Supplementary Online Content

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

eMethods. Overview of CVD Policy Model Structure and Analytic Approach

The Cardiovascular Disease Policy Model is a state-transition Markov model of cardiovascular disease (CVD) among US adults age 35-94.[1-6] The structure and transitions in the CVD Policy Model are shown in Appendix Figure S1. The base year of the current version is 2010, with inputs for population demographics, clinical characteristics, and cardiovascular disease incidence and prevalence defined by the US census and projections, the National Health Interview Survey, the National Health and Nutrition Examination Survey, the National Hospital Discharge Survey, along with numerous additional sources detailed below.[7-14] The Model is calibrated within 1% of targets for events and deaths for coronary heart disease and stroke as well as deaths from non-cardiovascular causes according to national hospital data and vital records collected in 2010 (Appendix Table 1). [9,10]

The Model separates the population into those without and those with existing CVD, which includes coronary heart disease (angina pectoris, myocardial infarction (MI), or arrest) and stroke (ischemic and hemorrhagic). In annual cycles, new 35 year olds enter, those who die or reach 95 years of age exit the modeled population, and those remaining alive transition among cells defined by age, sex, risk factors, and prior CVD history. Those without pre-existing CVD experience annual probabilities of incident coronary heart disease (CHD), stroke, or death from non-cardiovascular causes with rates dependent on age decile, sex, and values for cardiovascular risk factors. Following incident events, the Model characterizes the event type

and its sequelae for the first 30 days, after which survivors move into the CVD state aligned with initial and subsequent events and procedures occurring during the acute phase. Those with existing CVD experience annual probabilities of additional events and of death from CHD, stroke or non-cardiovascular causes, with the probability of transition from one state to another dependent on age decile, sex, and prior cardiovascular event history (Appendix Figure S1).

For this study, we developed a model representing the low-SES subpopulation in the US and a model representing the higher-SES subpopulation. Low-SES was defined as an income of <150% of the federal poverty level (FTP) and less than a high school education (measured by high school diploma or successful completion of the General Educational Development (GED) exam), while higher-SES was defined as either having an income ≥150% of the FTP or attainment of a high school diploma, GED, or higher level of education. We started from the structure and input values for the national CVD Policy Model and replaced inputs with values specific to each SES subgroup where available (Appendix Table 2).

National CVD Policy Model Technical Details

The present version of the CVD Policy Model includes data from prior versions as well as many updates and upgrades.[1-6] The Model is written in Fortran 95 and compiled using the Lahey Fortran 95 compiler V7.2 (Lahey Computer Systems, Incline Village, Nevada).

Population distribution according to age, sex, cardiovascular risk factors and disease history. The Model divides all adults in the US 35-94 years of age, quantified by the 2010 US Census,[13] into those without prior CVD and those with prior CVD. Age- and sex-stratified CVD prevalence in 2010 is calculated from National Health Interview Surveys from 2009-2011.[8]

The population without CVD is further stratified into cells representing all possible combinations of the following factors: age decile (six categories from 35-44 years to 85-94 years), sex (male or female), and levels of factors associated with CVD risk (up to three categories for each factor). This study includes the following risk factors and categories and, for continuous variables, the mean values associated with each age, gender, and risk category stratum:

- smoking status: active smoker (self-reports current smoking), non-smoker with exposure to environmental tobacco smoke (self-reports no active smoking and >=0.05 μg/mL cotinine), no smoke exposure (self-reports no active smoking and <0.05 μg/mL cotinine); for active smokers, the magnitude of exposure to tobacco smoke is captured as the average number of cigarettes smoked per day assessed by self-reported frequency and volume of smoking over a 30 day period

- systolic blood pressure (SBP): <130 mmHg, 130 to <140 mmHg, and ≥140 mmHg
- low-density lipoprotein cholesterol (LDL-c): <100 mg/dL, 100 to <130 mg/dL, ≥130 mg/dL
- high density lipoprotein cholesterol (HDL-c): <40 mg/dL, 40 to <60 mg/dL, ≥60 mg/dL
- diabetes status: yes (doctor diagnosis or fasting glucose > 125 mg/dL) or no (no doctor diagnosis and fasting glucose ≤ 125 mg/dL)
- body mass index (BMI): <25, 25-<30, ≥30 kg/m^2)

