

Supplementary Appendix

This appendix has been provided by the authors to give readers additional information about their work.

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**Appendix to “Reductions in Cardiovascular Disease Projected from Modest Reductions
in Dietary Salt” by Bibbins-Domingo, et al.**

General Overview of the CHD Policy Model

The CHD Policy Model is a computer-simulation, state-transition (Markov cohort) model of CHD incidence, prevalence, mortality, and costs in the U.S. population over age 35 years. In the version of the Model used for this analysis, the U.S. population age 35 to 85 years, without a history of CHD, was apportioned into 32,400 risk cells defined by six modifiable risk factors: systolic blood pressure (SBP) (<130, 130-139.9, ≥140 mmHg), low density lipoprotein (LDL) cholesterol (<2.6, 2.6-3.3, ≥3.4 mmol/L ;<100, 100-129.9, ≥130 mg/dL), high density lipoprotein (HDL) (1.0<,1.0-1.5,≥1.6 mmol/L; <40, 40-59.9, ≥60 mg/dL), smoking status (active smoker, non-smoker with exposure to environmental tobacco smoke, non-smoker without environmental exposure), diabetes mellitus (yes or no), and anti-hypertension medication use (yes or no) as well as by sex and ten-year age range. The population with prevalent CHD was apportioned into 1300 cells according to their age, sex, history of myocardial infarction (MI), arrest, angina, and/or revascularization.

CHD incidence and non-CHD deaths in the population without prior CHD were determined by logistic risk functions based on Framingham longitudinal data.¹ Transitions in the disease history component of the model were based on age-range specific event and case fatality rates estimated from national data, and literature-based relative risks of events among disease history subgroups (e.g., prior MI versus none). Non-CHD death rates in the population with CHD reflected the relative risk of non-CHD death for this population in the Framingham data. In the absence of evidence of a trend, all of these rates were assumed to remain constant. Absolute numbers of events vary with temporal changes in the population, the age-range distribution of

the population, and in response to user-defined interventions. All population distributions, risk factor levels, coefficients, event rates, case fatality rates, costs, and quality of life adjustments can be modified for forecasting simulations.

Population estimates

Population estimates for the adult US population over age 35 and older in 2000 were obtained from the US Census 2000 data by age and sex.² Estimates (2000-2007) and projections (2008-2050) of the 35 year-old population were also obtained from the US Census 2000.² US Census projections for 2010 to 2019 are used in this current study.

CHD prevalence

The background prevalence of CHD in 2000 was estimated from the National Health Interview Survey.³ The background prevalence of prior revascularization procedures was estimated from revascularizations before 2000 and estimated survival after revascularization from the National Hospital Discharge Survey (NHDS)⁴ and other studies.^{5, 6}

CHD deaths

Data on CHD deaths were obtained from the 2000 Vital Statistics Mortality Data.⁷ CHD deaths were estimated using the International Classification of Diseases (ICD) 10 codes I20-I25, I46 and 2/3 of I49, I50, and I51.^{7, 8} Other deaths were considered to be non-CHD deaths.

Arrest (sudden death) with resuscitation

The number who survive arrest to hospital discharge was estimated from NHDS⁴ for 1990-1999. Because the numbers are very small in any given year, we averaged the national estimates over the ten-year period. Estimates of pre-hospital arrest fatalities were based on Vital Statistics mortality data for selected causes by place of death.⁹ For ICD10 codes I20-I25, all emergency room deaths and those

dead on arrival were assumed to be deaths from arrests. All nursing home deaths were considered to be chronic CHD deaths. In-residence and "other place" deaths were estimated to have had resuscitation attempted based on reported resuscitation rates for witnessed¹⁰ or unwitnessed¹¹ arrest.

