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Review

The Role of Risk Equations in Primary Prevention of Atherosclerotic Cardiovascular Disease

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Abstract: Atherosclerotic cardiovascular disease (ASCVD) is the leading cause of death in the United States. The 2018 American Heart Association (AHA)/American College of Cardiology (ACC) blood cholesterol guideline recommends the use of the pooled cohort equations (PCE) for the assessment of 10-year ASCVD risk in participants aged 40–75 years without a history of ASCVD or diabetes, and low-density lipoprotein-cholesterol (LDL-C) ≥70 mg/dL and <190 mg/dL. Recently, AHA released the Predicting Risk of Cardiovascular Disease (CVD) EVENTs (PREVENT) risk equations that calculate both 10- and 30-year ASCVD, CVD, and heart failure (HF) risk in participants aged 30–79 years. This review provides an overview of cardiovascular risk assessment in primary prevention and performance of PREVENT compared to the PCE. PREVENT offers an enhanced approach to cardiovascular risk assessment by integrating markers of cardiovascular, kidney, and metabolic health along with social determinants of health, providing superior calibration and discrimination compared to PCE. PREVENT holds promise for refining statin eligibility and identifying individuals at risk for ASCVD. However, additional research is essential to define clinical thresholds and evaluate its potential in directing the use of emerging preventive therapies.

Keywords: primary prevention; risk prediction; atherosclerotic cardiovascular disease; atherosclerosis; PREVENT

1. Introduction

In the United States, approximately 400,000 deaths are recorded yearly attributed to atherosclerotic cardiovascular disease (ASCVD), making it the leading cause of death in the nation [1]. Preventing first-time and recurrent ASCVD events is a public health priority, with yearly national costs exceeding $120 billion—a figure expected to rise 2.5-fold from 2015–2035 [2]. The main driver of atherosclerosis is low-density lipoprotein-cholesterol (LDL-C), with multiple lines of evidence showing a strong and consistent causal relationship between LDL-C and ASCVD [3,4]. Thus, LDL-C remains a key target for lipid-lowering therapy [4]. The foundation for ASCVD prevention includes lifestyle modifications to optimize cardiovascular health, including a healthy diet, physical activity, sleep, and avoidance of nicotine [5]. In addition to lifestyle modifications, all individuals with a history of ASCVD, diabetes aged 40–75 years, or an LDL-C ≥190 mg/dL require LDL-C-lowering therapy [6]. For all other individuals aged 40–75 years with an LDL-C ≥70 mg/dL, statin allocation is based on a quantitative 10-year ASCVD risk estimation [6,7].

The 2018 American Heart Association (AHA)/American College of Cardiology (ACC) blood cholesterol guideline advocates using the pooled cohort equations (PCE) for assessing cardiovascular risk in primary prevention [6]. The PCE were established in 2013 to estimate the 10-year ASCVD risk in individuals with 40–75 years of age using demographic and risk factor data [6]. Since then, there have been dynamic changes in the

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landscape of cardiovascular disease, increasing the prevalence of cardiovascular disease (CVD) subtypes and the identification of novel CVD risk factors [8]. There has also been a need to assess risk in younger individuals, across diverse racial and ethnic groups, and over a horizon extending beyond 10 years [8]. The Predicting Risk of CVD EVENTs (PREVENT) risk equations were established in 2023 and include newer biomarkers and estimated risk not only for ASCVD, but also for CVD, and heart failure (HF) [8].

This review discusses the importance of risk estimation and clinical implementation of PREVENT risk equations in primary prevention. PubMed was searched for articles relating to the PREVENT risk calculator and PCE using terms "PREVENT risk calculator", "Pooled Cohort Equations," and "cardiovascular risk prediction." Relevant articles published in English were included based on their focus on risk equations in primary prevention of ASCVD. Articles published up to January 2025 were included.

2. PCE and Risk Assessment

The 2018 AHA/ACC blood cholesterol and 2019 ACC/AHA primary prevention guidelines recommend the application of PCE to determine the 10-year ASCVD risk to obtain a basis for adjusting the treatment intensity according the predicted risk [6,7]. Individuals with a history of ASCVD or severe primary hypercholesterolemia (LDL-C ≥190 mg/dL) all require lipid-lowering therapy [6]. Those with diabetes aged 40–75 years with an LDL-C ≥70 mg/dL require statin therapy as well, although PCE can assist in further refining risk [6]. For individuals without diabetes who are 40–75 years of age with an LDL-C ≥70 mg/dL and <190 mg/dL, a 10-year ASCVD risk estimation using the PCE should be calculated to guide primary prevention efforts [6].