The non-CVD population is distributed into 58,320 cells (60 ages * 2 sex groups* 3^5 (five risk factors with three levels) * 2 (one risk factor with 2 levels)) and each cell is assigned mean values for risk factors according to age, sex, and risk factor stratum. To determine the multivariable distribution of the population according to all risk factor level combinations, we used all continuous NHANES survey years (currently1999-2016) [7] and then used an iterative proportional fitting procedure to match marginal proportions for individual risk factors measured in more contemporary surveys (NHANES 2007-2010 for the 2010 base model).[7]

The population with prior CVD in 2010 is stratified into cells defined by age, sex, and CVD disease state (coronary heart disease, stroke, or both coronary heart disease and stroke) using prevalence values estimated from National Health Interview Survey data from 2009-2011,[8] assuming that the imperfect positive predictive value of survey data is offset by its imperfect sensitivity.[15-17] Age-specific prevalences for individual CVD states were fitted with polynomial or

spline functions of age to obtain smooth, monotonically increasing prevalences. The background prevalence of prior coronary revascularization was calculated from revascularizations before 2010 and estimated survival after revascularization, while model projections were used to infer the distribution of revascularization by CVD state.

Annual transition probabilities

Each annual cycle, the population without prior CVD experiences annual rates of incident CHD (angina, MI, or arrest), incident stroke (ischemic or hemorrhagic), and death from non-cardiovascular causes, as described below. The population remaining free of CVD each annual cycle transitions among cells with rates calculated to preserve age-range trends over time. The Age- and sex-stratified annual incidence of CHD and incidence of stroke is calculated from Framingham Heart Study and Framingham Offspring Study data ranging from 1988-2007,[18,19] and adjusted to reflect risk factor differences between Framingham cohorts and contemporary U.S. data measured in NHANES 2007-2010.[7] The Model incorporates annual rates of mortality from non-CVD causes calculated from 2010 US vital statistics, defined further below.[10] The relationship between annual changes in risk factors and incidence of CHD, stroke, and non-CVD death is determined by a risk function that incorporates age- and sex-specific alphas, risk factor-specific betas (β_k, k=1,2,3,…,6), and cell-specific risk factor means (m_k, k=1,2,3,…,6). The risk function is defined as:

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

Beta values are calculated from Cox Proportional hazards models, censoring at first event (CHD, stroke, or non-CVD death) using Framingham Heart Study examinations 13-28 and Framingham Offspring Study examinations 1-7.[18,19] The Model incorporates indirect effects of changes in BMI on modeled outcomes through changes in SBP, LDL-c, and HDL-c, with coefficients sourced through literature review,[20,21] and through changes in diabetes incidence as calculated from Cox proportional hazard models of Framingham data. [18,19]

Those in the previously CVD free population who experience CHD or stroke events in a given year are transitioned into the "bridge" portion of the model, a 30-day period with heightened probability of procedures, recurrent events, and cause-specific death. The risk of new onset CHD is assumed to be independent of the incidence of stroke in the same year. Those with incident CHD are first portioned into CHD category (angina pectoris, myocardial infarction, or arrest); risk factors are assumed to affect each category in proportion to overall CHD incidence, except for tobacco smokers who are assumed to have a higher relative risk for infarction and arrest ([22]; personal communication, Sean Coady, National Heart, Lung, and Blood Institute, February, 2006) and a proportionately lower coefficient for angina. Environmental tobacco exposure is assumed to carry a relative risk of 1.26 for myocardial infarction and cardiac arrest compared with non-exposed non-smokers[23] but not to influence angina.

CHD and stroke events and deaths

The number of patients with hospitalized myocardial infarctions was obtained from discharges coded as ICD-9 code 410 in the 2010 National Hospital Discharge Survey (NHDS)[9] adjusted for likely miscoding,[24] and excluding patients who were discharged alive after two days or fewer without a percutaneous coronary intervention and transfer patients. Case-fatality rates and rates of myocardial infarction in age/sex subgroups were estimated from national data[9] with subpopulation (e.g. prior MI versus not) relative rates derived from complementary sources.[25-27] Patients with pre-hospital arrest deaths were estimated from the U.S. Vital Statistics,[10] and patients with out-of-hospital cardiac arrests surviving to hospital discharge were estimated from national data.[9] Survival after a coronary heart disease events was estimated from national hospital data on status at discharge with rates reduced to reflect 30 day mortality using California data on the ratio of in-hospital survival to 30 day survival[28] and data from Medicare and from Emergency Medical Services in King County, Washington.[29,30] Rates of coronary revascularizations were estimated from the National Hospital Discharge Survey,[9] with mortality estimated from aggregated historical data.

