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Expansion of the National Salt Reduction Initiative: A Mathematical Model of Benefits and Risks of Population-level Sodium Reduction

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Abstract

Background—The National Salt Reduction Initiative, in which food producers agree to lower sodium to levels deemed feasible for different foods, is expected to significantly reduce sodium intake if expanded to a large sector of food manufacturers.

Objective—Given recent data on the relationship between sodium intake, hypertension and associated cardiovascular disease at a population level, we sought to examine risks and benefits of the program.

Design—To estimate the impact of further expanding the Initiative on hypertension, myocardial infarction and stroke incidence, and related mortality, given food consumption patterns across the US, we developed and validated a stochastic microsimulation model of hypertension, MI and stroke morbidity and mortality, using data from food producers on sodium reduction among foods, linked to 24-hour dietary recalls, blood pressure, and cardiovascular histories from the National Health and Nutrition Examination Survey.

Results—Expansion of the Initiative to ensure all restaurants and manufacturers reach agreed-upon sodium targets would be expected to avert between 0.9-3.0 MI's (a 1.6-5.4% reduction) and 0.5-2.8 strokes (a 1.1-6.2% reduction) per 10,000 Americans per year over the next decade, after incorporating consumption patterns and variations in the effect of sodium reduction on blood pressure among different demographic groups. Even high levels of consumer addition of table salt or substitution among food categories would be unlikely to neutralize this benefit. However, if recent epidemiological associations between very low sodium and increased mortality are causal, then older women may be at risk of increased mortality from excessively low sodium intake.

Conclusions—An expanded National Salt Reduction Initiative is likely to significantly reduce hypertension and hypertension-related cardiovascular morbidity, but may be accompanied by potential risks to older women.

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Keywords

sodium; hypertension; cardiovascular disease; health disparities

Introduction

While US Department of Agriculture guidelines recommend that Americans consume less than 2300 mg of sodium per day,¹ or equivalently 5750 mg of salt per day, adults in the United States consume an average of 3900 mg of sodium per day, 75% of which comes from packaged and prepared foods.² Because the majority of sodium intake is not from consumer addition of table salt, a key strategy to lower sodium intake has been to reformulate packaged and prepared foods. A growing number of national initiatives around the world aim to reduce sodium consumption through either voluntary or mandatory corporate commitment.³⁻⁷ For example, in the United Kingdom (UK), the nationwide salt reduction program is thought to have led to an estimated 15% reduction in population salt intake between the program's initiation in 2003 and follow-up data recorded in 2011.^{8, 9} Using the UK as a model, the New York City Health Department in 2010 announced the National Salt Reduction Initiative (NSRI),¹⁰ a pilot partnership of health organizations and corporations, which sought to reduce US sodium intake by 20% through voluntary corporate commitments to cut sodium in 62 categories of packaged and 25 categories of restaurant food items.^{11, 12} The 27 companies who have enrolled in the Initiative to date have pledged that specific food items included in the NSRI food target list (e.g., canned soup) will meet targets for salt content deemed feasible and appropriate for that food type. To date, the NSRI has reduced sodium among the enrolled group of food producers, but health outcomes from the program remain unclear.¹³

Previous studies have correlated lower sodium intake with lower blood pressure, and lower blood pressure with reduced cardiovascular disease (CVD) incidence and mortality among particularly hypertensive persons,^{8, 14-17} but epidemiological evidence also suggests an association between excessively low sodium and heightened mortality in some studies.^{18, 19} Hence, the “optimal” level of sodium that should be recommended has been unclear and heavily debated.

Prior mathematical modeling studies have found that large-scale sodium reduction would be expected to lower CVD.^{20, 21} Yet three major questions remain unresolved. First, how much total impact would be expected if food producers lowered sodium to meet the specific NSRI targets, which are set to levels of sodium considered feasible for each food type? Data on how much sodium reduction is possible for each type of food (while preserving quality, safety, and taste) have not been integrated with data on how much impact would be expected from such reductions given the physiology of salt-and-blood-pressure relationships. A second unresolved question is whether the dynamics of producer and consumer behavior will be likely to neutralize the NSRI effort, meaning that incomplete enrollment of food producers to the Initiative may allow consumers to substitute the newly lower-sodium foods for higher-sodium alternatives (e.g., if high-sodium tomato soup is still available on the market from uncommitted food producers, consumers may substitute lower-sodium soup for

the higher-sodium variety), and consumers may add more table salt after the Initiative. Would the level of sodium reduction considered feasible by food producers have significant impact on hypertension and hypertension-associated CVD, or be easily neutralized by non-adherent producer and consumer behaviors? A third question is whether the Initiative could produce perverse outcomes, given associations between low sodium excretion levels and a heightened risk of mortality.¹⁸ It remains unclear whether this association is causal or confounded by comorbidities. An Institute of Medicine committee found limited evidence that low salt intake may be associated with adverse health effects in subgroups of the population such as some patients with congestive heart failure, diabetes, or chronic kidney disease.²² Adopting a precautionary principle, it is prudent to identify which parts of the population may be pushed into a territory of increasing risk by the NSRI, if the association between very low sodium levels and higher mortality is indeed causal.

We sought to use extensive new data from food producers to address these questions, to examine the potential implications of a proposal to expand the NSRI from the current 27 food producers to all food producers in the US through incentivized voluntary agreements or potential legislative mandates. We used detailed tables of specific foods and their sodium content before and after participation in the NSRI, and linked this information to 24-hour dietary recalls from a nationally representative sample of 20,448 people among whom we also identified demographic features, cardiovascular event history, and blood pressure, to estimate how much sodium reduction and hypertension reduction would be experienced among different demographic groups. Using validated equations relating sodium intake and systolic blood pressure changes to the incidence and mortality from myocardial infarction (MI) and stroke, we estimated the cardiovascular impact of the expanded NSRI among different segments of the US population, and under varying possible producer and consumer responses to the Initiative. We also tracked whether any individuals were likely to cross thresholds of low sodium intake that have been correlated with increased mortality.

Methods

We constructed a discrete-time individual-level microsimulation model of MI and stroke, and associated mortality, in the US population, using input data from the NSRI and the National Health and Nutrition Examination Survey (NHANES, 2003-2010, the most recent years available with consistent sodium intake estimation procedures; $N = 20,448$). The Appendix contains all input data, equations, and complete technical details consistent with international model reporting guidelines,²³ along with a link to program code for replication and extension of our analysis. Here, we provide an overview of the model's key components (Figure 1 and Table 1).

