

ASCVD (Atherosclerotic Cardiovascular Disease) 2013 Risk Calculator from AHA/ACC

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Abstract

This calculator determines the 10-year risk of hard ASCVD, i.e. myocardial infarction, stroke, or death due to coronary heart disease or stroke, and can help guide decision-making on statin use.

I. HOW TO USE

When to Use

Patients at risk for atherosclerotic cardiovascular disease (ASCVD).

Pearls / Pitfalls

- In 2013 the American College of Cardiology (ACC) and the American Heart Association (AHA) released new guidelines for the evaluation and treatment of cholesterol in order to reduce the risk of atherosclerotic cardiovascular disease (ASCVD).
- This calculator provides a simplified way to follow the ASCVD treatment recommendations for patients without known ASCVD and with LDL levels between 70-189 mg/dL / 1.81-4.90 mmol/L). Our ASCVD Risk Algorithm is a step-wise approach for all adult patients – including those with known ASCVD.
- The treatment algorithm proposed by the ACC/AHA suggests aggressive treatment for many patients, but specifically notes that patients with known ASCVD and patients with extreme LDL levels (≥ 190 mg/dL / 4.92 mmol/L) are at the highest risk; it also provides the “intensity” of statin treatment based on patients’ predicted risk levels.

Points to keep in mind

- This score has been effectively superseded by the PREVENT risk prediction tool which was released in 2023.
- While the score was developed and validated in a large population from the United States, several studies have suggested that the risk calculator substantially over-estimates 10-year risk – even though some studies have suggested that its risk estimates are accurate. The score’s accuracy in patients outside the United States has also been brought into question.
- Statins are highly emphasized in the guidelines and recommendations, but lifestyle modifications are likely just as – if not more – important to ASCVD risk.
- Commonly referred to as the Pooled Cohort Equation.
- The PCE is not applicable to patients with atrial fibrillation or those older than 79 as they were excluded in the original derivation and validation study.

Why Use

The ASCVD Risk Estimate is a standardized guideline to predict risk and recommend management strategies for those at risk of hard ASCVD (i.e. myocardial infarction, stroke, or death due to coronary heart disease or stroke).

II. NEXT STEPS

Advice

When Considering Starting Statins

First, always engage in a clinician-patient discussion of the potential for ASCVD risk reduction, adverse effects, drug-drug interactions, and patient preferences. Consider:

- Potential for ASCVD risk-reduction benefits.
- Potential for adverse effects and drug-drug interactions, with special consideration for pre-existing conditions such as impaired liver/renal function.
- Heart-healthy lifestyle.
- Management of other risk factors.
- Patient preferences.

[See Section 5 of the relevant 2018 American guidelines for a discussion and recommendations about statin safety. Also see Table 3 of the same guidelines for summary of grossly equivalent statin intensities for different statins at different doses. \(Reproduced in the Facts and Figures section here.\)](#)

When Considering or Using High-Intensity Statins

The guidelines recommend treating clinicians consider:

- Multiple or serious comorbidities, such as impaired renal or hepatic function.
- A history of previous statin intolerance or muscle disorders.
- Unexplained elevated levels of alanine transaminase greater than three times the upper limit of normal.
- Patient characteristics or concomitant use of medications that affect statin metabolism.
- Age older than 75 years.

The risk of statin-related adverse effects are generally intensity-dependent. It is thus especially important to engage in shared decision-making with all patients who may require high-intensity statins.

Additional factors that are ASCVD risk enhancers per the 2018 American guidelines

- Family history of premature ASCVD
- Persistently elevated LDL-C levels at or above 160 mg/dL (4.1 mmol/L)
- Chronic kidney disease
- Metabolic syndrome
- Conditions specific to women (e.g. preeclampsia, premature menopause)
- Inflammatory diseases (especially rheumatoid arthritis, psoriasis, HIV)
- Ethnicity (e.g. South Asian ethnicity)

- Persistently elevated triglycerides levels at or above 175 mg/dL (2.0 mmol/L)
- And in selected individuals if measured:
- High-sensitivity C-reactive protein (hsCRP) levels at or above 2.0 mg/L
- Lp(a) levels above 50 mg/dL (125 nmol/L)
- ApoB at or above 130 mg/dL
- Ankle-brachial index <0.9

When Monitoring Statin Effects and Side Effects

- Assess adherence, response to therapy, and adverse effects within 4 to 12 weeks following statin initiation or change in therapy.
- Measure fasting lipid levels.
- Do not routinely monitor alanine transaminase or creatine kinase levels unless symptomatic.
- Screen and treat type 2 diabetes according to current practice guidelines; heart-healthy lifestyle habits should be encouraged to prevent progression to diabetes.