Proportion of arrests with no history of CHD

The CHD history of arrest patients is harder to ascertain than for MI because there is no national registry, because the numbers are smaller, and because fewer studies are available. We estimated the age range specific proportions of arrest with and without a history of CHD by a least squares fit to data from multiple sources.^{12, 13}

Myocardial infarction (MI)

Myocardial infarction (MI) target incidence was estimated as the average annual number of discharges coded as 410 in the NHDS 2000 data set. Records for MI with a hospital stay of less than 3 days and no acute revascularization in the same hospitalization were eliminated as likely "rule-out MI" cases. Remaining counts were reduced by the double count fraction reported by Westfall,¹⁴ and an additional 3% deduction was applied for miscoding, as reported by Petersen.¹⁵

MI case fatality rates

We used the NHDS 1996-2000 mean MI fatalities per adjusted total MI by age range for the older age-ranges (65-84years) and the National Registry of Myocardial Infarction (NRFMI) in-hospital case fatality rates for the younger age ranges (35-44 years).¹⁶ Studies of young MI patients estimate in-hospital mortality at 1-6% compared with 8-22% in older patients.¹⁷

We estimated in-hospital and 30-day case fatality rates from hospital discharge records from the State of California Office of Statewide Health Planning and Development (OSHPD) for the year 2000.¹⁸ The in-hospital case-fatality rate was based on unique person records (duplicate entries

eliminated by matching social security numbers (SSN)). A small number of records do not have SSNs and were omitted from the analysis. The overall rate ratio of 30-day case fatality rate to in-hospital case fatality rate was 1.28953 (12.07/9.36). This ratio was used to adjust national in-hospital case fatality rates to 30-day mortality rates.

Based on Rieves et.al.,¹⁹ we incorporated a mortality odds ratio of 1.6 for MI patients with prior MI and of 1.17 for patients with prior angina. Subset case fatality rates were calculated to reflect these odds ratios and preserve the overall estimated case fatality rate for MI.

Revascularization rates

The numbers of percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG) procedures were estimated from the NHDS for 2000. The revascularization rate was adjusted to reflect a repeat revascularization rate for PCI and for CABG within the first year. A trend in the ratio of PCI to CABG was estimated for 2000-2004. We assumed that PCI would be included as part of the treatment for MI in the same proportion observed in the NHDS data set for 2000, with emergency CABG complicating 2% of these procedures. We included reductions in mortality and re-MI rates for patients treated with PCI.^{20, 21}

The risk functions for incident CHD and non-CHD death

Incident CHD cases (MI, arrest, or angina) and non-CHD deaths in each risk factor cell for the at-risk US population were determined by risk functions r incorporating age/sex specific parameters α and risk factor specific betas $\{\beta_k, k = 1, 2, 3, \dots, 6\}$, which are constant over the time span of a simulation, and cell specific risk factor means $\{m_k, k = 1, 2, 3, \dots, 6\}$, which are altered by user-defined intervention:

$$r = e^{(\alpha + \sum_{k=1}^6 \beta_k m_k)} / (1 + e^{(\alpha + \sum_{k=1}^6 \beta_k m_k)})$$

Beta coefficients for CHD risk were determined for a 60-year-old, the average age of the first onset of CHD in individuals in examinations 9 to 13, 24, and 25 from the original Framingham cohort and 1-6 from the Framingham offspring cohort, for whom adequate data were available for a time-dependent logistic regression analysis.¹ For LDL cholesterol, the slope of the beta coefficient with age was determined from the interaction between LDL cholesterol and age, after adjusting for age and LDL cholesterol. For the other risk factors, the sex-specific slopes with age were calculated from the weighted average of the White/African American slopes in a large pooled analysis²² of multiple epidemiologic studies that reported beta coefficients for these risk factors. Coefficients for LDL cholesterol were not available for pooling, but coefficients for total cholesterol showed a similar down-sloping relationship with age as was found in Framingham for LDL cholesterol.²² The resulting coefficients were generally similar to those reported in various epidemiologic studies.

Beta coefficients for non-CHD death and for stroke were determined from the same exam sets of the Framingham cohort and offspring data used for the CHD betas, but with SBP, smoking, and diabetes as the only statistically significant covariates in the logistic regression analysis.