Simulated Population

We simulated a nationally representative sample of 10,000 adults aged 18 to 85 over the period 2015-2024. We chose to use a 10-year projection because we found that the uncertainty intervals around parameters were reasonable over this period. We would have to assume long-term stability of CVD incidence rates over unreasonably long periods beyond this. The simulated individuals were stratified by cohorts defined by age (18-39, 40-59, or

60-85 years old), sex, race/ethnicity (NHANES categories of non-Hispanic White, and non-Hispanic Black, and Mexican American), and income (<100% of the federal poverty level, 100-300%, and >300%) (eTable 1).

Initial systolic and diastolic blood pressure, hypertension medication use, history of MI or stroke, and daily sodium intake were assigned to each simulated individual by sampling from the probability distributions of each of these variables in NHANES, specific to each demographic characteristic listed above (eTables 2-6).²⁴ Hypertension status was defined as systolic or diastolic blood pressure greater than or equal to 140 mm Hg or 90 mm Hg, respectively, or use of antihypertensive medication. The probability distributions of daily sodium intake from NHANES were based on 24-hour dietary recall data corrected for within-person variations in consumption to estimate usual daily intake;²⁵ survey sample weights were used to generate a nationally-representative population.²⁶

Estimated sodium and blood pressure reductions from NSRI

The NSRI has announced sodium reduction agreements for each type of packaged and restaurant food.^{11, 12} For example, the NSRI target for packaged macaroni and cheese is to lower sodium from a current average of 531 mg per 100 grams of the food to 400 mg/100g. We simulated the proposed expansion of the program to all food manufacturers and restaurants in the US, and performed subanalyses simulating sodium reduction among packaged foods only, restaurant foods only, or both.

To estimate the effects of the NSRI on total daily per capita sodium intake for each individual in NHANES, food items included in the NSRI targets were matched to each food item in each 24-hour dietary recall in NHANES, using US Department of Agriculture (USDA) food codes and descriptions.²⁷ The USDA food code sequence associated with each food item in NHANES provided information on major commodity groups and more specific subgroups within the major groups. In order to provide conservative estimates of sodium reduction, within each food subgroup we only matched food items when the NHANES food description specifically contained the NSRI targeted food items. Next, given a person's daily reduction in sodium intake given the items in their dietary recall subjected to reduced sodium from the NSRI, we calculated the expected change in systolic blood pressure (SBP) using a previously validated non-linear equation derived from the Dietary Approaches to Stop Hypertension trial, which adjusts for differential blood pressure reduction by age for each gram reduction in sodium intake.^{21, 28, 29} The equation, detailed in the Appendix, estimates for example a reduction of 3.1 mm Hg in SBP given a 1g sodium reduction for 60-year olds, versus a reduction of 2.6 mm Hg in SBP given a 1g sodium reduction for 50-year olds. We conducted several sensitivity analyses, itemized below, to examine alternative sodium-blood-pressure relationships.

We estimated how much the relative risk of MI and stroke incidence and mortality would be reduced in each group, given their SBP reduction, accounting for the prevalence of prior MI or stroke and elevated risk of recurrent MI or stroke among those with a prior history of cardiovascular disease. To do so, we used the most recently updated equations derived from the Framingham Heart Study (1980 to 2003), which have been extensively validated in several recent cohorts containing more than 23,000 subjects, and subsequently validated

against a meta-analysis of prospective patient-level data on blood pressure and CVD mortality from more than 1 million adults^{21, 30-33} (Appendix Equations). The model validation involved comparing model results for new MI and stroke against data from Heart and Stroke statistics from the American Heart Association and ensuring the model was within 5% error of the independent estimates.³⁴ In all scenarios, we calculated how many people would potentially shift from above the level of sodium intake considered safe, to below 3g/day or 2g/day, which are thresholds variously debated as being associated with elevated mortality.¹⁸

We simulated the model 10,000 times using Monte Carlo sampling from the probability distributions of all input parameters for each cohort to capture uncertainties in our estimates, generating 95% confidence intervals around all outcomes. The model was implemented in the program *R* (v. 3.0.2, the R Project for Statistical Computing, Vienna) using the computer code linked in the Appendix.

Sensitivity and uncertainty analyses

Numerous alternative equations have been derived to capture the sodium-blood-pressure relationship. We considered each of these alternatives in sensitivity analyses. In our first sensitivity analysis, we considered how our results would change if the benefits of reduced sodium intake were principally among hypertensive rather than normotensive persons, with no greater salt sensitivity among older adults (i.e., per recent meta-analysis, a 1g/day decline in sodium intake would result in a 1.2 mm Hg lower SBP among hypertensive persons and 0.60 mm Hg lower SBP among normotensive persons).^{16, 20, 35} In a second sensitivity analysis, we modeled a larger response to salt reduction among the Black population than the non-Black population (-1.8 mm Hg in SBP among hypertensives and -1.2 mm Hg among normotensives persons from a 1g decline in daily sodium intake,^{20, 28, 36} versus -1.2mm Hg and -0.6 mm Hg, respectively, among non-Blacks).^{16, 20, 28, 36-39}

In a third sensitivity analysis, we assessed outcomes based on a recently published equation derived from international datasets adjusted for age, hypertensive status, and race.⁴⁰ In this equation, for a white, normotensive person at age 50, each 100 mmol per day sodium reduction lowered SBP by 3.7 mm Hg. For every year above or below age 50, there was 0.1 mm Hg larger or smaller blood pressure reduction, respectively. Additionally, there was 2.5 mm Hg greater reduction among Blacks and 1.9 mm Hg greater reduction among hypertensives.

