio would strengthen monitoring tools used for assessment of diet patterns, such as the USDA Food Patterns and Healthy Eating Index. Finally, clinicians should include both sodium and potassium in diet recommendations to achieve greater reductions in systolic blood pressure, while using the urinary sodium-potassium ratio as a screening tool for diet quality when available.

Table 1. Sodium and Potassium Recommendations

Organization	Guideline for daily sodium consumption (mg)	Guideline for daily potassium consumption (mg)
The Dietary Guidelines for Americans 2010[12]	<2300 for general population (set as upper limit by the DRI council) 1500 for older adults (≥51 years), children 18 and under, individuals with hypertension, chronic kidney disease, diabetes, or are African-American	4700 (derived from the adequate intake level set by the DRI Council)
American Heart Association/American College of Cardiology on Lifestyle Management to Reduce Cardiovascular Disease Risk Practice Guidelines[3]	≤2400 Further reduction to 1500 if possible Reduce by at least 1000 even if guidelines are not met	-
Institute of Medicine *Sodium Intake in Populations: Assessment of the Evidence*[31]	≤2300	-
The 7th Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure	≤2400	-
World Health Organization Sodium Intake for Adults and Children Guideline and Potassium Intake for Adults and Children Guideline[8,29]	<2000	≥3510

Table 2. Demographics of Participants, Multi-Ethnic Study of Atherosclerosis, Exam 5, 2010-2011

Age, mean (sd)	69.9 (9.5)
Age Category, n (%)	
45-54	76 (1.6)
55-64	1539 (32.6)
65-74	1480 (31.4)
75-84	1276 (27.1)
≥84	345 (7.3)
Gender	
Male	2202 (46.7)
Female	2514 (53.3)
Race/Ethnicity, n (%)	
European American	1926 (40.8)
Chinese American	541 (11.5)
African American	1250 (26.5)
Hispanic American	999 (21.2)
Body Mass Index, (kg/m^2), mean (sd)	28.5 (5.7)
Systolic blood pressure (mmHg), mean (sd)	124.1 (20.9)
Diastolic blood pressure (mmHg), mean (sd)	68.2 (10.1)
Hypertension, n (%)	2778 (59.7)
Diabetes, n (%)	921 (20.0)

Table 3. Usual sodium, potassium intake and the sodium-potassium ratio at Exam 5 by selected characteristics, Multi Ethnic Study of Atherosclerosis, April 2010-December 2011[1]

	Usual sodium intake (mg) Mean (SD)	Usual potassium intake (mg) Mean (SD)	Usual sodium-potassium ratio Mean (SD)
All	2430.0 (1403.1)	2580.1 (1329.9)	0.97 (0.32)
Age Category			
45-54	2651.5 (1512.5)	2609.1 (1471.4)	1.04 (0.29)
55-64	2711.6 (1516.0)	2704.2 (1406.0)	1.03 (0.32)
65-74	2333.7 (1343.3)	2529.9 (1295.7)	0.95 (0.31)
75-84	2210.6 (1278.7)	2482.1 (1259.6)	0.92 (0.31)
≥85	2303.2 (1314.0)	2580.6 (1297.2)	0.91 (0.29)
Gender			
Male	2637.6 (1419.4)	2666.1 (1362.2)	1.01 (0.33)
Female	2245.3 (1292.1)	2503.6 (1296.1)	0.93 (0.31)
Race/Ethnicity			
European American	2504.2 (1256.2)	2658.8 (1182.9)	0.96 (0.30)
Chinese American	1866.8 (1227.3)	2119.3 (1165.0)	0.89 (0.30)
African American	2437.1 (1497.3)	2441.7 (1410.4)	1.04 (0.36)
Hispanic American	2577.1 (1562.7)	2837.6 (1491.0)	0.93 (0.32)

