SUPPLEMENTAL MATERIAL

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METHODS AND SUPPLEMENTAL RESULTS SPECIFIC TO PRESENT ANALYSIS

Model structure

The Cardiovascular Disease (CVD) Policy Model is an established simulation model of coronary heart disease and stroke incidence, prevalence, mortality and costs in U.S. adults 35-94 years of age.[1-5] This analysis uses a CVD Policy Model that was adapted to represent cardiovascular disease in non-Hispanic Black US men (henceforth referred to as "Black men"), updated from previously published model.[6,7] In annual cycles, the model predicts incidence of coronary heart disease, stroke, and death from non-cardiovascular causes in the population without CVD as a function of age, sex, and conventional CVD risk factors (systolic blood pressure [BP]), smoking status, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, diabetes mellitus, body mass index, and anti-hypertensive medication use) (Supplemental Figure S1). In those who develop CVD, the model characterizes the initial event (myocardial infarction (MI), cardiac arrest, angina, or stroke) and its sequelae, including cardiovascular death, for 30 days. In the population with a history of CVD, the model predicts subsequent cardiovascular events, coronary revascularization procedures, and cardiovascular or non-cardiovascular mortality as a function of age, sex, and clinical history. Key model inputs for this analysis are summarized in Supplemental Table S1.

Core model inputs and calibration

The model platform includes the entire population of Black men in the US age 35-94 quantified using the US census and follows them until they die or turn 95 years of age, whichever comes first. [8,9] Cardiovascular disease risk factor distributions for Black men were estimated using population-weighted analysis National Health and Nutrition Examination Surveys (NHANES) from 2009-2016.[10] The relationship between unit changes in risk factors and incident coronary heart disease (CHD), stroke, and non-CVD death are determined by risk functions calculated from data pooled and harmonized from several National Heart Lung and Blood Institute (NHLBI) observational cohorts using competing risk Cox proportional hazard models.[11-16] Rates for prevalent and incident CVD, recurrent events, revascularization procedures, case fatality, and total mortality are estimated from national health surveys, hospitalization databases, vital statistics, observational cohort data, and published literature (Supplement Table S2).[10,14,17-39]

The model uses an iterative procedure based on the Newton-Raphson method to fit risk functions to incidence rate inputs.[40] Event and case fatality rates are adjusted until the model produces outcomes that come within less than 1% of annual hospitalized strokes and MIs and deaths from stroke, coronary heart disease, and all causes reported for Black men in the National Inpatient Sample (NIS) years 2012-2015 and vital statistics data from 2010-2015 (Supplemental Table S3 shows calibration results).[30,36]

In annual cycles, the model counts CHD and stroke events along with CVD and non-CVD deaths and assigns utilities and costs to each clinical event and health state (Supplemental Table S1). Cost inputs encompass acute and chronic care expenditures associated with healthcare utilization for CVD and non-CVD related causes and are calculated using survey-weighted procedures on datasets from the Medical Expenditure Panel Survey (MEPS) years 2006-2015 and the NIS years 2012-2015, along with complementary sources, and costs are indexed to 2019 US dollars using the Personal Consumption Expenditure Inflation Index from the Bureau of Economic Analysis.[30,41-43] Quality-of-life weights and acute tolls for cardiovascular events are estimated from the Global Burden of Disease 2010 study.[44-46]

The CVD Policy Model is programmed in Lahey Fortran 95. Monte Carlo simulations are programmed in Python. We analyzed outcomes using Python and Excel 2016 (Microsoft); and we performed statistical analyses using SAS version 9.4 (SAS Institute Inc) and R version 3.4 (R Foundation for Statistical Computing). Additional technical details about the CVD Policy Model are located on Supplement pages 14-22.

Target population for simulated interventions

Simulated interventions target Black men with characteristics matching eligibility criteria described in the Los Angeles Barber Blood Pressure Study (LABBPS).[47,48] We restricted treatment to Black men living in the US who were 35 to 79 years old in 2019 and allowed new 35-year-olds to enter the target population each year using projections from the Census

Bureau.[8,9] We treated everyone remaining alive through the end of 2028 and retained in the treatment population men who aged beyond 79 years during the 10-year simulations. We targeted men with a mean systolic BP of 140 mmHg or higher, estimated from NHANES years 2009-2016 (Supplemental Table S4).[10] We assumed that Barbershop-based pharmacist-led programs could reach 34% of all Black men with hypertension as follows. We assumed that the programs would only be offered in metropolitan areas (based on the available evidence of efficacy), where 85% of all Black men live (based on the 2016 American Community Survey).[49] We further reduced the simulated target population to include only regular patrons at specific barbershops (50%, based on data from a barbershop-based trial conducted in the Dallas, Texas area)[7] and, in the base-case, assumed that 80% of individuals offered the program would agree to participate (Supplemental Table S5). Note that varying the proportion of individuals enrolled would not be expected to alter the "value" of the program per enrolled participant but would affect the total population health impact of the program.

Intervention strategies

Key study-specific input parameters are shown in Supplemental Table S1. Individuals enrolled in the intervention arm of the LABPPS experienced a 20.8 mmHg (95% CI: 13.9 – 27.7 mmHg) reduction in systolic BP relative to the individuals in the control arm at 12 months.[47,48] We assumed that scaled-up barbershop programs may have variable effectiveness compared with the LABPPS, and therefore estimated the clinical and economic impact of pharmacist-led

hypertension management programs for a range of effectiveness levels (mean systolic BP reductions of 10, 15, 20, and 25 mm Hg).

In the control arm, the population experiences changes in systolic BP over time reflecting the demographic shifts that would be expected as the target population ages.[8-10] In the intervention arm, the population has a 10-25 mm Hg decline in systolic BP during the period of enrollment in the program.

Each 10-mm Hg reduction in systolic BP is assumed to reduce the risk of coronary heart disease-related major adverse cardiovascular events (relative risk 0.83 [95% CI: 0.78 - 0.88]) and stroke (relative risk 0.73 [95% CI: 0.68 – 0.77]), as observed in a meta-analysis of blood pressure treatment trials.[50] Note that although the above meta-analysis defined coronary heart disease as fatal and non-fatal myocardial infarction and sudden cardiac death (excluding silent myocardial infarction), we applied the observed hazard ratio to all coronary heart disease endpoints in the model (myocardial infarction, arrest, and stable and unstable angina) for consistency. This assumption is supported by our analysis of pooled NHLBI epidemiologic cohorts suggesting that the relationship between systolic BP and coronary heart disease events is unchanged with inclusion or exclusion of angina (Dr Yiyi Zhang, personal communication).