The PCE are a set of sex- and race-specific equations derived from four US-based cohorts that capture 10-year ASCVD risk [9,10]. The equations use the following parameters: age, sex, race, total cholesterol, high-density lipoprotein-cholesterol (HDL-C), systolic blood pressure, blood pressure medication use, diabetes, and tobacco use [9]. A 10-year ASCVD risk estimation <5%, 5–<7.5%, 7.5–<20%, and ≥20% correspond to low, borderline, intermediate, and high-risk, respectively [6,7]. A 10-year ASCVD risk ≥7.5% warrants statin therapy [6,7]. If uncertainty remains in those with borderline or intermediate risk, risk enhancers, such as elevated lipoprotein(a), can be considered to help refine risk [6,7]. Finally, the best tool for refining the risk in cases where there are uncertainties is coronary artery calcium (CAC), a low-dose radiation, and non-contrast computed tomography [6,7]. Several studies have documented the "power of 0," such that the absence of CAC is associated with low ASCVD risk [11–13]. In a multi-ethnic atherosclerosis study by Nasir et al., the authors found that a CAC score of 0 reclassified 49% of individuals in borderline or intermediate risk groups based on PCE to below the threshold for statin initiation [14]. Although a CAC score of zero is associated with good short- to intermediate-term prognosis, individuals with a CAC score of zero who have cardiovascular risk factors—such as diabetes, smoking, or a family history of premature ASCVD—remain at risk for incident events over the long term. This observation underscores the importance of addressing modifiable risk factors even in the absence of detectable coronary calcification. Thus, the 2018 AHA/ACC blood cholesterol guideline supports delaying statin initiation in those aged 40–75 with an LDL-C ≥70 mg/dL and <190 mg/dL and a 10-year ASCVD risk ≥5% and <20%, without diabetes, tobacco use, or strong family history of premature ASCVD, when uncertainty regarding statin initiation exists [6,7].

Even though PCE has been validated and widely adopted, there are concerns regarding their performance in risk estimation and use in clinical practice. The PCE can only be used in those aged 40–75 years and there is a need for estimation earlier in adulthood, given that data has indicated that longer exposure to elevated LDL-C correlates with a higher risk of ASCVD [3]. Additionally, in multiple cohorts, the PCE can overestimate the risk [15,16]. PCE derivation used cohort data that enrolled participants from 1968 to 1990 and does not appropriately capture modern CVD and CVD risk factor prevalence, likely explaining the overestimation of risk [16]. Furthermore, the PCE were established using only white and black populations and may over or underestimate risk in non-white and non-black populations [9,10]. Finally, the PCE fail to consider social determinants of health, which are tightly linked with ASCVD risk and overall cardiovascular health [15,17]. Therefore, studies have shown that PCE underestimates the risk in populations with lower socioeconomic status [15]. To address these concerns regarding risk prediction using the PCE, the AHA developed a contemporary cardiovascular risk prediction tool for primary prevention named as PREVENT [8].

3. PREVENT Risk Calculator

AHA's sex-specific and race-free PREVENT risk calculator incorporates comprehensive measures of cardiovascular, kidney, and metabolic health. PREVENT calculates 10- and 30-year CVD, ASCVD, and HF risk for individuals 30–79 years of age [8]. Moreover, a place-based social disadvantage score was added to highlight the importance of capturing social determinants of health [8]. The following parameters are included in the

PREVENT risk equation: age, sex, total cholesterol, HDL-C, lipid-lowering medication use, systolic blood pressure, anti-hypertensive medication use, body mass index, estimated glomerular filtration rate, diabetes, and current smoking [8]. Additionally, urine albumin creatinine ratio, hemoglobin A1c, and social deprivation index (SDI), can be included to enhance risk prediction if available [8]. Incorporating the SDI into PREVENT is an important addition highlighting the interplay between socioeconomic factors and cardiovascular risk. By leveraging place-based social disadvantage scores, PREVENT estimates risk more accurately in underserved populations and addresses disparities in ASCVD outcomes. These advances underscore the need for equitable prevention strategies that consider environmental and social contexts, potentially guiding resource allocation and public health interventions to high-risk communities.