We estimated overall incidence of CHD and non-CHD death by age-range and sex for 2000 by adjusting the Framingham incidence estimates for 1986 to take into account the trends in risk factor means from 1986 to 2000. The corresponding values of the intercepts were estimated by iterative fitting of the risk function to the overall incidence. Incidence of arrest without prior CHD was estimated using the proportion of arrest without prior CHD in Olmsted County;¹² incidence of MI was estimated using the proportion of MI without prior CHD in several published studies

analyzing data from the National Registry of Myocardial Infarction 2,²³ Cardiovascular Cooperative Project,²⁴ and the Worcester Heart Attack study.^{16, 25}

All risks and rates were assumed to be constant over time, in the absence of evidence of a trend. Trends, for example in the utilization of revascularization between 2000 and the present, were incorporated as they become apparent, but were not projected into the future. The CHD and non-CHD death risk functions are applied to every state in every year of a simulation, so that the competing risk of these two outcomes is accommodated naturally over time.

Incident CHD event allocation

Risk factors were assumed to affect the incidence of myocardial infarction, arrest, and angina in proportion to overall incidence, except smoking was assumed to have a higher relative risk for infarction and arrest²⁶ and a proportionately lower coefficient for angina. Environmental tobacco exposure was assumed to carry a relative risk of 1.26 for myocardial infarction and cardiac arrest compared with non-exposed non-smokers²⁷ but not to influence angina.

Risk factor prevalence and correlations between risk factors

The prevalence of each risk factor level and correlations among risk factors (and thus the apportionment of the U.S. population without CHD into the 3240 risk cells described above) were estimated from the National Health and Nutrition Examination Survey (NHANES),²⁸ survey years 1999-2006.

Transitions between risk factor levels

Transfers from one risk factor level to another were included to preserve the NHANES proportions of the population with each risk factor level. For example, the proportion of 35-44 year old men with low (<2.6 mmol/L (<100 mg/dL)) LDL cholesterol is 0.215. For 45-54 year old

men the proportion is 0.133. The shift toward higher LDL cholesterol levels is most likely caused by increasing LDL levels as people age. In higher age ranges, this trend reverses, so that by age 75-84, the proportion is 0.314. The change in the upper age ranges is most likely due to a more complex array of factors, including the fact that people with higher risk are more likely to die. Annual transfer rates between risk factor levels were calculated to reduce the low risk population from 0.215 to 0.133 over 10 years, without regard to the reason for the change, but taking into account the effect of the Model's CHD incidence and non-CHD death rates in the base case.

Costs

Total health care costs, from the perspective of the healthcare system, were estimated using national data.²⁹ The CHD cost component was estimated using California data,¹⁸ deflated using cost to charge ratios³⁰ and the ratio of the U.S. national average costs to the California average,³¹ and then inflated to 2006 dollars using the Bureau of Labor Statistics Consumer Price Index for Medical Care Costs.³² Health-related quality-of-life weights were based on observational data.³³ Costs and QALY's were discounted at 3% per year.

Quality control and validation

The CHD Policy Model was calibrated to reproduce national data on risk factor distributions, total CHD deaths, acute MI, witnessed sudden cardiac death, and revascularization procedures in the base year. Validation of projections into the future is an ongoing effort in which the Model's results under a broad range of scenarios were compared with data from studies, clinical trials, and surveys, obtained from public sources or by personal communication. Validation required reasonable agreement in outcomes when the conditions that produced the data were incorporated.

For example, simulations of the US population aged 45-64, imposing the pre- and post- LDL cholesterol and HDL cholesterol levels recorded in the West of Scotland Study (WOSCOPS),³⁴ resulted in similar results for the cumulative percentage of the cohort to have died of CHD or have had a first MI, and for the ratio of events in participants who were and were not treated with statins (Appendix Table 1).