[1]*Participants with energy intake <400kcal/day or >6000kcal/day were excluded*

Table 4. Usual sodium-potassium ratio by selected characteristics, Multi-Ethnic Study of Atherosclerosis, 2010-2011*

	Sample N	# participants meeting ratio ≤ 0.32 (%)	# participants meeting ratio <0.49 (%)	# participants meeting ratio ≤1.0 (%)
All	4388	14 (0.32)	180 (4.1)	2546 (58)
Age Category				
45-54	73	0 (0)	1 (1.4)	35 (48)
55-64	1,457	4 (0.27)	36 (2.5)	719 (49)
65-74	1,400	5 (0.36)	66 (4.7)	845 (60)
75-84	1,151	4 (0.35)	63 (5.5)	744 (65)
≥85	307	1 (0.33)	14 (4.6)	203 (66)
Gender				
Male	2,066	4 (0.19)	64 (31)	1,059 (51)
Female	2,322	10 (0.43)	116 (5)	1,487 (59)
Race/Ethnicity				
European American	1,813	7 (0.39)	59 (3.3)	1060 (58)
Chinese-American	500	0 (0)	31 (6.2)	345 (69)
African American	1,128	2 (0.18)	37 (3.3)	557 (49)
Hispanic American	947	5 (0.53)	53 (5.6)	584 (62)

** Excluding participants with energy intake <400 kcal/day or >6000 kcal/day*

Table 5. Demographics of Participants, Multi-Ethnic Study of Atherosclerosis, Exam 1, 2000-2002

Age, mean (sd)	61.2 (10.2)
Age Category, n (%)	
45-54	1,905 (28.4)
55-64	1,853 (27.6)
65-74	1,986 (29.7)
75-84	955 (14.3)
Gender, n (%)	
Male	3160
Female	3545
Race/Ethnicity, n (%)	
European American	2,596 (38.7)
Chinese American	799 (11.9)
African American	1,839 (27.4)
Hispanic American	1,471 (21.9)
Body Mass Index, (kg/m^2), mean (sd)	28.3 (5.5)
Systolic blood pressure, (mmHg), mean (sd)	126.6 (21.5)
Diastolic blood pressure, (mmHg), mean (sd)	71.9 (10.3)
Diabetes, n (%)	837 (12.5)
Take Medication for Hypertension, n (%)	2,497 (37.3)
European American	858 (33.1)
Chinese American	228 (28.5)
African American	929 (50.6)
Hispanic American	482 (32.8)

Table 6. Usual sodium, potassium intake and the sodium-potassium ratio at Exam 1 by selected characteristics, Multi Ethnic Study of Atherosclerosis, 2000 - 2002

	Usual sodium intake (mg) Mean (SD)	Usual potassium intake (mg) Mean (SD)	Usual sodium-potassium ratio Mean (SD)
All	2147.2 (1255.5)	2628.7 (1310.9)	0.84 (0.30)
Age Category			
45-54	2181.9 (1224.8)	2625.3 (1280.6)	0.85 (0.30)
55-64	2132.8 (1219.3)	2594.5 (1257.9)	0.85 (0.30)
65-74	2175.8 (1329.4)	2667.6 (1368.2)	0.83 (0.30)
75-84	2185.8 (1357.4)	2580.3 (1287.9)	0.86 (0.32)
Gender			
Male	2439.6 (1328.8)	2812.2 (1344.5)	0.89 (0.29)
Female	1880.9 (1108.3)	2469.6 (1262.3)	0.79 (0.29)
Race/Ethnicity			
European American	2143.2 (1246.5)	2615.0 (1283.8)	0.84 (0.30)
Chinese American	2089.8 (1211)	2611.8 (1293.5)	0.81 (0.29)
African American	2156.7 (1300.7)	2659.3 (1374.7)	0.83 (0.30)
Hispanic American	2172.8 (1239.3)	2624.5 (1288.8)	0.85 (0.29)

