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PREVENT Equations in Young Adults

Fairness, Calibration, and Performance Across Racial and Ethnic Groups

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ABSTRACT

BACKGROUND Cardiovascular disease (CVD) is increasing among young adults. The American Heart Association's PREVENT (Predicting Risk of Cardiovascular Disease Events) equations estimate risk of CVD, atherosclerotic cardiovascular disease (ASCVD), and heart failure (HF) for primary prevention. Augmented equations additionally include zip code-based social deprivation index (SDI) to address adverse social exposures.

OBJECTIVES We assessed performance and algorithmic fairness of base and SDI-augmented PREVENT equations in young adults aged 30 to 39 years, defining fairness as similar performance across racial and ethnic groups. An exploratory analysis was conducted among young adults aged 20 to 29 years.

METHODS We included Kaiser Permanente Southern California members aged 20 to 39 years without prior CVD between 2008 and 2009, followed through 2019. We compared 10-year predicted and observed CVD, ASCVD, and HF events for base and SDI-augmented PREVENT models. Performance (Harell's C, calibration slopes, mean calibration) and fairness (concordance imparity, fair calibration) were estimated by race and ethnicity and age group (30-39 years [primary analysis], 20-29 years [exploratory analysis]).

RESULTS Among 161,202 young adults aged 30 to 39 years (60.0% women; 51.7% Hispanic, 26.9% non-Hispanic White, 12.5% Asian/Pacific Islander, 8.9% non-Hispanic Black), 10-year CVD incidence was 0.7%. Race-specific Harrell's C-statistics for the base PREVENT CVD model ranged from 0.68 to 0.72, yielding low concordance imparity (0.04; 95% CI: 0.02-0.22) which implies fair discrimination. Mean calibration showed underprediction in non-Hispanic Black participants (0.54; 95% CI: 0.48-0.65) vs other groups (range: 0.96-1.07). In fair calibration testing, prediction errors differed across racial and ethnic groups. Results were similar for ASCVD and HF. Adding SDI did not improve performance or fairness despite disparities across groups. In exploratory analyses among 80,978 individuals aged 20 to 29 years, performance and fairness results were similar.

CONCLUSIONS This large, diverse cohort of young adults demonstrates how the PREVENT equations may perform when applied in real-world clinical settings, reflecting the true operational environment faced by large health systems. Applications of PREVENT in clinical patient care, eg, early initiation of preventive strategies, should consider variations in model performance across age, race, and ethnicity. (JACC. 2026;■:■-■) © 2026 by the American College of Cardiology Foundation.

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ABBREVIATIONS AND ACRONYMS

ASCVD = atherosclerotic cardiovascular disease

BMI = body mass index

CVD = cardiovascular disease

eGFR = estimated glomerular filtration rate

HDL-C = high-density lipoprotein cholesterol

HF = heart failure

KPSC = Kaiser Permanente Southern California

PREVENT = Predicting Risk of Cardiovascular Disease Events

SBP = systolic blood pressure

SDI = social deprivation index

Cardiovascular disease (CVD) is a leading cause of morbidity and mortality in the United States.[1] Declines in cardiovascular health begin in childhood and adolescence and accelerate through young adulthood, resulting in increasing CVD risk early in the life-course.[2-4] Recent data suggest stable or increasing CVD rates among specific subgroups of young adults in the United States, an age group that account for a growing proportion of CVD events.[5-9] In parallel, persistent disparities in CVD trends across racial and ethnic groups are exacerbated by long-standing inequities in social and structural determinants of health.[5,10] These patterns underscore the need for effective and equitable prevention strategies targeted toward young adults, particularly across diverse racial and ethnic populations.

To guide CVD primary prevention efforts, multisociety guidelines recommend matching the intensity of CVD preventive interventions to a patient's estimated absolute CVD risk, as estimated by risk prediction models.[11,12] In 2023, the American Heart Association published Predicting Risk of Cardiovascular Disease Events (PREVENT), a set of risk prediction equations that estimate 10- and 30-year risk of heart failure (HF), atherosclerotic cardiovascular disease (ASCVD), and their composite of total CVD. PREVENT expanded age ranges from previous risk prediction, which began at 40 years, to include individuals as young as 30 years. Yet, no studies have directly evaluated the performance of PREVENT exclusively in young adults aged 30 to 39 years.

The use of race in clinical risk assessment has been criticized because of concerns regarding algorithmic fairness, which could result in race-related variations in treatment and potentially exacerbate disparities.[13-17] Accordingly, the PREVENT models strive to achieve fairness across racial groups through unawareness, because race and ethnicity are not predictors.[18] However, some contend that the use of race as a predictor variable is a proxy for upstream social and structural determinants, including systemic racism, that may influence the risk for CVD.[19] To account for contributions from adverse social exposures, PREVENT includes an optional augmented model that incorporates residential zip code-level social deprivation index (SDI)[20] to account for risk related to adverse or protective social determinants of health (SDOH) that contribute to health outcomes.[21-23] Yet, whether the removal of race achieves equity in practice, even after incorporating SDI, has not been examined in a large, diverse real-world cohort. This is of particular importance because PREVENT is already being incorporated into guidelines and clinical decision support.

In this study, we sought to assess performance and algorithmic fairness, defined as consistent discrimination and calibration across groups, of the PREVENT base and SDI-augmented equations to predict total CVD, ASCVD, and HF in >160,000 young adults aged 30 to 39 years across racial and ethnic groups in an integrated health system. We further assessed the utility of PREVENT among even younger adults aged 20 to 29 years as an exploratory analysis, because clinicians inevitably apply risk scores outside of their derivation ranges, particularly among those who may benefit most from early intervention.

METHODS

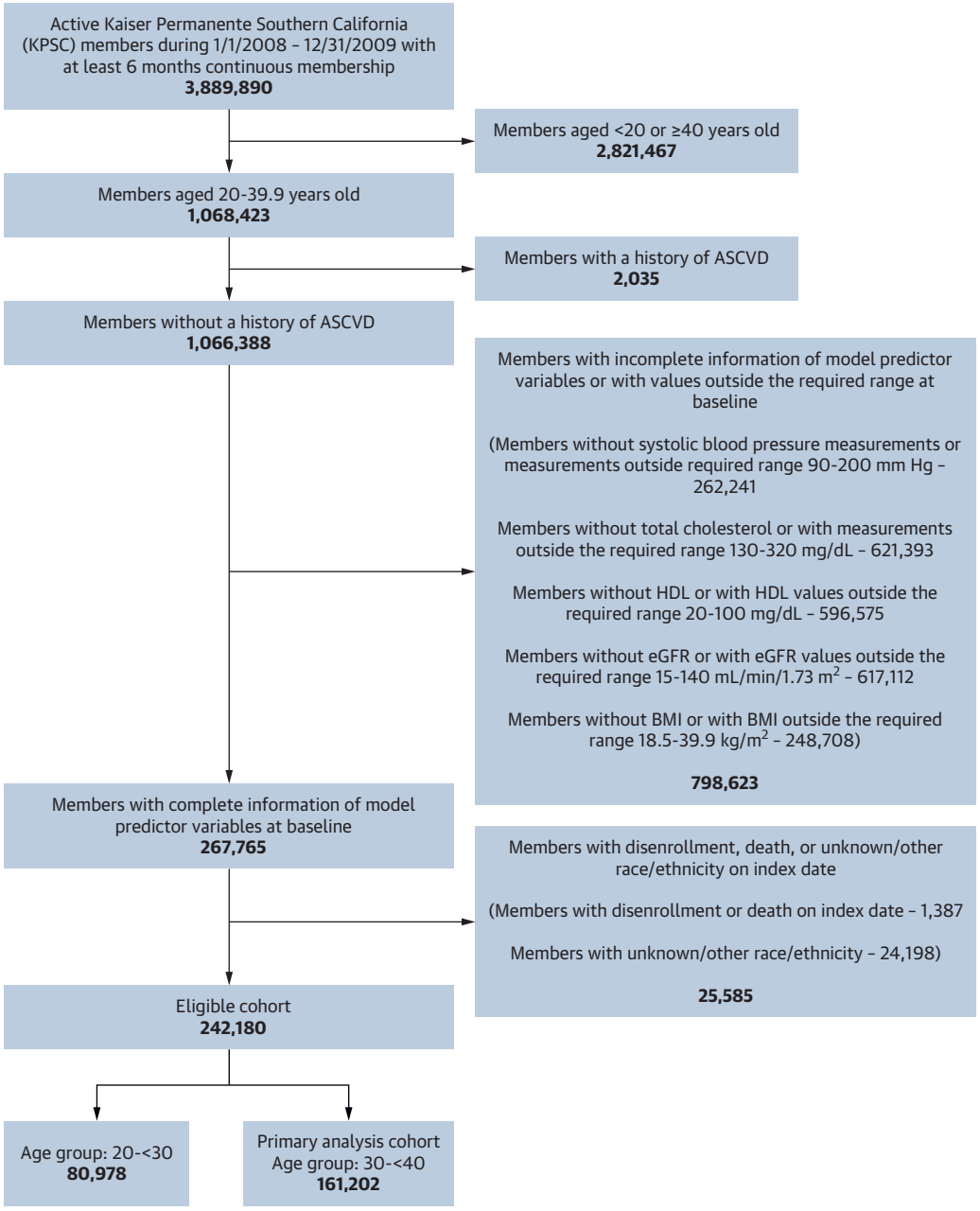
This study's protocols complied with the tenets of the Declaration of Helsinki and was approved by the Institutional Review Board at Kaiser Permanente Southern California (KPSC) (#13699). Informed consent was waived given the retrospective nature of the study.