The number of hospitalized strokes was also obtained from the 2010 NHDS.[9] We applied positive predictive values of specific ICD-9 stroke hospital diagnosis codes (inclusive of ICD 9 codes 430-438) according to methods described in Williams et al (1999),[31] which involved pooling published data from four cohort studies of stroke incidence that compared hospital diagnoses with a gold standard.[32-35] The positive predictive values were applied to age- and sex-

specific NHDS cases in order to estimate total stroke event rates (inclusive of first-ever and recurrent stroke events). Applying 30-day case fatality rates based on the Atherosclerosis in Communities Study[35,36] yielded annual mortality rate estimates within the range of stroke rates reported by the U.S. Centers for Disease Control (CDC Wonder) for 2010.[10] Incidence calibration assumed that 77% of all strokes are incident (first ever),[37] but it was assumed that the proportion first ever/total diminished with age (i.e., >90% of all strokes are first strokes in 35-44 year olds and 50% are first strokes in 85-94 year olds). The resulting incidence of hospitalized stroke approximated age- and sex- specific stroke incidence rates observed in U.S. stroke cohort and surveillance studies. The annual probabilities of CHD in stroke patients and stroke following myocardial infarction were based on natural history studies.[38-44]

Deaths from coronary heart disease and stroke in 2010 were extracted from U.S. Vital Statistics.[10] Deaths were categorized according to the International Classification of Diseases (ICD) 10 codes:[45] I20-I25 and two-thirds of I49, I50, and I51 were used to estimate coronary heart disease deaths,[46] I60-I69 were used to estimate stroke deaths, and all other deaths were considered non-CVD deaths.

Detailed Inputs and Analytic Approach for Current Study

National estimates of CHD death rate for low- and higher-SES adults

We used data from US Vital Statistics to evaluate national evidence for a differential rate of CHD mortality according to SES.[11] Because income data are not collected on death certificates, we used educational attainment as a proxy to stratify deaths by low-SES (no high school diploma or GED) and higher-SES (either a high school diploma or GED). We classified deaths with ICD-10 codes I20 through I25 and two-thirds of I49 through I51 as CHD deaths.[45,46] We restricted our sample to the 48 states that used the 2003 version of the US death certificate in 2015,[47] on which measurement of education by degree attainment aligns with the American Community Survey[48] used to define population totals for rate denominators. We excluded from the count of CHD deaths (numerator) and population (denominator) data for Alabama and West Virginia, states that used the 1989 version of the death certificate in 2015,[49] due to quality concerns relating to measurement of education.[50]

CVD Policy Models for the Low- and Higher-SES populations

Appendix Table 2 shows key inputs for the present analysis, including inputs used to adjust the national CVD Policy Model in order to represent CHD in the low- and higher-SES populations in the US. We gathered inputs from nationally representative databases where measures of income and education were available for stratifying data by our study definition of low- and higher-SES, and otherwise conducted reviews of the literature to inform inputs.

We calculated the size of the low- and higher-SES populations using the 2015 American Community Survey.[48] The National Health and Nutrition Examination Survey (NHANES) waves 1999-2016 were used to calculate the multivariate distribution of CHD risk factors for each SES group.[7] We then used NHANES waves 2011-2016 to define the contemporary subgroup-specific risk factor means and proportions for SBP, HDL-c, and BMI and waves 2011-2014 to define distributions for smoking, LDL-c, and diabetes, representing most contemporary data available at the time of analysis.[7] Annual transitions among risk factor levels in the non-CVD population were calculated separately for low- and higher-SES models, with rates computed to maintain age- and gender-specific risk factor distributions over time in each group.

Annual rates of incident CHD, incident stroke, and non-CVD death in those without prior CVD along with rates of recurrent events and deaths in those with CVD history were adjusted from the national CVD Policy Model rates to account for differences in CVD risk factor distributions between the US population and the low-SES and higher-SES populations.[7] These adjustments resulted in overall lower rates of CVD events and deaths for the higher-SES population model and higher rates for the lower-SES population model relative to national model inputs.