The effect of systolic BP changes on the risk of non-CVD death, assumed to occur because of the effects of BP reduction on heart failure outcomes, were calculated from NHLBI pooled cohort data (further detailed on Supplement page 15).[11-16]

Intervention costs and side effects

For this analysis, the model assumed the healthcare sector perspective. Thus, it included all direct healthcare-related costs, regardless of who paid for them.

Treatment costs included clinical encounters with pharmacists, prescribed antihypertensive medications, and healthcare costs resulting from serious adverse events (SAEs) during the course of anti-hypertensive treatment. For pharmacist costs, we estimated the number of hours pharmacists spent in clinical encounters (in person or by phone) using data collected during the LABBPS study.[51] We then multiplied average hours of time spent per participant per year by the mean national salary for licensed pharmacists in the US to estimate the annual cost of pharmacist time for clinical encounters.[52] Our inputs reflect that pharmacists spent more time with participants in year 1 compared to years 2 and beyond (Supplemental Table S1). To estimate the incremental cost of prescribed medications, we applied the cost of generic medications ($4 per prescription per month as commonly available at large retailers) to the average number of additional antihypertensive prescriptions used in the intervention arm of the LABBPS compared with the control arm.[53]

For SAE rates, we pooled data from a meta-analysis of 21 anti-hypertension treatment trials and the Systolic Blood Pressure Intervention Trial (SPRINT) to estimate the probability of experiencing an SAE when taking >2 full standard doses of antihypertensive medications (1.31%; 95% CI: 1.08%, 1.66%) compared with taking ≤2 full standard doses of hypertensive

medications (0.85%; 95% CI: 0.64%, 1.04%).[39,54] We then applied these rates to the arms of the LABBPS to derive the incremental rate of SAEs associated with program enrollment (further described on Supplement page 24). We assumed that the rate of adverse events would vary with the magnitude of BP reduction and lowered SAE rates for scenarios modeling lower systolic BP effects than observed in the LABBPS using the approach described previously.[55,56]

We assumed each SAE resulted in one hospitalization and one 40-minute outpatient follow-up visit, with costs estimated from Healthcare Cost and Utilization Project (H-CUP) and Medicare Services Physicians Fee Schedule respectively. [30,57] We estimated the probability that a patient would die following a SAE ($p = 0.017$) from discharge status data in the 2014 NIS online query system available from the Healthcare Cost and Utilization Project.[30] For those surviving a SAE, we assumed a quality-of-life decrement of 0.1 lasting 30 days for participants with acute kidney injury and a decrement of 0.1 lasting 14 days for those experiencing hypertension, syncope, bradycardia, or electrolyte abnormalities based, with frequencies of each type sourced from the SPRINT trial.[54]

Simulations do not include costs or quality-of-life penalties for less severe adverse events (e.g., non-life-threatening side effects from specific medication formulations) under the assumption that such events would promptly be resolved in consultation with the program's pharmacist with, for example, a change in medication.

Main outcomes and measures

We projected the population health impact of scaled-up barbershop-based pharmacist-led BP control programs on health outcomes, defined as the number of Major Adverse Cardiovascular Events (MACE, a composite of cardiovascular death, non-fatal coronary heart disease, and non-fatal stroke) averted and number of quality-adjusted life years (QALYs) gained. We computed annualized outcomes from 10-year projections. Given that real-world implementation of barbershop-based pharmacist-led BP control programs may be tailored to each location, we estimated the cost per patient-year of enrollment at which the program would be cost-effective, assuming cost-effectiveness thresholds of $50,000 per QALY gained, $100,000 per QALY gained, and $150,000 per QALY gained. In order to do so, we projected incremental healthcare costs (associated with pharmacist clinical time, increased use of BP medications, and resulting adverse drug events, and cost-savings from averted cardiovascular events). These were estimated over a 10-year horizon and then annualized to estimate average annual costs and benefits in a dynamic cohort (as new 35-year-olds can enter the program over time, as can individuals developing hypertension in later years of program implementation).

Sensitivity analyses

Probabilistic sensitivity analyses simultaneously varied multiple input parameters across pre-specified statistical distributions in 1000 iterations (Supplemental Table S1 shows the range and type of distribution used for each parameter). The results of these simulations captured the

uncertainty in key outcomes presented as 95% uncertainty intervals (UIs). As is recommended, we used beta distributions for probabilities and quality-of-life estimates (as these are bounded by 0 and 1), and log-normal distributions for costs.[58]

In one-way sensitivity analyses, we varied key input parameters one at a time while holding others constant at their base-case estimates. We used lower and upper bounds from 95% confidence intervals for the effect of changes in systolic BP on coronary heart disease and on stroke event rates and for costs of hospitalized MIs and strokes (Supplemental Table S1).[30,50] We tested the sensitivity of results to the base case assumption of generic medication pricing by assuming instead costs of anti-hypertensive medications calculated from MEPS data ($344 per patient per year; estimation described on Supplement page 23).[10,43] While our base case assumes no decrease in quality of life associated with taking new prescription medications every day, we tested the effect of a pill disutility of 0.001 in sensitivity analyses.[59]

Supplemental results

Supplemental Table S6 shows the average annual number of Black men treated for each systolic BP effectiveness scenario and the control arm over the simulation years 2019-2028. The average number of people treated annually varies from year to year as new 35-year-olds enroll in the program over time and the proportion of the population developing hypertension in later years grows as a result of population aging. Additionally, the average number of enrolled individuals varies by program effectiveness, with larger BP reductions leading to fewer

individuals dying of CHD and stroke and, in turn, higher numbers of patients treated over 10 year simulations.

Supplemental Figure S2 shows results for the one-way sensitivity analyses for the scenario assuming a 15 mm Hg reduction in systolic BP, evaluating willingness-to-pay outcomes for the cost-effectiveness threshold of $100,000 per QALY gained.

Study oversight and data sharing

The authors of the manuscript attest to the completeness and accuracy of the data and analysis. The institutional Review Board at the University of California, San Francisco approved research undertaken with the CVD Policy Model.

The Cardiovascular Disease Policy Model is available to interested reasearchers who submit a 1- to 2-page research proposal and collaboration plan to Dr. Bibbins-Domingo (email, Kirsten.bibbins-domingo@ucsf.edu) and sign the Creative Commons agreement available at http://tiny.ucsf.edu/CVDpolicymodel, pending approval by the model team. Data for this study come from sources detailed throughout this document. Data from health surveys, vital statistics, and hospitalization databases are publically available at web links provided in the Supplemental References. Information on how to access to data from National Heart, Lung, and Blood Institute observational cohorts can be found at https://biolincc.nhlbi.nih.gov.