STUDY SAMPLE. This study included members of KPSC, a large diverse U.S. integrated health care system. All medical care provided within the KPSC system is captured in comprehensive electronic health records (EHRs); medical care received outside the system is captured via insurance claims.

We included young adults aged 30 to 39 years (main study population) and 20 to 29 years (exploratory analyses) who were active members in KPSC between January 1, 2008, and December 31, 2009, with at least 6 months of continuous prior membership (**Figure 1**). Those with history of CVD or out-of-range and/or incomplete model predictors at baseline were excluded. Study baseline was defined as the earliest eligible date individuals met inclusion/exclusion criteria. Supplemental Table 1 compares included members to those excluded because of out-of-range predictors, including systolic blood pressure (SBP) (<90 or >200 mm Hg), total cholesterol (<130 or >320 mg/dL), high-density lipoprotein cholesterol (HDL-C) (<20 or >100 mg/dL), estimated glomerular filtration rate (eGFR) (<15 or >140 mL/min/1.73 m^2), or body mass index (BMI) (<18.5 or >40.0 kg/m^2).

ASCVD, HF, AND CVD EVENTS. The main outcomes of interest were incident ASCVD, defined as nonfatal myocardial infarction, fatal coronary heart disease, and fatal or nonfatal stroke; incident HF; and their composite, total CVD. Supplemental Table 2 displays details and International Classification of Diseases

FIGURE 1 Kaiser Permanente Southern California Analytic Sample Derivation



This study included members of Kaiser Permanente Southern California (KPSC), a large diverse U.S. integrated health care system. All medical care provided within the KPSC system is captured in comprehensive electronic health records; medical care received outside the system is captured via insurance claims. The study population included young adults aged 20 to 39 years who were active members in KPSC between January 1, 2008, and December 31, 2009, with at least 6 months of continuous prior membership. Those with history of cardiovascular disease (CVD) or out-of-range and/or incomplete model predictors at baseline were excluded. The final primary sample included 161,202 young adults who were aged 30 to <40 years. The supplementary analysis included a final sample of 80,978 young adults who were aged 20 to <30 years. ASCVD = atherosclerotic cardiovascular disease; BMI = body mass index; eGFR = estimated glomerular filtration rate; HDL = high-density lipoprotein.

codes used for ascertainment. Study participants were followed until the earliest of outcomes of interest, end of KPSC membership, death, or end of the 10-year prediction window. Supplemental Table 3 compares baseline characteristics of young adults with at least 10 years of membership and young adults with <10 years of membership. Supplemental Table 4 summarizes available follow-up by race and ethnicity among all KPSC members aged 30 to 39 or 20 to 29 years and among those included after applying exclusion criteria.

RISK PREDICTION. Predicted risk was calculated using the base and SDI-augmented PREVENT equations for 10-year risk of total CVD, ASCVD, and HF using baseline age in years, sex, total cholesterol (mg/dL), HDL-C (mg/dL), SBP (mm Hg), BMI (kg/m^2), eGFR (mL/min/1.73 m^2) calculated using the Chronic Kidney Disease Epidemiology Collaboration 2021 creatinine equation,[24] diabetes, current smoking, antihypertensive medication, and statin use.[25] Age, sex, race, ethnicity, and smoking status were based on both self-report and administrative data. Although PREVENT was derived and validated for adults aged 30 to 79 years, we used individuals' actual age for the risk calculation for adults aged <30 years. Total cholesterol, HDL-C, and eGFR were obtained from outpatient laboratory data within 6 years before the index date to account for laboratory testing intervals, because KPSC clinical guidelines recommend testing every 4 to 6 years in healthy individuals aged 20 to 39 years (Supplemental Figure 1). SBP was obtained from outpatient data within 2 years before the index date. For these laboratory measures and SBP, the values before and closest to the index date were selected. Height and weight measurements from clinical encounters before and closest to the index date were used to calculate BMI. Antihypertension medication and statin use were determined using outpatient pharmacy records within 1 year before the index date. SDI-augmented PREVENT equations included deciles of SDI based on residential billing zip code at the index date, which we mapped via geocoding to 2008-2012 U.S. census data at the tract level.[20]

PERFORMANCE AND ALGORITHMIC FAIRNESS METRICS. Performance and algorithmic fairness of the base and SDI-augmented PREVENT equations centered around discrimination and calibration. Broadly, discrimination indicates how well a model differentiates between individuals at higher vs lower risk of an event.[26] Calibration is a goodness-of-fit measure reflecting the extent to which a model correctly estimates absolute risk.[26]

For the discrimination performance metric, we used Harrell's C, which ranges from 0 to 1 with higher scores indicating better discrimination. To evaluate calibration performance in each group, we used mean calibration, or "calibration-in-the-large," which measures systematic overprediction and underprediction and was estimated as the ratio of predicted risk to observed risk. A mean calibration of 1 indicates perfect calibration (predicted outcomes equal to observed outcomes); <1, underestimation (predicted outcomes less than observed outcomes); and >1, overestimation (predicted outcomes greater than observed outcomes). We also calculated calibration slopes, intercepts, and their 95% CIs using linear regression. Calibration was also assessed by plotting predicted risk vs observed risk by decile of predicted risk and via the Hosmer-Lemeshow goodness-of-fit test statistic.