We then further adjusted the incidence of CHD to reflect the elevated risk of low-SES compared to higher-SES that is independent of traditional CHD risk factors, a finding observed in several observational cohorts identified through literature review.[51-55] We assumed a relative risk of

1.58 (95% CI: 1.31, 1.9) for the effect of low- compared to higher-SES (defined identically to the present analysis) computed in the Atherosclerotic Risk in Communities (ARIC) Study cohort;[51] and calibrated CHD incidence to match overall US incidence in each age- and gender- stratum while also matching the assumed independent relative risk between the two SES strata. A similar relative risk for the independent effect of SES on incident CHD was observed in the REasons for Geographic And Racial Differences in Stroke study, which we derived to equal 1.63 for low- compared to higher-SES adults after inverse variance weighting four published income/education strata for those under age 65 reported by Lewis and colleagues.[53]

We further incorporated evidence of an elevated rate of death following MI in low- versus higher-SES, also independent of traditional CHD risk factors,[52,56,57] using an estimate for 28-day case fatality reported from analysis of ARIC data.[52] Foraker and colleagues (2013)[52] reported odds ratios for fatality following MI for 6 strata: 3 levels of SES (low, medium, and high income) and 2 levels of race (black and white), with high income whites used as the referent. We generated relative risk inputs that aligned with our dichotomous SES variable by first converting published odds ratios into relative risks assuming the risk of MI case fatality in the higher-SES group of 5%.[9] After weighting stratum-specific estimates by the income/race distribution of the US population,[58] we combined relative risks from the six strata to generate one estimate representing our low-SES definition (combining estimates for low income blacks and low income whites) and one representing our higher-SES definition (combining estimates for middle income blacks, high income blacks, middle income whites, and high income whites). Ultimately,

the MI case fatality rate was adjusted up for our low SES model (relative to the national model) using a relative risk of 1.31 (95% CI: 0.98, 1.66) and adjusted down for our higher-SES model using a relative risk of 0.83 (95% CI: 0.64, 0.99), equating to a low- verses higher-SES relative risk of 1.58 (95% CI: 0.97, 2.60). We generated confidence intervals for these relative risk inputs using simulations written in R that varied strata-specific odds ratios (based on published 95% confidence intervals)[52] and the underlying rate of MI case fatality (based on distributions calculated from national data).[9,52]

Model Simulations

We used dynamic population analyses to simulate contemporary rates of MI and CHD death in low- and higher-SES adults 35-64 years of age, and to parse events in the low SES population into attributable factors. For dynamic population simulations, we included all adults who were 35-64 anytime from the start of 2015 to the end of 2024, incorporating new 35 year olds (projected from the Census)[12] and removing those who either died (based on model rates described above) or reached the age of 65 each annual cycle. We used closed cohort simulations to predict the cumulative incidence of CHD (including angina, MI or arrest) in low SES adults from the age of 35 to 65, including only those who were 35 years old in 2015 and following them for incident CHD through the end of the year 2044. CHD risk factor distributions remained in steady state over time for each age- and gender-stratum (though distributions were specific for the low- and higher-SES populations), due to the annual risk factor transitions calculated as described above. As such, we assumed no temporal risk factor trends over the

course of simulation years. We also held constant over time the elevated rate of incident CHD and MI case fatality independent of traditional CHD risk factors assumed for low- compared to higher-SES adults as described above.[51,52]

We designed simulations to compare the projected impact of fully addressing the elevated, independent CHD risk associated with low- compared to higher-SES (i.e. closing the gap on the independent effect) to simulations of interventions designed to improve traditional modifiable CHD risk factors (i.e., smoking, SBP, LDL-c, and diabetes) to ideal levels. We evaluated risk factors one at a time, first measuring the projected effect of improving sex- and age-stratified risk factor measurements in low SES adults to levels observed for higher-SES adults in 2011-2016 NHANES data[7] and then further improving risk factors to ideal levels for those whose NHANES measurements were out of range. To improve risk factors to ideal levels, we simulated achieving SBP of 110 mmHg in all adults with values > 110 mmHg,[59] LDL-c to 70 mg/dL for diabetics and those with a history of CVD and to 100 mg/dL for all others with values exceeding these targets,[60] removing exposure to cigarette smoke for anyone with active or passive exposure, and resetting the prevalence of diabetes to zero.

