Sick Populations and Sick Subpopulations

Reducing Disparities in Cardiovascular Disease Between Blacks and Whites in the United States

BACKGROUND: Cardiovascular disease (CVD) death rates are much higher in blacks than whites in the United States. It is unclear how CVD risk and events are distributed among blacks versus whites and how interventions reduce racial disparities.

METHODS: We developed risk models for fatal and for fatal and nonfatal CVD using 8 cohorts in the United States. We used 6154 adults who were 50 to 69 years of age in the National Health and Nutrition Examination Survey 1999 to 2012 to estimate the distributions of risk and events in blacks and whites. We estimated the total and disparity impacts of a range of population-wide, targeted, and risk-based interventions on 10-year CVD risks and event rates.

RESULTS: Twenty-five percent (95% confidence interval [CI], 22–28) of black men and 12% (95% CI, 10–14) of black women were at ≥6.67% risk of fatal CVD (almost equivalent to 20% risk of fatal or nonfatal CVD) compared with 10% (95% CI, 8–12) of white men and 3% (95% CI, 2–4) of white women. These high-risk individuals accounted for 55% (95% CI, 49–59) of CVD deaths among black men and 42% (95% CI, 35–46) in black women compared with 30% (95% CI, 24–35) in white men and 18% (95% CI, 13–22) in white women. We estimated that an intervention that treated multiple risk factors in high-risk individuals could reduce black-white difference in CVD death rate from 1659 to 1244 per 100 000 in men and from 1320 to 897 in women. Rates of fatal and nonfatal CVD were generally similar between black and white men. In women, a larger proportion of women were at ≥7.5% risk of CVD (30% versus 19% in whites), and an intervention that targeted multiple risk factors among this group was estimated to reduce black-white differences in CVD rates from 1688 to 1197 per 100 000.

CONCLUSIONS: A substantially larger proportion of blacks have a high risk of fatal CVD and bear a large share of CVD deaths. A risk-based intervention that reduces multiple risk factors could substantially reduce overall CVD rates and racial disparities in CVD death rates.

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Clinical Perspective

What Is New?

- We investigated how risk of fatal and fatal plus nonfatal cardiovascular disease (CVD), estimated with a risk prediction model, is distributed among whites and blacks in the United States and how population-wide or targeted interventions on CVD risk factors would reduce these racial disparities.
- We used a nationally representative sample of adults 50 to 69 years of age in the United States and a CVD risk prediction model that was recalibrated separately for blacks and whites.

What Are the Clinical Implications?

- Our results indicated that there are substantial disparities in risk of fatal CVD.
- A large proportion of fatal CVD events among blacks were concentrated among a small proportion of the population; in contrast, racial disparities in risk of fatal and nonfatal CVD were only noticeable among women.
- Population-wide and targeted interventions on single risk factors did not reduce black-white disparities in fatal CVD risk substantially.
- An intervention that focused on high-risk individuals and reduced multiple risk factors simultaneously could reduce black-white disparities in fatal CVD risk by a quarter in men and a third in women.
- Focusing preventive interventions on high-risk individuals has a large potential to improve overall CVD health and to reduce racial disparities.

Cardiovascular diseases (CVDs) are the leading causes of death in the United States, with substantially higher death rates among blacks than whites.[1,2] Previous research has shown that up to three-quarters of absolute disparities between blacks and whites in CVD mortality may be due to differences in classic risk factors (ie, raised blood pressure and serum cholesterol, diabetes mellitus, obesity, and smoking).[3,4] Therefore, interventions that reduce these risk factors are expected to reduce disparities in CVD mortality between blacks and whites, but it is not clear which types of interventions, population-wide or targeted, can reduce racial disparities. Population-wide interventions can have large impacts on overall disease burden,[5] but their impact on disparities depends on how they change risk factors in different subgroups of the population. For example, health education may reduce or widen disparities, depending on how it is delivered.[6–8] The disparity impact of interventions that target high-risk individuals (identified with the use of a single risk factor or a combination of risk factors) will depend on whether the worse-off group has more or fewer high-risk individuals. Therefore, it is essential to have information on not only the average CVD risk and events but also how CVD risk and events are distributed in better-off and worse-off subgroups of the population.

Some studies have qualitatively or quantitatively assessed the impacts of current risk factor exposures or scenarios of reducing risk factors on disparities in CVD or total mortality.[3,4,9–14] Most of these studies have considered hypothetical risk factor reductions as opposed to interventions that could be implemented in practice. Other studies have used inconsistent or incomparable data and methods for calculating mortality effects across different risk factors, therefore reducing comparability. Furthermore, no study has assessed the disparity impact of risk-based prevention that is recommended by recent clinical guidelines[15,16] because information on distributions of absolute CVD risk by race was not available. Here, we analyzed the total and disparity impacts of a range of population-wide, targeted, and risk-based interventions on 10-year CVD risks and rates using consistent methods and data. We hypothesized that a much larger proportion of blacks are at high risk of CVD than whites and hence that the disparity in high-risk subgroup is responsible for a large part of disparity in event rates between races.

METHODS

Overview

We estimated the effects of 3 types of interventions on CVD risk and events and their disparities between blacks and whites: population-wide interventions (alone or in combination), interventions to lower risk-factor level among individuals with high levels for a single risk factor, and a risk-based intervention that targeted individuals with high predicted 10-year CVD risk and treated several risk factors simultaneously (Table 1[15,17–28]). We first estimated the 10-year risk and events of both fatal and fatal and nonfatal coronary heart disease (CHD) or stroke for a representative sample of blacks and whites in the United States. Risks were predicted on the basis of systolic blood pressure (SBP), serum total cholesterol (TC), diabetes mellitus, and smoking with the use of risk prediction equations that were recalibrated for each age-sex-race group.[29] We then assessed how each intervention changed the predicted risk and events for each age-sex-race group.

Data on Risk Factors

We used data on risk factors from 7 rounds of the NHANES (National Health and Nutrition Examination Survey) 1999 to 2012 to have stable estimates for each age-sex-race subgroup. We included black or white participants who were 50 to 69 years old and did not have a history of CHD or stroke. We excluded participants ≥70 years of age to focus on the age range commonly considered for premature event and mortality.

We accounted for complex survey design to make estimates of risk factor, predicted risk, and events representative of the national population. We used TC as opposed to low-density lipoprotein cholesterol because low-density lipoprotein cholesterol was measured in only half of the participants. Diabetes mellitus was defined as having a fasting plasma

Table 1. Selected Risk Factors, Their Exposure Metrics, and Examples of Population-Wide, Single Raised Risk Factor, and Risk-Based Interventions

Risk Factors	Exposure Metric (Unit)	Population-Wide Interventions	Single Raised Risk Factor Interventions	Multiple Population-Level Interventions	Risk-Based Interventions
High blood pressure	SBP (mm Hg)	Reducing salt intake in packaged and prepared food*	2 Antihypertensive drugs at standard dose if diabetic or SBP ≥140 mm Hg for nondiabetic adults <60 y of age or SBP ≥150 mm Hg for nondiabetic adults ≥60 y of age†	Multiple risk factor intervention at the population level, including reducing salt intake, dietary improvement, tobacco control, and increasing the price of sugar-sweetened beverages	Multiple risk factor intervention, including antihypertensive and lipid-lowering medications, smoking cessation, and lifestyle modification if 10-y risk of fatal CVD ≥2.5% (or total CVD risk ≥7.5%)‡
High serum cholesterol	Serum TC (mmol/L)	Community-based dietary improvement to reduce dairy fat and replace saturated with unsaturated fats and to increase vegetable and fruit consumption§	High-intensity statin if 10-y ASCVD risk ≥7.5%, or LDL cholesterol ≥4.9 mmol/L, moderate-intensity statin if diabetic 40–75 y of age and 10-y ASCVD risk <7.5% ‖		
Tobacco smoking	Current smoker prevalence (percentage)	Tobacco control package to ban smoking in indoor workplaces, offer cessation treatment in general stores, put warning on packages, ban advertisements, and increase tobacco tax¶	Referral to smoking cessation intervention such as group behavioral therapy#		
Diabetes mellitus	Diabetes prevalence (percentage)	Increase in price of sugar-sweetened beverages**	Intensive lifestyle modification intervention if diabetic††		

ASCVD indicates atherosclerotic cardiovascular disease; CVD, cardiovascular disease; LDL, low-density lipoprotein; SBP, systolic blood pressure; and TC, total cholesterol.

*We assumed a moderate salt reduction of about 1.4 g/d based on the United Kingdom's successful experience in reducing salt intake at the population level.[17] The effects of modest sodium reduction on blood pressure vary significantly by age, race and hypertensive status. We calculated the SBP reduction using the regression equation from a large meta-analysis of >100 randomized, controlled trials.[18]

†This intervention is based on the clinical guideline from the Eighth Joint National Committee.[25] We calculated the SBP reduction by 0.09×P–5.67, where P is the pretreatment SBP value (mm Hg).[26]

‡The multiple risk factor intervention included 2 antihypertensive drugs at standard dose, statin treatment, referral to smoking cessation intervention and lifestyle-modification intervention if the 10-year risk of fatal CVD ≥2.5% (or fatal and nonfatal CVD risk ≥7.5%).

§The dietary improvement intervention is based on the success of the North Karelia Project in Finland, which includes substituting vegetable oil margarine for butter, nonfat or low-fat milk for fatty milk, and lean meat for meat high in saturated fat; using vegetable oil for cooking; and increasing vegetable and fruit consumption. We assumed that such an intervention would reduce the population mean TC by 0.5 mmol/L, the average decline over 5 years in Finland.[19] Similar dietary intervention was also found to improve the population mean TC level in New Zealand.[20]

‖This intervention is based on the 2013 American College of Cardiology/American Heart Association guidelines for the treatment of cholesterol to reduce atherosclerotic cardiovascular risk in adults.[15] We used atorvastatin 80 mg/d as high-intensity statin and atorvastatin 20 mg/d for moderate-intensity statin.

¶The tobacco control package is based on the World Health Organization MPOWER tobacco control policies, which include banning smoking in all indoor workplaces, providing nicotine replacement therapy and bupropion in general stores or pharmacies with prescription, putting bold and graphic warning to cover at least 50% of the package, banning all direct advertisements, and increasing the retail price of cigarette by 10% in taxes. We assumed a 11% reduction in smoking prevalence based on a previous policy evaluation report.[21]

#Evidence suggested that group behavioral therapy is one of the most effective strategies for smoking cessation, with a risk ratio of 2.71, which translates into 13% reduction in smoking prevalence.[27]

**We modeled a 50% increase in price of sugar-sweetened beverages, which we estimated would reduce consumption by ≈50% on the basis of the experience in Mexico, where raising prices by 10% decreased consumption by 12%.[22] Halving consumption in the United States would translate into a 0.5-serving/d reduction because average sugar-sweetened beverage intake in adults is ≈1 serving per day.[23] Evidence shows that consumption of 1 to 2 servings per day of sugar-sweetened beverage compared with none or <1 serving/mo is associated with 26% increased risk of type 2 diabetes mellitus.[24] Therefore, a 50% increase in the price of sugar-sweetened beverages is associated with ≈12% decreased risk of type 2 diabetes mellitus with a linear effect on log scale.