III. EVIDENCE

Evidence Appraisal

The 2013 ACC/AHA ASCVD risk score was developed to provide, at the time, an updated ASCVD risk stratification tool that uses commonly available risk factor variables and that makes conscious efforts to better consider / represent African Americans. Commonly referred to as the Pooled Cohort Equation (PCE), the score was developed from 5 large prospective cohort studies in the United States, with a total sample size of 24,626 in the pooled validation cohort. External validation was performed on 3 similar studies, with a total pooled sample size of 37,759. Patients aged 40 to 79 who were apparently healthy, African American or White, and free of a previous history of MI (recognized or unrecognized), stroke, congestive heart failure, percutaneous coronary intervention, coronary bypass surgery, or atrial fibrillation were included. Importantly, those who were older than 79 and those who had a known history of atrial fibrillation were excluded, meaning that the estimates are not applicable to these patient groups. While the former age-based exclusion is widely known, clinicians may not be as aware of the latter – this serves as a reminder to be especially cautious with the use and interpretation of PCE in these patients.

The authors provided detailed discussions about the choice of outcome (a composite of myocardial infarction, stroke, and death due to coronary heart disease or stroke) in the original paper's supplementary materials. In particular, heart failure was explicitly mentioned to have been considered as a component of the outcome, but was even-

tually dropped due to heterogeneity in outcome definition / adjudication among the included cohorts. The authors also mentioned that additional covariates, e.g. BMI, diastolic blood pressure, eGFR, and statin use, were initially explored for inclusion in the model, but were dropped as they did not significantly improve discrimination when added to the model.

While the choice of outcome was sensible at the time that the PCE was developed and was grossly consistent with the definition of major adverse cardiovascular events used in many trials, more contemporary tools such as the European SCORE2 family of risk scores and the American PREVENT risk tool have both opted for broader cardiovascular outcomes. Thus, as clinicians transition from PCE to PREVENT / other newer risk tools, this distinction in the predicted outcome must be noted.

Notably, the outcome led to a competing risk scenario where deaths due to causes other than coronary heart disease and stroke would prevent observation of further events and therefore constitute a competing event. Despite this, the model was constructed using a Cox proportional hazards model, essentially taking a cause-specific approach to this competing risk scenario. This was probably due to limitations with computing power at the time of development, but the cause-specific approach is more appropriate for etiological questions, instead of the prognostic question – which is the case here and which usually requires more computationally intensive approaches such as the Fine and Gray sub-distribution model. One important consequence of this is a predisposition to overestimation of risks – something which would subsequently prove to be an issue.

In the original paper, internal validation of the score was performed using a 10x10 cross-validation, achieving C-statistics between 0.713 and 0.818 in different subsets (stratified by ethnicity [White vs African Americans] and sex [males vs females]), with calibration plots showing slopes close to 1 for all subsets. In external validation, discrimination was markedly worse for all subsets across all validation cohorts (C-statistics between 0.556 and 0.768), with overestimation of event risks observed in all subsets across all validation cohorts.

Subsequently, multiple studies have externally validated the PCE. [A systematic review and meta-analysis published in 2019](#) pooled 61 validations (30 in men and 31 in women), demonstrating a pooled C-statistic of 0.74 in women and 0.70 in men – both of which were higher than other Framingham-based scores (ATP III and Wilson score). However, pooled analysis of calibration demonstrated significant overestimation of risks in both sexes, with pooled observed-expected (O/E) ratios of 0.66 in men and 0.76 in women. This has continued to be echoed by later, more contemporary studies, including a [2020 study by Khera et al](#) using data from 8 US cohorts – they observed that calibration was especially bad in high-risk patients and those with high BMI. Nonetheless, there

were also studies that found the opposite, i.e. underestimation of risks, such as a [2019 Austrian study by Wallich et al](#), although these studies appeared to be far fewer and less common than those that showed overestimation. However, reassuringly though, a [decision curve analysis by Qureshi et al](#) has shown that there was net benefit in replacing the older Framingham cohort-based scores with the PCE.