ADDITIONAL TECHNICAL DETAILS FOR THE CARDIOVASCULAR DISEASE POLICY MODEL

Population and risk factors

We use the 2010 census to define the number of Black men each age from 35 to 94 years who are living in the US and census projections to quantify the number of new 35-year-olds entering the model population each annual cycle from 2011 until the end of the simulated timescale.[8,9] In the base year (2010), the model separates the population into those without pre-existing CVD and those with a history of CVD based on analysis of data collected from the National Health Interview Surveys from 2009-2011.[37] The population of Black men without pre-existing CVD is further stratified into cells defined by age and risk factor levels, with risk factors categorized into two or three strata as shown below. The prevalence of CVD risk factors were estimated using survey design-weighted analysis of National Health and Nutrition Examination Surveys (NHANES) from 2009-2016:[10]

- **systolic blood pressure (BP):** <140 mmHg, 140 to <160 mmHg, or ≥160 mmHg
- **current use of anti-hypertensive medications:** yes (self-reports currently taking a medication prescribed for high blood pressure) vs. no (not currently taking a medication to treat high blood pressure)
- **low density lipoprotein cholesterol (LDL-C):** <100 mg/dL, 100 to <130 mg/dL, or ≥130 mg/dL
- **high density lipoprotein cholesterol (HDL-C):** <40 mg/dL, 40 to <60 mg/dL, or ≥60 mg/dL

- **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), or no smoke exposure (self-reports no active smoking and <0.05 µg/mL cotinine)
- **diabetes status:** yes (self-reports doctor diagnosis or fasting glucose > 125 mg/dL) vs. no (no doctor diagnosis and fasting glucose ≤ 125 mg/dL)
- **body mass index (BMI)**: <25 kg/m^2, 25-<30 kg/m^2, or ≥30 kg/m^2

Using multivariate distributions estimated using NHANES years 1999-2016,[10] the model distributes Black men without CVD into 58,320 cells representing all combinations of the seven risk factor levels (60 ages * 3^5 (six risk factors with three levels) * 2^2 (two risk factors with 2 levels)). The model then uses an iterative proportional fitting procedure to match marginal proportions for individual risk factors measured in more contemporary survey years (NHANES 2009-2016) in Black men self-reporting no history of CVD.[10] The model assigns each cell the age-, sex-, and race-ethnic-specific mean values for all risk factor levels represented by the cell and estimated from NHANES 2009-2016.[10]

Model risk functions

Each annual cycle, a proportion of the CVD-free population experiences an incident CVD event. The remaining population, remaining free of CVD, transitions among cells at rates that preserve age-specific risk factor trends over time. Incident events occurring in the CVD-free population

are characterized as coronary heart disease (stable and unstable angina, hospitalized myocardial infarction (MI), or arrests occurring outside or inside the hospital), stroke (hospitalized ischemic or hemorrhagic stroke), or death from a cause other than cardiovascular disease. Annual rates of incident events are defined by risk functions that include age- and sex-specific beta coefficients, which determine the relationship between CVD risk factors and incident events; and alpha coefficients, which are generated by fitting to annual incidence rates for coronary heart disease, stroke, and non-CVD death (detailed below). The risk for each outcome is then calculated for every cell using alpha and beta coefficients, along with mean values for each CVD risk factor level represented by the cell, using the following equation:

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

Where *r* represents risk, with separate risks for coronary heart disease, stroke, and non-CVD death; α represents age- and sex-specific intercepts for each risk function determined by the model when fitting to incidence rates in the base year; β represents the effect on risk for one-unit changes in a given CVD risk factor; *m* represents mean values for a given risk factor; and k represents a counter over all six CVD risk factors that have an effect on coronary heart disease, stroke, and/or non-CVD death risk (i.e., systolic BP, LDL-C, HDL-C, smoking, diabetes, and BMI).

We estimated risk function beta coefficients using competing risk Cox proportional hazards analysis of data collected from Black adults enrolled in observational cohort studies funded by

the National Heart, Lung, and Blood Institute. Cohorts included the Atherosclerosis Risk in Communities Study,[11] the Cardiovascular Health Study,[12] the Coronary Artery Risk Development in Young Adults Study,[13] the Framingham Offspring study,[14] the Health, Aging and Body Composition Study,[15] and the Multi-Ethnic Study of Atherosclerosis.[16]

Incidence rates

We derived inputs for coronary heart disease incidence by first estimating annual incidence of coronary heart disease for men included in the Framingham Heart Study and Framingham Offspring Study data from 1988-2007 and subsequently adjusting the incidence to account for differences in risk factor distributions measured in Framingham participants compared to Black men surveyed by NHANES from 2009-2016.[10,14,38] For stroke incidence, we first estimated the overall annual rate of stroke hospitalizations by age for Black men in the NIS years 2012-2015 and then reduced the resulting rates of total stroke by assuming that: (1) 77% of total strokes are first-ever strokes, with the proportion of first-to-total strokes inversely related to 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); and (2) the rate of incident strokes occurring in those with prior MI is higher than the rate of first strokes in CVD-free adults. [21,26-28,30] We confirmed that our coronary heart disease and stroke incidence inputs align with rates reported for observational studies that evaluated the effect of race on CVD outcomes.[18,21,60,61]

Characterization of incident events

The population experiencing an incident coronary heart disease or stroke event in any given year transitions into the "bridge" submodel, a 30-day period with increased healthcare utilization and cause-specific mortality. The model apportions incident coronary heart disease according to event type (angina pectoris, MI, or arrest), assuming that smokers have a higher risk of MI and arrest relative to non-smokers.[17,19] Environmental tobacco exposure is assumed to carry a relative risk of 1.26 for MI and cardiac arrest compared with non-exposed non-smokers but not to influence angina.[20] Relative to inputs for our U.S. model, we assumed that Black men with incident coronary heart disease are more likely to have a fatal MI or arrest as their first event based on analysis of fatal versus non-fatal incident events by race in observational cohort studies.[18]

Event and case fatality rates in CVD states

Those who survive the 30 days following incident coronary heart disease or stroke events transition into cells corresponding to their cardiovascular event history, age, and sex. The model assumes a higher rate of recurrent CVD events, procedures, and deaths in the first year following a new event compared to years 2 and beyond, with annual rates of CVD events and deaths dependent on age, sex, and CVD history. To develop CVD state transition rates for the CVD Policy Model representing Black men, we started with inputs developed for our calibrated US model and adjusted rates where evidence indicates variation over

race/ethnicity.[3,4,18,21,26,34,60-63] Final event and case fatality rates inputs are iteratively adjusted during calibration (Supplement page 20 further describes death rates and calibration targets).