For validation of cost and cost-effectiveness aspects of the model, the model was used to duplicate a cost-effectiveness analysis of secondary prevention based on the 4S study.³⁵ With discounting set to 5%, no quality of life adjustments, and CHD and drug costs only (excludes non-CHD health care costs), the model produced a cost-effectiveness ratio of \$13,100/life year for statins overall in the 35-74 year-old secondary prevention population, as compared with age- and sex-specific analyses from 4S that estimated ratios ranging from \$4,700/QALY (70 year-old men) to \$18,800/QALY (35 year-old women).³⁵

Probabilistic sensitivity analyses of model parameters

Monte Carlo simulations were used to determine the uncertainty around outcomes. Beta coefficients for the associations of systolic blood pressure, LDL and HDL cholesterol, smoking and diabetes with both CHD and non-CHD events were assumed to have a normal probability distribution with standard errors derived from the fitted regressions from Framingham data (described above). We generated covariance matrices for each of these beta coefficients; based on the evidence for minimal correlation among these factors, we assumed these effects to be independent. We also varied annual quality of life measures for the angina, MI and arrest states (based on variability observed in the Beaver Dam study).³³

Salt reduction simulations

For this analysis we used versions of the Model to estimate effects in the entire adult US population and separate models to estimate effects in the black and non-black US sub-populations. Risk function betas for the black and non-black populations were assumed to be the same as in the US population. The proportion of the entire adult US population and the black and non-black sub-populations with each of the six risk factors by age decade and sex is derived from NHANES 1999-2004. Appendix Table 2 lists the proportion using anti-hypertensive medications in NHANES in these years.

We modeled the linear effect of reducing daily salt intake by 0-3 grams/day¹⁴ using a lower estimate for the effect of salt reduction on SBP based on a large meta analysis^{3, 15} and a higher estimate based on clinical trial data.^{16, 17} We modeled an accentuated response to salt reduction among blacks, persons with hypertension, and persons 65 years or older.^{16, 18-21} We assumed proportionately greater response for a given reduction in dietary salt in these groups and linear effects across the range of salt reduction tested. We conducted simulations in the entire US population and among black and non-black subgroups and estimated annual reductions in incident CHD, total MI, incident stroke, and death from any cause as a result of reductions in dietary salt for the entire population and separately by age, sex and race. We conducted sensitivity analyses varying the impact of salt reduction on changes in cardiovascular risk based on estimates suggesting that treating blood pressure through salt reduction or medication use does not lower cardiovascular risk to the same level as native blood pressure.²⁶ We also performed sensitivity analyses assuming no additional salt sensitivity due to race and using race-specific beta coefficients as have been published from an analysis of the Atherosclerosis Risk In Communities (ARIC) study.⁴⁶

In the manuscript we report these results for the 3 gm/day reduction in dietary salt. Here we report the results for the 1 gm/day reduction. Relative reductions in incident CHD, total MI, incident stroke, and death from any cause as a result of a 1 gm/day reduction in dietary salt by age, sex, and race subgroups are presented in the Appendix Figure. Overall reductions in rates per 10,000 in the entire US population and in the black and non-black subpopulation are presented in Appendix Table 3, as are the results of the sensitivity analyses. Rates presented here are age-adjusted to the US population to allow for comparisons of the relative effect across these different populations.

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Appendix Table 1. Cumulative percentage of persons with a first CHD events (MI or CHD death)

Year	WOSCOPS*			CHD Policy Model		
	Placebo	Intervention	Ratio	Baseline	Intervention	Ratio
1	1.7%	1.2%	0.73	1.6%	1.1%	0.67
2	3.2%	2.2%	0.68	3.3%	2.2%	0.67
3	4.9%	3.3%	0.68	5.1%	3.4%	0.67
4	6.5%	4.3%	0.67	7.0%	4.6%	0.66
5	9.2%	6.4%	0.70	8.8%	5.9%	0.67

* With Kaplan-Meier survival adjustment for censored data WOSCOPS- West of Scotland Study³⁴

Appendix Table 2. Prevalence of anti-hypertension medication use estimated from the National Health and Nutrition Examination Survey 1999-2004

Age range, in years	Prevalence of anti-hypertension medication use by race and sex			
	Non-Black Population		Black Population	
	Men	Women	Men	Women
35-44	9.6%	9.2%	12.1%	19.5%
45-54	20.4%	21.2%	29.4%	37.7%
55-64	33.2%	39.6%	46.7%	63.7%
65-74	46.9%	51.3%	61.4%	65.7%
75-84	42.3%	57.9%	57.6%	75.4%