In a fourth sensitivity analysis, we considered the possibility of incomplete participation of food producers in the Initiative, such that consumers could preferentially choose the highest-sodium foods from uncommitted food producers that are still on the market after the Initiative (e.g., the highest-sodium macaroni and cheese after the NSRI), to examine what levels of enrollment of food producers would be necessary to still ensure significant population-level benefits of an NSRI under a worst-case scenario of consumer substitution to the highest-sodium products for each food type. We varied the proportion of foods consumed covered by the NSRI to identify the critical percentage of food items that need to be covered under the NSRI such that even if consumers are substituting for the highest-sodium food products in each food category, the overall systolic blood pressure, myocardial

infarction, and stroke incidence and mortality rates in the US would still be significantly lowered by the Initiative. We also estimated how much table salt would need to be added by the consumer to fully neutralize the NSRI benefits, and whether such addition was consistent with observed ranges of consumer salt addition.⁴¹

Results

We estimate that if there were no change to current levels of sodium intake, the US population aged 18 to 85 years old would be expected to experience an annual rate of approximately 40.0 new MIs (95% CI: 39.8 -40.2) and 34.3 strokes (95% CI: 34.0-34.5) per 10,000 persons, consistent with the current incidence estimates of 40.0 new MIs and 34.5 new strokes per 10,000 persons aged 35 to 74 years old.^{34, 42, 43}

The expanded NSRI covering both packaged and restaurant food items would be expected to reduce mean daily sodium intake by 447 mg per person per day on average, or 13.0% (95% CI: 12.9-13.1) (Figure 2, eTables 7 and 8), which would correspond to a reduction in the prevalence of hypertension by 3.0% (95% CI: 2.7-3.3); and MI and stroke incidence by 5.4% (95% CI: 4.9-5.9) and 6.3% (95% CI: 5.7-6.9), respectively (Table 2). The Initiative would be expected to lower MI and stroke mortality by 5.1% (95% CI: 3.8-6.3) and 4.9% (95% CI: 3.2-6.7), respectively.

If the NSRI included only restaurant food items, the program would lower MI and stroke mortality by an estimated 2.7% (95% CI: 1.4-4.0) and 2.1% (95% CI: 0.4-3.9), respectively (Tables 2 and 3). Hence, most of the benefit from the program would likely be due to sodium changes among packaged foods.

Implications for health disparities

We estimated that individuals in all demographic cohorts would benefit from sodium reduction from the expanded NSRI (Figure 3), but the projected benefits in MI and stroke incidence would be largest among non-Hispanic White males in the 40-59 year old age cohort (experiencing a 5.5% (95% CI: 5.1-5.9) decline in MI incidence, and a 7.8% (95% CI: 7.2-8.3) decline in stroke incidence), after accounting for their typical food choice preferences and NSRI-associated sodium reductions. Mexican males, aged 60-85, had a slightly but not significantly lower percentage change in incident MI than non-Hispanic White males, aged 40-59. Mortality rates also decreased the most in this cohort, by 5.6% (95% CI: 5.0-6.3) for MI and by 8.0% (95% CI: 6.3-9.7) for stroke.

The expanded NSRI would not be expected to significantly narrow the disparity in overall hypertension prevalence between non-Hispanic Blacks and Whites. Before the implementation of the NSRI, the difference in overall hypertension prevalence between non-Hispanic Black and White people was 12.6% (95% CI: 12.4-12.8), while after the implementation, the gap was estimated to be 12.8% (95% CI: 12.6-13.0) (Table 2).

In the simulations including both restaurant and packaged foods in the NSRI, we estimated that large numbers of people, 1,217 (95% CI: 1205-1229) per 10,000 persons, would move from above the threshold of 3 grams/day of sodium recently correlated to increased

mortality,¹⁸ to below that threshold due to the NSRI. Approximately 901 (95% CI: 893-909) per 10,000 persons would be expected to lower sodium consumption below the threshold of 2 grams/day due to the NSRI. The group at particular risk for moving below this threshold was older women (Figure 2).

Sensitivity analyses

In our first sensitivity analysis, we examined what would happen if reductions in sodium intake were mostly beneficial to hypertensive persons and not differentially beneficial among older adults, as observed in the DASH trial (Table 3). Under the scenario in which the NSRI was implemented in both restaurant and packaged food items, annual incidence rates of MI and strokes, respectively, decreased by 0.9 per 10,000 persons and by 0.5 per 10,000 persons from the pre-NSRI scenario (a 1.6% (95% CI: 1.1-2.1) and 1.1% (95% CI: 0.5-1.7) reduction, respectively (Table 3)).

In our second sensitivity analysis, we simulated a larger blood pressure impact of reduced sodium intake among Blacks than non-Blacks, and observed that the annual incidence of MI decreased by 1.0 per 10,000 persons and stroke incidence decreased by 0.7 per 10,000 persons from the pre-NSRI scenario (a 1.9% (95% CI: 1.4-2.4) and 1.5% (95% CI: 0.8-2.1) reduction, respectively (Table 3)).

In our third sensitivity analysis, we evaluated the impact of reduced sodium intake on blood pressure based on recently derived equations from an international meta-analysis. The annual incidence of MI and stroke decreased by 2.2 per 10,000 and 1.0 per 10,000 (a 4.0% (95% CI: 3.5-4.5) and 2.3% (95% CI: 1.7-3.0) reduction, respectively (Table 3)).

In our fourth sensitivity analysis, we simulated scenarios in which only a certain fraction of food producers was committed to the NSRI and consumers preferentially chose the highest-sodium product for each food type. The NSRI was expected to significantly reduce the incidence and mortality of MI and stroke at the population level (at the $p < 0.05$ threshold) when at least 65% of foods in each USDA food category were included in the NSRI (Table 3). Furthermore, while discretionary salt use currently provides 5 to 10 percent of total sodium intake in the US, adding even as much as 15% of current daily sodium intake at the table or while cooking in the form of table salt would not neutralize the benefits of the NSRI: annual MI and stroke incidence would still decrease by 1.5% (95% CI: 0.3-1.5) and 1.6% (95% CI: 0.9-2.2), respectively. It would require the addition of 768 grams of salt per person per day at the table or during cooking (a rise in table salt use to 20% of current discretionary intake) to fully neutralize the NSRI (Table 3).

Discussion

Using a mathematical model of dietary sodium intake, blood pressure and subsequent MI and stroke incidence and mortality in the US, we found that if all food manufacturers and restaurants in the US committed to meet the current NSRI targets, hypertension prevalence would be expected to decrease by 3.0% by 2024, from 37.1% to 36.0%. Yet the benefits may be heterogeneous among different groups. The projected benefits in terms of MI and stroke incidence and mortality would be expected to be largest among non-Hispanic White

males in the 40-59 year old age cohort given their food consumption profiles and given the amount of sodium that is expected to be lowered in each type of food according to NSRI targets. However, in light of recent epidemiological associations between very low sodium intake and mortality, we tracked whether individuals were likely to cross thresholds below 3g/day or 2g/day of sodium. We found a significant increase in the number of people falling below these thresholds due to the NSRI among older women, when accounting for observed patterns of consumption and existing sodium intake across the country.

Among those expected to benefit from the NSRI, we found that even high levels of substitution from lowered-sodium to highest-sodium products in each food category would be unlikely to neutralize the benefits of an expanded NSRI, nor would high levels of consumer sodium addition. This has been a major question for nutritional modifications at the population level.³⁷ To ensure statistically significant population-level reductions in MI and stroke incidence and mortality, we estimate that at least 65% of foods in each USDA food category would need to be included in the NSRI, in the worst-case scenario in which consumers preferentially substituted the newly lower-sodium food items within the highest-sodium food products in each food category.