Sensitivity analyses

We used Monte Carlo simulations to evaluate the sensitivity of our results to the uncertainty around several key study inputs considered simultaneously (Appendix Table 2). We conducted 1000 iterations of each Model simulation, with each iteration selecting new values for the

following inputs based on their probability distributions. We varied mean values and proportions of CHD risk factors measured for low- and higher-SES models including smoke exposure, SBP, LDL-c, HDL-c, diabetes, and BMI, assuming a normal distribution and standard errors calculated from NHANES waves 2011-2016.[7] We varied beta values from the Model's risk function defining the relationship between one-unit changes smoke exposure, SBP, LDL-c, HDL-c, and diabetes and the incidence of CHD, assuming a normal distribution and standard errors computed from our analysis of Framingham Heart Study exams 13-28 and Offspring Cohort exams 1-7.[18,19] The increased independent risk of CHD in low- compared to higher-SES adults was varied by selecting values from the published 95% confidence interval and adjusting CHD risk values in the low- and higher-SES models to achieve the selected relative risk,[51] assuming a normal distribution for the log relative risk. We similarly assumed normality for the relative risk of fatality following MI in the low- and higher-SES population models, with case fatality inputs varied for each iteration based on relative risks selected from the 95% confidence intervals we derived from published values, as described in detail above.[52] The Monte Carlo program, written in Python, stores results for each of the 1000 iterations, from which 95% uncertainty intervals are calculated finding the lower 2.5% and the upper 97.5% bounds for each outcome using Microsoft Excel 2010.

eTable 1. Comparisons of Selected Cardiovascular Disease Policy Model Simulation Outputs for 2010 (Model Base Year) With National Targets for 2010

Age and sex category	Total myocardial infarctions Target source: NHDS		Total strokes Target source: NHDS		CHD deaths Target source: national vital statistics		Stroke deaths Target source: national vital statistics		All-cause deaths Target source: national vital statistics	
	Target	**Model**	**Target**	**Model**	**Target**	**Model**	**Target**	**Model**	**Target**	**Model**
Males										
35-44	13,979	13,839	16,535	16,553	4,783	4,862	1,027	1,031	43,345	43,335
45-54	56,129	55,811	43,493	43,710	19,489	19,594	3,298	3,301	111,981	111,933
55-64	77,992	77,395	67,863	68,497	38,032	38,065	6,159	6,133	190,845	190,629
65-74	75,804	75,689	79,450	79,239	45,700	46,096	9,350	9,265	231,327	231,231
75-84	62,982	63,063	76,205	76,436	64,610	65,097	16,215	16,240	312,778	312,873
85-94	37,568	37,483	38,943	39,247	64,071	63,958	15,318	14,742	264,705	263,235
Females										
35-44	6,259	6,144	6,390	6,387	1,710	1,822	873	875	26,538	26,619
45-54	17,071	17,035	36,952	37,083	6,858	6,969	2,609	2,764	71,145	71,352
55-64	40,246	40,403	42,966	43,222	15,122	15,265	4,622	4,605	122,502	122,546
65-74	43,843	43,898	69,473	69,659	24,964	25,137	8,504	8,308	178,530	178,342
75-84	60,097	60,043	93,040	93,434	53,247	53,600	21,492	21,541	313,803	313,894
85-94	57,661	57,403	77,481	77,883	99,680	98,988	35,416	36,233	448,864	447,244
Deviation from target	-0.26%		0.39%		0.27%		0.12%		-0.14%	

Abbreviations: CHD = coronary heart disease; NHDS = National Hospital Discharge Survey

eTable 2. Key Input Values for the Low- and Higher-SES CVD Policy Models, With Measures of Variability Indicated for Inputs That Were Varied in Monte Carlo Simulations*