We assume similar rates of coronary heart disease events and revascularization procedures in Black men with a history of CVD compared with all US men with CVD, which were estimated for the national model using hospitalization databases, natural history studies, and complementary sources.[22-26,29,32-35,64-66] We assume a higher probability of fatal MIs and out-of-hospital coronary arrests for Black men compared with all US men as evidenced in published results from observational cohorts.[18] We used 2012-2015 NIS data to estimate age-stratified annual rates of stroke recurrence along with first strokes in those with prior coronary heart disease for Black men, which we apportioned after removing strokes assumed to occur in CVD-free men (described on Supplement page 17). [21,26-28,30]

For the national CVD Policy Model, age- and sex-specific in-hospital MI case-fatality rates were estimated from the 2010 National Hospital Discharge Survey (NHDS) and adjusted rates for specific health states (e.g., fatality from first MI vs. recurrent MI) using relative risks sourced from published literature.[4,22,29,32,33] We estimated pre-hospital arrest deaths from U.S. Vital Statistics and out-of-hospital cardiac arrests surviving to hospital from the NHDS.[22,36] We extended coronary heart disease case fatality inputs to 30 days using in-hospital to 30-day mortality ratios calculated from California hospital and mortality data along with published studies.[31,34,35] For the current model representing Black men, we adjusted MI case fatality inputs using age-stratified relative rates of in-hospital death following MI for Black men

compared to all US men in NIS 2012-2015 data along with evidence from Medicare for race/ethnic differences in 30-day mortality post discharge.[30,63] Whereas MI and arrest case fatality rates represent 30 days following index events, stroke case fatality inputs represent deaths in the first year following stroke events. We calculated age-stratified stroke fatality rates by dividing annual deaths from stroke reported for Black men in vital statistics data by annual stroke hospitalizations for Black men observed in the NIS. [30,36] Final 1-year stroke death rates were adjusted during the calibration process, described below, and compared for consistency with published data.[21,67]

Death rates and calibration targets

For calibration targets, we used the NIS from years 2012-2015 to estimate age-stratified numbers of Black men hospitalized for MI (ICD-9 code 410) and stroke (ICD 9 codes 430-438) annually.[30] We used US vital statistics data from 2010-2015 to generate annual age-stratified rates of Black men who died from coronary heart disease (ICD-10 codes I20-I25 and two-thirds of I49, I50, and I51), stroke (ICD-10 codes I60-I69), and all causes (Supplemental Table S3).[36]

We calculated age-stratified inputs for the annual rate of non-CVD death in Black men by subtracting from the count of all deaths in vital statistics from 2010-2015 those due to coronary heart disease or stroke and dividing the remaining number by the total population represented over years 2010-2015.[36] The model assumes that non-CVD mortality occurs at the same rate among those with or without prior CVD.

Healthcare utilization costs

We estimated hospitalization costs for coronary heart disease and stroke events using survey-weighted data from NIS years 2012-2015.[30] Hospitalization cost inputs vary over age, sex, event type (i.e., MI, arrest, heart failure, unstable angina, or stroke), and survival status. Costs for isolated revascularization procedures and stroke rehabilitation were estimated using 2008 data from the California Office of Statewide Health Planning and Development (OSHPD), which were deflated by a factor of 1.15 to represent national costs before inflating to 2019 US dollars.[31,68] Hospitalization records were selected using ICD-9 or ICD-10 codes located in the first coding position and survival status was assessed using discharge indicators (Supplemental Table S7). We converted hospital charges to costs using cost-to-charge ratios calculated for the respective datasets and increased hospital facility costs by a factor of 1.264 to capture professional fees and services delivered during the hospitalization but not captured by hospitalization databases.[30,31,68] In order to convert admissions costs to 30-day costs, we used ratios of costs for admissions estimated in OSHPD to costs over 30-days following discharge measured in the Medical Expenditure Panel Survey (MEPS) in 1999-2008.[31,43] The model assumes that healthcare utilization costs are higher during the first year following acute events compared to years two and beyond, with chronic CVD costs for relevant time periods estimated using MEPS data.[43]

We estimated age- and sex-specific annual healthcare costs for non-CVD care (i.e., "background costs") using data from MEPS 2006-2015 along with complementary sources that provide

estimates of long-term care costs not captured by MEPS.[43,69-72] We estimated mean background healthcare costs by calculating total healthcare expenditures minus the costs for office visits and treatments relating to CVD, hypertension or hyperlipidemia.[43] We then calculated the mean background expenditures in MEPS using a two-part model.[73] The first part uses survey-weighted multivariable logistic regression to predict the probability of non-zero background healthcare costs, adjusting for covariates. The second part, among individuals with non-zero background healthcare costs, uses a survey-weighted multivariable generalized linear model with a log link and gamma distribution to predict background healthcare costs, adjusting for the same covariates as in the logistic regression. We used the combined two-part model to calculate age- and sex-specific mean background costs, excluding MEPS participants with CVD-related emergency department visits or hospitalizations in the prior year. We estimated average annual costs for long-term care by combining utilization data reported from the National Center for Health Statistics in 2013-2014 with cost estimates reported in the 2018 US Renal Data System Annual Report and other published sources.[69-72]

ADDITIONAL TECHNICAL DETAILS

Measurement of systolic BP

We estimated systolic BP distributions for Black men in the US from NHANES 2009-2016.[10] Trained and certified NHANES examiners took three BP readings using a mercury-gravity sphygmomanometer after participants are instructed to rest in a seated position for five minutes. Examiners took a fourth reading if any of the first three measures were interrupted or incomplete. For our analysis, we dropped the first of three readings and calculated the average of the remaining two to assign each participant a systolic BP. We then categorized Black men as having systolic BP <140 mmHg, 140 to <160 mmHg, or ≥160 mmHg and calculated survey-weighted prevalence and mean values for Black men stratified on age and cardiovascular disease history status to use as inputs for systolic BP distributions (Supplemental Table S4).