††The lifestyle modification intervention included a healthy low-calorie, low-fat diet and physical activity of moderate intensity such as brisk walking for at least 150 min/wk. The effect of lifestyle intervention is based on the Diabetes Prevention Intervention, [28] which reduces the diabetes incidence by 58%.

glucose ≥126 mg/dL, hemoglobin A_{1c} ≥6.5%, history of diagnosis by a health professional, or use of insulin or oral hypoglycemic agents.

Data on CVD Deaths

In our primary analysis, we used fatal CVD as the outcome because data on nonfatal events, which are required for risk equation recalibration, are not available for the national US population (see below for methods to estimate fatal and nonfatal rates by race). We used mortality data from the National Center for Health Statistics to calculate death rates in 2011. We defined CVD death as death resulting from CHD (*International Classification of Diseases, 10th Revision* codes I20–I25) or stroke (codes I60–I69).

Effect Sizes for Interventions

We obtained the effects of interventions on risk factors from meta-analyses of randomized controlled trials, observational studies, or policy evaluation analyses, as detailed in Table 1. We used a larger effect size for the impact of salt reduction on blood pressure among blacks versus whites[18] but used the same effect size between blacks and whites for all other interventions because proportional effects have been found to be generally similar by race.[30–33] Under the risk-based intervention scenario, we used individuals' absolute CVD risks to determine whether they were affected by the interventions and assigned interventions (eg, antihypertensive and statin treatments) only to individuals who were not already receiving them. We applied smoking cessation to smokers regardless of their absolute CVD risks. We note that the level of evidence supporting interventions varies; for example, the impact of population-wide interventions has been estimated only in observational studies,[18,21,24] whereas the effect of statins on CVD has been consistently shown in many randomized trials.[34] We also note that an individual may receive both population-wide and targeted interventions in practice, although these 2 types of interventions were analyzed separately here.

Statistical Analysis

We used risk prediction equations (or risk scores) for fatal CVD and for total CVD developed from 8 prospective cohorts in the United States, as described elsewhere.[29] Briefly, the models use 4 inputs to estimate individual-level 10-year risk: the participants' risk factor levels, coefficients (ie, log hazard ratios) for each risk factor estimated from the cohorts, mean risk factor level for the same age-sex-race subgroup as the index participant, and average CVD event rate for the same age-sex-race subgroup as the index participant. The risk factors in the model were SBP, TC, diabetes mellitus, and smoking. We used this new risk predication equation because it is based on data from multiple cohorts, allows a straightforward recalibration by sex and race, allows the age pattern of CVD risk to vary across race-sex subgroups, and includes interactions between age and SBP, TC, diabetes mellitus, and smoking and interaction between sex and diabetes mellitus to account for the fact that the proportional effects of these risk factors on CVD vary by age and sex.[35–39] For this application, we modified the risk scores to separate current from former smokers. The coefficients of the risk scores and the validation methods and results are presented in Tables I and II in the online-only Data Supplement. We assumed the same proportional associations between risk factors and fatal CVD risk for blacks and whites on the basis of previous evidence.[30–33] We relaxed this assumption by using race-specific coefficients in a sensitivity analysis (Table III in the online-only Data Supplement).

In the primary analysis, we first recalibrated the risk score by replacing the CVD event rate and mean risk factor levels with the observed age-sex-race–specific rates in the US population. We then used the recalibrated risk score and individual-level data from NHANES to estimate the 10-year risk of fatal CVD for each participant under the current risk factor levels. We report the mean predicted risk and number of events and their relative or absolute differences between blacks and whites. We also present how the population and events were distributed by risk level in each sex-race subgroup. We further report the proportions of population and events at fatal CVD risk ≥2.5%, hereafter referred to as moderate risk, and ≥6.67%, hereafter referred to as high risk. Because almost one-third of CVD events are fatal in the United States,[40] these risk thresholds approximately correspond to ≥7.5% (the American College of Cardiology/American Heart Association threshold[15]) and ≥20% (the Adult Treatment Panel III threshold[16]) for fatal and nonfatal CVD. In our secondary analysis, we used fatal and nonfatal CVD events as outcome. We calculated the age-sex-race–specific event rate of fatal and nonfatal CVD (CHD and stroke) using the corresponding death rate multiplying by the race-specific ratio of total to fatal events. We used the ratios of total to fatal events for CHD and stroke as reported in the REGARDS (Reasons for Geographic and Racial Differences in Stroke) cohort[40,41] to account for the higher case fatality rates in blacks. We reported the proportions of population and events at total CVD risk ≥7.5% and ≥20%. In a sensitivity analysis, we calculated the 10-year CVD risk using the American College of Cardiology/American Heart Association 2013 Pooled Cohort Equations.[42]

To estimate the effects of interventions on CVD risk and events, we first estimated their effects on risk factor(s) and then recalculated the 10-year risk and events using the postintervention risk factor levels. We chose this approach instead of directly applying the impact of interventions on CVD risk because for many of the interventions analyzed here, the outcome of epidemiologic studies is risk factor level. For example, we estimated the impact of reducing the incidence of diabetes mellitus from the Diabetes Prevention Program and combined that with evidence on the effect of diabetes mellitus on CVD from meta-analyses of observational studies.[28,35] Because no studies have shown a direct impact of diabetes prevention on CVD mortality, we conducted a separate analysis by removing diabetes prevention from the risk-based multiple risk factor intervention.

We quantified uncertainty by sampling repeated draws of different inputs to analysis, as described in the online-only Data Supplement. All analyses were conducted with Stata 12.0 (StataCorp, College Station, TX) and R 3.02. The study was approved by the institutional review board of the Harvard School of Public Health (Boston, MA).

RESULTS

We included 6154 blacks and whites from 7 rounds of NHANES (Figure I in the online-only Data Supplement). About one-third of participants were black. TC levels

were similar between blacks and whites, whereas other risk factor levels were higher in blacks (Table IV in the online-only Data Supplement).

Mean 10-year risk of fatal CVD was 5.1% in black men versus 3.4% in white men (risk ratio, 1.49) and 3.0% in black women versus 1.7% in white women (risk ratio, 1.79). This was equivalent to a 10-year CVD death rate of 5052 per 100 000 in black men versus 3393 in white men (rate difference, 1659 per 100 000) and 2989 in black women versus 1669 in white women (rate difference, 1320).

The distributions of both population and events by 10-year risk of fatal CVD were shifted to the right among blacks compared with whites; the distribution of events had a heavier tail than that of population because most of the events arise from the high-risk individuals (Figure 1). As a result, 25% (95% confidence interval [CI], 22–28) of black men were at high risk (≥6.67% risk of fatal CVD in 10 years) compared with only 10% (95% CI, 8–12) of white men (Table 2 and Figure II in the online-only Data Supplement). This high-risk subgroup accounted for 55% (95% CI, 49–59) of CVD deaths in black men compared with 30% (95% CI, 24–35) in white men. For women, 12% (95% CI, 10–14) of blacks versus 3% (95% CI, 2–4) of whites at high risk accounted for 42% (95% CI, 35–46) of CVD deaths in blacks versus 18% (95% CI, 13–22) in whites. Compared with the results of fatal CVD, black-white disparities in total CVD were substantially smaller for men (Figure 2, Table 3, and Figure III in the online-only Data Supplement). In women, disparities were only noticeable for those with ≥7.5% CVD risk; 30% (95% CI, 27–33) of black women versus 19% (95% CI, 18–21) of white women accounted for 61% (95% CI, 57–63) of CVD events in blacks versus 46% (95% CI, 44–49) in whites.

Population-wide interventions (ie, salt reduction, improving diet, World Health Organization's MPOWER tobacco control policies, increasing price of sugar-sweetened beverages) and targeted interventions on single risk factors (ie, antihypertensive and statin treatments, referral for quitting smoking, diabetes prevention program) were estimated to reduce the 10-year CVD death rate by at most 440 per 100 000 in men and 290 in women. The risk-based multiple risk factor intervention was estimated to reduce the average CVD death rate by 1086 per 100 000 in men and 669 in women for