Our model enhances the findings from previous models of sodium reduction in the US^{20, 21} by incorporating data on the feasible sodium reduction levels for each type of food, integrated with data on how consumers behave in terms of current food preferences, to calculate expected benefits from sodium reduction through food modification. Nevertheless, our analysis has limitations inherent to modeling based on secondary data sources. First, we modeled the effects of sodium reduction on blood pressure based on published data, assuming that the health benefits of sodium reduction were mediated through changes in blood pressure observed in such trials. The differential effects of sodium reduction according to hypertension status, age and race were modeled in accordance with observational data, and there is uncertainty around these estimates when applied to the total population.^{8, 38, 40} The relationship between sodium intake and CVD risks requires further assessment across various countries to evaluate differential impacts based on the baseline sodium intake and CVD risk among diverse populations. However, in sensitivity analyses, our results were robust to wide variations in these assumptions. Second, we used data from NHANES, which are subject to the limitations of survey studies, including recall biases, acceptability biases, and underreporting.⁴⁷ Because we relied on dietary recall data that may underestimate the baseline sodium intake, our study may provide conservative results of expanded NSRI benefits. Third, we did not account for potential effects of sodium reduction that are not directly associated with blood pressure reduction, nor hypertension-related pathologies such as chronic kidney disease, which may garner additional benefits from the program. Finally, validated equations are not available to account for simultaneous potassium intake changes that also have been correlated with cardiovascular mortality. High sodium excretion is most strongly associated with increased blood pressure in persons with lower potassium excretion. Derivation of relevant equations to estimate the impact of these interrelationships is an important topic for future study.

Our model used a 10-year projection because this is the duration over which uncertainty in the model remains manageable. For potential future research, one could combine our

approach with demographic models, such as the Lee-Carter model, to identify how overall population size, total morbidity, and long-term health care may shift in ways that could either increase or decrease costs in the future.⁴⁸ Also, because the National Salt Reduction Initiative is a partnership of health organizations and corporations through voluntary corporate commitments, the program does not have publicly available information on how much the product reformulations cost for individual companies, nor on observed changes in demand that lead to differences in corporate revenue that would be included in a cost-effectiveness analysis conducted from a societal perspective. Once the information on the cost of the program becomes publicly available, future cost-effectiveness analysis of the program would be valuable.

Our model was validated against independent MI and stroke incidence estimates. However, such estimates are not themselves a real population registry, but rather are largely from surveys. Hence, our model cannot be thought of as calibrated or validated retrospectively against a real population. A potential amendment to this problem is prospective validation and further refinement of the model against emerging datasets that will provide some further insights.

In sum, consistent with the positive impact of national salt reduction initiatives implemented in other countries,^{3-7, 49} our findings indicate that expansion of the NSRI would be likely to significantly lower hypertension and hypertension-related cardiovascular disease in the US, given food producers' negotiated estimates of how much sodium can be reduced in each food, even in the context of complex producer and consumer responses to the Initiative. Yet the benefits and potential risks would be unevenly distributed; younger men would be expected to benefit most, while older female adults may be among those who might be subjected to higher risks from very low sodium levels. This suggests that careful consideration should be made of how to target such large-scale population-wide sodium reduction initiatives so as to minimize adverse effects among the most vulnerable.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Appendix - Equations

A. Equations used to estimate change in blood pressure and relative risk of MI or stroke

1. Equations used to estimate change in blood pressure with sodium reduction

Main simulation²¹:

Change in SBP = $-0.0598(x/23) - 0.0431 \times \text{age in years}$

(x = change in mg of sodium intake)

Sensitivity analyses:

i. Estimated change in SBP associated with 1g/day sodium reduction²⁰

Group	No increased salt sensitivity by age	No increased salt sensitivity by age Increased salt sensitivity in Black population
Persons with hypertension	1.20	1.80
All Others	0.60	1.20

ii. Estimated change in SBP associated with 100mmol/day sodium reduction⁴⁰

Change in SBP = $-3.735 \cdot (x/2300) - 0.105 \cdot (\text{age in years} - 50) - 1.874 \cdot \text{Htn} - 2.489 \cdot \text{Black}$

(x = change in daily mg of sodium intake; Htn: hypertensive vs. normotensive; Black: Black vs. non-Black)

2. Equations used to estimate relative risk reduction of MI and stroke²¹

(x = age in years)

Slope MI = $-1.1009 \text{E} - 05x^2 + 8.6305 \text{E} - 04x + 3.5176 \text{E} - 02$

Relative risk of MI = $2^{(\text{change in SBP} \cdot \text{slope MI})}$

Slope stroke = $-2.5946 \text{E} - 05x^2 + 2.3052 \text{E} - 03x + 2.2168 \text{E} - 02$

Relative risk of stroke = $2^{(\text{change in SBP} \cdot \text{slope stroke})}$

B. Equations used to model monthly probability of MI or stroke ²¹

(x = age in years)

Given no history of MI,

Male: $y = 1 \text{E} - 04 \cdot e^{0.0312x}$

Female: $y = 8 \text{E} - 06 \cdot e^{0.0599x}$

Given no history of stroke,

Male: $y = 9 \text{E} - 06 \cdot e^{0.0622x}$

Female: $y = 3 \text{E} - 06 \cdot e^{0.0741x}$

Given history of CVD,

The risk of MI or stroke without a history of CVD was multiplied by a constant with a mean of 2, standard deviation 1.0204, gamma distribution (shape=3.84166, scale=0.520608)

C. Equations used to model probability of mortality after MI or stroke ²¹

(x = age in years)

After MI,

$$\text{Male: } y = 0.0289 * e^{0.0269x}$$

$$\text{Female: } y = 0.0004 * e^{0.0706x}$$

After stroke,

$$\text{Male: } y = 0.0003 * e^{0.0782x}$$

$$\text{Female: } y = 0.0034 * e^{0.0428x}$$

D. Company Commitments to NSRI Targets¹⁰

The NSRI applauds the companies that have agreed to pursue NSRI targets, and we encourage all manufacturers, restaurants, supermarkets and other food companies to follow their example.