Calculation of medication costs for one-way sensitivity analysis

We assumed average US drug pricing as an upper bound in place of our base case assumption of $4 per prescription per month (generic pricing) in a one-way sensitivity analysis. For the sensitivity inputs, we used MEPS 2015-2017 data to estimate survey-weighted national average costs for each class of antihypertensive medication reported in the treatment and control arms of the trial.[43,47] In order to generate stable cost estimates for classes less frequently reported in MEPS, we pooled data for potassium-sparing diuretics, loop diuretics, alpha-1 blockers, alpha

agonists, and direct vasodilators. We applied the class-specific average costs estimated in MEPS to the distribution of medications prescribed to participants in the intervention and control arms of the barbershop trial and used the mean difference in drug cost between arms to represent the annual average medication cost for each participant in our simulated intervention. [43,47]

Estimation of serious adverse event inputs

We estimated the annual rate of SAEs for anti-hypertensive medications by combining information from a meta-analysis of 21 antihypertensive drug treatment trials, data from the Systolic Blood Pressure Intervention Trial (SPRINT), and medication use at 12 months from the LABBPS.[39,54] We defined SAEs as "fatal or life threatening" events resulting in "clinically significant or persistent disability," prolonging a hospitalization, or representing "clinically significant hazard or harm possibly requiring intervention".[54] Adverse events reported in SPRINT included hypotension, syncope, bradycardia, electrolyte abnormality, and acute kidney injury. We pooled the data from the meta-analysis and SPRINT to estimate the probability of experiencing an SAE when taking >2 full standard doses of antihypertensive medications (1.31%; 95% CI: 1.08%, 1.66%) and ≤2 (0.85%; 95% CI: 0.64%, 1.04%).[39,54]

We determined the average cost of SAE hospitalizations from the 2014 NIS online query system available from the Healthcare Cost and Utilization Project.[30] We collected mean costs using ICD-9 codes as follows: hypotension (ICD-9 codes 458.0, 458.29, 458.8, 458.9), syncope (ICD-9 code

780.2), bradycardia (ICD-9 codes 427.81, 427.89), electrolyte abnormality (ICD-9 codes 791.9, 276.*), and acute kidney injury (ICD-9 codes 584.*, 586.*). We then weighted the mean hospitalization cost for each category of SAE by the frequency distribution of SAE types reported in SPRINT to generate an average overall hospitalization cost for SAEs associated with hypertension management. To capture professional fees associated with the hospitalization but not captured in the NIS database, we increased the mean hospitalization cost by a factor of 1.264 for a final mean hospitalization cost of $10,818.[68] We assumed that patients who survive hospitalization will have one follow-up outpatient appointment with a mean cost of $148, which is the 2019 non-facility price for procedure code 92115 listed in the Centers for Medicaid and Medicare Services Physicians Fee Schedule.[57]

We estimated the probability that a patient would die from a SAE (p = 0.017) using the discharge status for SAE hospitalizations from the 2014 NIS online query system available from the Healthcare Cost and Utilization Project.[30] For those surviving a SAE, we assumed a quality-of-life decrement of 0.1 lasting 30 days for participants with acute kidney injury and a decrement of 0.1 lasting 14 days for those experiencing hypertension, syncope, bradycardia, or electrolyte abnormalities based, with frequencies of each type sourced from the SPRINT trial.[54]

Supplemental Tables

Supplemental Table S1. Key input parameters for current analysis					
			Monte Carlo simulations		
Input Parameter	**Base case values**	**Range for sensitivity analyses**	**Distributions**	**Parameters**	**Source**
Intervention effect sizes					
Scenarios for reduction in SBP from Barbershop intervention, mm Hg	10 15 20 25	-	-	-	Assumed range for real-world implementation of Victor et al (2019)[47]
Relative risk of CHD per 10 mmHg SBP	0.83	(0.78-0.88)	Log normal	mu = -0.186 sigma = 0.031	Ettehad (2016)[50]
Relative risk of stroke per 10 mmHg SBP	0.73	(0.68-0.77)	Log normal	mu = -0.315 sigma = 0.032	Ettehad (2016)[50]
Relative risk of non-CVD death 10 mmHg SBP[†]	0.97	(0.94-0.99)	Log normal	mu = -0.035 sigma = 0.011	Pooled cohort data from NHLBI[‡11-16]
Adverse events from anti-hypertensive therapies					
Serious adverse events, %	0.38[¥]	(0.31-0.48)	β	alpha= 47.22 beta= 77.05	LABBPS participant data; Xie (2016);[39] SPRINT Research Group (2015) [54]
Death following serious adverse event, %	1.72	-	-		AHRQ H-CUP[30]
Treatment costs, 2019 USD					
Anti-hypertensive medications	$52	($42-63)	Normal	Mean = $52 SE = $5	$4 generic medications,[53] LABBPS participant data

Pharmacist year 1	$519	($415-$623)	Normal	Mean = $519 SE = $53	LABBPS participant data; Bureau of Labor Statistics[52]
Pharmacist years 2+	$295	($236 - $354)	Normal	Mean = $295 SE = $30	LABBPS participant data, Bureau of Labor Statistics[52]
Serious adverse event hospitalization	$10,818	-	-	-	National Inpatient Sample,[30] Peterson (2015)[68]
Follow-up clinic visit for SAE (code 99215)	$148	n/a	n/a	n/a	Physician Fee Schedule[57]
Cardiovascular costs					
Costs of CHD care, 2019 USD*					
Acute fatal MI hospitalization	$64,115	($60,348-67,841)	Log normal	mu= 11.070 sigma= 0.030	National Inpatient Sample,[30] Peterson et al (2015)[68]
Acute nonfatal MI hospitalization	$40,579	($40,140-$41,031)	Log normal	mu= 10.611 sigma= 0.006	National Inpatient Sample, [30] Peterson et al (2015)[68]
Acute MI posthospitalization year 1 costs	$11,748	($10,265-$14,000)	Log normal	mu= 9.368 sigma= 0.081	Medical Expenditure Panel Survey[43]
CHD costs, subsequent years	$2,451	($2,079-$2,862)	Log normal	mu= 7.800 sigma= 0.081	Medical Expenditure Panel Survey[43]
Costs of heart failure care, 2019 USD*					
Heart failure hospitalization	$20,038	($19,481-$20,605)	Log normal	mu= 9.877 sigma= 0.240	National Inpatient Sample,[30] Peterson et al (2015) [68]
Costs of stroke care, 2019 USD*					

Fatal stroke hospitalization	$34,058	($32,140-$36,068)	Log normal	mu= 10.436 sigma= 0.029	National Inpatient Sample, [30] Peterson et al (2015) [68]
Nonfatal stroke hospitalization	$20,996	($20,601-$21,388)	Log normal	mu= 9.952 sigma= 0.010	National Inpatient Sample,[30] Peterson et al (2015) [68]
Poststroke cost, months 2-11	$18,569	($15,504-$21,853)	Log normal	mu= 9.825 sigma= 0.087	Medical Expenditure Panel Survey[43]
Poststroke cost, annual, subsequent years	$5,160	($4,288-$6,121)	Log normal	mu= 8.545 sigma= 0.090	Medical Expenditure Panel Survey[43]
Utility Weights					
No history of cardiovascular disease	1.00				
History of angina	0.9064	(0.8710-0.9360)	β	alpha= 278.77 beta= 28.79	Moran et al (2014),[44,45] Murray et al (2012)[46]
History of MI	0.9648	(0.9513-0.9764)	β	alpha= 798.21 beta= 29.12	Moran et al (2014),[44,45] Murray et al (2012)[46]
History of stroke	0.8835	(0.8456-0.9133)	β	alpha= 304.00 beta= 40.09	Moran et al (2014),[44,45] Murray et al (2012)[46]
History of MI and stroke	0.8524	(0.8083-0.8987)	β	alpha= 247.86 beta= 42.92	Moran et al (2014),[44,45] Murray et al (2012)[46]
Transient utility tolls for acute events					
Angina	0.0936 for 30 days	(0.0621-0.1351)	β	alpha= 25.76 beta= 3276.66	Moran et al (2014),[44,45] Murray et al (2012)[46]
Percutaneous revascularization	0.1168 for 30 days	(0.0499-0.2336)	β	alpha= 6.14 beta= 633.63	Kazi et al (2014)[74]
Surgical revascularization	0.2336 for 30 days	(0.1168-0.4818)	β	alpha= 6.15 beta= 314.37	Kazi et al (2014)[74]