Table 7. Excreted sodium and potassium at Exam 1 by selected characteristics, Multi-Ethnic Study of Atherosclerosis, 2000 - 2002

	Sodium [mg/dL (SD)]	Potassium [mg/dL (SD)]	Sodium-potassium ratio (SD)
All	2366.49 (1169.86)	2144.24 (1153.33)	1.30 (0.72)
Age Category			
45-54	2379.19 (1197.87)	2098.23 (1133.31)	1.31 (0.69)
55-64	2379.72 (1199.54)	2154.18 (1142.47)	1.29 (0.72)
65-74	2336.42 (1130.86)	2122.75 (1154.57)	1.30 (0.71)
75-84	2401.32 (1218.0)	2141.99 (1214.43)	1.33 (0.73)
Gender			
Male	2589.57 (1148.83)	2279.51 (1128.82)	1.32 (0.70)
Female	2176.10 (1151.92)	2032.57 (1164.57)	1.27 (0.73)
Race/Ethnicity			
European American	2335.88 (1171.91)	2113.50 (1175.86)	1.31 (0.74)
Chinese American	2367.55 (1161.8)	2148.15 (1145.99)	1.31 (0.75)
African American	2374.21 (1185.11)	2154.9 (1140.15)	1.29 (0.70)
Hispanic American	2410.33 (1151.04)	2183.14 (1133.05)	1.28 (0.68)

Table 8. Urinary sodium-potassium ratio by selected characteristics, Multi-Ethnic Study of Atherosclerosis, 2000 - 2002

	Sample N	# participants meeting ratio ≤ 0.32 (%)	# participants meeting ratio <0.49 (%)	# participants meeting ratio ≤1.0 (%)
All	6,705	189(2.8)	597(8.9)	2588 (38.6)
Age Category				
45-54	1,905	35 (1.8)	122 (6.4)	549 (28.8)
55-64	1,853	31(1.7)	106 (5.7)	561 (30.3)
65-74	1,986	33(1.7)	123 (6.2)	509 (25.6)
75-84	955	16(1.7)	36 (3.8)	147 (15.4)
Gender				
Male	3160	67 (2.1)	231 (7.3)	1148(36.3)
Female	3545	122 (3.4)	366 (10.3)	1440(40.6)
Race/Ethnicity				
European American	2,596	58(2.2)	232 (8.9)	999 (38.5)
Chinese-American	799	37 (4.6)	78 (9.8)	309 (38.7)
African American	1,839	51 (2.8)	162 (8.8)	692 (37.6)
Hispanic American	1,471	39 (2.7)	113 (7.7)	551 (37.5)

Table 9. Usual sodium-potassium ratio compared to spot urine collection of sodium and potassium at Exam 1, 2000 - 2002

	Exam 1 FFQ Sodium-potassium ratio (SD)	Exam 1 Spot Urine Sodium-potassium ratio (SD)
All	0.84 (0.30)	1.30 (0.72)
Age Category		
45-54	0.85 (0.30)	1.31 (0.69)
55-64	0.85 (0.30)	1.29 (0.72)
65-74	0.83 (0.30)	1.30 (0.71)
75-84	0.86 (0.32)	1.33 (0.73)
Gender		
Male	0.89 (0.29)	1.32 (0.70)
Female	0.79 (0.29)	1.27 (0.73)
Race/Ethnicity		
European American	0.84 (0.30)	1.31 (0.74)
Chinese American	0.81 (0.29)	1.31 (0.75)
African American	0.83 (0.30)	1.29 (0.70)
Hispanic American	0.85 (0.29)	1.28 (0.68)

Table 10. Sodium and potassium intake reported by FFQ by race/ethnicity at Exam 1 and Exam 5, Multi-Ethnic Study of Atherosclerosis, 2000 - 2002 and 2010- 2011