- Au Bon Pain
- Bertucci's Italian Restaurant
- Black Bear European Style Deli
- Boar's Head Provisions Co.
- Butterball
- Campbell Soup Company
- Delhaize America
- Dietz & Watson
- FreshDirect
- Furmano's
- Goya Foods
- Hain Celestial
- Heinz
- Ken's Foods
- Kraft Foods
- LiDestri Foods/Francesco Rinaldi
- Mars Food US
- McCain Foods
- Premio
- Red Gold, Inc.
- Snyder's-Lance, Inc.
- Starbucks Coffee Company
- Subway®

- Target Corporation
- Unilever
- Uno Chicago Grill
- White Rose

E. Simulation code available at <https://sdr.stanford.edu>

References

1. United States Department of Agriculture. Dietary Guidelines for Americans. 2010. Available from: <http://www.cnpp.usda.gov/DGAs2010-PolicyDocument.htm>
2. Mattes RD, Donnelly D. Relative contributions of dietary sodium sources. J Am Coll Nutr. 1991; 10(4):383–93. [PubMed: 1910064]
3. European Commission. Survey on member states' implementation of the European Salt Reduction Framework. 2012. Available from: http://ec.europa.eu/health/nutrition_physical_activity/docs/salt_report1_en.pdf
4. Food Safety Authority of Ireland. Salt commitments and updates. 2010. Available from: http://www.fsai.ie/science_and_health/salt_commitments_and_updates.html
5. UK Food Standards Agency. Salt. 2007. Available from: <http://food.gov.uk/healthiereating/salt/>
6. Webster JL, Dunford EK, Hawkes C, Neal BC. Salt reduction initiatives around the world. J Hypertens. 2011; 29(6):1043–50. [PubMed: 21546876]
7. World Action on Salt and Health. Japan salt action summary. 2010. Available from: <http://www.worldactiononsalt.com/action/asia.htm>
8. He FJ, MacGregor GA. Salt reduction lowers cardiovascular risk: meta-analysis of outcome trials. Lancet. 2011; 378(9789):380–2. [PubMed: 21803192]
9. He FJ, Pombo-Rodrigues S, Macgregor GA. Salt reduction in England from 2003 to 2011: its relationship to blood pressure, stroke and ischaemic heart disease mortality. BMJ Open. 2014; 4(4):e004549.
10. New York City Department of Health and Mental Hygiene. National Salt Reduction Initiative. 2010. Available from: <http://www.nyc.gov/html/doh/html/diseases/salt.shtml>
11. New York City Department of Health and Mental Hygiene. National Salt Reduction Initiative Packaged Food Categories and Targets. 2010. Available from: <http://www.nyc.gov/html/doh/downloads/pdf/cardio/packaged-food-targets.pdf>
12. New York City Department of Health and Mental Hygiene. National Salt Reduction Initiative Restaurant Food Categories and Targets. 2010. Available from: <http://www.nyc.gov/html/doh/downloads/pdf/cardio/cardio-salt-nsri-restaurant.pdf>
13. New York City Department of Health and Mental Hygiene. National Salt Reduction Initiative Corporate Commitments. 2014. Available from: <http://www.nyc.gov/html/doh/downloads/pdf/cardio/nsri-corporate-commitments.pdf>
14. World Health Organization. Effect of reduced sodium intake on blood pressure, renal function, blood lipids and other potential adverse effects. 2014. Available from: <http://apps.who.int/iris/handle/10665/79325>
15. Aburto NJ, Ziolkovska A, Hooper L, Elliott P, Cappuccio FP, Meerpohl JJ. Effect of lower sodium intake on health: systematic review and meta-analyses. BMJ. 2013; 346:f1326. [PubMed: 23558163]
16. He FJ, MacGregor GA. Effect of longer-term modest salt reduction on blood pressure. Cochrane Database Syst Rev. 2013; (3):CD004937. [PubMed: 23633321]
17. He FJ, MacGregor GA. Reducing population salt intake worldwide: from evidence to implementation. Prog Cardiovasc Dis. 2010; 52(5):363–82. [PubMed: 20226955]

18. O'Donnell M, Mente A, Rangarajan S, McQueen MJ, Wang X, Liu L, et al. Urinary sodium and potassium excretion, mortality, and cardiovascular events. *N Engl J Med*. 2014; 371(7):612–23. [PubMed: 25119607]
19. O'Donnell MJ, Yusuf S, Mente A, Gao P, Mann JF, Teo K, et al. Urinary sodium and potassium excretion and risk of cardiovascular events. *JAMA*. 2011; 306(20):2229–38. [PubMed: 22110105]
20. Bibbins-Domingo K, Chertow GM, Coxson PG, Moran A, Lightwood JM, Pletcher MJ, et al. Projected effect of dietary salt reductions on future cardiovascular disease. *N Engl J Med*. 2010; 362(7):590–9. [PubMed: 20089957]
21. Smith-Spangler CM, Juusola JL, Enns EA, Owens DK, Garber AM. Population strategies to decrease sodium intake and the burden of cardiovascular disease: a cost-effectiveness analysis. *Ann Intern Med*. 2010; 152(8):481–7. W170–3. [PubMed: 20194225]
22. Strom, BL.; Yaktine, AL.; Oria, M., editors. Sodium Intake in Populations: Assessment of Evidence. Washington (DC): Institute of Medicine, National Academy Press; 2013.
23. Rahmandad H, Sterman JD. Reporting guidelines for simulation-based research in social sciences. *Syst Dyn Rev*. 2012; 28(4):396–411.
24. Centers for Disease Control and Prevention. National Health and Nutrition Examination Survey, 2009 - 2010 Data Documentation, Codebook, and Frequencies. 2011. Available from: http://www.cdc.gov/nchs/nhanes/nhanes2009-2010/BPX_F.htm
25. Tooze JA, Midthune D, Dodd KW, Freedman LS, Krebs-Smith SM, Subar AF, et al. A new statistical method for estimating the usual intake of episodically consumed foods with application to their distribution. *J Am Diet Assoc*. 2006; 106(10):1575–87. [PubMed: 17000190]
26. Centers for Disease Control and Prevention. Continuous NHANES web tutorial. 2013. Available from: http://www.cdc.gov/nchs/tutorials/Nhanes/index_continuous.htm
27. United States Department of Agriculture. The USDA Food and Nutrient Database for Dietary Studies, 5.0. 2013. Available from: http://www.ars.usda.gov/SP2UserFiles/Place/12355000/pdf/fndds/fndds5_doc.pdf
28. Bray GA, Vollmer WM, Sacks FM, Obarzanek E, Svetkey LP, Appel LJ, et al. A further subgroup analysis of the effects of the DASH diet and three dietary sodium levels on blood pressure: results of the DASH-Sodium Trial. *Am J Cardiol*. 2004; 94(2):222–7. [PubMed: 15246908]
29. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension*. 2003; 42(6):1206–52. [PubMed: 14656957]
30. D'Agostino RB Sr, Grundy S, Sullivan LM, Wilson P, CHD Prediction Group. Validation of the Framingham coronary heart disease prediction scores: results of a multiple ethnic groups investigation. *JAMA*. 2001; 286(2):180–7. [PubMed: 11448281]
31. D'Agostino RB Sr, Vasan RS, Pencina MJ, Wolf PA, Cobain M, Massaro JM, et al. General cardiovascular risk profile for use in primary care: the Framingham Heart Study. *Circulation*. 2008; 117(6):743–53. [PubMed: 18212285]
32. Lewington S, Clarke R, Qizilbash N, Peto R, Collins R, Prospective Studies C. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet*. 2002; 360(9349):1903–13. [PubMed: 12493255]
33. National Heart Lung and Blood Institute. Incidence and prevalence: Chart book on cardiovascular and lung diseases, 2006. 2009. Available from: http://www.nhlbi.nih.gov/resources/docs/cht-book_ip.htm
34. Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Blaha MJ, et al. Heart disease and stroke statistics--2014 update: a report from the American Heart Association. *Circulation*. 2014; 129(3):e28–e292. [PubMed: 24352519]
35. He FJ, MacGregor GA. How far should salt intake be reduced? *Hypertension*. 2003; 42(6):1093–9. [PubMed: 14610100]
36. Vollmer WM, Sacks FM, Ard J, Appel LJ, Bray GA, Simons-Morton DG, et al. Effects of diet and sodium intake on blood pressure: subgroup analysis of the DASH-sodium trial. *Ann Intern Med*. 2001; 135(12):1019–28. [PubMed: 11747380]