Input Parameter	Men		Women		Source
	Low SES	Higher SES	Low SES	Higher SES	
Population size in 2015					
Age 35-44	5,314,757	15,078,422	5,711,236	14,895,399	2015 American Community Survey[48]
Age 45-54	5,171,440	16,098,546	5,203,791	16,669,624	
Age 55-64	4,707,787	14,980,341	5,058,896	16,101,156	
Age 35 only	617,169	1,652,977	666,762	1,561,003	
	Men		**Women**		
Mean values(SE) for traditional CHD risk factors in adults without prior cardiovascular disease*	**Low SES**	**Higher SES**	**Low SES**	**Higher SES**	
Active smoking prevalence*					National Health and Nutrition Examination Survey waves 2011-2016 for SBP, HDL-c, and BMI and waves 2011-2014 for smoking, LDL-c and diabetes[7]
Ages 35-44	0.36 (0.03)	0.18 (0.02)	0.26 (0.02)	0.15 (0.02)	
Ages 45-54	0.38 (0.03)	0.15 (0.02)	0.32 (0.04)	0.18 (0.03)	
Ages 55-64	0.34 (0.04)	0.18 (0.02)	0.21 (0.04)	0.11 (0.02)	
SBP (mmHg)*					
Ages 35-44	122.9 (0.7)	120.9 (0.7)	115.3 (0.6)	114.1 (0.5)	
Ages 45-54	125.1 (0.8)	123.1 (0.7)	123.3 (0.7)	119.4 (0.8)	
Ages 55-64	128.8 (1.8)	128.6 (0.9)	128.5 (0.9)	124.1 (0.8)	
LDL-c (mg/dL)*					
Ages 35-44	123.5 (2.7)	124.5 (2.3)	112.4 (3.0)	114.5 (3.1)	
Ages 45-54	124.7 (5.1)	117.8 (3.3)	118.9 (3.7)	126.0 (3.4)	
Ages 55-64	112.7 (4.6)	115.0 (3.2)	125.5 (4.6)	127.8 (2.4)	
HDL-c (mg/dL)*					
Ages 35-44	46.5 (0.7)	46.4 (0.6)	54.5 (0.8)	59.7 (0.8)	
Ages 45-54	47.8 (0.8)	47.5 (0.8)	55.2 (1.0)	61.3 (0.9)	
Ages 55-64	48.4 (1.2)	50.1 (0.9)	56.7 (0.9)	64.2 (1.3)	
Diabetes prevalence*					
Ages 35-44	0.08 (0.02)	0.03 (0.01)	0.10 (0.02)	0.07 (0.02)	
Ages 45-54	0.21 (0.04)	0.16 (0.04)	0.22 (0.04)	0.06 (0.02)	
Ages 55-64	0.19 (0.03)	0.13 (0.03)	0.25 (0.04)	0.16 (0.04)	
BMI (kg/m^2)*					
Ages 35-44	29.6 (0.3)	29.5 (0.3)	30.5 (0.4)	28.7 (0.3)	
Ages 45-54	28.6 (0.3)	29.5 (0.3)	30.9 (0.5)	29.6 (0.4)	
Ages 55-64	28.5 (0.4)	29.3 (0.4)	30.6 (0.5)	30.2 (0.4)	
Beta (SE) values for risk function relating risk factors to CHD*	**Men**		**Women**		
Smoking 1 additional cigarette/day, β (SE)* †					Cox proportional hazards models using
Ages 35-44	0.008 (0.001)		0.015 (0.001)		