It is important to note that the PCE included ethnicity (African American or not) in the model. This was intentional as the authors noted that previous, Framingham-based scores underrepresented African Americans. However, other ethnic minorities in the US were not given specific consideration, and some studies, such as a [2022 study by Mantri et al](#), demonstrated that risks may be significantly underestimated in some ethnicities such as south Asians in the United States. Overall, however, there has been criticism on the inclusion of race/ethnicity in PCE and other clinical algorithms. PCE tends to produce higher risk estimates in African Americans and thus may lead to over-intervention which, considering that race is a social construct, may be undesirable and contribute to health inequities. Some studies have also suggested that disparities in social determinants of health may be a critical driver of racial differences in cardiovascular risks. Race and ethnicity were therefore removed from the latest cardiovascular risk score developed by the AHA and ACC (the 2023 PREVENT risk prediction tool), instead including an optional social deprivation variable instead. Nonetheless, it is important to bear in mind that studies showing issues with calibration in non-American cohorts (e.g. [a Malaysian study by Chia et al](#)) may not actually be due to ethnic differences in cardiovascular risks (which exist), but rather be due to geographical differences in cardiovascular risks which are often prominent. The latter was explicitly considered by the European Society of Cardiology's SCORE2 family of risk scores. Probably as the PCE and PREVENT were designed with a single country (the United States) in mind, this was not considered by either PCE or PREVENT. This does, however, mean that clinicians outside of the United States should exercise caution when interpreting PCE estimates for their local patients.

Formula

Scoring information is available in [Appendix 7 in the Goff, et al. 2013 paper](#).

Facts & Figures

- These estimates may underestimate the 10-year risk for some race/ethnic groups, including American Indians, some Asian Americans (e.g., of south Asian ancestry), and some Hispanics (e.g., Puerto Ricans).

- It may overestimate the risk for some Asian Americans (e.g., of east Asian ancestry) and some Hispanics (e.g., Mexican Americans).
- Because the primary use of these risk estimates is to facilitate the very important discussion regarding risk reduction through lifestyle change, the imprecision introduced is small enough to justify proceeding with lifestyle change counseling informed by these results.

Optimal Risk Factors

For the comparison of optimal risk factors, these were defined by the following specific risk factor numbers for an individual of the same age, sex and race:

- Total cholesterol of 170 mg/dL
- HDL-cholesterol of 50 mg/dL
- *Untreated* systolic blood pressure of 110 mm Hg
- No diabetes history
- Not a current smoker

US Preventive Services Task Force (USPSTF) Guidelines

In 2016, the US Preventive Services Task Force (USPSTF) made similar but slightly different recommendations for adults without a history of cardiovascular disease (CVD) to use a low- to moderate-dose statin for the prevention of CVD events and mortality when all of the following criteria are met:

1. Age 40 to 75 years
2. 1 or more CVD risk factors (ie, dyslipidemia, diabetes, hypertension, or smoking)
3. Calculated 10-year risk of a cardiovascular event of 10% or greater (B recommendation)

The USPSTF gave a B recommendation—indicating high certainty that the benefit is moderate or moderate certainty that the benefit is moderate to substantial—for starting low- to moderate-dose statins in adults ages 40 to 75 years without a history of cardiovascular disease (CVD) who have one or more CVD risk factors and a 10-year CVD risk of 10% or greater.

The USPSTF dropped its level of endorsement to C for adults with a lower 10-year risk (7.5%-10%) with 1 or more CVD risk factors and made no recommendations for adults 76 years of age and older, explaining that there was insufficient evidence for this age group.

[These recommendations have subsequently been maintained in the 2022 version.](#)

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LITERATURE

Original/Primary and initial validation

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Intensity of Statin Therapy

Type of Statin	Taken Daily, Average LDL Lowering Effect	Types of Medication
High-intensity statin therapy	Approximately $\geq 50\%$	Atorvastatin 40–80 mg
		Rosuvastatin 20–40 mg
Moderate-intensity statin therapy	Approximately 30% to $< 50\%$	Atorvastatin 10–20 mg
		Rosuvastatin 5–10 mg
		Simvastatin 20–40 mg
		Pravastatin 40–80 mg
		Lovastatin 40 mg
		Fluvastatin XL 80 mg
		Fluvastatin 40 mg
Low-intensity statin therapy	Approximately $< 30\%$	BID Pitavastatin 2–4 mg
		Simvastatin 10 mg
		Pravastatin 10–20 mg
		Lovastatin 20 mg
		Fluvastatin 20–40 mg
		Pitavastatin 1 mg

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