37. Cappuccio FP, Markandu ND, Carney C, Sagnella GA, MacGregor GA. Double-blind randomised trial of modest salt restriction in older people. *Lancet*. 1997; 350(9081):850–4. [PubMed: 9310603]
38. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. *N Engl J Med*. 2001; 344(1):3–10. [PubMed: 11136953]
39. Swift PA, Markandu ND, Sagnella GA, He FJ, MacGregor GA. Modest salt reduction reduces blood pressure and urine protein excretion in black hypertensives: a randomized control trial. *Hypertension*. 2005; 46(2):308–12. [PubMed: 15983240]
40. Mozaffarian D, Fahimi S, Singh GM, Micha R, Khatibzadeh S, Engell RE, et al. Global sodium consumption and death from cardiovascular causes. *N Engl J Med*. 2014; 371(7):624–34. [PubMed: 25119608]
41. United States Department of Agriculture. Sodium and Potassium, Dietary Guidelines for Americans. 2005. Available from: <http://www.health.gov/dietaryguidelines/dga2005/document/html/chapter8.htm>
42. Centers for Disease Control and Prevention. Heart disease facts. 2014. Available from: <http://www.cdc.gov/heartdisease/facts.htm>
43. Centers for Disease Control and Prevention. Stroke facts. 2014. Available from: <http://www.cdc.gov/stroke/facts.htm>
44. Roger VL, Go AS, Lloyd-Jones DM, Adams RJ, Berry JD, Brown TM, et al. Heart disease and stroke statistics--2011 update: a report from the American Heart Association. *Circulation*. 2011; 123(4):e18–e209. [PubMed: 21160056]
45. CDC. Heart Disease Facts. Available from: <http://www.cdc.gov/heartdisease/facts.htm>
46. CDC. Stroke Facts. Available from: <http://www.cdc.gov/stroke/facts.htm>
47. Freedman L, Schatzkin A, Midthune D, Kipnis V. Dealing with dietary measurement error in nutritional cohort studies. *J Natl Cancer Inst*. 2011; 103(14):1086–92. [PubMed: 21653922]
48. Lee R, Miller T. Evaluating the performance of the Lee-Carter method for forecasting mortality. *Demography*. 2001; 38(4):537–49. [PubMed: 11723950]
49. He FJ, Brinsden HC, MacGregor GA. Salt reduction in the United Kingdom: a successful experiment in public health. *J Hum Hypertens*. 2014; 28(6):345–52. [PubMed: 24172290]

Figure 1.

Microsimulation model of the expanded National Salt Reduction Initiative (NSRI). The US population is simulated by age, sex, race/ethnicity and income. Each simulated individual is given a dietary profile based on the 24-hour dietary recalls in the National Health, Nutrition and Examination Survey, specific to their demographic characteristics, and an associated systolic blood pressure (SBP), hypertension status (Htn status), and history of cardiovascular disease in the form of myocardial infarction or stroke (CVD history), which affects their risk of recurrent MI or stroke. Based on their dietary profile, the model examines which foods they consume that are included in the NSRI targets, and reduces their daily sodium intake in correspondence to the quantity of those foods in their consumption profile. Their sodium intake from non-NSRI foods is unchanged. Based on their sodium reduction, we calculate the change in their SBP, and the associated change in their risk of MI or stroke.

Figure 2. Estimated daily sodium intake by demographic characteristic (gender, age, race, and income) before and after an NSRI affecting packaged foods only (“packaged”), restaurant foods only (“restaurant”), or both (“both”).

Figure 3.

Projected reductions in cardiovascular disease (myocardial infarctions (MIs) and stroke) and related deaths by age, gender, and race/ethnicity after an expanded NSRI.

Table 1
Model Parameters and Sources

Parameters	Source
Population size of demographic cohorts (Appendix eTable 1)	NHANES 2003-2010
Risk of myocardial infarction (MI) or stroke by demographic group (Appendix Equations)	Model-based estimates from meta-analysis data ^{21, 32, 33}
Baseline MI prevalence (Appendix eTable 2)	NHANES 2003-2010
Baseline stroke prevalence (Appendix eTable 3)	NHANES 2003-2010
Baseline diastolic blood pressure (Appendix eTable 4)	NHANES 2003-2010
Baseline systolic blood pressure (Appendix eTable 5)	NHANES 2003-2010
Baseline hypertension treatment prevalence (Appendix eTable 6)	NHANES 2003-2010
Systolic blood pressure reduction from sodium reduction (Appendix Equations)	Model-based estimates from meta-analysis and trial ^{8, 20, 36, 40} data
Relative risk reduction in MI/stroke from each mm Hg reduction in systolic blood pressure (Appendix Equations)	Model calibration to national data ²¹
MI or stroke mortality rate (Appendix Equations)	Model calibration to national data ^{21, 32, 33}