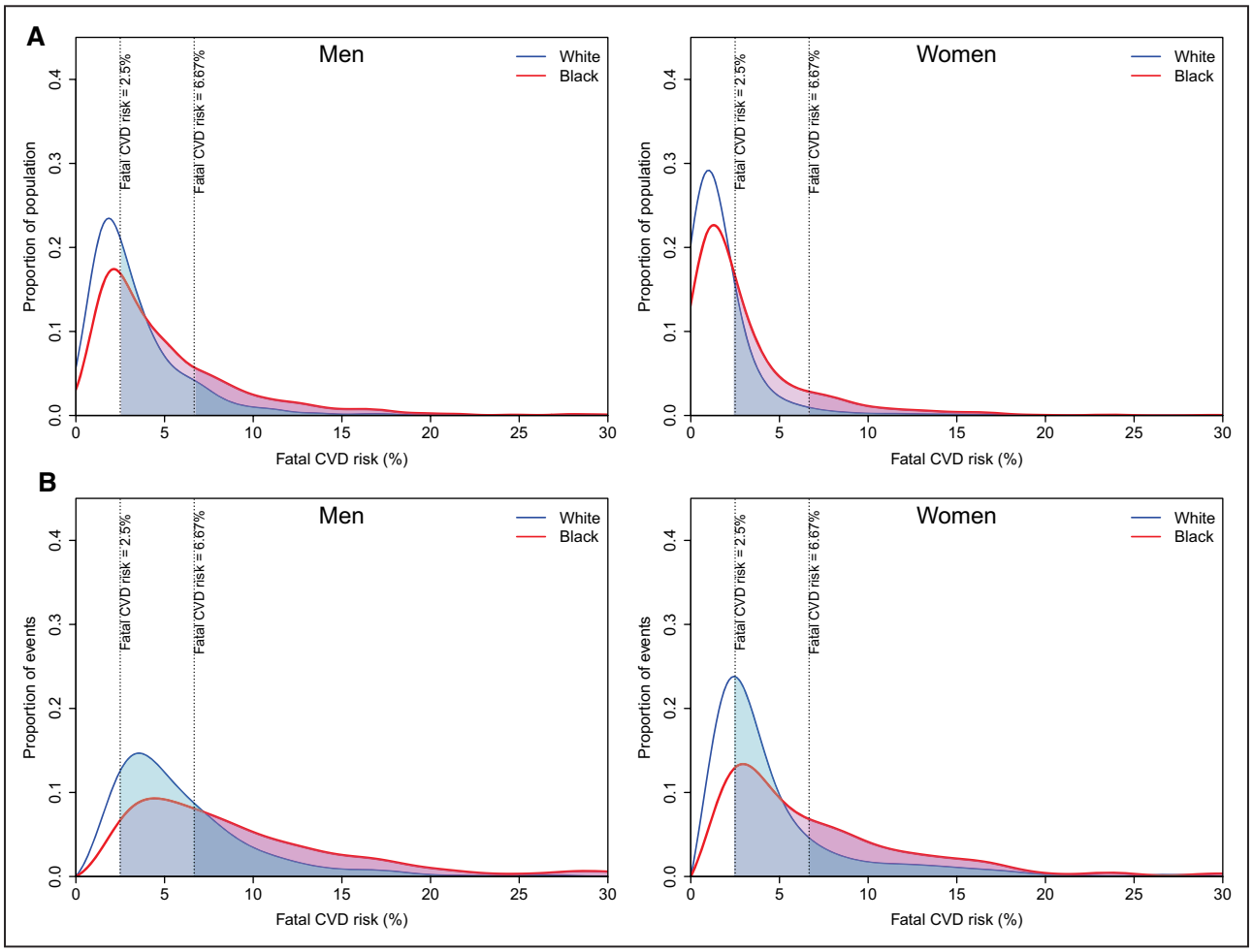

Figure 1. Distributions of predicted 10-year risk of fatal cardiovascular disease (CVD) in the population (A) and among cases (B).

Table 2. Proportion of Population and Proportion of Fatal CVD Events Occurring Among High-Risk Individuals by Sex and Race

	Fatal CVD Risk ≥2.5%*		Fatal CVD Risk ≥6.67%*	
	Proportion of Population, %	Proportion of Events, %	Proportion of Population, %	Proportion of Events, %
Men				
White	50 (47–52)	77 (75–78)	10 (8–12)	30 (24–35)
Black	66 (62–69)	88 (87–90)	25 (22–28)	55 (49–59)
Women				
White	17 (15–18)	49 (46–52)	3 (2–4)	18 (13–22)
Black	36 (33–39)	74 (72–76)	12 (10–14)	42 (35–46)

CVD indicates cardiovascular disease.

*These risk thresholds are approximately equal to ≥7.5% (the American College of Cardiology/American Heart Association threshold[15]) and ≥20% (the Adult Treatment Panel III threshold[16]) for fatal and nonfatal CVD because almost one-third of CVD events are fatal in the United States.[40]

blacks, and 671 per 100 000 in men and 246 in women for whites (Tables 4 and 5).

Population-wide interventions and targeted interventions on single risk factors did not reduce substantially the proportion of population at high risk of fatal CVD (≥6.67% risk of fatal CVD in 10 years) in either whites or blacks (Figure 3). In contrast, the risk-based multiple risk factor intervention was estimated to reduce

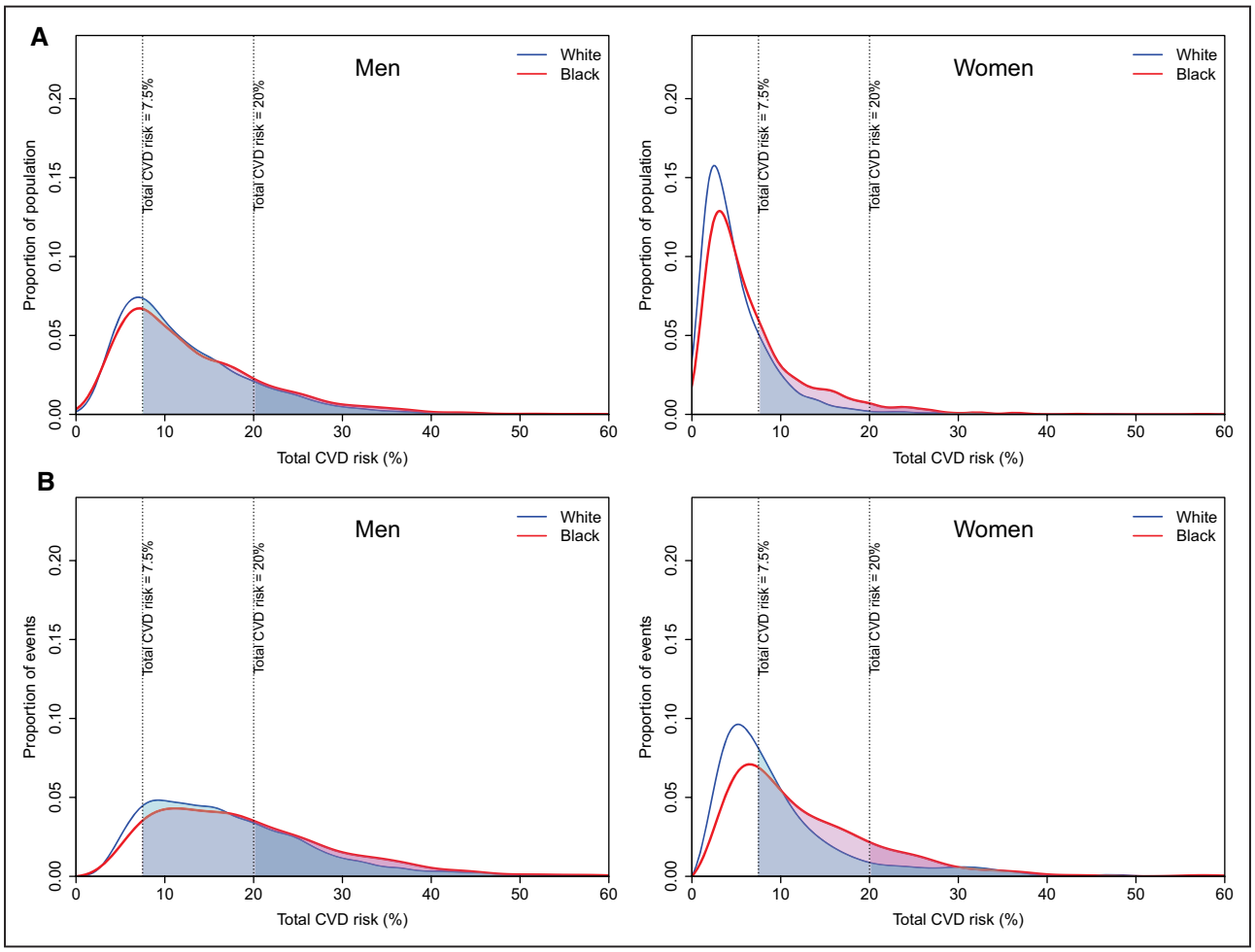

Figure 2. Distributions of predicted 10-year risk of fatal and nonfatal cardiovascular disease (CVD) in the population (A) and among cases (B).

Table 3. Proportion of Population and Proportion of Fatal and- Nonfatal CVD Events Occurring Among High-Risk Individuals by Sex and Race

	Fatal and Nonfatal CVD Risk ≥7.5%		Fatal and Nonfatal CVD Risk ≥20%	
	Proportion of Population, %	Proportion of Events, %	Proportion of Population, %	Proportion of Events, %
Men				
White	68 (66–71)	86 (85–87)	15 (13–16)	32 (29–34)
Black	70 (66–73)	87 (86–89)	18 (15–20)	37 (32–40)
Women				
White	19 (18–21)	46 (44–49)	2 (1–3)	10 (6–13)
Black	30 (27–33)	61 (57–63)	4 (3–5)	16 (12–19)

CVD indicates cardiovascular disease.

the proportion of high-risk population by at most 12 percentage points for men and 6 percentage points for women. Results were similar for the moderate-risk group (≥2.5% risk of fatal CVD in 10 years), for which the risk-based multiple risk factor intervention was estimated to reduce the moderate- or high-risk proportion by at most 13 percentage points for men and 9 percentage points for women compared with at most 6

Table 4. Impact of Interventions on 10-Year Rate of Fatal CVD Among Men by Race

	10-y CVD Death Rate, per 100 000		Rate Ratio (Black vs White)	Rate Difference (Black minus White), per 100 000	Absolute Change in Rate Difference, per 100 000	Relative Change in Rate Difference, %
	Black	White				
Current, n	5052	3393	1.49	1659	NA	NA
Population-wide interventions						
Salt reduction	4939	3329	1.48	1610	–49	–3
Community-based dietary improvement to reduce serum cholesterol	4906	3290	1.49	1616	–44	–3
MPOWER package for smoking	4939	3356	1.47	1583	–77	–5
Increasing price of sugar-sweetened beverages for diabetes prevention	4972	3362	1.48	1609	–50	–3
Multiple interventions (all the above)	4612	3151	1.46	1461	–198	–12
Single raised risk factor interventions						
Treatment for hypertension	4885	3290	1.48	1595	–64	–4
Treatment for dyslipidemia	4687	3143	1.49	1544	–116	–7
Referral for quitting smoking	4875	3342	1.46	1533	–126	–8
Diabetes prevention program	4704	3187	1.48	1517	–142	–9
Risk-based interventions						
Multiple risk factors (all the single raised risk factor interventions if risk of CVD death ≥2.5%)*	3966	2722	1.46	1244	–415	–25
Multiple risk factors (all the single raised risk factor interventions except diabetes prevention)	4296	2882	1.49	1414	–246	–15

CVD indicates cardiovascular disease; MPOWER, World Health Organization MPOWER tobacco control policies, which include banning smoking in all indoor workplaces, providing nicotine replacement therapy and bupropion in general stores or pharmacies with prescription, putting bold and graphic warning to cover at least 50% of the package, banning all direct advertisements, and increasing the retail price of cigarette by 10% in taxes.

*The eligibility for each intervention was defined on the basis of risk of CVD death ≥2.5% except for smoking cessation. Smoking cessation was provided to all current smokers regardless of his/her predictive risk.