We defined algorithmic fairness as similar discrimination and calibration across racial and ethnic groups.[27] The algorithmic fairness metric for discrimination, concordance imparity, was the maximum difference in Harrell's C, calculated as the difference between the highest and lowest Harrell's C across racial and ethnic groups, with lower concordance imparity indicating a fairer model reflecting a smaller range of C statistics across racial and ethnic groups.[27] We also used the statistical fairness metric of fair calibration (**Figure 2**), which measures whether the model creates probability value-based disparity systematically.[27] First, we assessed representation consistency, or whether the predicted risks for each racial and ethnic group were representative of the observed risks. For each racial and ethnic group, we split predicted probabilities into deciles and calculated the Hosmer-Lemeshow goodness-of-fit test statistic. If the associated P value was >0.05 in each racial and ethnic group, fair calibration was considered met because of representation consistency. If representation was inconsistent, we next evaluated difference consistency, defined as whether the difference between predicted risk and observed risk was accordant across racial and ethnic groups. We ran pairwise Wilcoxon signed-rank tests between each racial and ethnic group. If all associated P values were >0.05, fair calibration was considered met because of difference consistency. If neither representation consistency nor difference consistency was met, model calibration was classified as unfair.

In addition to calibration and discrimination, we evaluated sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) at risk cutoffs of 3.5%, 5%, and 7.5%, consistent with

currently used or proposed clinical thresholds. We calculated sensitivity and specificity 95% CIs using the normal approximation,[28] and PPV and NPV 95% CIs using the Mercaldo et al[29] formula. We also calculated Brier score as a measure of accuracy. Brier score is calculated as the mean squared difference between predicted probabilities and actual outcomes and ranges from 0 to 1, with a score of 0 indicating perfect accuracy.

STATISTICAL ANALYSIS. Baseline characteristics were described by race and ethnicity using means and SDs for continuous variables and percents for categorical variables.

Kaplan-Meier product limit estimators were used to estimate survival free from CVD, adjusting for right censoring of participants, and subtracted from 1 to estimate the observed risk of total CVD, ASCVD, and HF events within 10 years. Predicted risk was calculated using the PREVENT base and SDI-augmented equations for 10-year risk of CVD, ASCVD, and HF.[30] Performance and algorithmic fairness metrics were estimated for 10-year risk of CVD, ASCVD, and HF using base and SDI-augmented PREVENT equations by racial and ethnic group among individuals aged 30 to 39 years.

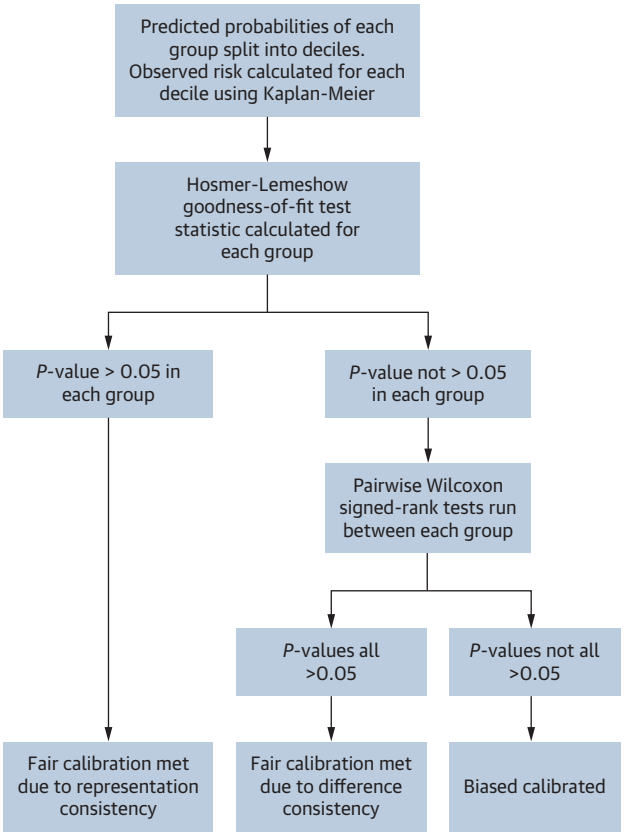
We performed 2 sensitivity analyses. First, as an exploratory stress test, we applied PREVENT and performed all analyses in individuals aged 20 to 29 years. Second, we reran analyses restricting our sample to young adults with laboratory data measured within 2 years of their index date.

Analyses were performed using SAS version 9.4. Statistical significance was set a priori at $\alpha = 0.05$. All 95% CIs were calculated via the nonparametric bootstrap with 750 resamples unless otherwise stated.

RESULTS

PRIMARY ANALYSIS: INDIVIDUALS AGED 30 TO 39 YEARS. Of 161,202 young adults aged 30 to 39 years included, 60.0% were women, 51.7% Hispanic, 26.9% non-Hispanic White, 12.5% Asian/Pacific Islander, and 8.9% non-Hispanic Black (Table 1). Non-Hispanic Black young adults more frequently used antihypertensive medications and had lower kidney function than other groups. Hispanic young adults were more likely to live in neighborhoods with low educational attainment. Asian/Pacific Islander and non-Hispanic White young adults more frequently lived in areas with median household incomes >$80,000 and lower SDI deciles, while Hispanic and non-Hispanic Black young adults more frequently lived in areas with median household incomes <$50,000 and higher SDI deciles.

FIGURE 2 Flow Chart Describing Evaluation of Fair Calibration Across Racial and Ethnic Groups



Fair calibration metric developed by Zhang and Weiss measures whether the model creates probability value-based disparity systematically.[27] First, we assessed representation consistency, or whether the predicted risks for each racial and ethnic group were representative of the observed risks. For each racial and ethnic group, we split predicted probabilities into deciles and calculated the Hosmer-Lemeshow goodness-of-fit test statistic. If the associated *P* value was >0.05 in each racial and ethnic group, fair calibration was considered met caused by representation consistency. If representation was inconsistent, we next evaluated difference consistency, or whether differences between predicted and observed risk were similar across racial and ethnic groups. We ran pairwise Wilcoxon signed-rank tests between each racial and ethnic group. If all associated *P* values were >0.5, fair calibration was considered met because of difference consistency. If neither representation consistency nor difference consistency was met, model calibration was classified as unfair.

Among individuals aged 30 to 39 years, mean follow-up time ranged from 6.8 to 7.5 years across racial and ethnic groups. There were 512, 328, 203, and 143 incident CVD events among Hispanic, non-Hispanic White, non-Hispanic Black, and Asian/Pacific Islander young adults, respectively (Table 2). Non-Hispanic Black individuals more frequently experienced CVD events (1.4%) compared with the other groups (0.6%-0.7%). This increased risk was reflected across all outcomes (Table 2, Figure 3).

TABLE 1 Sample Characteristics Overall and by Racial and Ethnic Group Among KPSC Young Adults Aged 30 to 39 Years (N = 161,202)