Acute MI	0.0961 for 30 days	(0.0621-0.1363)	β	alpha= 25.56 beta= 3210.08	Moran et al (2014),[44,45] Murray et al (2012)[46]
Acute stroke	0.1375 for 30 days	(0.1022-0.1874)	β	alpha= 93.58 beta= 3463.06	Moran et al (2014),[44,45] Murray et al (2012)[46]
Acute kidney injury associated with treatment	0.1000 for 30 days	-	-	-	Assumed
Serious adverse events other than acute kidney injury	0.1000 for 14 days	-	-	-	Assumed

† the effect of changes in systolic BP on risk of non-CVD declines over age; the effect shown here is for men age 55-64 years

‡ from harmonized and pooled data collected among Black adults in the Atherosclerosis Risk in Communities Study,[11] the Cardiovascular Health Study,[12] the Coronary Artery Risk Development in Young Adults Study,[13] the Framingham Offspring study,[14] the Health, Aging and Body Composition Study,[15] and the Multi-Ethnic Study of Atherosclerosis[16]

* Healthcare cost inputs vary over age decile; costs for men age 55-64 years (at the time of event or in a chronic state) are presented here for reference.

¥ We assumed that the rate of adverse events would vary with the amount of blood pressure reduction. The value reported here (0.38%) corresponds to a 20.8 mm Hg reduction in systolic BP observed in the LABBPS. For scenarios assuming lower systolic BP reductions, we decreased the rates of SAEs using the approach described previously.[55,56]

Supplemental Table S2. Core inputs for the CVD Policy Model representing Black men living in the US	
Model inputs	**Sources**
Population size	US Census (2010)[8]
Population projections	US Census projections (2017 release)[9]
Cardiovascular disease prevalence	National Health Interview Survey (2009-2011) [37]
Cardiovascular risk factor distributions	National Health and Nutrition Examination Surveys (2009-2016)[10]
Risk functions for CHD, stroke, and non-CVD death (baseline risk)	Data pooled and harmonized from Black participants in the following NHLBI cohorts: the Atherosclerosis Risk in Communities Study,[11] the Cardiovascular Health Study,[12] the Coronary Artery Risk Development in Young Adults Study,[13] the Framingham Offspring study,[14] the Health, Aging and Body Composition Study,[15] and the Multi-Ethnic Study of Atherosclerosis[16]
CHD incidence and characterization	Framingham Heart Study and Framingham Offspring Study (1988-2007);[14,38] National Health and Nutrition Examination Surveys (2009-2016); [10] Coady et al (2006);[17] Colantonio et al (2017) [18]; Parish et al (2005);[19] Law et al (1997) [20]
Stroke incidence	National Inpatient Sample (2010-2015);[30] Virani et al (2020)[21]
Rates of CVD events and procedures in those with prior CVD	National Inpatient Sample (2010-2015);[30] National Hospital Discharge Survey (2000-2010); [22] Appelros et al (2011);[23] Lakshminarayah et al (2011);[24] Prosser et al (2007);[25] Virani et al (2020);[21] Witt (2005)[26] Merkler et al (2018);[27] Sundboll (2016);[28] Vaccarino et al (2009);[29] Virani et al (2020)[21]
MI 30-day case fatality rates	National Inpatient Sample (2010-2015);[30] National Hospital Discharge Survey (2010);[22] California Office of Statewide Health Planning and Development;[31] Canto et al (2021);[32] Colantonio et al (2017)[18] Rieves et al (2000);[33] Vaccarino et al (2009)[29]
Arrest 30-day case fatality rates	National Hospital Discharge Survey (2000-2010); [22] California Office of Statewide Health Planning and Development;[31] Groeneveld et al (2003);[34] Rea et al (2003)[35]

Rates of mortality from revascularization procedures	National Hospital Discharge Survey (2000-2010) [22]
Annual rates of CHD death from causes other than MI, arrest, or revascularization	National Hospital Discharge Survey (2000-2010);[22] National Vital Statistics (2010-2015)[36]
Stroke 1-year fatality rates	National Vital Statistics (2010-2015);[36] National Inpatient Sample (2012-2015)[30]
Calibration targets	**Sources**
Total annual hospitalizations for MI and stroke	National Inpatient Sample (2012-2015)[30]
Total annual deaths from CHD, stroke, and non-CVD causes	National Vital Statistics (2010-2015) [36]

Abbreviations: CHD = coronary heart disease; CVD = cardiovascular disease; MI = myocardial infarction

Supplemental Table S3. Results of calibration exercise comparing selected CVD Policy Model simulation outputs for 2010 with targets for Black men measured in national databases

	CHD deaths		Stroke deaths		All-cause deaths		Total MI		Total strokes	
	Target source: national vital statistics (2010-2015)		Target source: national vital statistics (2010-2015)		Target source: national vital statistics (2010-2015)		Target: National Inpatient Sample (2012-2015)		Target: National Inpatient Sample (2012-2015)	
Age category	**Target***	**Model**	**Target***	**Model**	**Target***	**Model**	**Target***	**Model**	**Target***	**Model**
35-44	870	869	281	281	8,184	8,183	2,579	1,744	3,550	3,537
45-54	3,016	3,011	864	858	18,661	18,645	6,747	7,091	11,172	11,109
55-64	5,399	5,422	1,467	1,467	29,925	29,929	9,204	8,333	15,283	15,275
65-74	5,162	5,160	1,515	1,516	27,366	27,330	6,374	6,368	11,598	11,705
75-84	4,881	4,864	1,563	1,552	25,198	25,092	3,732	4,458	7,449	7,391
85-94	2,904	2,909	846	850	14,279	14,217	1,395	2,178	2,580	2,588
Deviation from target	0.01%		-0.18%		-0.18%		0.25%		0.01%	