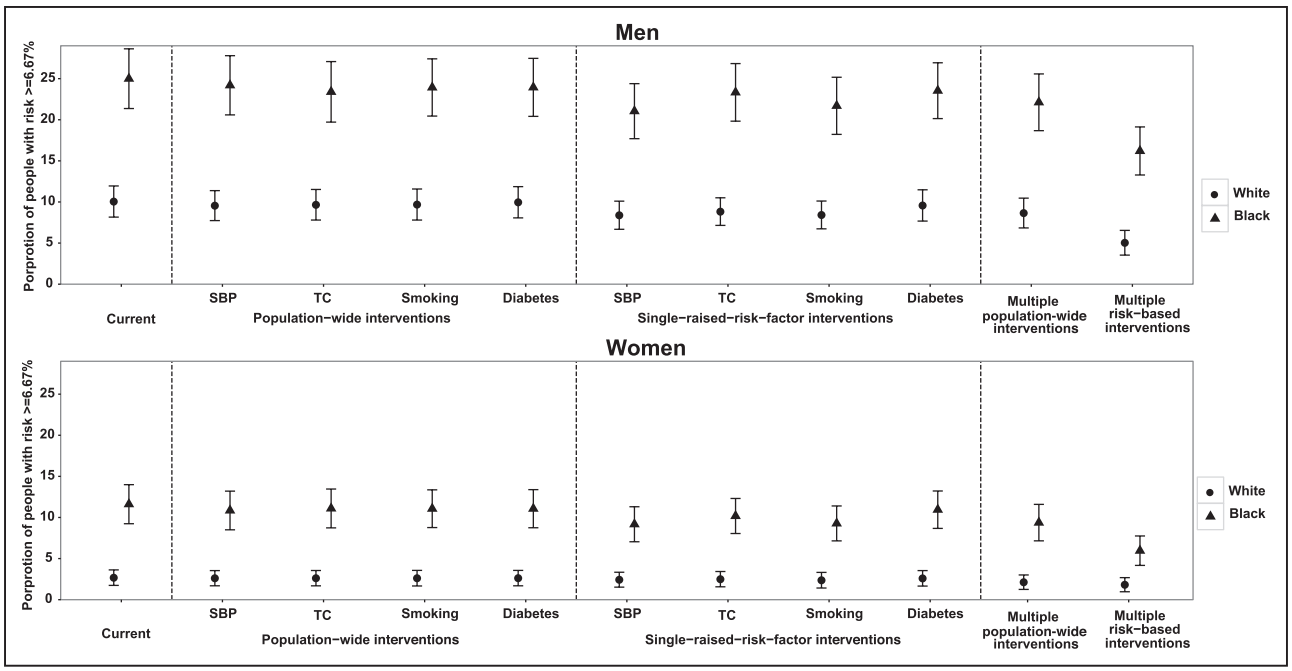

Figure 3. Impact of population-wide, single raised risk factor, and risk-based interventions on proportion of population with ≥6.67%* 10-year risk of fatal cardiovascular disease (CVD).

CI indicates confidence interval; SBP, systolic blood pressure; and TC, total cholesterol. *This threshold approximately equals to ≥20% for risk of fatal and nonfatal CVD given that one third of CVD events are fatal in the United States.[40]

and 4 percentage points in population-wide or targeted interventions (Figure 4). Our sensitivity analysis using separate fatal CVD risk scores for blacks and whites showed similar results (Figures IV and V in the online-only Data Supplement).

None of the interventions analyzed here had a potential to reduce black versus white fatal CVD rate ratios (Tables 4 and 5). When we considered disparities in absolute CVD rates, combining the 4 selected population-wide interventions was estimated to reduce black-white

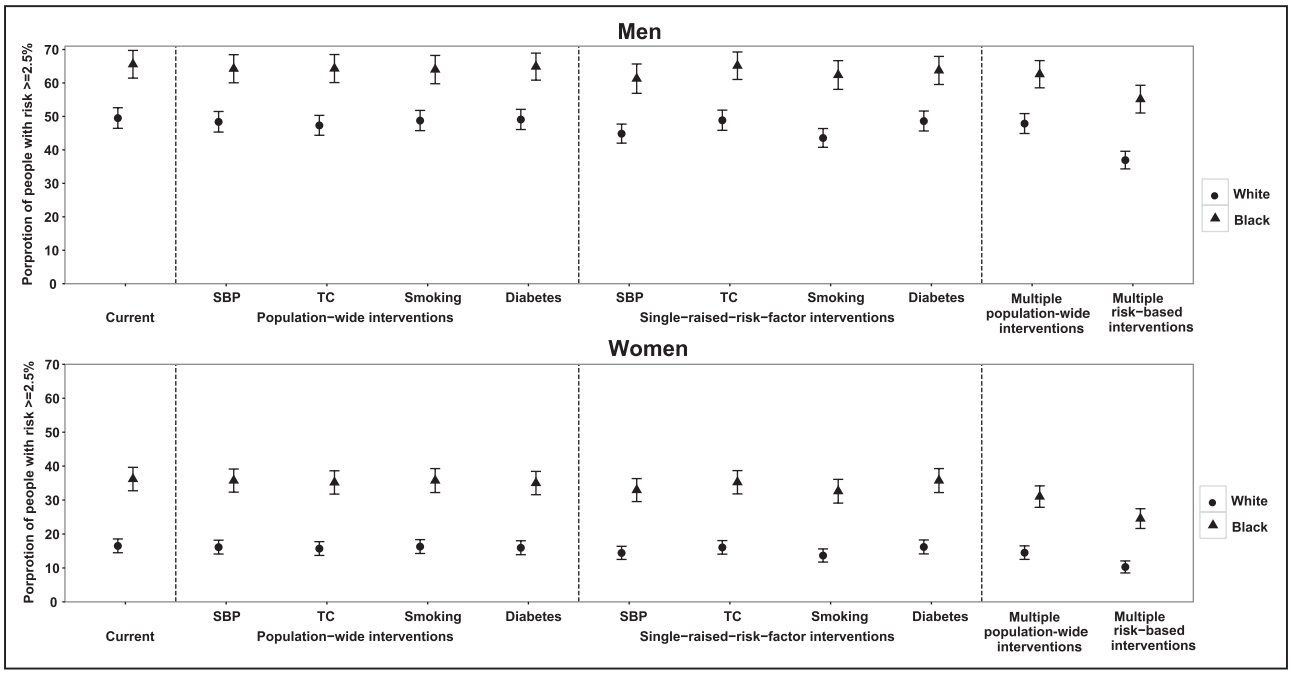

Figure 4. Impact of population-wide, single raised risk factor, and risk-based interventions on proportion of population with ≥ 2.5%* 10-year risk of fatal cardiovascular disease (CVD).

CI indicates confidence interval; SBP, systolic blood pressure; and TC, total cholesterol. *This threshold approximately equals to ≥7.5% for fatal and nonfatal CVD given that one third of CVD events are fatal in the United States.[40]

Table 5. Impact of Interventions on 10-Year Rate of Fatal CVD Among Women by Race

	10-y CVD Death Rate, per 100 000		Rate Ratio (Black vs White)	Rate Difference (Black minus White), per 100 000	Absolute Change in Rate Difference, per 100 000	Relative Change in Rate Difference, %
	Black	White				
Current, n	2989	1669	1.79	1320	NA	NA
Population-wide interventions						
Salt reduction	2945	1644	1.79	1301	–19	–1
Community-based dietary improvement to reduce serum cholesterol	2893	1618	1.79	1276	–45	–3
MPOWER package for smoking	2934	1647	1.78	1287	–34	–3
Increasing price of sugar-sweetened beverages for diabetes prevention	2931	1651	1.77	1280	–41	–3
Multiple interventions (all the above)	2729	1549	1.76	1179	–141	–11
Single raised risk factor interventions						
Treatment for hypertension	2906	1629	1.78	1277	–43	–3
Treatment for dyslipidemia	2731	1554	1.76	1177	–144	–11
Referral for quitting smoking	2926	1643	1.78	1283	–38	–3
Diabetes prevention program	2699	1551	1.74	1148	–173	–13
Risk-based interventions						
Multiple risk factors (all the single raised risk factor interventions if risk of CVD death ≥2.5%)*	2320	1423	1.63	897	–423	–32
Multiple risk factors (all the single raised risk factor interventions except diabetes prevention)	2601	1495	1.74	1106	–214	–16

CVD indicates cardiovascular disease; MPOWER, World Health Organization MPOWER tobacco control policies, which include banning smoking in all indoor workplaces, providing nicotine replacement therapy and bupropion in general stores or pharmacies with prescription, putting bold and graphic warning to cover at least 50% of the package, banning all direct advertisements, and increasing the retail price of cigarette by 10% in taxes.

*The eligibility for each intervention was defined on the basis of risk of CVD death ≥2.5% except for smoking cessation. Smoking cessation was provided to all current smokers regardless of his/her predictive risk.

disparities by 198 per 100 000 (12% of total absolute disparity) in men and 141 (11%) in women.

Among targeted single-risk interventions, the diabetes prevention program had the largest potential, with an estimated reduction in absolute disparity by 142 (9%) in men and 173 (13%) in women. The risk-based multiple risk factor intervention had much larger potential and could reduce absolute disparities by 415 per 100 000 (25%) in men and 423 (32%) in women. Removing diabetes prevention from the risk-based multiple risk factor intervention reduced the estimated impact of risk-based intervention by 41% to 50%, but this intervention still had the largest potential for reducing absolute black-white disparities.

For fatal and nonfatal CVD rates, there were no significant disparities between blacks and whites in men (Table 6). The estimated black-white disparities in women could be reduced by 217 per 100 000 (13% of total disparity in absolute risk) through a combination of 4 population-wide interventions. Implementing a diabetes prevention program alone was estimated to reduce disparities by 412 (24%) and the risk-based multiple risk factor intervention by 491 (29%; Table 7). The sensitivity analysis using American College of Cardiology/American Heart Association 2013 Pooled Cohort Equations also showed results consistent with the main analysis (Tables V and VI in the online-only Data Supplement).