Table 2
Projected Estimates of Hypertension Prevalence and Annual Incidence and Mortality of Cardiovascular Disease (Myocardial Infarction [MI] and Stroke) per 10,000 Persons, and Reduction from the Baseline, Mean (SE)

	Hypertension, %			MI Incidence, n			Stroke Incidence, n			MI Mortality, n			Stroke Mortality, n		
	Restaurant	Packaged	Both	Restaurant	Packaged	Both	Restaurant	Packaged	Both	Restaurant	Packaged	Both	Restaurant	Packaged	Both
All persons	36.7(0.0) [*] -0.4(0.0) [*]	36.1(0.0) [*] -1.0(0.0) [*]	36.0(0.0) [*] -1.1(0.0) [*]	54.4(0.1) -1.5(0.1)	53.4(0.1) -2.5(0.1)	52.9(0.1) -3.0(0.1)	43.0(0.1) -1.4(0.1)	41.7(0.1) -2.7(0.1)	41.6(0.1) -2.8(0.1)	8.3(0.0) [*] -0.2(0.0) [*]	8.2(0.0) [*] -0.3(0.0) [*]	8.1(0.0) [*] -0.4(0.0) [*]	4.6(0.0) [*] -0.1(0.0) [*]	4.5(0.0) [*] -0.2(0.0) [*]	4.5(0.0) [*] -0.2(0.0) [*]
Age															
18-39	7.5(0.0) [*] -0.2(0.0) [*]	7.2(0.0) [*] -0.5(0.0) [*]	7.1(0.0) [*] -0.6(0.0) [*]	18.2(0.1) -0.0(0.1)	17.7(0.1) -0.4(0.1)	17.6(0.1) -0.5(0.1)	5.3(0.0) [*] -0.0(0.0) [*]	5.3(0.0) [*] -0.0(0.0) [*]	5.1(0.0) [*] -0.2(0.0) [*]	1.0(0.0) [*] -0.0(0.0) [*]	1.0(0.0) [*] -0.0(0.0) [*]	1.0(0.0) [*] -0.0(0.0) [*]	0.0(0.0) [*] -0.0(0.0) [*]	0.0(0.0) [*] -0.0(0.0) [*]	0.0(0.0) [*] -0.0(0.0) [*]
40-59	35.6(0.0) [*] -0.3(0.0) [*]	35.0(0.0) [*] -0.9(0.0) [*]	34.7(0.0) [*] -1.2(0.0) [*]	40.0(0.1) -0.8(0.1)	38.8(0.1) -2.0(0.1)	38.7(0.1) -2.1(0.1)	21.2(0.1) -0.3(0.1)	20.2(0.1) -1.3(0.1)	20.2(0.1) -1.3(0.1)	3.9(0.0) [*] -0.0(0.0) [*]	3.8(0.0) [*] -0.1(0.0) [*]	3.7(0.0) [*] -0.2(0.0) [*]	0.6(0.0) [*] -0.0(0.0) [*]	0.5(0.0) [*] -0.1(0.0) [*]	0.5(0.0) [*] -0.1(0.0) [*]
60-85	6.9(0.0) [*] -1.1(0.0) [*]	66.3(0.0) [*] -1.7(0.0) [*]	66.1(0.0) [*] -1.9(0.0) [*]	104.9(0.2) -4.0(0.2)	103.5(0.2) -5.4(0.2)	102.6(0.2) -6.3(0.2)	102.4(0.2) -4.0(0.2)	99.7(0.2) -6.7(0.2)	99.6(0.2) -6.8(0.2)	20.0(0.1) -0.6(0.1)	19.6(0.1) -1.0(0.1)	19.5(0.1) -1.1(0.1)	13.1(0.1) -0.4(0.1)	12.8(0.1) -0.7(0.1)	12.8(0.1) -0.7(0.1)
Gender															
Male	36.6(0.0) -0.4(0.1)	35.5(0.0) [*] -1.1(0.0) [*]	35.7(0.0) [*] -1.3(0.0) [*]	72.4(0.2) -1.9(0.2)	70.8(0.2) -3.5(0.2)	70.3(0.2) -4.0(0.2)	48.8(0.1) -1.6(0.1)	47.2(0.1) -3.2(0.1)	47.0(0.1) -3.4(0.1)	13.6(0.1) -0.3(0.0) [*]	13.3(0.1) -0.6(0.0) [*]	13.2(0.1) -0.7(0.0) [*]	5.8(0.0) [*] -0.1(0.0) [*]	5.6(0.0) [*] -0.3(0.0) [*]	5.6(0.0) [*] -0.3(0.0) [*]
Female	36.8(0.1) -0.4(0.1)	36.4(0.1) -0.8(0.1)	36.3(0.1) -0.9(0.1)	36.4(0.1) -1.2(0.1)	35.9(0.1) -1.7(0.1)	35.6(0.1) -2.0(0.1)	37.2(0.1) -1.2(0.1)	36.3(0.1) -2.1(0.1)	36.2(0.1) -2.2(0.1)	3.2(0.0) [*] -0.1(0.1)	3.0(0.0) [*] -0.2(0.1)	3.0(0.0) [*] -0.2(0.1)	3.3(0.0) [*] -0.2(0.0) [*]	3.3(0.0) [*] -0.2(0.0) [*]	3.3(0.0) [*] -0.2(0.0) [*]
Race/Ethnicity															
Mexican	31.7(0.1) -0.5(0.1)	31.1(0.1) -1.1(0.1)	31.0(0.1) -1.2(0.1)	53.7(0.2) -1.4(0.2)	52.9(0.2) -2.2(0.2)	52.3(0.2) -2.8(0.2)	42.3(0.2) -1.2(0.2)	41.2(0.2) -2.3(0.2)	41.1(0.2) -2.4(0.2)	8.0(0.1) -0.1(0.1)	8.0(0.1) -0.2(0.1)	7.8(0.1) -0.4(0.1)	4.5(0.0) [*] -0.0 [*] (0.1)	4.3(0.0) [*] -0.1(0.1)	4.3(0.0) [*] -0.1(0.1)
NH White	32.8(0.1) -0.4(0.1)	32.2(0.1) -1.0(0.1)	32.1(0.1) -1.1(0.1)	56.0(0.1) -1.4(0.1)	54.7(0.1) -2.7(0.1)	54.3(0.1) -3.1(0.1)	42.6(0.1) -1.8(0.1)	41.4(0.1) -3.0(0.1)	41.2(0.1) -3.2(0.1)	8.9(0.1) -0.2(0.1)	8.6(0.0) [*] -0.5(0.1)	8.6(0.0) [*] -0.5(0.1)	4.5(0.0) [*] -0.2(0.0) [*]	4.4(0.0) [*] -0.3(0.0) [*]	4.3(0.0) [*] -0.4(0.0) [*]
NH Black	45.5(0.1) -0.3(0.1)	44.8(0.1) -0.8(0.1)	44.9(0.1) -0.9(0.1)	53.6(0.2) -1.8(0.2)	52.4(0.2) -3.0(0.2)	52.2(0.2) -3.2(0.2)	44.0(0.2) -1.3(0.2)	42.7(0.2) -2.6(0.2)	42.7(0.2) -2.6(0.2)	8.0(0.1) -0.3(0.1)	7.8(0.1) -0.5(0.1)	7.8(0.1) -0.5(0.1)	4.8(0.1) -0.1(0.1)	4.6(0.1) -0.3(0.1)	4.6(0.1) -0.3(0.1)
Income															
Low	37.4(0.1) -0.4(0.1)	36.9(0.1) -0.9(0.1)	36.8(0.1) -1.0(0.1)	55.9(0.2) -1.7(0.2)	55.1(0.2) -2.5(0.2)	54.5(0.2) -3.1(0.2)	43.9(0.2) -1.3(0.2)	42.7(0.2) -2.5(0.2)	42.6(0.1) -2.6(0.2)	8.7(0.1) -0.3(0.1)	8.5(0.1) -0.5(0.1)	8.5(0.1) -0.5(0.1)	4.8(0.1) 0.0 [*] (0.1)	4.6(0.1) -0.2(0.1)	4.6(0.1) -0.2(0.1)
Middle	37.9(0.1) -0.4(0.1)	37.4(0.1) -0.9(0.1)	37.3(0.1) -1.0(0.1)	54.5(0.2) -1.5(0.2)	53.4(0.2) -2.7(0.2)	53.1(0.2) -3.0(0.2)	43.2(0.2) -1.6(0.2)	42.0(0.1) -2.8(0.1)	41.9(0.1) -2.9(0.1)	8.4(0.1) -0.3(0.1)	8.3(0.1) -0.4(0.1)	8.2(0.1) -0.5(0.1)	4.6(0.0) [*] -0.2(0.0) [*]	4.5(0.0) [*] -0.3(0.0) [*]	4.5(0.0) [*] -0.3(0.0) [*]