EXAM 1	Sodium [mg (SD)]	Potassium [mg (SD)]	Sodium-potassium ratio (SD)	HTN Medication (%)
All	2147.2 (1255.5)	2628.7 (1310.9)	0.84 (0.30)	37.3
European American	2143.2 (1246.5)	2615.0 (1283.8)	0.84 (0.30)	33.1
Chinese American	2089.8 (1211)	2611.8 (1293.5)	0.81 (0.29)	28.5
African American	2156.7 (1300.7)	2659.3 (1374.7)	0.83 (0.30)	50.6
Hispanic American	2172.8 (1239.3)	2624.5 (1288.8)	0.85 (0.29)	32.8
EXAM 5				
All	2396.6 (1417.9)	2535.0 (1323.4)	0.97 (0.32)	54.6
European American	2339.6 (1387.3)	2489.0 (1275.1)	0.96 (0.31)	43.5
Chinese American	2385.1 (1533.6)	2505.7 (1391.0)	0.96 (0.32)	88.6
African American	2479.8 (1443.3)	2589.6 (1364.4)	0.99 (0.33)	45.5
Hispanic American	2429.3 (1345.7)	2591.1 (1311.5)	0.96 (0.31)	57.2

Table 11. Odds ratio between the sodium-potassium ratio and hypertension medication usage at Exam 1 and Exam 5, 2000 - 2002 and 2010- 2011[1]

	Hypertension medication usage		
	Odds ratio	95% confidence interval	p-value
Exam 1			
Sodium-potassium ratio	1.03	0.85-1.25	0.76
Exam 5			
Sodium-potassium ratio	1.24	1.01-1.53	0.04

[1] Covariates include age, gender, and race

Table 12. Association between sodium, potassium, sodium-potassium ratio, and systolic blood pressure at Exam 1, 2000 - 2002 [1]

	Systolic blood pressure (mmHg)	
	β-coefficient (95% confidence interval) [2]	p-value
Sodium excretion (mg/dL)	1.18 (0.73 – 1.62)	<0.0001
Potassium excretion (mg/dL)	-2.43 (-2.87 to - 2.00)	<0.0001
Sodium-potassium ratio	3.43 (2.78 – 4.09)	<0.0001

[1] *Model covariates include age, gender, race/ethnicity, and hypertension medication usage*
[2] *β-coefficient demonstrates difference in blood pressure associated with 1000mg difference in absolute sodium and potassium excretion*

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Appendix I

Usual sodium and potassium intake to usual energy intake by selected characteristics, Multi-Ethnic Study of Atherosclerosis, April 2010-December 2011

	Mean sodium to energy ratio [(mg/d)/(kcal/d)]			Mean potassium to energy ratio [(mg/d)/(kcal/d)]		
	Crude	Outliers excluded*	Energy truncated**	Crude	Outliers excluded*	Energy truncated**
All	1.50	1.50	1.50	1.64	1.65	1.64
Age Category						
45-54	1.58	1.58	1.58	1.59	1.58	1.59
55-64	1.54	1.54	1.53	1.57	1.57	1.57
65-74	1.49	1.49	1.49	1.66	1.66	1.66
75-84	1.47	1.47	1.47	1.71	1.71	1.70
≥85	1.44	1.45	1.45	1.70	1.70	1.69
Gender						
Male	1.50	1.50	1.50	1.56	1.56	1.56
Female	1.49	1.50	1.50	1.72	1.72	1.72
Race/Ethnicity						
European American	1.49	1.49	1.49	1.63	1.63	1.63
Chinese American	1.51	1.53	1.52	1.78	1.79	1.78
African American	1.48	1.48	1.48	1.53	1.52	1.52
Hispanic American	1.53	1.52	1.52	1.74	1.74	1.74

** Excluding upper and lower 1% participants*

*** Excluding participants with energy intake <400 kcal/day or >6000 kcal/day*

Appendix II

Table 13. Usual sodium and potassium intake by selected characteristics, excluding outliers*, Multi-Ethnic Study of Atherosclerosis, April 2010-December 2011