DISCUSSION

We found that a substantially larger proportion of blacks (25% of men and 12% of women) in the United States had a high risk of fatal CVD than their white counterparts (10% of men and 3% of women). These high-risk individuals bore about half of the burden of fatal CVD events in the population. An intervention that could identify high-risk individuals and treat multiple risk factors could both deliver large total benefits and substantially

Table 6. Impact of Interventions on 10-Year Rate of Fatal and Nonfatal CVD Among Men by Race

	10-y CVD Event Rate, per 100 000		Rate Ratio (Black vs White)	Rate Difference (Black minus White), per 100 000	Absolute Change in Rate Difference, per 100 000	Relative Change in Rate Difference, %
	Black	White				
Current	13 082	12 343	1.06	739	NA	NA
Population-wide interventions						
Salt reduction	12 881	12 189	1.06	691	–48	–6
Community-based dietary improvement to reduce serum cholesterol	12 703	11 965	1.06	739	–1	0
MPOWER package for smoking	12 852	12 224	1.05	629	–110	–15
Increasing price of sugar-sweetened beverages for diabetes prevention	12 962	12 264	1.06	697	–42	–6
Multiple interventions (all the above)	12 204	11 613	1.05	590	–149	–20
Single raised risk factor interventions						
Treatment for hypertension	12 829	12 144	1.06	686	–54	–7
Treatment for dyslipidemia	12 147	11 347	1.07	800	61	8
Referral for quitting smoking	12 816	12 190	1.05	626	–113	–15
Diabetes prevention program	12 449	11 868	1.05	581	–158	–21
Risk-based interventions						
Multiple risk factors (all the single raised risk factor interventions if risk of fatal and nonfatal CVD ≥7.5%)*	10 791	10 246	1.05	546	–193	–26
Multiple risk factors (all the single raised risk factor interventions except diabetes prevention)	11 430	10 654	1.07	776	37	5

CVD indicates cardiovascular disease; MPOWER, World Health Organization MPOWER tobacco control policies, which include banning smoking in all indoor workplaces, providing nicotine replacement therapy and bupropion in general stores or pharmacies with prescription, putting bold and graphic warning to cover at least 50% of the package, banning all direct advertisements, and increasing the retail price of cigarette by 10% in taxes.

*The eligibility for each intervention was defined on the basis of risk of total CVD ≥7.5% except for smoking cessation. Smoking cessation was provided to all current smokers regardless of predicted risk.

reduce the absolute black-white disparities. Population-wide and targeted interventions on single risk factors had smaller potential on reducing racial disparities in CVD compared with a risk-based intervention on multiple risk factors. Total CVD risks were similar in black and white men, and the disparity between black and white women could be substantially reduced by a risk-based multiple risk intervention.

Our results on the disparities in risk factor exposure and in their role as a cause of racial disparities in CVD are consistent with those of previous analyses.[1,3,4,9,11,13] A previous study proposed that population-wide interventions have a larger effect on health disparities than interventions that target high-risk individuals, but the 2 scenarios were only qualitatively compared.[10] Other studies quantified the effects of hypothetical risk factor reductions on disparity in mortality without considering specific interventions.[3,4,9,11,13] In addition, previous studies often used a single risk factor to identify high-risk individuals and considered interventions on 1 risk factor at a time.[11,43]

A key strength of our analysis is that we have assessed not only the aggregate risk and events within each group but also how risk and events were distributed, providing important information on who needs intervention and what the expected impact of intervention is. In addition, we compared the total and disparity impacts of a wide range of population-wide and targeted interventions using consistent methods and data. Risk factor distributions were from a nationally representative survey; mortality data were from a vital registration system; and effect sizes for interventions were obtained from large meta-analyses of randomized trials or observational studies that had adjusted for important confounders. We also systematically quantified the uncertainty as a result of the sampling variability in the national surveys and the uncertainty of coefficients from the risk prediction equations. Finally, our primary model included age interaction between risk factors and CVD, incorporating evidence from many prospective studies.[35]

Our study has some limitations. First, although we estimated the risk distributions for both fatal CVD and

Table 7. Impact of Interventions on 10-Year Rate of Fatal and Nonfatal CVD Among Women by Race

	10-y CVD Event Rate, per 100 000		Rate Ratio (Black vs White)	Rate Difference (Black minus White), per 100 000	Absolute Change in Rate Difference, per 100 000	Relative Change in Rate Difference, %
	Black	White				
Current	6868	5179	1.33	1688	NA	NA
Population-wide interventions						
Salt reduction	6791	5123	1.33	1668	–20	–1
Community-based dietary improvement to reduce serum cholesterol	6645	5011	1.33	1634	–55	–3
MPOWER package for smoking	6748	5144	1.31	1604	–84	–5
Increasing price of sugar-sweetened beverages for diabetes prevention	6736	5151	1.31	1585	–104	–6
Multiple interventions (all the above)	6358	4886	1.30	1471	–217	–13
Single raised risk factor interventions						
Treatment for hypertension	6739	5106	1.32	1633	–56	–3
Treatment for dyslipidemia	6329	4819	1.31	1510	–179	–11
Referral for quitting smoking	6719	5130	1.31	1589	–99	–6
Diabetes prevention program	6290	5013	1.25	1277	–412	–24
Risk-based interventions						
Multiple risk factors (all the single raised risk factor interventions if risk of fatal and nonfatal CVD ≥7.5%)*	5740	4542	1.26	1197	–491	–29
Multiple risk factors (all the single raised risk factor interventions except diabetes prevention)	6135	4683	1.31	1452	–236	–14

CVD indicates cardiovascular disease; MPOWER, World Health Organization MPOWER tobacco control policies, which include banning smoking in all indoor workplaces, providing nicotine replacement therapy and bupropion in general stores or pharmacies with prescription, putting bold and graphic warning to cover at least 50% of the package, banning all direct advertisements, and increasing the retail price of cigarette by 10% in taxes.

*The eligibility for each intervention was defined on the basis of risk of total CVD ≥7.5% except for smoking cessation. Smoking cessation was provided to all current smokers regardless of predicted risk.

total CVD, reliable national data on total CVD incidence are not available for model recalibration, especially by race. Recent data from a large prospective cohort (REGARDS) show that black men have higher incidence of fatal CHD and lower incidence of nonfatal CHD than white men, resulting in similar incidences of total CHD for black and white men.[40] Using estimates of case fatality rates from REGARDS to recalibrate model for total CVD risk eliminated much of the racial disparity in total CVD risk; thus, it is expected that the interventions evaluated here would have minimal impact on racial disparities in total CVD risk. Second, our analysis focused on primary prevention of CVD. However, patients with a history of CVD have a high risk of subsequent cardiovascular events and should receive treatments for risk factors. In the United States, 9% of blacks and 6% of whites 50 to 69 years of age in the 2011 to 2012 NHANES survey had a history of CVD. Were these proportions to be added to our estimates of prevalence of high-risk status, disparities would be slightly larger than our estimates. Our analysis did not include patients with CVD because existing risk scores for these patients require data on predictors such as electrocardiography results, coronary imaging, and biomarkers that are not measured in NHANES.[44,45] Third, we assumed that compliance with interventions would be similar to that observed in the randomized trials and observational studies used to generate the intervention effects, which may lead to overestimation of the impact of interventions on black-white disparities. Although compliance may vary by race, prior work suggested that noncompliance is likely due to barriers to access to and poor quality of health care.[46,47] If insurance coverage and healthcare quality were similar across races, it is unlikely that compliance would differ substantially, as has been observed for antiretroviral therapy.[48] Fourth, smoking cessation interventions have been shown to affect disadvantaged populations more strongly. However, detailed data on the differential impacts of smoking cessation by

race are not available. Therefore, our estimates for the impact of smoking cessation on black-white disparities in CVD risk should be considered conservative. Fifth, there is limited evidence on the direct impact of diabetes prevention on CVD, and it remains unclear whether the Diabetes Prevention Program prevents or delays the onset of diabetes mellitus in healthy individuals. Our sensitivity analyses of removing diabetes prevention from the risk-based multiple risk factor intervention confirmed that the largest impact on reducing the absolute back-white disparities still came from the risk-based intervention. Finally, the effects of some interventions (eg, reducing salt in package food, World Health Organization's MPOWER tobacco control policies) may be cumulative over decades. Our analyses did not incorporate the cumulative effects and hence may underestimate the long-term effect of these interventions.

CONCLUSIONS

Although prevention and treatment have helped reduce CVD rates over the past few decades in the United States, mortality rates remain higher in blacks than whites.[1,2,40] Eliminating racial disparities in health is one of the overarching goals of the Healthy People 2020 agenda.[49] Because disparities in CVD are caused by disparities in broader social, economic, and environmental determinants, policies and strategies are needed to address these factors and to facilitate healthy lifestyle and environment. Meanwhile, our findings suggest that a much larger proportion of blacks are at high risk of fatal CVD than whites and that this high-risk subpopulation is bearing almost half of the deaths in the population. Therefore, by targeting this sick subpopulation with combination risk-based therapy, we can reduce a large share of events. Such an approach has been advocated for the US population as a whole.[15] However, achieving its potential as a means to reduce racial disparities will require increasing health insurance coverage and a strong primary care system that is equipped with well-trained health workers and appropriate infrastructure to provide low-cost essential drugs. Previous research has shown that universal health insurance for individuals >65 years of age in the United States is associated with lower racial differences in cardiovascular risk factors.[50] An accessible and high-quality primary care program also has successfully reduced cardiovascular health inequality in other countries.[51] The window of opportunity for addressing cardiovascular health disparity lies in the Affordable Care Act of 2010,[52] which has already shown promise in improving access to primary care services[53] and commits to eliminate barriers to health for disadvantaged communities, along with the new guideline for risk-based multidrug treatment for CVD.[15] Their intersection could help identify important opportunities to improve the access and affordability of risk-based treatment for CVD in underserved population and finally improve cardiovascular health for all.

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None.

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FOOTNOTES

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Sick Populations and Sick Subpopulations: Reducing Disparities in Cardiovascular Disease Between Blacks and Whites in the United States

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SUPPLEMENTAL MATERIAL

Sick populations and sick subpopulations: reducing disparities in cardiovascular disease between blacks and whites in the United States

Supplemental Text

Quantifying the uncertainty of prevalence of high CVD risk

There were two sources of uncertainty in predicted prevalence of high CVD risk, which include 1) the sampling uncertainty in NHANES; and 2) the uncertainty of the regression coefficients from the risk prediction equation.

To capture the sampling uncertainty, we first calculated the effective sample size (ESS) for NHANES. ESS is different from the actual sample size because sampled individuals are from clusters that do not cover the whole country, thus they contain less information than they would from a random sample of the population. We systematically quantified the uncertainty of prevalence of high CVD risk using a simulation approach. In each of the 1000 simulation iteration, we randomly drew

1. N observations from NHANES (N = ESS), sampling with replacement and with each observation having the probability of being selected proportional to its sample weight. These N observations from the survey could be treated as simple random sample in terms of their representativeness and their true information content measured by ESS.
2. A set of regression coefficients from their corresponding uncertainty distributions while accounting for their correlations. We used a bootstrapping approach to draw samples of logHRs from their multivariable Normal distributions. We accounted for correlations between logHRs by incorporating variance-covariance matrix into the sampling process.

For each set of draw, we predicted the 10-year risk for each individual in the sample, and calculated the prevalence of high fatal CVD risk under current risk factor levels as well as under different population-based and targeted prevention programs. We repeated this process

1000 times and quantified the variability across all 1000 simulation estimates. We used the median of these 1000 estimates as the point estimate of the quantities of interest, and its 2.5th and 97.5th percentiles as the 95% confidence interval.