CHD = coronary heart disease; CVD = Cardiovascular disease; MI = myocardial infarction; NIS = National Inpatient Sample

* Mortality and event rates from nationally representative datasets were pooled over years and annualized

Supplemental Table S4. Prevalence and mean values of systolic blood pressure for Black men stratified by cardiovascular disease history status in the CVD Policy Model, estimated from NHANES 1999-2016*

Age group	Systolic BP stratum	Prevalence		Mean value (mmHg)
		No history of CVD	History of CVD	
Age 35-44	<140 mmHg	0.820	0.603	120.3
	140 to <160 mmHg	0.139	0.244	148.6
	≥ 160 mmHg	0.041	0.153	171.2
Age 45-54	<140 mmHg	0.777	0.639	122.1
	140 to <160 mmHg	0.169	0.226	147.3
	≥ 160 mmHg	0.054	0.135	179.5
Age 55-64	<140 mmHg	0.655	0.654	123.8
	140 to <160 mmHg	0.254	0.217	148.6
	≥ 160 mmHg	0.091	0.129	173.2
Age 65-74	<140 mmHg	0.633	0.617	124.7
	140 to <160 mmHg	0.253	0.226	147.9
	≥ 160 mmHg	0.114	0.157	173.2
Age 75-84	<140 mmHg	0.522	0.652	124.1
	140 to <160 mmHg	0.303	0.230	150.0
	≥ 160 mmHg	0.175	0.118	175.0
Incoming, Age 35‡	<140 mmHg	0.871	0.666	-
	140 to <160 mmHg	0.103	0.219	-
	≥ 160 mmHg	0.026	0.115	-

CVD = cardiovascular disease; NHANES = National Health and Nutrition Examination Survey; SBP = systolic blood pressure

* NHANES years 2009-2016 used for the population without pre-existing CVD and years 1999-2016 used for those reporting a history of CVD

‡ incoming 35-year-olds are assigned the mean value of SBP shown for ages 35-44 years

Supplemental Table S5. Description of target population in simulated barbershop-based intervention	
Characteristics (cumulative down rows)	**Average annual number from 2019-2028**
All Black men 35-79 years old in the US	9,847,000
+ SBP >=140 mmHg	2,767,000
+ live in metropolitan area	2,352,000
+ regular patron at a barbershop	1,176,000
+ enroll in program **(treatment population)**	**941,000**

Supplemental Table S6. Average annual persons treated by effectiveness scenario	
Mean reduction in systolic BP	**Average Annual of People Treated**
No change (control arm)	921,000
10 mm Hg	932,000
15 mm Hg	937,000
20 mm Hg	941,000
25 mm Hg	945,000

Supplemental Table S7. Incremental Classification of Diseases 9th (ICD-9) and 10th (ICD-10) Revision codes for identification of coronary heart disease and stroke events in vital statistics and hospitalization records

Outcome category	Event type	Coding system	Code number	Description
Coronary Heart Disease	**Acute Myocardial Infarction**	**ICD-9**	410	Acute myocardial infarction
		ICD-10	I21.x	Acute myocardial infarction
			I22.x	Subsequent ST elevation (STEMI) and non-ST elevation (NSTEMI) myocardial infarction
	Cardiac arrest	**ICD-9**	427	Cardiac dysrhythmias
		ICD-10	I46.0	Cardiac arrest with successful resuscitation
			I46.1	Sudden cardiac death, so described
			I46.9	Cardiac arrest, unspecified
	Other acute CHD	**ICD-9**	411	Other acute and subacute forms of ischemic heart disease
		ICD-10	I24.1	Dressler syndrome
			I24.8	Other forms of acute ischaemic heart disease
			I24.9	Acute ischaemic heart disease, unspecified
	Angina	**ICD-9**	413	Angina pectoris
		ICD-10	I20.0	Unstable angina
			I20.1	Angina pectoris with documented spasm
			I20.8	Other forms of angina pectoris
			I20.9	Angina pectoris, unspecified
	Other chronic CHD	**ICD-9**	412	Old myocardial infarction
			413	Angina pectoris
			414	Other forms of chronic ischemic heart disease
		ICD-10	I25.0	Atherosclerotic cardiovascular disease, so described
			I25.1	Atherosclerotic heart disease
			I25.2	Old myocardial infarction
			I25.3	Aneurysm of heart
			I25.4	Coronary artery aneurysm
			I25.5	Ischaemic cardiomyopathy
			I25.6	Silent myocardial ischaemia
			I25.8	Other forms of chronic ischaemic heart disease
			I25.9	Chronic ischaemic heart disease, unspecified
	Heart failure and other heart disease*	**ICD-10**	I49.0	Ventricular fibrillation and flutter
			I49.1	Atrial premature depolarization
			I49.2	Junctional premature depolarization
			I49.3	Ventricular premature depolarization
			I49.4	Other and unspecified premature depolarization
			I49.5	Sick sinus syndrome
			I49.8	Other specified cardiac arrhythmias
			I49.9	Cardiac arrhythmia, unspecified
			I50.0	Congestive heart failure

			I50.1	Left ventricular failure
			I50.9	Heart failure, unspecified
			I51.8	Other ill-defined heart diseases
			I51.9	Heart disease, unspecified
Stroke	**Stroke inputs represent total stroke**	**ICD-9**	430	Subarachnoid hemorrhage
			431	Intracerebral hemorrhage
			432	Other and unspecified intracranial hemorrhage
			433	Occlusion and stenosis of precerebral arteries
			434	Occlusion of cerebral arteries
			435**	Transient cerebral ischemia
			436	Acute, but ill-defined, cerebrovascular disease
			437	Other and ill-defined cerebrovascular disease
			438	Late effects of cerebrovascular disease
		ICD-10	I60.X	Nontraumatic subarachnoid hemorrhage
			I61.X	Nontraumatic intracerebral hemorrhage
			I62.X	Other and unspecified nontraumatic intracranial hemorrhage
			I63.X	Cerebral infarction
			I65.X	Occlusion and stenosis of precerebral arteries, not resulting in cerebral infarction
			I66.X	Occlusion and stenosis of cerebral arteries, not resulting in cerebral infarction
			I67.X	Other cerebrovascular diseases
			I68.X	Cerebrovascular disorders in diseases classified elsewhere
			I69.X	Sequelae of cerebrovascular disease

* Two-thirds of codes I49-I51 considered ischemic heart failure and include in counts of coronary heart disease deaths from US vital statistics

** Note that the CVD Policy Model definition of stroke incidence and recurrence does not include transient ischemic attacks; we applied positive predictive values from Williams et al. (1999) to ICD-9 codes for cerebrovascular disease (including code 435 indicating transient cerebral ischemia) to estimate hospitalizations for stroke