		Racial and Ethnic Group			
	Overall (N = 161,202)	Asian/Pacific Islander (n = 20,137)	Hispanic (n = 83,347)	Non-Hispanic Black (n = 14,333)	Non-Hispanic White (n = 43,385)
Sociodemographics					
Age, y	35.0 ± 2.9	35.0 ± 2.8	34.9 ± 2.8	35.2 ± 2.9	35.1 ± 2.9
Women, %	60.0	60.5	61.0	66.1	55.9
Insurance type, %					
Medicaid	2.3	0.9	2.1	6.4	2.1
Commercial	91.1	91.0	94.0	90.4	85.9
Private	6.5	8.1	3.8	3.0	12.0
Other	0.1	0.1	0.1	0.1	0.1
Medium household income (USD)					
≤$49,900	29.3	18.5	36.8	37.1	17.3
$50,000-$64,900	21.7	18.9	24.0	22.1	18.6
$60,000-$79,900	16.9	17.6	16.0	14.7	18.9
≥$80,000	27.0	41.2	18.3	18.0	40.1
Unknown	5.1	3.7	4.9	8.1	5.1
Low neighborhood educational attainment (<75% with high school education or higher)	33.9	19.7	48.6	31.0	13.3
Neighborhood poverty >25%	7.0	2.7	9.6	11.1	2.4
Unemployment rate >10%	8.7	4.2	10.6	12.1	5.9
PREVENT predictors					
Systolic blood pressure, mm Hg	119.1 ± 13.8	117.1 ± 14.1	118.4 ± 13.5	122.7 ± 14.7	120.2 ± 13.5
Total cholesterol, mg/dL	192.8 ± 34.0	193.0 ± 33.4	193.4 ± 34.0	188.8 ± 33.6	192.9 ± 34.3
High-density lipoprotein cholesterol, mg/dL	50.2 ± 12.9	52.1 ± 13.2	48.7 ± 12.2	53.4 ± 13.2	51.1 ± 13.7
Body mass index, kg/m^2	28.4 ± 4.9	25.7 ± 4.4	29.1 ± 4.7	29.6 ± 5.1	27.9 ± 5.0
Diabetes, %	4.1	3.6	4.7	4.3	3.0
Current smoking, %	7.4	5.7	5.8	8.9	10.7
Antihypertensive treatment, %	6.7	7.5	5.5	12.7	6.7
Lipid-lowering treatment, %	4.5	5.0	4.6	4.3	4.4
Estimated glomerular filtration rate, mL/min/1.73 m^2	106.0 ± 15.5	108.4 ± 14.4	109.6 ± 14.1	94.6 ± 16.9	101.8 ± 15.3
SDI score decile					
1	10.2	17.2	5.4	5.3	17.7
2	9.8	14.1	6.1	8.9	15.1
3	10.1	13.0	7.7	7.6	14.1
4	9.9	10.2	9.2	8.4	11.6
5	8.7	8.0	8.6	8.5	9.2
6	9.9	10.6	10.7	9.0	8.3
7	9.2	7.6	10.9	9.5	6.7
8	10.7	7.8	12.9	12.8	6.8
9	9.4	5.9	12.3	12.3	4.4
10	8.9	3.5	12.8	13.0	2.7
Unknown	3.3	2.2	3.4	4.7	3.2

Values are mean ± SD or %.

Mean age of CVD onset ranged from 38.3 to 40.4 years, ASCVD onset from 38.8 to 40.7 years, and HF onset from 37.0 to 40.1 years across groups.

Across racial and ethnic groups, Harrell's C using the base PREVENT model ranged from 0.68 (95% CI: 0.65-0.71) to 0.72 (95% CI: 0.68-0.77) for CVD, 0.68 (95% CI: 0.63-0.72) to 0.75 (95% CI: 0.69-0.80) for ASCVD, and 0.65 (95% CI: 0.55-0.75) to 0.76 (95% CI: 0.70-0.83) for HF (Table 2). Discrimination was highest for CVD among Asian/Pacific Islander and Non-Hispanic Black young adults, for ASCVD among Asian/Pacific Islander young adults, and for HF among Non-Hispanic Black young adults. The maximum difference in discrimination (concordance imparity) across groups was 0.04 (95% CI: 0.02-0.22) for CVD, 0.07 (95% CI: 0.03-0.29) for ASCVD, and 0.11

TABLE 2 Performance Metrics of Base and SDI-Augmented PREVENT-Estimated 10-Year Risk of Total CVD, ASCVD, and HF in Young Adults 30 to 39 Years in KPSC (N = 161,202)

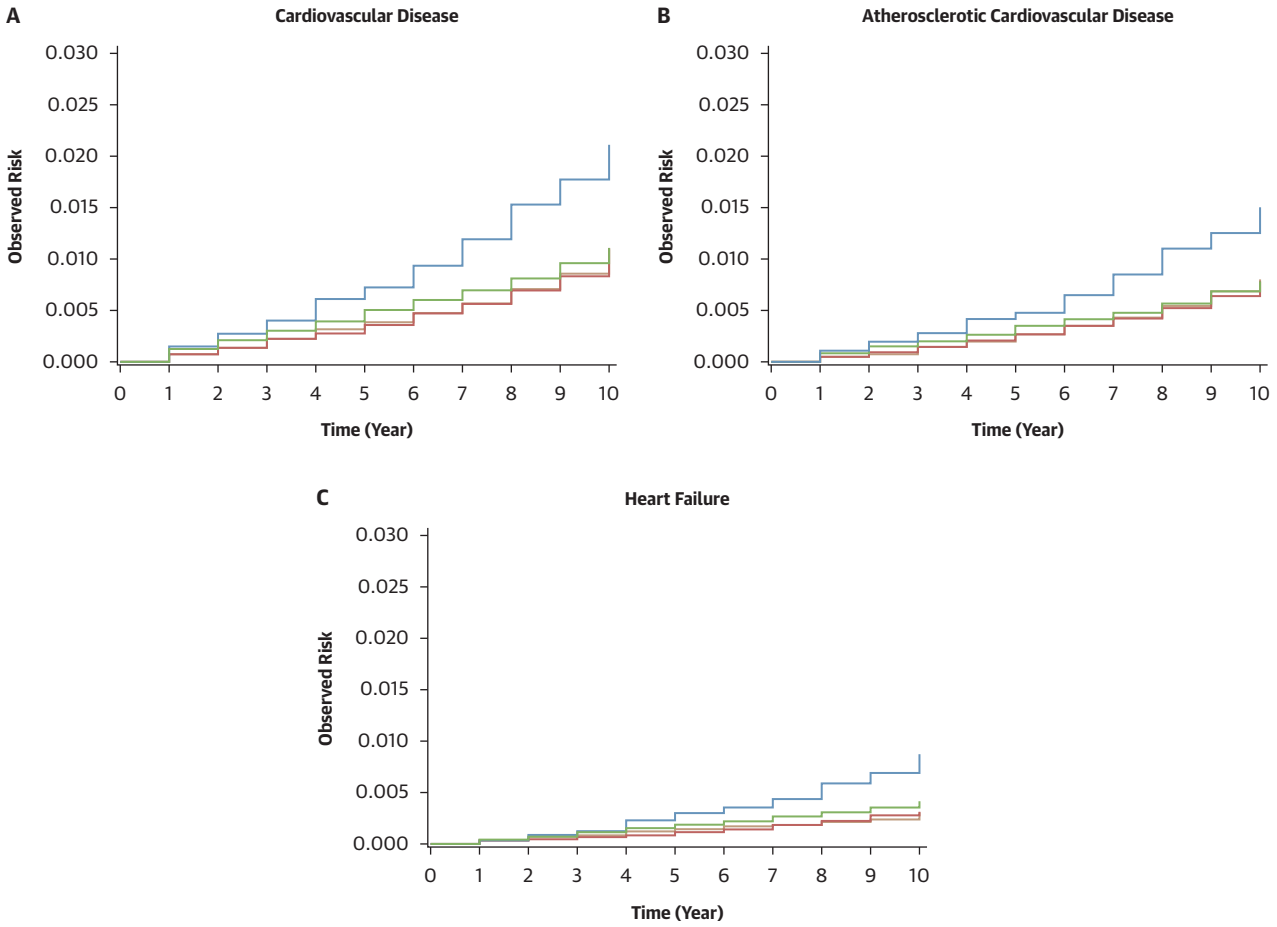
				Discrimination				Calibration					
				Harrell's C		Concordance Imparity		Calibration Slope[a]		Mean Calibration		Fair Calibration	
	n	Events	Person-y	Base	SDI	Base	SDI	Base	SDI	Base	SDI	Base	SDI
Cardiovascular disease						0.04 (0.02-0.22)	0.04 (0.02-0.20)					Biased	Biased
Asian/Pacific Islander	20,137	143	150,475	0.72 (0.66-0.77)	0.72 (0.66-0.76)			1.08 (0.94-1.22)	1.07 (0.87-1.27)	0.96 (0.59-1.13)	0.94 (0.57-1.10)		
Hispanic	83,347	512	562,889	0.70 (0.67-0.72)	0.70 (0.67-0.72)			0.98 (0.84-1.11)	0.93 (0.83-1.03)	1.07 (0.64-1.16)	1.10 (0.65-1.20)		
Non-Hispanic Black	14,333	203	101,611	0.72 (0.68-0.77)	0.73 (0.68-0.77)			1.84 (1.57-2.10)	1.93 (1.56-2.30)	0.54 (0.48-0.65)	0.55 (0.48-0.66)		
Non-Hispanic White	43,385	328	301,085	0.68 (0.65-0.71)	0.68 (0.65-0.72)			0.92 (0.72-1.11)	0.98 (0.81-1.15)	0.96 (0.66-1.07)	0.92 (0.64-1.03)		
Atherosclerotic cardiovascular disease						0.07 (0.03-0.29)	0.07 (0.03-0.27)					Biased	Biased
Asian/Pacific Islander	20,137	113	150,475	0.75 (0.69-0.80)	0.74 (0.69-0.80)			1.25 (0.97-1.53)	1.20 (0.91-1.49)	0.82 (0.41-1.00)	0.81 (0.40-0.98)		
Hispanic	83,347	392	562,889	0.69 (0.65-0.72)	0.69 (0.65-0.72)			1.06 (0.89-1.23)	1.00 (0.85-1.14)	0.97 (0.44-1.07)	1.00 (0.46-1.10)		
Non-Hispanic Black	14,333	144	101,611	0.71 (0.67-0.76)	0.71 (0.66-0.76)			1.84 (1.50-2.18)	1.90 (1.50-2.18)	0.52 (0.43-0.61)	0.53 (0.44-0.63)		
Non-Hispanic White	43,385	230	301,085	0.68 (0.63-0.72)	0.68 (0.64-0.72)			0.98 (0.77-1.19)	1.01 (0.83-1.19)	0.95 (0.46-1.07)	0.91 (0.45-1.04)		
Heart failure						0.11 (0.04-0.31)	0.10 (0.03-0.29)					Biased	Biased
Asian/Pacific Islander	20,137	42	150,475	0.65 (0.55-0.75)	0.66 (0.57-0.76)			1.07 (0.82-1.33)	1.11 (0.83-1.39)	1.14 (0.20-1.57)	1.15 (0.20-1.59)		
Hispanic	83,347	164	562,889	0.73 (0.68-0.77)	0.73 (0.68-0.78)			0.99 (0.90-1.07)	0.93 (0.84-1.03)	1.13 (0.22-1.34)	1.19 (0.23-1.40)		
Non-Hispanic Black	14,333	82	101,611	0.76 (0.70-0.83)	0.76 (0.69-0.82)			1.98 (1.67-2.29)	1.99 (1.65-2.33)	0.54 (0.28-0.67)	0.56 (0.29-0.70)		
Non-Hispanic White	43,385	123	301,085	0.70 (0.65-0.75)	0.71 (0.66-0.75)			0.98 (0.75-1.21)	1.03 (0.81-1.26)	0.88 (0.23-1.07)	0.87 (0.23-1.05)		