Supplemental Table 1: Coefficients of the fatal CVD risk function and corresponding hazard ratios

Variable	Coefficient (LogHR) *		HR (95% CI) †
	Main term (P value)	Age interaction term (P value)	
Systolic blood pressure (per 10 mmHg)	0.523 (<0.001)	-0.005 (<0.001)	1.24 (1.22-1.26)
Total cholesterol (per 1 mmol/L)	0.587 (<0.001)	-0.007 (<0.001)	1.17 (1.12-1.21)
Diabetes	1.715 (<0.001)	-0.013 (0.010)	2.28 (1.99-2.61)
Former smoker	-0.384 (0.290)	0.006 (0.222)	1.02 (0.90-1.15)
Current smoker	1.132 (0.003)	-0.008 (0.171)	1.87 (1.67-2.09)
Diabetes and female ‡	0.467 (<0.001)	-	1.60 (1.24-2.04)

* We validated the risk score for fatal CVD in the US general population using the first round of NHANES III (1988-1991) with linked mortality data in 2006. We used Harrell's C statistic to assess the ability of the risk score to predict shorter versus longer survival times,[1] and used a modified Hosmer-Lemeshow Chi-square test with 9 degrees of freedom to compare the predicted and observed number of events over 10 years by deciles of risk. The fatal CVD risk score performed well in the US general population. The Harrell's C statistic was 76% (95% confidence interval 71-80) and the calibration Chi-square statistic was 15.3 (degree of freedom of 9, P value of 0.08).

† Hazard ratios for systolic blood pressure, total cholesterol, diabetes, and smoking calculated at median age of CVD death, which is 66 years. Hazard ratio for diabetes is for men, and its interaction with sex shows the additional risk among women.

‡ We included an interaction for sex with diabetes based on evidence from meta-analyses of prospective cohorts.[2] Hajifathalian et al[3] also had interaction for sex with smoking, but we did not include here as the risk score has separated current from former smokers and the interactions were no longer consistently significant.

Supplemental Table 2: Coefficients of the fatal-and-nonfatal CVD risk function and corresponding hazard ratios

Variable	Coefficient (LogHR)		HR (95% CI) *
	Main term (P value)	Age interaction term (P value)	
Systolic blood pressure (per 10 mmHg)	0.307 (<0.001)	-0.002 (<0.001)	1.18 (1.16-1.19)
Total cholesterol (per 1 mmol/L)	0.618 (<0.001)	-0.007 (<0.001)	1.19 (1.16-1.22)
Diabetes	1.464 (<0.001)	-0.013 (0.001)	1.89 (1.72-2.07)
Former smoker	0.198 (0.395)	-0.017 (0.617)	1.09 (1.02-1.18)
Current smoker	1.778 (<0.001)	-0.019 (<0.001)	1.77 (1.65-1.89)
Diabetes and female †	0.391 (<0.001)	-	1.48 (1.27-1.72)

***** Hazard ratios for systolic blood pressure, total cholesterol, diabetes, and smoking calculated at median age of total CVD event, which is 64 years. Hazard ratio for diabetes is for men, and its interaction with sex shows the additional risk among women.

† We included an interaction for sex with diabetes based on evidence from meta-analyses of prospective cohorts.[2] Hajifathalian et al[3] also had interaction for sex with smoking, but we did not include here as the risk score has separated current from former smokers and the interactions were no longer consistently significant.

Supplemental Table 3: Coefficients of the race-specific fatal CVD risk function and validation results

Race	Variable	Coefficient		HR (95% CI) *
		Main component (P value)	Age interaction (P value)	
White	SBP (per 10 mmHg)	0.432 (0.001)	-0.004 (<0.001)	1.20 (1.17-1.23)
	TC (per 1 mmol/L)	0.634 (<0.001)	-0.007 (0.001)	1.18 (1.11-1.23)
	Diabetes	0.534 (<0.001)	-	1.71 (1.36-2.14)
	Former smoker	0.065 (0.427)	-	1.07 (0.91-1.26)
	Current smoker	0.693 (<0.001)	-	2.00 (1.70-2.34)
	Diabetes*sex	0.515 (0.003)	-	1.68 (1.19-2.34)
Black	SBP (per 10 mmHg)	0.580 (0.009)	-0.005 (0.107)	1.27 (1.19-1.37)
	TC (per 1 mmol/L)	0.242 (<0.001)	-	1.27 (1.13-1.43)
	Diabetes	1.176 (<0.001)	-	3.24 (2.20-4.77)
	Former smoker	0.050 (0.766)	-	1.05 (0.76-1.46)
	Current smoker	0.754 (<0.001)	-	2.12 (1.53-2.94)
	Diabetes*sex	0.198 (0.499)	-	1.22 (0.69-2.16)

		Validation	
Race	**Sex**	**Chi2 for Hosmer Lemeshow test**	**Harrell's C**
White	Men	15.8	64(60-67)
	Women	14.1	70(64-75)
Black	Men	4.9	76(69-82)
	Women	7.8	87(83-91)

***** Hazard ratios for systolic blood pressure and total cholesterol (for white only) calculated at median age of event, which is 66 years. Hazard ratio for diabetes is for men, and its interaction with sex shows the additional risk among women.

Supplemental Table 4: Age-standardized mean or prevalence (standard deviation) of systolic blood pressure, total cholesterol, diabetes and smoking by sex and race *

	Systolic blood pressure (mmHg)	Total cholesterol (mmol/L)	Diabetes (%)	Former smoker (%)	Current smoker (%)
Men					
White	126.5 (16.1)	5.3 (1.1)	16.1 (0.8)	41.4 (1.2)	21.2 (1.0)
Black	132.9 (19.9)	5.1 (1.0)	28.4 (1.5)	31.6 (1.8)	33.1 (1.8)
Women					
White	126.0 (18.3)	5.6 (1.0)	11.0 (0.8)	31.0 (1.5)	16.6 (1.0)
Black	133.3 (21.9)	5.4 (1.1)	28.4 (1.7)	21.4 (1.6)	20.7 (1.6)

* The estimates are age-standardized to the 2010 US population.

Supplemental Table 5: Proportion of population and proportion of ASCVD events occurring among high-risk individuals by sex and race, using ACC/AHA 2013 Pooled Cohort Equations

	ASCVD risk ≥ 7.5%		ASCVD risk ≥ 20%	
	Proportion of population (%)	**Proportion of event (%)**	**Proportion of population (%)**	**Proportion of event (%)**
Men				
White	63 (61-66)	84 (83-85)	13 (11-14)	30 (27-33)
Black	78 (75-81)	92 (91-93)	23 (20-26)	45 (41-49)
Women				
White	21 (19-22)	49 (47-51)	2 (1-3)	9 (6-12)
Black	51 (47-54)	82 (80-83)	14 (12-16)	40 (35-44)

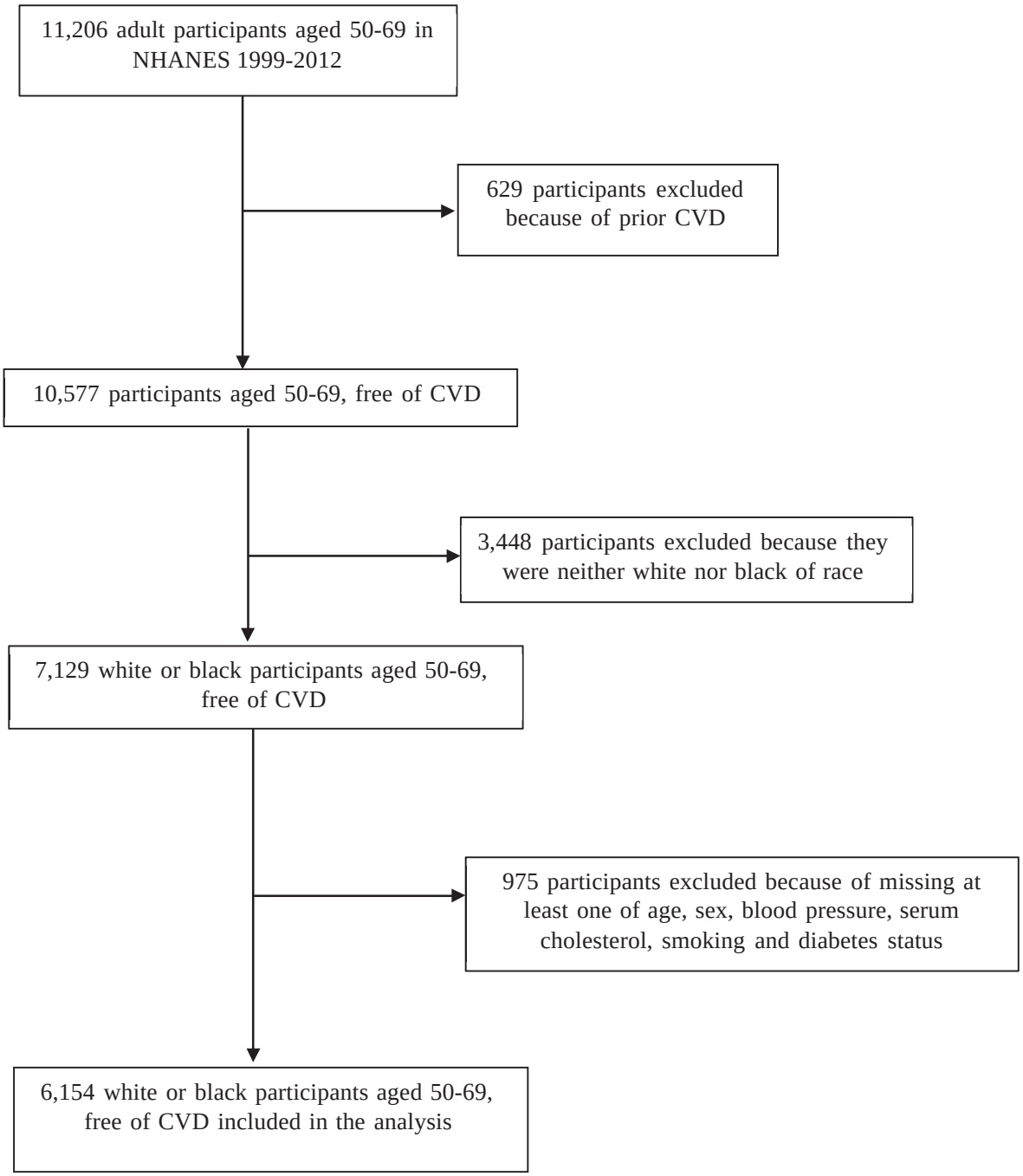
ASCVD = atherosclerotic cardiovascular disease, ACC= American College of Cardiology, AHA= American Heart Association