The derivation sample for PREVENT included 3,281,919 participants from 25 data sets and the validation cohort consisted of 3,330,085 participants from an additional 21 data sets [8]. Over a follow-up of almost 5 years, there were a total of 106,661 and 104,854 CVD events, 66,503 and 67,902 ASCVD events, and 59,350 and 55,966 HF events, in the derivation and validation samples, respectively [8]. The PREVENT model performed quite well for total CVD events with a median C statistic of 0.794 and 0.757 and calibration slope of 1.03 and 0.94 for females and males, respectively [8]. Comparatively, the median C statistic for total CVD events using PCE was 0.789 and 0.747 and calibration slope was 0.84 and 0.67 for females and males, respectively (Table 1) [8]. Similar findings were seen for ASCVD and HF outcomes and across different races and ethnicities [8]. The lower calibration slope for PCE highlights the tendency for PCE to overestimate risk, while the calibration for PREVENT was close to 1, indicating a well-calibrated model [8].

Table 1. Comparison of PREVENT versus PCE.

Characteristic	PREVENT	PCE
Year [6,9]	2023	2013
Guideline Endorsed [6]	Not currently	AHA/ACC Blood Cholesterol Guideline
Variables [6,8]	Age, sex, total cholesterol, HDL-C, lipid-lowering medication use, SBP, anti-hypertensive medication use, BMI, eGFR, diabetes, and current smoking	Age, sex, race, total cholesterol, HDL-C, SBP, blood pressure medication use, diabetes, and tobacco use
Optional Variables [8]	uACR, HbA1c, SDI	None
C-statistic and calibration slope (CVD) [8]	Female: C-statistic 0.794, calibration slope 1.03 Male: C-statistic 0.757, calibration slope 0.94	Female: C-statistic 0.789, calibration slope 0.84 Male: C-statistic 0.747, calibration slope 0.67
C-statistic and calibration slope (ASCVD) [8]	Female: C-statistic 0.774, calibration slope 1.09 Male: C-statistic 0.736, calibration slope 1.04	Female: C-statistic 0.772, calibration slope 0.54 Male: C-statistic 0.733, calibration slope 0.50
C-statistic and calibration slope (HF) [8]	Female: C-statistic 0.830, calibration slope 1.00 Male: C-statistic 0.809, calibration slope 0.89	Female: C-statistic 0.810, calibration slope 0.51 Male: C-statistic 0.791, calibration slope 0.37

Differences between PREVENT and PCE risk equations. C-statistic presented as median. PREVENT, Predicting Risk of CVD EVENTs; PCE, pooled cohort equations; AHA, American Heart Association; ACC, American College of Cardiology; HDL-C, high-density lipoprotein-cholesterol; SBP, systolic blood pressure; BMI, body mass index; eGFR, estimated glomerular filtration rate; uACR, urine albumin creatinine ratio; HbA1c, hemoglobin A1c; SDI, social deprivation index; CVD, cardiovascular disease; ASCVD, atherosclerotic cardiovascular disease; HF, heart failure, and C-statistic, concordance-statistic.

Since the release of the PREVENT risk calculator, there has been ongoing research assessing the performance of PREVENT using the National Health and Nutrition Examination Survey (NHANES) [18]. In a cross-sectional study by Anderson et al. that included 3795 participants from NHANES, the authors found the mean 10-year ASCVD risk to be 8.0% and 4.3% using PCE and PREVENT equations, respectively [18]. In this study, 17.3 million adults would no longer qualify for statin therapy using the PREVENT risk calculator, who would otherwise be eligible based on PCE [18]. The reduced mean 10-year ASCVD risk estimation with PREVENT represents an intentional recalibration that reflects contemporary epidemiologic data [8]. Although this recalibration may exclude millions of individuals from statin eligibility, it is consistent with the model's enhanced calibration and discrimination metrics [8,18]. However, this shift raises questions about whether new thresholds for initiating preventive therapies should be established to balance precision and population-level benefits. Clinical trials and

real-world studies are needed to validate these thresholds and determine their impact on ASCVD outcomes, particularly in individuals classified as low or borderline risk by PREVENT, but high risk by PCE. Such research will be critical in avoiding unintended consequences, such as undertreatment of at-risk populations.