	Usual sodium (mg/d)		Usual potassium (mg/d)	
	Mean	% ≤ 1500mg	Mean	% ≥ 4700mg
All	2371.3 (1300.5)	27.6	2521.7 (1229.4)	6.2
Age Category				
45-54	2651.5 (1512.5)	23.3	2509.2 (1206.9)	8.3
55-64	2627.4 (1346.6)	20.9	2628.9 (1223.4)	6.5
65-74	2292.9 (1269.9)	29.4	2491.9 (1250.6)	6.4
75-84	2152.7 (1203.1)	33.1	2430.1 (1207.0)	5.4
≥85	2286.5 (1318.5)	31.9	2504.9 (1219.9)	6.1
Gender				
Male	2560.3 (1355.9)	22.9	2604.9 (1246.0)	6.9
Female	2201.6 (1219.5)	31.9	2448.3 (1210.1)	5.5
Race/Ethnicity				
European American	2482.9 (1223.7)	20.7	2634.0 (1143.4)	5.4
Chinese American	1816.1 (1191.3)	50.78	2073.8 (1174.8)	3.5
African American	2357.0 (1341.3)	30.2	2374.7 (1262.0)	6.6
Hispanic American	2475.9 (1373.2)	25.3	2727.3 (1296.9)	8.6

** Excluding upper and lower 1% of data*

Table 14. Usual intake of sodium and potassium by selected characteristics with energy truncation*, Multi-Ethnic Study of Atherosclerosis, April 2010-December 2011

	Usual sodium (mg/d)		Usual potassium (mg/d)	
	Mean	% ≤ 1500mg	Mean	% ≥ 4700mg
All	2430.0 (1403.1)	26.8	2580.1 (1329.9)	6.8
Age Category				
45-54	2651.5 (1512.5)	23.3	2609.1 (1471.4)	9.6
55-64	2711.6 (1516.0)	20.3	2704.2 (1406.0)	7.5
65-74	2333.7 (1343.3)	28.7	2529.9 (1295.7)	6.8
75-84	2210.6 (1278.7)	31.8	2482.1 (1259.6)	5.8
≥85	2303.2 (1314.0)	31.3	2580.6 (1297.2)	6.8
Gender				
Male	2637.6 (1419.4)	42.2	2666.1 (1362.2)	3.7
Female	2245.3 (1292.1)	42.4	2503.6 (1296.1)	3.3
Race/Ethnicity				
European American	2504.2 (1256.2)	20.4	2658.8 (1182.9)	5.7
Chinese American	1866.8 (1227.3)	49.2	2119.3 (1165.0)	3.6
African American	2437.1 (1497.3)	29.3	2441.7 (1410.4)	7.4
Hispanic American	2577.1 (1562.7)	24.3	2837.6 (1491.0)	10.0

** Excluding participants with energy intake <400kcal / d or >6000kcal / d*

Appendix III

Table 15. Correlation between sodium, potassium, sodium-potassium ratio, and systolic blood pressure

EXAM 1 (n = 6702)		
	r-value	p-value
Urinary sodium (mg/dL) and systolic blood pressure (mmHg)	0.0097	0.43
Urinary potassium (mg/dL) and systolic blood pressure	-0.13	0.0
Urinary ratio and systolic blood pressure (mmHg)	0.15	0.0
EXAM 5 (n = 4386)		
Usual sodium (mg) and systolic blood pressure (mmHg)	-0.059	0.0001
Usual potassium (mg) and systolic blood pressure (mmHg)	-0.033	0.03
Usual sodium-potassium ratio and systolic blood pressure (mmHg)	-0.036	0.02
EXAM 1 & EXAM 5		
Usual sodium intake at Exam 1 and usual sodium intake at Exam 5	0.46	<0.0001
Usual potassium intake at Exam 1 and usual potassium intake at Exam 5	0.44	<0.0001