[a]Calibration intercept ranged from −0.001 to 0.001 across all outcomes and racial and ethnic groups.
SDI = social deprivation index.

(95% CI: 0.04-0.31) for HF. Discrimination and subsequent concordance imparity for the SDI-augmented model were similar to the base model (Table 2).

There were considerable differences in calibration of the PREVENT base model. The lowest mean calibration for CVD was among non-Hispanic Black young adults (0.54 [95% CI: 0.48-0.65]) indicating significant underestimation of risk, up to 0.96 (95% CI: 0.59-1.13) among Asian/Pacific Islander and non-Hispanic White young adults, and 1.07 (95% CI: 0.64-1.16) among Hispanic young adults (Table 2, Figure 4). Calibration slopes also reflected this pattern, ranging from 0.92 (95% CI: 0.72-1.11) to 1.08 (95% CI: 0.94-1.22) in Asian/Pacific Islander, Hispanic, and non-Hispanic White young adults, highlighting good calibration, compared with 1.84 (95% CI: 1.57-2.10) in non-Hispanic Black young adults, highlighting large underestimation of risk in the base CVD model. These trends were consistent for ASCVD and HF outcomes. After restricting the sample to young adults with laboratory data measured within 2 years before their index date, findings remained consistent (Supplemental Table 5). Brier scores followed a similar pattern, with similar accuracy in all groups except non-Hispanic Black young adults, whose accuracy was markedly lower (Supplemental Table 6). Results did not meaningfully differ for SDI-augmented equations (Table 2, Figure 4).

FIGURE 3 Observed Risk by Racial and Ethnic Group Among Young Adults Aged 30 to 39 Years in KPSC (N = 161,202)