Supplemental Figures

Supplemental Figure S1

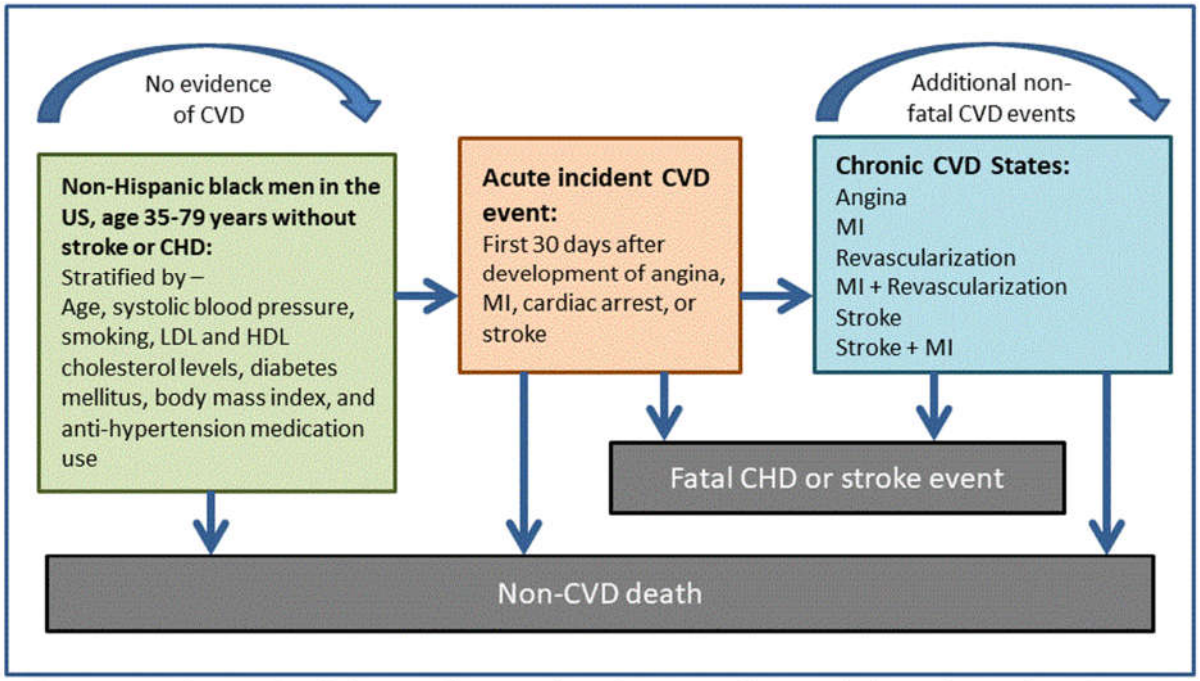
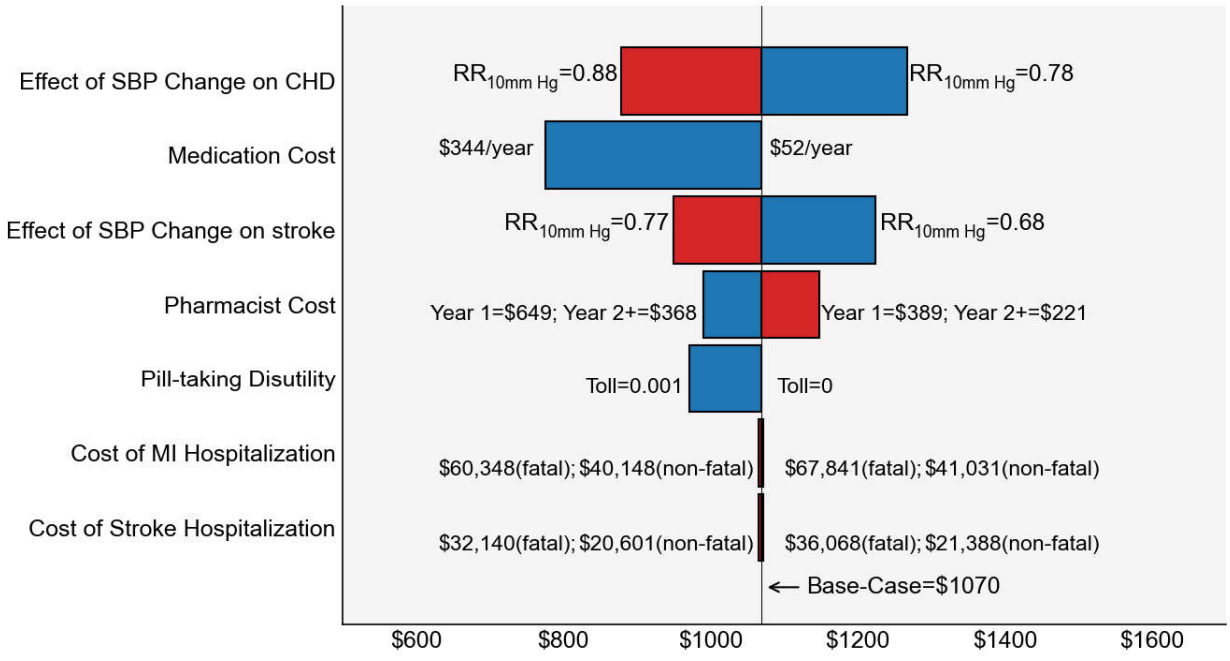


Figure S1. Schematic of the Cardiovascular Disease Policy Model as adapted to the present analysis. Abbreviations: CHD, coronary heart disease; CVD, cardiovascular disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MI, myocardial infarction.

Supplemental Figure S2.Tornado diagram. The figure below, called a tornado diagram because of its appearance, depicts the results of key one-way sensitivity analyses (i.e., analyses in which one parameter is varied at a time while holding all others at their base-case value). These analyses quantify the effect of uncertainty on the study findings. For this analysis, enrollment in the program is assumed to result in a 15mm Hg reduction in systolic BP (lower than the 20.8 mm Hg observed in the LABPPS but within the 95% confidence interval of that estimate), and patients are assumed to experience no pill-related disutility for increased use of BP meds in the intervention arm. For each parameter, red represents the lower end of the parameter value used in sensitivity analyses and blue represents the upper end of the parameter value. The figure demonstrates that, in the base case, the program would have to cost $1070 or less to be cost-effective at a threshold of $100,000 per QALY gained. This threshold price is sensitive to effect of a 10mm Hg change in SBP on CHD and stroke, medication costs, cost of the pharmacist's clinical time, and inclusion of a disutility for taking medication each day.



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