Another slightly larger cross-sectional study of 7765 participants from NHANES found the mean 10-year ASCVD risk to be 9.0% and 4.6% using PCE and PREVENT equations, respectively [19]. Approximately 14.3 million adults recommended for statin therapy by PCE would no longer qualify based on PREVENT risk equations [19]. The authors concluded that the lower treatment eligibility for primary prevention efforts based on PREVENT could lead to 107,000 incident myocardial infarctions or strokes for 10 years [19]. While analyses of NHANES provide valuable insights into the implications of adopting PREVENT, its cross-sectional design limits causal inference. Furthermore, differences in population characteristics, follow-up duration, and methodology between derivation and validation cohorts underscore the need for longitudinal studies.

A prognostic study from NHANES investigated the performance of PREVENT and PCE in detecting fatal CVD events using 24,582 participants over a 10-year follow-up [20]. In this study, both PREVENT and PCE had excellent discrimination, with a C statistic of 0.890 and 0.880, respectively [20]. PREVENT had slight underfitting of the model, while PCE exhibited overfitting, with a calibration slope of 1.13 and 0.77, respectively [20]. The study used the adjusted CVD mortality from the National Death Index; and thus, nonfatal CVD events were not included, which may explain underfitting of the PREVENT model. Inclusion of adjudicated fatal and non-fatal events in longitudinal studies could better elucidate the long-term benefits and potential unintended consequences of adopting PREVENT over PCE in clinical practice.

Recently, the prevalence of sodium-glucose cotransporter-2 inhibitors (SGLT2i) and glucagon-like peptide-1 receptor agonists (GLP-1RA) in NHANES was assessed using PREVENT [21]. The authors included 1668 adults with diabetes in primary prevention, aged 30–79 years, and found the overall use of SGLT2i and/or GLP-1RA to be 8.9% [21]. Interestingly, the authors found the absolute percent of SGLT2i and/or GLP-1RA use in those with a 10-year CVD PREVENT risk of <7.5%, 7.5–<20%, and ≥20% risk to be 9.9%, 10.9%, and 4.3%, respectively [21]. In this study, the overall use of SGLT2i and GLP-1RA were relatively low and use was paradoxically higher in lower-risk groups [21]. These results underscore the need for improved prevention efforts in high-risk individuals with diabetes [21]. The PREVENT risk calculator may help guide prevention efforts, especially with newer preventative therapies.

Future studies are needed to classify the 10- and 30-year PREVENT risk categories and thresholds for initiating primary prevention therapies for CVD, ASCVD, and HF. Additionally, more data is needed to recommend newer preventative therapies for CVD, ASCVD, and HF, such as SGLT2i and GLP-1RA, based on PREVENT risk. CAC testing has proven indispensable in refining ASCVD risk in intermediate-risk populations using PCE, particularly through the ‘power of zero.’ However, its potential role within PREVENT’s risk stratification framework remains unexplored. Future studies should assess whether CAC can enhance the predictive value of PREVENT, particularly in borderline and intermediate-risk categories. Such investigations could establish whether a zero CAC score retains its derisking capacity in populations assessed with PREVENT and identify thresholds that may justify advanced imaging modalities.

4. Conclusion

PREVENT represents a significant advancement in cardiovascular risk prediction by broadening the scope of assessed risk factors, incorporating social determinants of health, and providing both short- and long-term risk estimates. Compared to PCE, its enhanced calibration and discrimination metrics suggest it may better tailor preventive strategies for diverse populations. However, the clinical implications of its lower statin eligibility thresholds warrant careful consideration to balance undertreatment risks. Future studies should focus on establishing optimal thresholds for therapeutic interventions, evaluating the cost-effectiveness of using PREVENT in routine care, and exploring its integration with CAC scoring to refine risk stratification further. Finally, although risk equations provide valuable guidance, clinical assessment is needed to tailor the preventive strategies to the state of individual patients. Although these equations are useful for determining when to start statin therapy, they should complement, not replace, personalized, patient-centered discussions. For instance, younger patients with prolonged exposure to elevated LDL-C or those with strong family histories of premature ASCVD may benefit from preventive therapies despite falling below standard thresholds. Similarly, older adults with competing risks might require a more conservative approach. Shared decision-making must take into account patient preferences, socioeconomic factors, and family history to ensure that preventive strategies are personalized and aligned with individual needs and priorities. This personalized approach not only fosters better patient engagement but also enhances the overall effectiveness of preventive care.

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Data Availability Statement

This study did not require institutional review board approval as it is a review article and did not involve human subjects research or the collection of new data.

Conflicts of Interest

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Use of AI and AI-Assisted Technologies

These were not used by the authors submitting this paper.

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