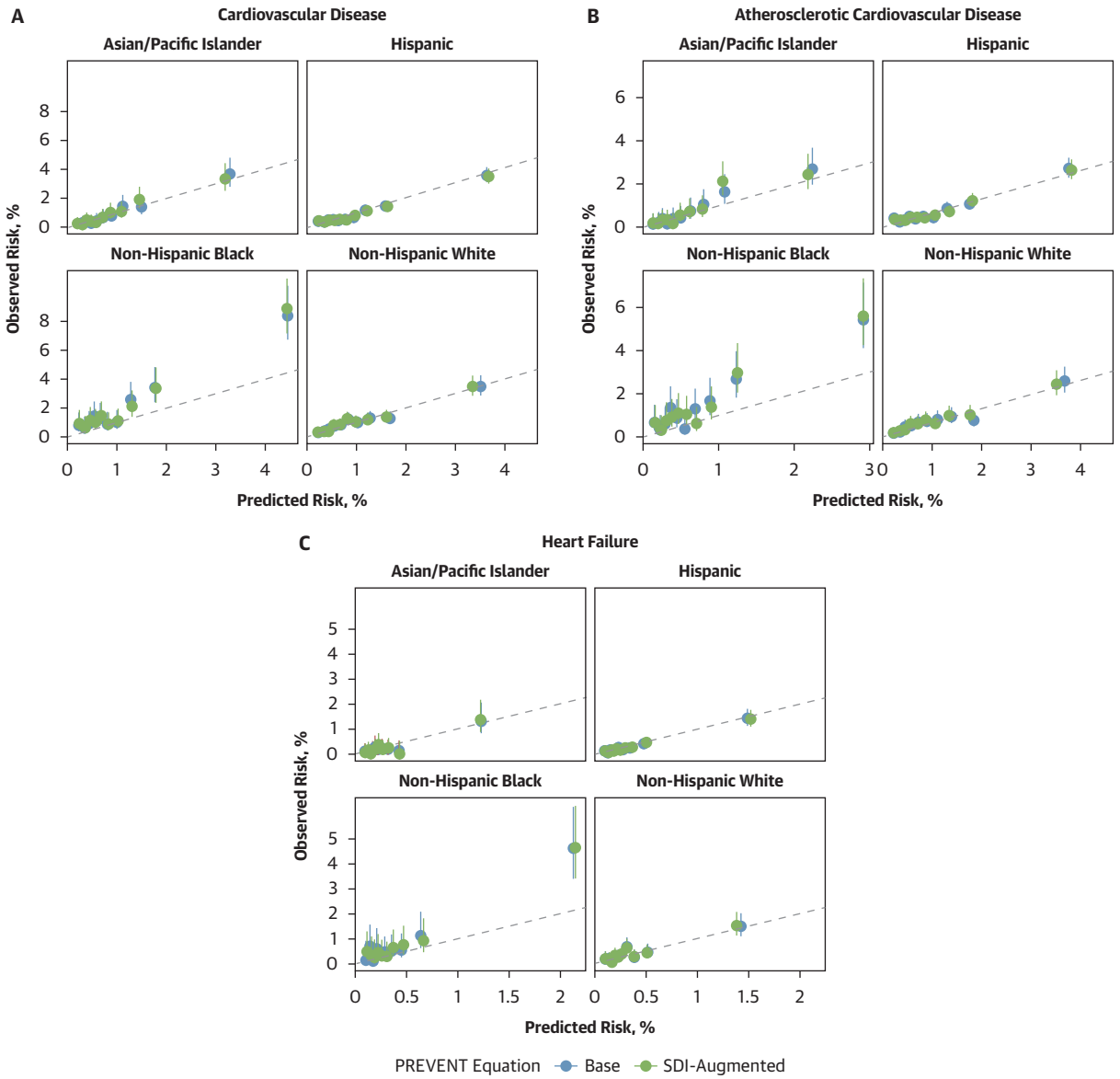


Brown lines indicate Asian/Pacific Islander young adults, red, Hispanic young adults; blue, non-Hispanic Black young adults; and green, non-Hispanic White young adults. Kaplan-Meier product limit estimators were used to estimate survival, adjusting for right censoring of participants, and subtracted from 1 to estimate the observed risk of (A) total cardiovascular disease, (B) atherosclerotic cardiovascular disease, and (C) heart failure events within 10 years.

The first component of fair calibration indicated low representation consistency, where most groups demonstrated disagreement between predicted and observed CVD risk (*P* values from Hosmer-Lemeshow tests were <0.05 across all groups except for Asian/Pacific Islander young adults) (Supplemental Table 7). Difference consistency then was assessed using pairwise Wilcoxon signed-rank tests. Prediction errors were similar across all groups except for non-Hispanic Black young adults, for whom prediction errors were significantly different in all pairwise comparisons (Supplemental Table 7). These results were consistent across all outcomes in both the base and SDI-augmented models.

IMPLICATIONS FOR CLINICAL DECISION MAKING AMONG YOUNG ADULTS. Sensitivity, specificity, PPV, and NPV at prespecified thresholds of base PREVENT-estimated 10-year predicted risk of CVD varied by racial and ethnic group and risk cutoff (**Figure 5**). Across all groups and cutoffs, sensitivity was generally low, ranging from 0.14 to 0.25 for a cutoff of 3.5% and 0.03 to 0.12 for a cutoff of 7.5%. Sensitivity was highest among non-Hispanic Black young adults across all 3 cutoffs. Specificity was generally low to modest, ranging from 0.08 to 0.50 for a cutoff of 3.5% and 0.09 to 0.51 for a cutoff of 7.5%. Specificity was consistently lowest among non-Hispanic Black young adults and highest among Hispanic young adults. PPV was low (0.04-0.08 for a cutoff of 3.5% and 0.06-0.18 for a cutoff of 7.5%), reflecting the low prevalence of events in this young population. NPV was high across all racial and

FIGURE 4 Calibration of Base and SDI-Augmented PREVENT-Estimated 10-Year Predicted Risk of Total CVD, ASCVD, and HF Among Young Adults Aged 30 to 39 Years (KPSC, N = 161,202)

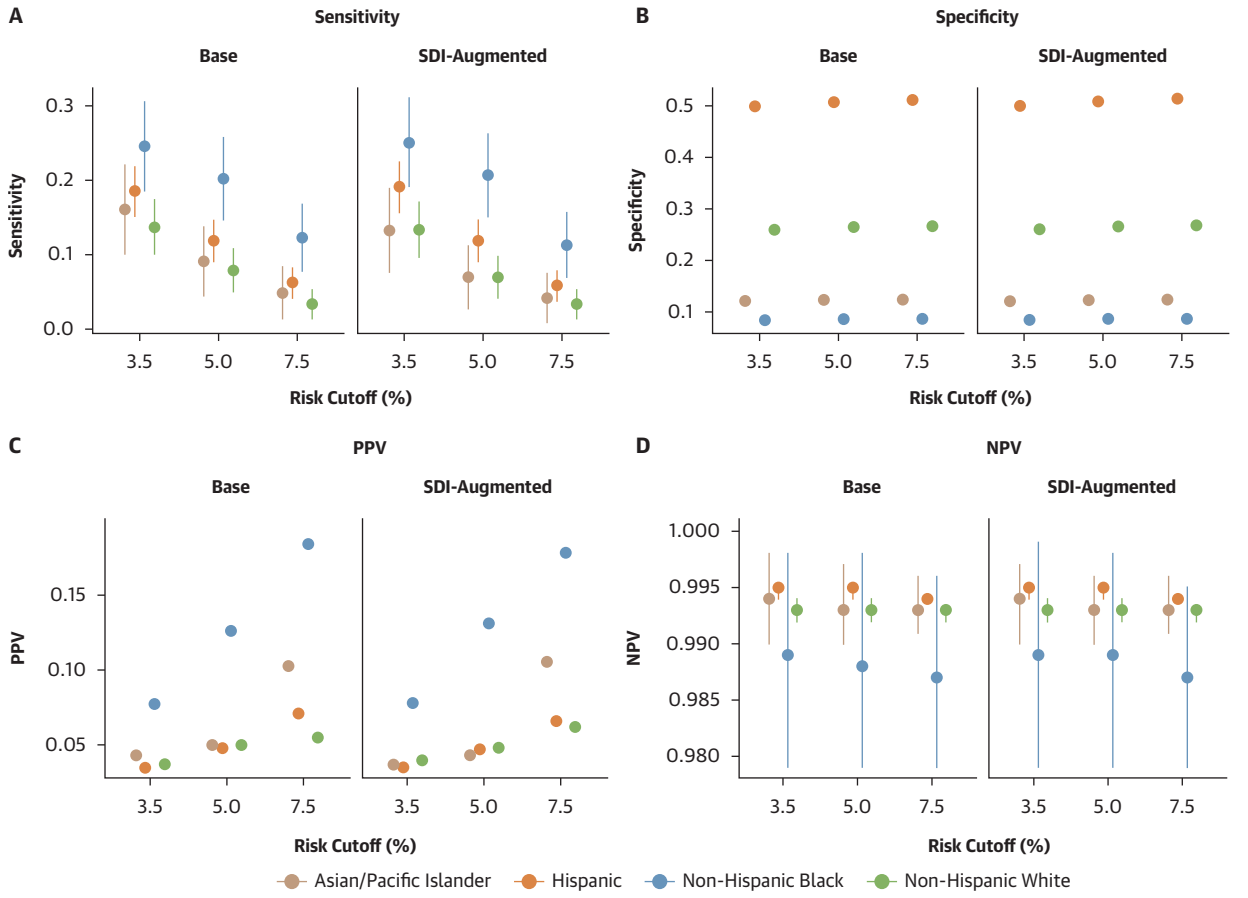


Base and social deprivation index (SDI)-augmented PREVENT-predicted 10-year risk for each outcome ([A] cardiovascular disease, [B] atherosclerotic cardiovascular disease, and [C] heart failure) was split into deciles within each racial and ethnic group. Kaplan-Meier product limit estimators were used to estimate survival, adjusting for right censoring of participants, and subtracted from 1 to estimate the observed risk within each decile. Mean predicted risk in each decile was plotted against observed risk. Vertical lines represent 95% CIs. Base in blue; SDI-Augmented in green.

ethnic groups and cutoffs (0.988-0.995). Results were similar in the SDI-augmented CVD PREVENT model (**Figure 5**) and in the other outcomes (Supplemental Figure 2).