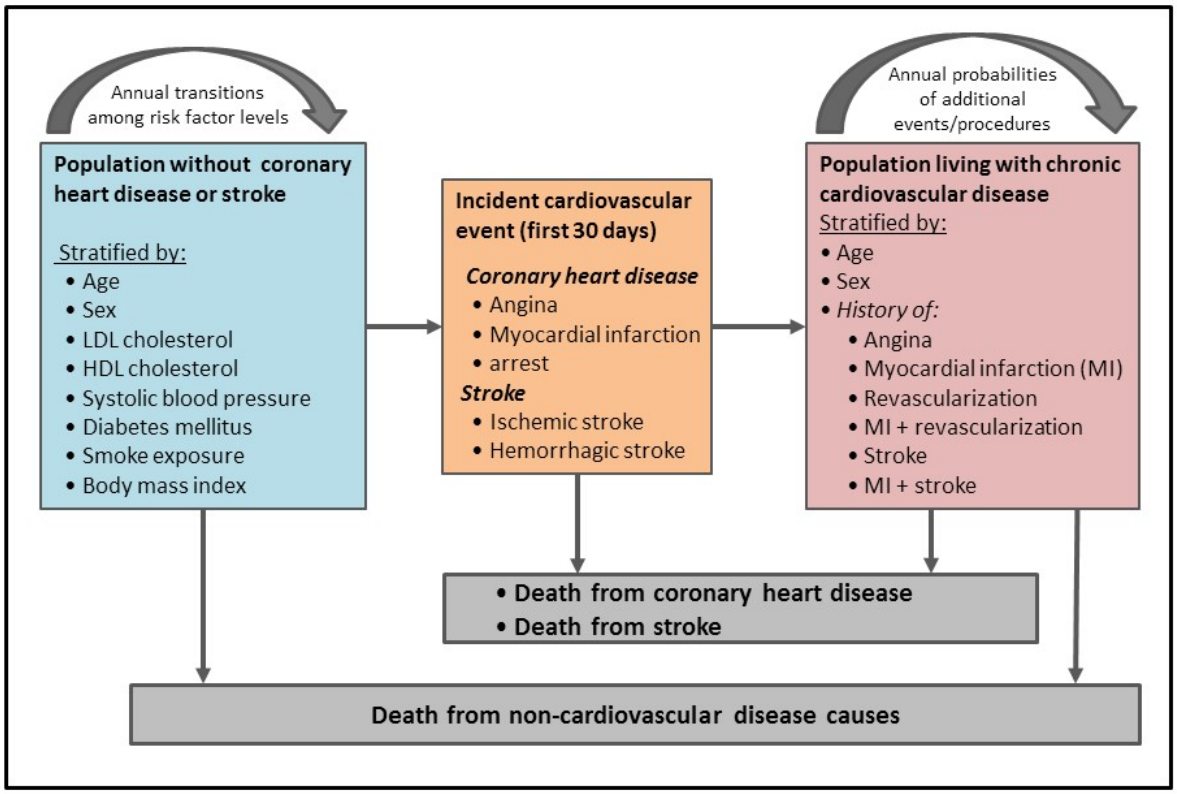
Ages 45-54	0.017 (0.002)		0.020 (0.002)		data from Framingham Heart Study exams 13-28 and Offspring Cohort exams 1-7[18,19]
Ages 55-64	0.016 (0.002)		0.029 (0.002)		
Effect of 1 mmHg SBP, β (SE)*					
Ages 35-44	0.021 (0.003)				
Ages 45-54	0.017 (0.002)				
Ages 55-64	0.013 (0.002)				
Effect of 1 mg/dL LDL, β (SE)*					
Ages 35-44	0.013 (0.001)				
Ages 45-54	0.010 (0.001)				
Ages 55-64	0.008 (0.001)				
Effect if 1 mg/dL HDL-c, β (SE)*					
Ages 35-44	-0.042 (0.004)				
Ages 45-54	-0.036 (0.003)				
Ages 55-64	-0.030 (0.002)				
Effect of those with compared to without diabetes, β (SE)*					
Ages 35-64	0.606 (0.075)				
Effect of 1 kg/m^2 increase in BMI	**Men**		**Women**		
SBP change (mmHg)	1.43 mmHg		1.24 mm Hg		Wilsgaard 2000[21]
LDL-c change (mg/dL)	2.75 mg/dL		2.24 mg/dL		Wilsgaard 2004[20]
HDL-c change (mg/dL)	-1.55 mg/dL		-0.77 mg/dL		
Diabetes risk					Cox proportional hazards analysis of Framingham data as above[18,19]
Ages 35-44	0.22				
Ages 45-54	0.17				
Ages 55-64	0.16				
Relative risk (95% CI) of CHD and mortality following MI associated with SES status	**Men**		**Women**		
	Low SES	**Higher SES**	**Low SES**	**Higher SES**	
Incident CHD, RR (95% CI)*	1.58 (1.31, 1.90)				Fischella 2009[51]
28-day fatality following MI, RR (95% CI)*	1.31 (0.98, 1.66)	0.83 (0.64, 0.99)	1.31 (0.98, 1.66)	0.83 (0.64, 0.99)	Relative risks and 95% CIs simulated based on Foraker 2013[52]

Abbreviations: CHD = coronary heart disease; CI = confidence interval; HDL-c = high-density lipoprotein cholesterol; LDL-c = low-density lipoprotein cholesterol; MI = myocardial infarction; SE = standard error; SES = socioeconomic status; SBP = systolic blood pressure

* Key inputs were varied probabilistically using Monte Carlo simulations, where we ran 1000 iterations each choosing varied values for each designated input from its probability distribution defined by the mean (SE) for CHD risk factors, beta (SE) for beta coefficients, and RR (95% CI) for the independent effect of low- compared to higher-SES for incident CHD and MI case fatality.

† The beta values relating cigarettes smoked per day to incident CHD result from Cox proportional hazards model analyses of Framingham data and subsequent calibration along with CHD event calibration to reproduce the relative risk of MI and CHD death for smokers.[22]

eFigure. Schematic of the Cardiovascular Disease Policy Model



Note that this study modeled separately dynamic cohorts of the US population representing the risk factor distributions and independent risk of CHD associated with those of low and of higher socioeconomic status, as defined through analysis of national surveys and literature review.

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