Appendix Table 3. Annual rate differences in cardiovascular rate reductions for 1 gm/day reduction in dietary salt by assumptions of differential salt sensitivity by age and race*

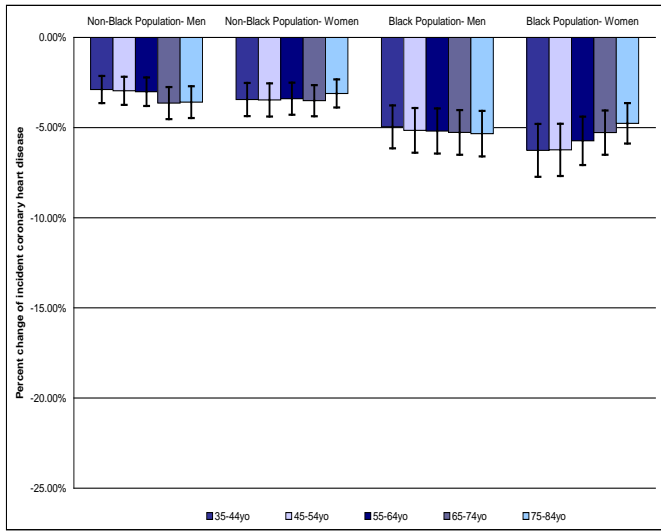
	Change in rate per 10,000 (SD) (percent change)							
	Incident CHD		Total MI		Incident Stroke		All-Cause Mortality	
	Low	High	Low	High	Low	High	Low	High
Main Simulation								
US Population	-1.6 (0.1) -2.1 %	-2.6 (0.2) -3.4%	-1.3 (0.1) -2.7%	-2.1 (0.2) -4.3%	-0.8 (0.1) -1.8%	-1.3 (0.2) -2.8%	-1.1 (0.2) -0.9%	-1.8 (0.3) -1.5%
Non-Black Population	-1.5 (0.1) -2.1%	-2.4 (0.2) -3.4%	-1.2 (0.1) -2.6%	-1.9 (0.2) -4.2%	-0.7 (0.1) -1.7%	-1.2 (0.2) -2.7%	-1.0 (0.1) -0.9%	-1.7 (0.2) -1.5%
US Blacks	-2.7 (0.2) -3.4%	-4.4 (0.4) -5.6%	-2.0 (0.2) -4.1%	-3.3 (0.3) -6.7%	-1.4 (0.2) -2.6%	-2.3 (0.3) -4.3%	-1.8 (0.2) -1.5%	-3.0 (0.4) -2.5%
Sensitivity analysis								
Diminished cardiovascular risk reversal with blood pressure lowering[†]								
US Population	-1.1 (0.1) -1.4%	-1.8 (0.2) -2.3%	-0.9 (0.1) -1.8%	-1.4 (0.1) -2.8%	-0.5 (0.1) -1.2%	-0.9 (0.1) -1.9%	-0.8 (0.1) -0.6%	-1.2 (0.2) -1.0%
No increased salt sensitivity due to age								
US Population	-1.5 (0.1) -2.0%	-2.5 (0.2) -3.3%	-1.2 (0.1) -2.5%	-2.0 (0.2) -4.0%	-0.8 (0.1) -1.6%	-1.2 (0.2) -2.6%	-1.1 (0.2) -0.9%	-1.8 (0.2) -1.4%
No increased salt sensitivity due to race								
US Blacks (no increase by race)	-1.5 (0.2) -1.9%	-2.4 (0.2) -3.0%	-1.1 (0.1) -2.3%	-1.8 (0.2) -3.6%	-1.1 (0.2) -0.9%	-1.7 (0.3) -1.4%	-1.0 (0.1) -1.8%	-1.5 (0.2) -2.8%
Black-specific beta coefficients based on the ARIC Study[‡]								
US Blacks (alternate betas)	-2.4 (1.3) -3.0%	-3.8 (1.9) -4.8%	-1.7 (1.0) -3.4%	-2.7 (1.4) -5.4%	-1.4 (0.3) -2.5%	-2.3 (0.4) -4.2%	-1.7 (0.6) -1.4%	-2.7 (0.9) -2.2%

