Although atherosclerotic cardiovascular disease (ASCVD) is the leading cause of death among US Hispanic individuals, there is no validated Hispanic ASCVD risk assessment tool. Current guidelines recommend the non-Hispanic White pooled cohort equation (PCE) for 10-year ASCVD risk estimation among Hispanic individuals, while the non-Hispanic Black PCE is recommended when African ancestry is present. The performance of this approach is unknown, and there is no guidance on its clinical implementation.

We evaluated the PCE performance among participants from 2 contemporary, population-based, multi-ethnic US cohorts: the DHS (Dallas Heart Study; ages 18–65 years) and the MESA (Multi-Ethnic Study of Atherosclerosis; ages 45–84 years). Informed consent was obtained, and institutional review boards approved both studies. Participants were stratified by self-reported race of non-Hispanic White, non-Hispanic Black, and Hispanic individuals. MESA Hispanic individuals were stratified by self-reported heritage into a group consisting of Mexican and Central and South American individuals and another consisting of Puerto Rican, Dominican, and Cuban individuals based on hypothesized African ancestry. The DHS did not include Hispanic heritage group classification. We included participants aged 40 to 75 years without prior ASCVD or diabetes mellitus, with low-density lipoprotein cholesterol 70 to 189 mg/dL who were followed for incident adjudicated ASCVD events: fatal and nonfatal myocardial infarctions, strokes, or coronary heart disease deaths. We excluded participants with baseline statin use or missing PCE data. Data will not be made available for purposes of reproducing the results.

We assessed risk discrimination using c-statistics reflecting the area under the receiver operating characteristic curve. We compared mean 10-year PCE-predicted ASCVD incidence to observed 10-year ASCVD events across baseline 10-year risk categories: <5%, 5% to <7.5%, 7.5% to <20%, and ≥20%. We assessed calibration by predicted-to-observed risk (P/O) ratios using the Grønnesby-Borgan goodness-of-fit test with an ideal P/O ratio of 1. We compared P/O ratios using unpaired t-tests. We estimated 95% CI for c-statistics and P/O ratios by dropping the 2.5% smallest and largest values from 1000 sorted non-parametric bootstrap estimates. As an exploratory analysis, we compared risk discrimination using both non-Hispanic White and non-Hispanic Black PCEs among MESA Hispanic heritage groups described...
above. We used SAS 9.4 (SAS Institute Inc., Cary, NC), with 2-sided \( P<0.05 \) considered statistically significant.

We included 1065 Hispanic individuals (86% MESA; mean, 57 years; 53% women), 2159 non-Hispanic White individuals (79% MESA; mean, 56 years; 53% women), and 1674 non-Hispanic Black individuals (65% MESA; mean, 57 years, 58% women). At 10 years, there were 295 incident ASCVD events (Hispanic individuals \( n=61 \), non-Hispanic White individuals \( n=124 \), non-Hispanic Black individuals \( n=110 \)). Similar discrimination was seen with either non-Hispanic White or non-Hispanic Black PCE with no significant sex differences in Hispanic individuals (non-Hispanic White PCE

**Figure.** Baseline PCE variables and risk calibration of the non-Hispanic White and the non-Hispanic Black PCE among participants from the DHS and MESA.

There were statistically significant differences \( P<0.05 \) in mean age, sex, mean systolic BP, median total cholesterol, median HDL cholesterol, smoking prevalence, and antihypertensive use across groups. The non-Hispanic White PCE led to similar risk overprediction in non-Hispanic White individuals and in Hispanic individuals (A and B). The non-Hispanic Black PCE led to similar risk overprediction in non-Hispanic Black individuals and Hispanic individuals (C and D). ASCVD indicates atherosclerotic cardiovascular disease; BP, blood pressure; DHS, Dallas Heart Study; HDL, high-density lipoprotein; MESA, Multi-Ethnic Study of Atherosclerosis; N-H, non-Hispanic; and PCE, pooled cohort equation.
Our analysis was limited by few events in some risk categories and in Hispanic heritage groups. Additionally, Hispanic individuals in the MESA/DHS do not entirely encompass the diversity of Hispanic heritage in the United States, and this may limit the generalizability of our findings. The impact of medication initiation during follow-up is not accounted for.

In conclusion, the PCE performance on Hispanic individuals shows good risk discrimination but suboptimal risk calibration, similar to its performance among non-Hispanic White and non-Hispanic Black individuals. As acknowledged in recent guidelines, better understanding of Hispanic ancestral heterogeneity may lead to more appropriate cardiovascular risk estimates in US Hispanic individuals.

**ARTICLE INFORMATION**

Received October 5, 2020; accepted February 8, 2021.

**Affiliations**

From the Division of Cardiology, Department of Medicine, Duke University Hospital, Durham, NC (K.F.R.); Division of Cardiology, Department of Medicine, Emory University School of Medicine, Atlanta, GA (A.M.); Division of Cardiology, Department of Medicine, University of Texas Southwestern, Dallas, TX (C.A., P.E.G., A.P., A.K., P.H.J.); Section of Cardiovascular Medicine, Department of Internal Medicine, Yale School of Medicine, New Haven, CT (R.K.); Center for Outcomes Research and Evaluation, Yale-New Haven Hospital, New Haven, CT (R.K.); Department of Epidemiology and Population Health, Albert Einstein College of Medicine, Bronx, NY (R.K., C.J.R.); Division of Public Health Sciences, Fred Hutchinson Cancer Research Center, Seattle, WA (R.K.); Johns Hopkins Ciccarone Center for the Prevention of Cardiovascular Disease, Baltimore, MD (M.J.B., R.S.B.); Department of Cardiology, Debailey Heart & Vascular Center, Houston Methodist Hospital, Houston, TX (K.N.); and Division of Cardiology, Montefiore Medical Center, Bronx, NY (C.J.R.).

**Acknowledgments**

The authors thank the other investigators, the staff, and the participants of the MESA and the DHS for their valuable contributions. A full list of MESA investigators and institutions can be found at http://www.mesa-nhlbi.org.

**Sources of Funding**

The Multi-Ethnic Study of Atherosclerosis is supported by contracts N01-HC-95159, N01-HC-95160, N01-HC-95161, N01-HC-95162, N01-HC-95163, N01-HC-95164, N01-HC-95165, N01-HC-95166, N01-HC-95167, N01-HC-95168, N01-HC-95169, and HHSN268201500003I from the National Heart, Lung, and Blood Institute and by grants UL1-RR-024156 and UL1-RR-024156 from the National Center for Research Resources. The Dallas Heart Study was funded by a grant from the Donald W. Reynolds Foundation.

**Disclosures**

Dr Joshi reports grant support from the American Heart Association, NovoNordisk, National Aeronautics and Space Administration; consulting from Regeneron and Bayer; and Equity G3 Therapeutics. The remaining authors have no disclosures to report.

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