EXPLORATORY ANALYSIS: INDIVIDUALS AGED 20 TO 29 YEARS. Of 80,978 young adults aged 20 to 29 years included in the exploratory analysis, 63.9% were women, 50.7% Hispanic, 30.4% non-Hispanic White, 10.2% Asian/Pacific Islander, and 8.6% non-Hispanic Black (Supplemental Table 8). Distributions of sociodemographic and clinical characteristics across racial and ethnic groups were similar to those in the main analysis sample. Non-Hispanic Black young adults were more likely to experience CVD and HF events (Supplemental Figure 3).

FIGURE 5 Sensitivity, Specificity, Positive Predictive Value, and Negative Predictive Value (KPSC, N = 161,202)



Sensitivity, specificity, positive predictive value, and negative predictive value at prespecified thresholds of base and SDI-augmented PREVENT-estimated 10-year predicted risk of total CVD by racial and ethnic group among young adults aged 30-39 years (KPSC, N = 161,202). Specificity and PPV 95% CIs were ≤±0.01 across all groups and cutoffs. NPV = negative predictive value; PPV = positive predictive value; SDI = social deprivation index.

Discrimination among young adults aged 20 to 29 years was similar to discrimination among those aged 30 to 39 years (Supplemental Table 9). Concordance imparity was higher in the 20- to 29-year-old group compared with the 30- to 39-year-old group across all outcomes (max difference between Harrell's C of 0.12 for the base CVD equation, 0.17 for the base ASCVD equation, and 0.15 for the base HF equation). Calibration slopes were highest in non-Hispanic Black young adults (1.51 [95% CI: 1.20-1.82]) for the base and SDI-augmented CVD equations, indicating underestimation consistent with the primary analysis. Calibration slopes were low for the ASCVD equations across all racial and ethnic groups, although these estimates were imprecise. HF calibration slopes were high for Asian/Pacific Islander (2.12 [95% CI: 1.22-3.01] for base HF equation; 2.58 [95% CI: 1.84-3.32] for SDI-augmented HF equation) and non-Hispanic Black young adults (1.94 [95% CI: 0.88-3.00] for base HF equation; 2.36 [95% CI: 1.32-3.40] for SDI-augmented HF equation) (Supplemental Figure 4). Sensitivity, specificity, PPV, and NPV at prespecified thresholds of base and SDI PREVENT-estimated 10-year predicted risk of CVD, ASCVD, and HF varied by racial and ethnic group and risk cutoff (Supplemental Figure 5). Across all groups and cutoffs, sensitivity was generally low (range: 0.00-0.13) and specificity was generally low-to-modest (range: 0.10-0.51). Specificity was consistently lowest among non-Hispanic Black and Asian/Pacific Islander young adults, and highest among Hispanic young adults. PPV was low (0.00-0.25 across outcomes and cutoffs), reflecting the low prevalence of events, especially ASCVD, in this young population.

NPV was high across all racial and ethnic groups and cutoffs (0.997-0.999).

DISCUSSION

This evaluation of the PREVENT equations in a large, diverse cohort of young adult members of KPSC aged 30 to 39 years demonstrates how these models may perform when broadly applied in real-world health system settings. In this analysis, the PREVENT equations for 10-year risk of total CVD, ASCVD, and HF demonstrated adequate and fair discrimination across racial and ethnic groups, whereby individuals who experienced cardiovascular events had higher predicted risks than those who did not. However, the model demonstrated unfair calibration: risk was slightly underestimated across racial and ethnic groups, with more pronounced underestimation among non-Hispanic Black young adults. These results and calibration challenges persisted in exploratory analyses among young adults aged 20 to 29 years (**Central Illustration**).

The addition of SDI, a measure of area-level social risk factors and one proxy for underlying factors such as structural racism, to the base model did not meaningfully improve performance or algorithmic fairness despite greater prevalences of higher deprivation among Hispanic and non-Hispanic Black young adults compared with Asian/Pacific Islander and non-Hispanic White young adults. The use of race and ethnicity in clinical risk prediction algorithms is heavily debated.[13-17,21,31-37] Self-identified race is a social construct and variably and often imprecisely correlates with biological factors such as genetics, health behaviors, or the experiences of racism.[15] Racial and ethnic disparities in cardiovascular health and disease largely arise because of differences in upstream social exposures and foundational structural determinants that may propagate racism.[38,39] Previous studies demonstrated substantial variation in model performance in the race-specific Pooled Cohort Equations across racial and ethnic subpopulations.[40,41] Although the addition of SDI as an optional predictor in the PREVENT model was an effort to incorporate consideration of SDOH,[30] in our sample the addition of SDI did not impact model performance or improve model fairness metrics despite varying levels of deprivation by race and ethnicity. This finding was consistent with another study,[42] and suggests that using area-level SDI alone may insufficiently capture excess risk caused by individual-level social and structural disadvantage,[43] as SDI may not operationalize the full range of social and structural dimensions for which race often serves as a proxy.[37] Area-level proxies can mask variation of social conditions within neighborhoods. Regions defined by zip code are administrative and do not necessarily reflect individual-level social or community experience within the zip code boundary, and consequently individual privilege or disadvantage may be inadequately captured.[44] A previous study found adding individual-level SDOH to the SDI-augmented equations modestly improved calibration in White and Black participants,[45] suggesting using both area-level SDOH and richer individual-level SDOH variables, such as education, income, or access to care, may more accurately capture a person's lived experience and the mechanisms by which SDOH affect health outcomes. Future work should explore individual-level SDOH that best capture the influence of social adversity to improve model performance and fairness.

Although PREVENT was developed for aged 30 to 79 years, clinicians may inevitably apply it more broadly. In our exploratory analysis applying PREVENT to young adults aged 20 to 29 years, model discrimination was comparable to that seen in the aged 30 to 39 group; however, concordance imparity was higher in the aged 20 to 29 group across all outcomes, suggesting wider disparities in predictive performance between racial and ethnic groups in this younger cohort. Additionally, the calibration slopes indicated substantial underestimation of risk among non-Hispanic Black young adults in CVD and HF outcomes, and among Asian/Pacific Islander young adults in HF outcomes. These findings highlight the brittleness and potential for biased risk predictions when extrapolating PREVENT outside its intended derivation population.

The systematic differences in calibration observed in our study suggest that a global recalibration may be warranted, wherein race and ethnicity-specific validation measures are incorporated into the model selection process. Although local recalibration, where risk prediction model coefficients are refit using a local population, has been suggested as a potential solution to miscalibration,[46-49] there are several issues with its implementation. Insufficient events, particularly in younger subpopulations, present a major limitation. Local recalibration also requires substantial time, resources, and technical expertise for implementation and validation, and is vulnerable to errors such as inconsistent variable ascertainment from EHRs. In some cases, recalibration may not result in improved calibration or performance,[50] or could lead to unequal application of treatment, leading to differential undertreatment or

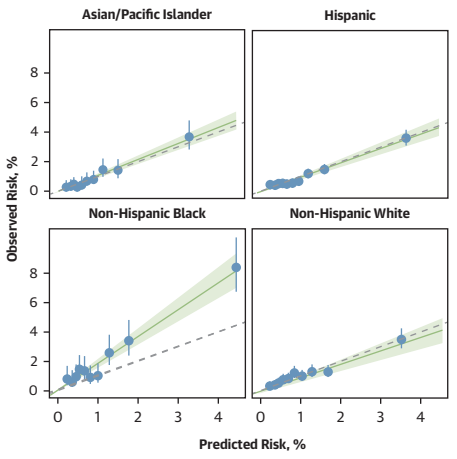
CENTRAL ILLUSTRATION **Performance and Algorithmic Fairness of Base and SDI-augmented PREVENT Equations by Racial and Ethnic Group**

This Analysis Including 161, 202 Members of KPSC Aged 30 to 39 Years Found PREVENT Demonstrated Fair Discrimination Across Racial and Ethnic Groups but Underestimated Risk in Non-Hispanic Black Young Adults, Leading to Unfair Calibration.