* Rates are age-adjusted to the US population

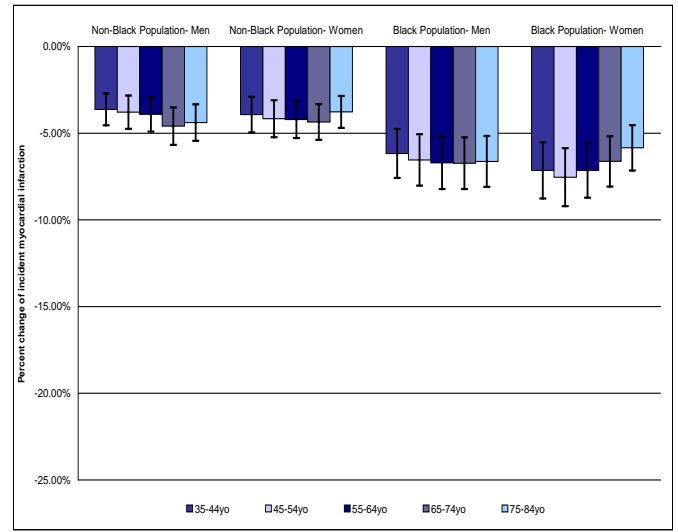
[†] Assumes cardiovascular benefit of 2/3 of the native blood pressure^{44,45}

[‡] Uses beta coefficients for all of the CHD risk factors based on an analysis of the Atherosclerosis Risk in Communities (ARIC) study⁴⁶

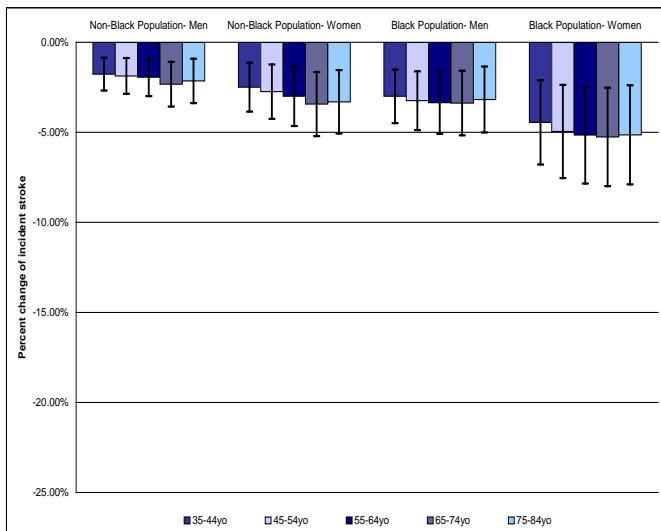
Appendix Figure Legend. Percent change in cardiovascular events with 1 gm/day reduction in dietary salt by US sub-populations. a) incident coronary heart disease, b) new and recurrent myocardial infarctions, c) incident stroke, d) death from any cause.



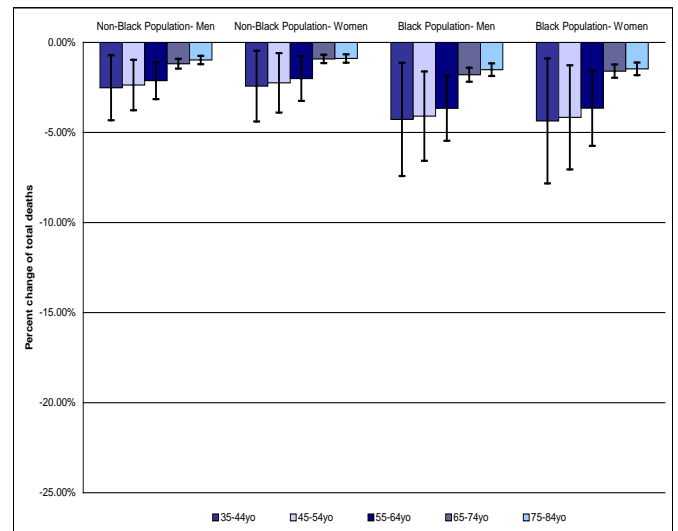
a.



b.



c.



d.