	Hypertension, %			MI Incidence, n			Stroke Incidence, n			MI Mortality, n			Stroke Mortality, n		
	Restaurant	Packaged	Both	Restaurant	Packaged	Both	Restaurant	Packaged	Both	Restaurant	Packaged	Both	Restaurant	Packaged	Both
High	34.6(0.1) -0.5(0.1)	34.0(0.1) -1.1(0.1)	33.9(0.1) -1.2(0.1)	52.8(0.2) -1.4(0.2)	51.6(0.2) -2.6(0.2)	51.3(0.2) -2.9(0.2)	41.8(0.1) -1.5(0.1)	40.5(0.1) -2.8(0.1)	40.4(0.1) -2.9(0.1)	7.7(0.1) -0.3(0.1)	7.7(0.1) -0.3(0.1)	7.5(0.1) -0.5(0.1)	4.3(0.0) -0.1(0.0)	4.2(0.0) -0.2(0.0)	4.2(0.0) -0.2(0.0)

* Values less than 0.05.

Table 3
Sensitivity Analyses: Simulated Model Projections of Hypertension Prevalence and Annual Incidence of Cardiovascular Disease (Myocardial Infarction [MI] and Stroke) and Related Deaths per 10,000 Persons, Mean (SE)

	Hypertension %	Incidence of MI	Incidence of Stroke	Deaths from MI	Deaths from Stroke
Main Simulation					
Baseline	37.08 (0.04)	55.95 (0.11)	44.40 (0.10)	8.52 (0.04)	4.68 (0.03)
Restaurant foods only	36.67 (0.04)	54.42 (0.10)	42.97 (0.10)	8.29 (0.04)	4.58 (0.03)
Packaged foods only	36.11 (0.04)	53.36 (0.10)	41.73 (0.09)	8.16 (0.04)	4.45 (0.03)
Packaged and restaurant foods	35.99 (0.04)	52.95 (0.10)	41.63 (0.09)	8.09 (0.04)	4.45 (0.03)
Sensitivity Analyses[†]					
<i>Salt Sensitivity</i>					
No increased salt sensitivity with advanced age	36.80 (0.04)	55.04 (0.10)	43.92 (0.10)	8.45 (0.04)	4.66 (0.03)
Increased salt sensitivity in Black population, not with age	36.73 (0.04)	54.91 (0.10)	43.79 (0.10)	8.43 (0.04)	4.63 (0.03)
Increased salt sensitivity in Black, hypertensive, and advanced age	35.97 (0.04)	53.73 (0.10)	42.36 (0.10)	8.25 (0.04)	4.52 (0.03)
<i>Table Salt Addition</i>					
1/12 teaspoon (192 mg)	36.33 (0.04)	53.65 (0.10)	42.31 (0.10)	8.19 (0.04)	4.51 (0.03)
1/6 teaspoon (384 mg)	36.67 (0.04)	54.38 (0.10)	43.01 (0.10)	8.29 (0.04)	4.58 (0.03)
1/4 teaspoon (576 mg)	37.03 (0.04)	55.12 (0.10)	43.71 (0.10)	8.39 (0.04)	4.64 (0.03)
1/3 teaspoon (768 mg)	37.39 (0.04)	55.87 (0.11)	44.42 (0.10)	8.49 (0.04)	4.70 (0.03)
<i>Percentage of Consuming Highest Sodium Foods</i>					
5% substitution	36.16 (0.04)	53.31 (0.10)	41.97 (0.10)	8.14 (0.04)	4.48 (0.03)
15% substitution	36.51 (0.04)	54.05 (0.10)	42.63 (0.10)	8.25 (0.04)	4.54 (0.03)
25% substitution	36.90 (0.04)	54.86 (0.10)	43.38 (0.10)	8.36 (0.04)	4.60 (0.03)
35% substitution	37.29 (0.04)	55.78 (0.10)	44.11 (0.10)	8.49 (0.04)	4.67 (0.03)

[†] Sensitivity analyses assumed sodium reduction in both packaged and restaurant foods.