Supplemental Table 6: Impact of interventions on 10-year rate of atherosclerotic cardiovascular disease (per 100,000) by sex and race, using ACC/AHA 2013 Pooled Cohort Equations

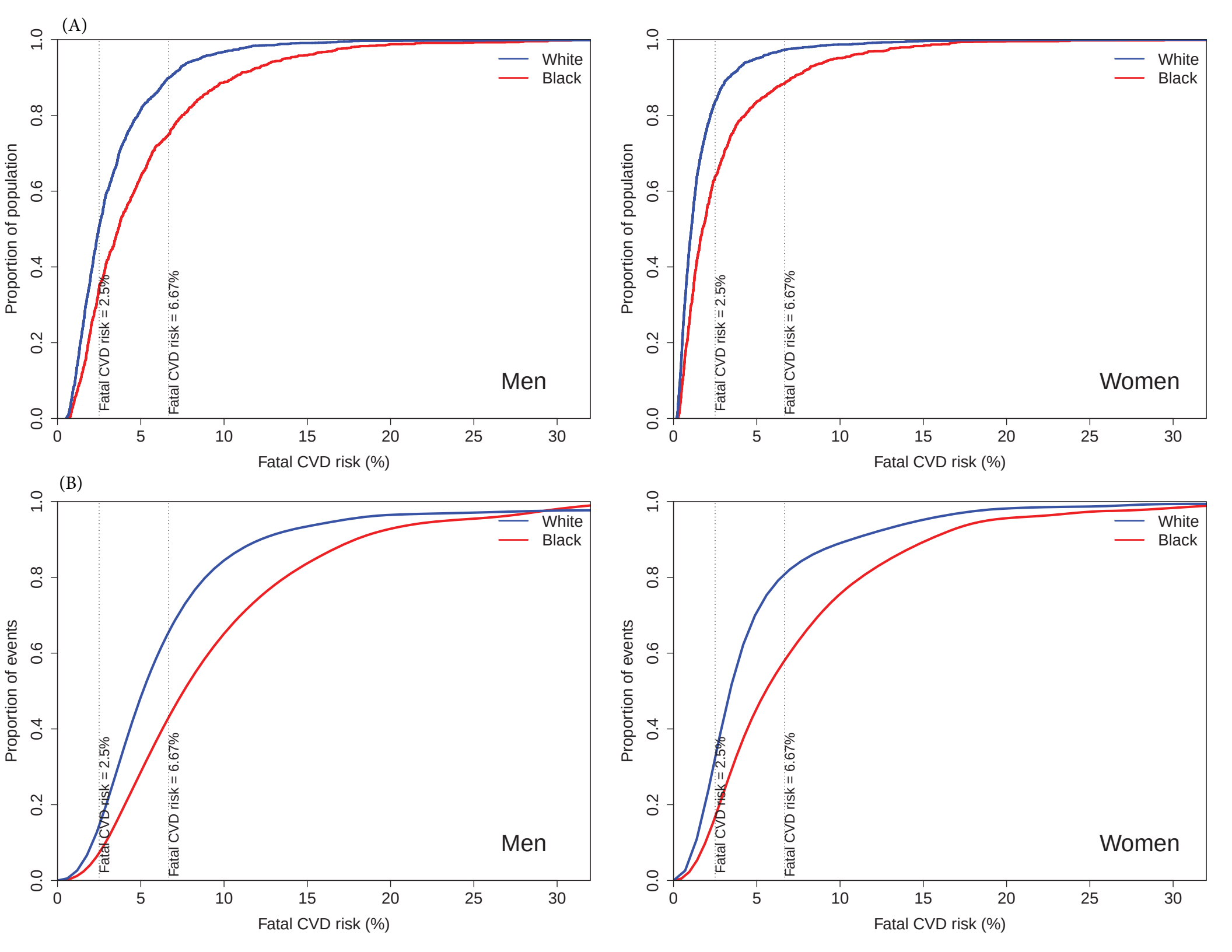
	Men						Women					
	10-year ASCVD event rate		Rate ratio (black vs. white)	Rate difference	Absolute change in rate difference	Relative change in rate difference	10-year ASCVD event rate		Rate ratio (black vs. white)	Rate difference	Absolute change in rate difference	Relative change in rate difference
	Black	White	-	-			Black	White				
Current	15,017	11,701	1.28	3,316	NA	NA	10,788	5,215	2.07	5,573	NA	NA
Population-wide interventions												
1) Salt reduction	14,858	11,585	1.28	3,272	-43	-1%	10,651	5,166	2.06	5,485	-88	-2%
2) Community-based dietary improvement to reduce serum cholesterol	14,871	11,320	1.31	3,551	236	7%	10,425	5,069	2.06	5,356	-216	-4%
3) MPOWER package for smoking	14,868	11,573	1.28	3,294	-22	-1%	10,660	5,139	2.07	5,521	-51	-1%
4) Increasing price of sugar-sweetened beverages for diabetes prevention	14,939	11,633	1.28	3,306	-10	0%	10,591	5,178	2.05	5,413	-159	-3%
5) Multiple interventions (1-4)	14,474	11,018	1.31	3,456	140	4%	9,986	4,921	2.03	5,065	-507	-9%
Single raised risk factor interventions												
6) Treatment for hypertension	14,853	11,577	1.28	3,277	-39	-1%	10,579	5,165	2.05	5,414	-158	-3%
7) Treatment for dyslipidemia	14,621	10,684	1.37	3,938	622	19%	9,700	4,891	1.98	4,809	-764	-14%
8) Referral for quitting smoking	14,827	11,553	1.28	3,274	-42	-1%	10,629	5,124	2.07	5,504	-68	-1%
9) Diabetes prevention program	14,359	11,238	1.28	3,120	-196	-6%	10,033	5,039	1.99	4,994	-579	-10%
Risk−based interventions												
10) Multiple risk factors (6-9 if risk of CVD death ≥ 2.5%) *	12,365	8,620	1.43	3,745	430	13%	7,407	4,353	1.70	3,054	-2,518	-45%
11) Multiple risk factors (10 without diabetes prevention)	13,016	8,955	1.45	4,061	745	22%	7,956	4,446	1.79	3,510	-2,063	-37%

* The eligibility for each intervention was defined based on risk of ASCVD ≥ 7.5% except for smoking cessation. Smoking cessation was provided to all current smokers irrespective of his/her predictive risk. ASCVD = atherosclerotic cardiovascular disease, ACC= American College of Cardiology, AHA= American Heart Association

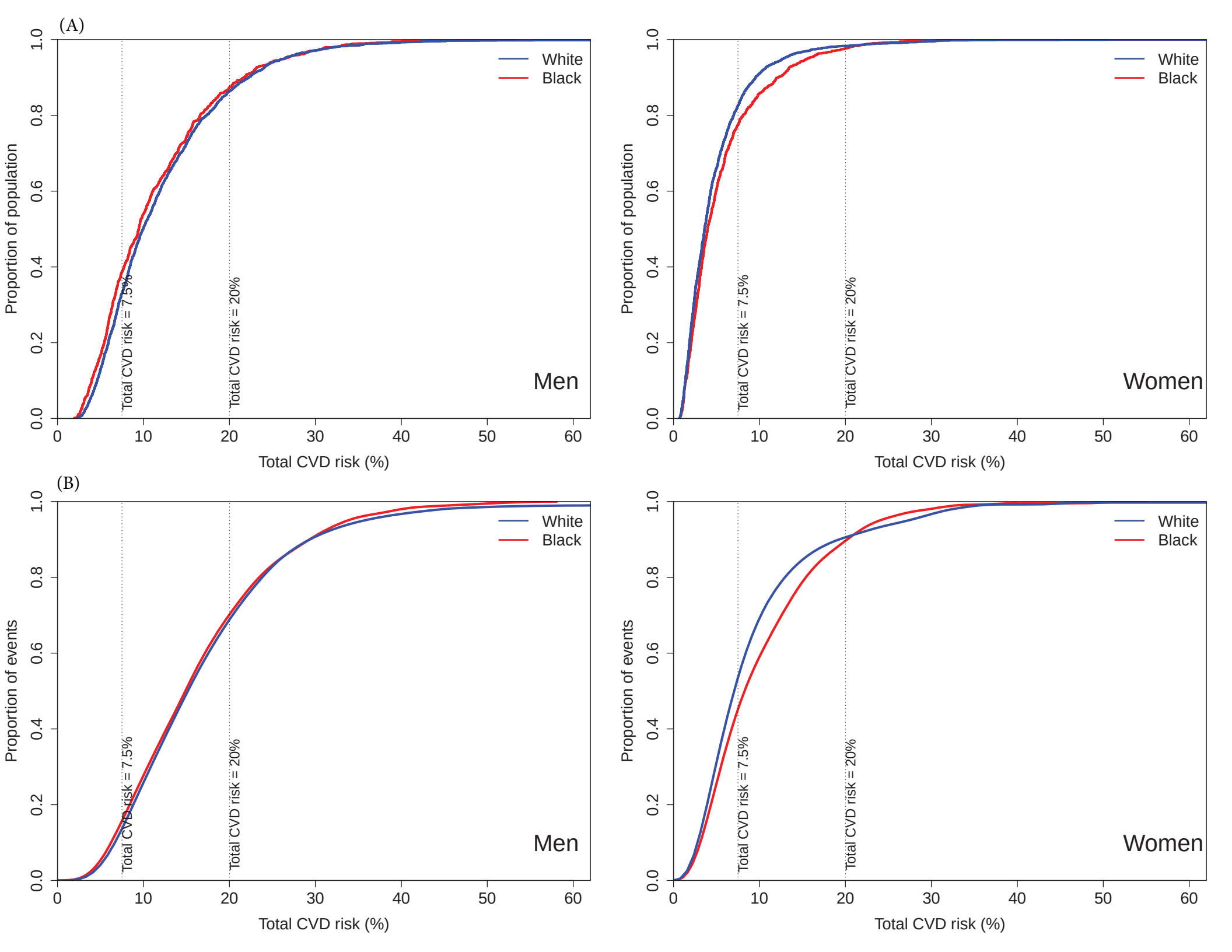
Supplemental Figure 1: Flowchart for selecting study population in the National Health and Nutrition Examination Survey (NHANES) 1999-2012