Performance and Fairness Metrics of Base PREVENT-Estimated 10-Year Predicted Risk of Total CVD Among Young Adults 30 to 39 Years

	Asian/Pacific Islander	Hispanic	Non-Hispanic Black	Non-Hispanic White
Discrimination	Harrell's C			
	0.72 (0.66, 0.77)	0.70 (0.67, 0.72)	0.72 (0.68, 0.77)	0.68 (0.65, 0.71)
	Concordance Imparity			
	Algorithmically Fair 0.04 (0.02, 0.22)			
Calibration	Calibration Slope			
	1.08 (0.94, 1.22)	0.98 (0.84, 1.11)	1.84 (1.57, 2.10)	0.92 (0.72, 1.11)
	Fair Calibration			
	Algorithmically Unfair (Difference Inconsistency)			

Calibration of Base PREVENT-Estimated 10-Year Risk of Total CVD Among Young Adults 30 to 39 Years



Augmenting the Base PREVENT model with Social Deprivation Index (SDI) did not meaningfully change these results despite differences in SDI across racial and ethnic groups. These results were consistent for the 80,978 members aged 20 to 29 years, with particular under-estimation of 10-year risk of heart failure among 20 to 29-year-old Non-Hispanic Black young adults.

Gauen AM, et al. JACC. 2026;■(■):■-■.

Discrimination was evaluated using Harrell's C. The discrimination fairness metric, concordance imparity, is the difference between the highest and lowest Harrell's C across racial and ethnic groups, with lower concordance imparity indicating a fairer model. Calibration was evaluated using calibration slopes. We plotted calibration by splitting predicted risk into deciles within each racial and ethnic group, then estimating observed risk within each decile using Kaplan-Meier product limit estimators to estimate survival and subtracting from 1; mean predicted risk was plotted against observed risk. We used the statistical fairness metric of fair calibration, which measures whether the model creates probability value-based disparity systematically.[27] Within this metric, difference consistency tests whether the difference between predicted and observed risk was accordant across racial and ethnic groups via pairwise Wilcoxon signed-rank tests. CVD = cardiovascular disease; KPSC = Kaiser Permanente Southern California; SDI = social deprivation index.

overtreatment in at-risk populations. Ultimately, this approach does not address the underlying causal mechanisms of miscalibration, which may stem from differences in health behaviors, clinical practice patterns, or how outcomes are identified and coded.[51] Studies like this one are needed to evaluate the performance of PREVENT and advise on thresholds for treatment that align with actual event occurrence.

Cardiovascular risk prediction models are important for guiding early prevention strategies that can reduce cumulative burden of exposure and premature disease and lower lifetime cardiovascular risk.[11,12,52,53] These prediction models provide a global assessment of cardiovascular health and are incorporated into national guidelines to recommend preventive treatments such as statins or antihypertensive therapy (eg, prescribe statins if 10-year ASCVD risk is >7.5%).[11,52] Systematic cardiovascular risk underestimation can result in missed opportunities to initiate preventive treatment, compounding long-term disparities in cardiovascular outcomes. In our study, calibration, or the extent to which a model correctly estimates absolute risk,[26] was generally good except among non-Hispanic Black young adults, where the model substantially underestimated risk across all 3 outcomes. This group-specific miscalibration reflects algorithmic unfairness that could widen existing disparities in cardiovascular outcomes through undertreatment or less aggressive

prevention strategies in higher-risk populations. As non-Hispanic Black adults experience a far greater burden of cardiovascular events throughout the life course, it is essential that the risk prediction tools we use to dictate treatment decisions are well-calibrated, so current risk thresholds reliably identify high-risk individuals in all groups and support equitable treatment decisions. Beyond this, discrimination, not calibration, may be a more clinically relevant metric for health systems with fixed budgets for preventive services that need to make resource allocation decisions.

Algorithmic fairness metrics may vary across populations. In our sample, PREVENT substantially underestimated risk among non-Hispanic Black young adults but only slightly underestimated risk in the other groups, leading to unfair calibration. However, the PREVENT derivation and validation samples reported similar calibration performance across racial and ethnic groups.[25] Other external evaluations, which included adults 30 to 79 years, found less pronounced calibration differences by race and ethnicity.[42,54,55] These findings suggest that fairness assessments, particularly for calibration, may be context-dependent and influenced by population characteristics. In contrast, discrimination was consistent and fair across racial and ethnic groups in our sample, which aligns with prior evaluations of PREVENT,[25,42,54-56] suggesting fair discrimination may be more robust across settings.

STUDY STRENGTHS AND LIMITATIONS. Strengths of this study include size and diversity of the sample, rigorous event ascertainment, and longitudinal follow-up data. In addition, our algorithmic fairness metrics provide a statistical framework for quantifying differences in model performance across groups while accounting for right-censoring, a limitation of popular fairness metrics such as equalized odds and statistical parity.[27] However, several limitations warrant consideration. First, we could only estimate 10-year risk of total CVD, ASCVD, and HF because of a maximum follow-up time of 15 years. Second, individual-level SDOH were not captured; neighborhood-level indexes capture only certain aspects of SDOH, and their variability may be limited when using regional data such as from KPSC. Third, receipt of care abroad not reported in the United States is not captured. Fourth, we cannot classify individuals as U.S. citizens or residents, limiting our ability to study non-U.S. born populations who may be more transitory. Fifth, EHR-based samples like this one may over-represent young adults with consistent access to care and long-term engagement with the same health system or who remain in the same state, potentially capturing a more privileged subset who may differ systematically from those who relocate or change health systems more frequently. Sixth, although copayment for preventive services may vary by insurance type, all young adults were members of KPSC; therefore, the findings may not be generalizable to populations such as uninsured young adults. Seventh, because of the small proportion of Pacific Islander young adults, we aggregated Asian and Pacific Islander young adults into one racial and ethnic category. However, it is well recognized that Pacific Islanders experience important, clinically significant heterogeneity in health and disease outcomes.[57,58] Future studies should evaluate the performance and fairness of the PREVENT models specifically in Pacific Islander populations. Last, laboratory values were ascertained from testing done at routine annual check-ups; as this is not a mass screening program, it is possible that laboratories are more available among individuals who have a clinical history or individuals more health conscious. Importantly, these data limitations are not unique to this sample, but rather reflect the operational realities large health systems will encounter when PREVENT is implemented in clinical decision support.

CONCLUSIONS

In summary, this large, diverse cohort of young adults demonstrates how the PREVENT equations may perform when applied in real-world clinical settings, reflecting the true operational environment faced by large health systems. Although PREVENT demonstrated fair discrimination, it substantially underestimated cardiovascular risk among non-Hispanic Black young adults but not in other racial and ethnic groups, indicating unfair calibration. These results highlight the need for caution in universally applying fixed thresholds to absolute 10-year CVD risk to guide treatment among young adults. Moreover, the addition of SDI to the PREVENT base model did not improve fairness, despite disparities in SDI across groups, and extrapolation below age 30 years revealed further fragility caused by paucity of events. Guidelines for preventive treatment should consider age and the potential for differential impacts across racial and ethnic groups in their recommendations to encourage early initiation of preventive strategies for longer, healthier lives.

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KEY WORDS algorithmic fairness, cardiovascular disease, epidemiology, primary prevention, risk prediction

APPENDIX For supplemental tables and figures, please see the online version of this paper.