11,206 adult participants aged 50-69 in NHANES 1999-2012
629 participants excluded because of prior CVD
10,577 participants aged 50-69, free of CVD
3,448 participants excluded because they were neither white nor black of race
7,129 white or black participants aged 50-69, free of CVD
975 participants excluded because of missing at least one of age, sex, blood pressure, serum cholesterol, smoking and diabetes status
6,154 white or black participants aged 50-69, free of CVD included in the analysis

Supplemental Figure 2: Cumulative distributions of predicted 10-year risk of fatal CVD in the population (A) and among cases (B)

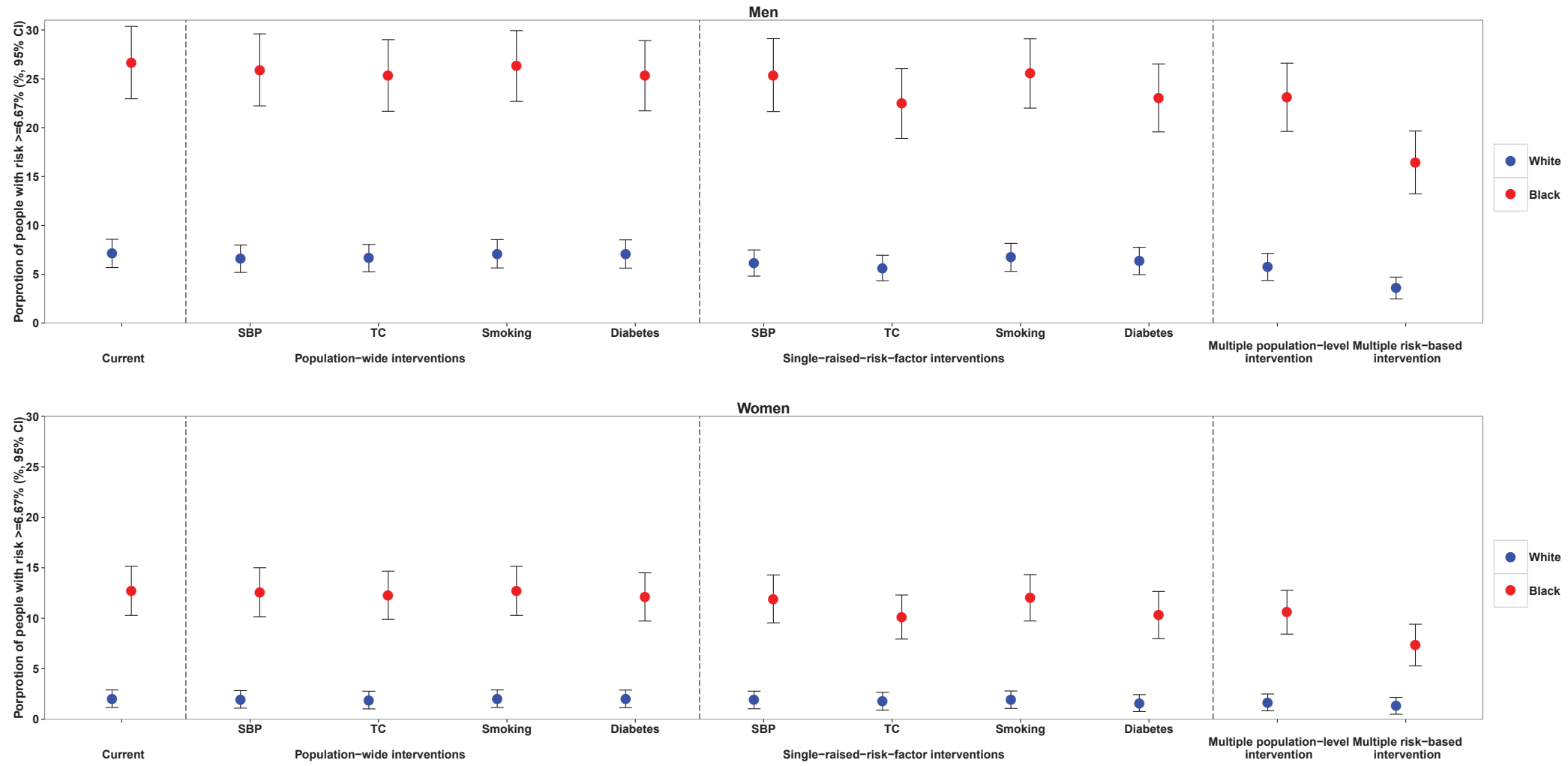

(A)
Proportion of population
Fatal CVD risk (%)
White
Black
Fatal CVD risk = 2.5%
Fatal CVD risk = 6.67%
Men
Women
(B)
Proportion of events

Supplemental Figure 3: Cumulative distributions of predicted 10-year risk of fatal-and-nonfatal CVD in the population (A) and among cases (B)


(A)
Proportion of population
Total CVD risk (%)
Total CVD risk = 7.5%
Total CVD risk = 20%
White
Black
Men
Women
(B)
Proportion of events

Supplemental Figure 4: Impact of population-wide, single raised risk factor, and risk-based interventions on proportion of population with ≥ 6.67%[*] 10-year risk of fatal CVD using race-specific risk scores [†]

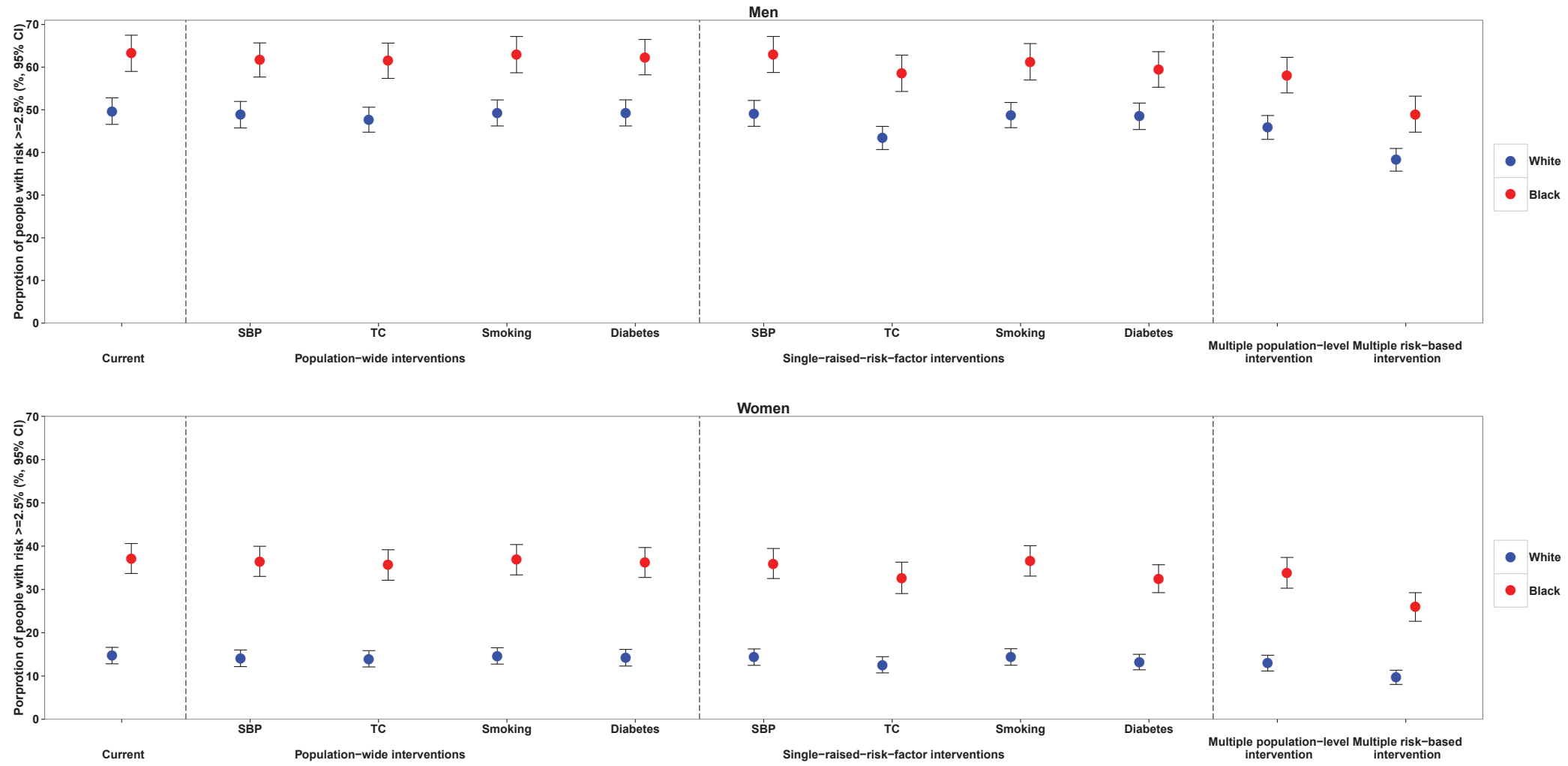
***** This threshold approximately equals to ≥ 20% for risk of fatal-and-nonfatal CVD given one third of CVD events are fatal in the US.[4]


Men
Porprotion of people with risk >=6.67% (%, 95% CI)
0
5
10
15
20
25
30
SBP
TC
Smoking
Diabetes
SBP
TC
Smoking
Diabetes
Current
Population−wide interventions
Single−raised−risk−factor interventions
Multiple population−level intervention
Multiple risk−based intervention
White
Black

Women
Porprotion of people with risk >=6.67% (%, 95% CI)
0
5
10
15
20
25
30
SBP
TC
Smoking
Diabetes
SBP
TC
Smoking
Diabetes
Current
Population−wide interventions
Single−raised−risk−factor interventions
Multiple population−level intervention
Multiple risk−based intervention
White
Black

Supplemental Figure 5: Impact of population-wide, single raised risk factor, and risk-based interventions on proportion of population with ≥ 2.5%* 10-year risk of fatal CVD using race-specific risk scores

***** This threshold approximately equals to ≥ 7.5% for risk of fatal-and-nonfatal CVD given one third of CVD events are fatal in the US.[4]


Men
Porprotion of people with risk >=2.5% (%, 95% CI)
0
10
20
30
40
50
60
70
SBP
TC
Smoking
Diabetes
SBP
TC
Smoking
Diabetes
Current
Population−wide interventions
Single−raised−risk−factor interventions
Multiple population−level intervention
Multiple risk−based intervention
White
Black

Women
Porprotion of people with risk >=2.5% (%, 95% CI)
0
10
20
30
40
50
60
70
SBP
TC
Smoking
Diabetes
SBP
TC
Smoking
Diabetes
Current
Population−wide interventions
Single−raised−risk−factor interventions
Multiple population−level intervention
Multiple risk−based intervention
White
Black

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