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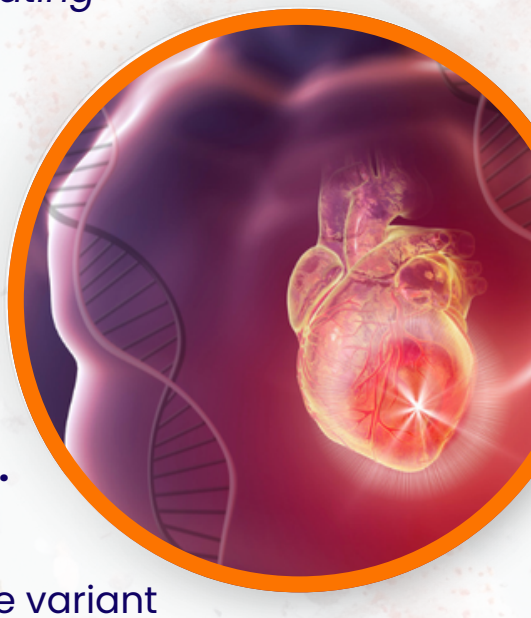
OMNIINSIGHT™ CORONARY ARTERY DISEASE PROGRAM

*A comprehensive, MENA-focused approach integrating **polygenic risk scores (PRS)**, **monogenic variant testing, clinical factors, and lifestyle** to deliver individualized coronary artery disease prevention strategies.*



WHY PRS MATTERS

- Traditional genetic testing **alone is not enough.**
- PRS modulates the penetrance of **single-gene mutations,** meaning two patients with the same variant may have very different levels of risk.
- Uses a clinically validated multi-ancestry PRS, calibrated for diverse populations including **the MENA region.**

PERSONALIZED PREVENTION

Results are translated into **a clinically actionable care plan** aligned with best-practice guidelines, enabling personalized recommendations for **screening, lifestyle modification, medical interventions, and genetic counseling.**

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MONOGENIC RISK COVERAGE

Monogenic Risk Coverage identifies high-impact pathogenic variants across **NCCN** recommended genes, including: **LDLR, APOB, PCSK9, LDLRAP1.**

POWERED BY BLENDED GENOME EXOME

The innovative genomic technology is developed in collaboration **with Broad Clinical Labs at Harvard and MIT**, the program combines **95.5× Whole-Exome Sequencing (WES)** with **genome-wide lcWGS** to detect both rare mutations and polygenic risk with high precision.

Testing is performed through **CAP/CLIA-accredited laboratories,** with analytics support from **Allelica**, (MGB/LMM), and regional collaboration with **Novo Genomics.**

Equipped with most precise risk assessment available.

SCAN ME

Get started today
info@avigena.com
+1 617 412 5580

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Avigena OmniInsight™(AVI-OI™)

8 Cardiovascular Genomic Risk Stratification (PRS-Based)

Genomics-guided risk prediction and prevention

Avigena OmniInsight™ – Eight (8) Cardiovascular Diseases (AVI-OI-CVD™) **IPC: AVI-OI-CVD-01**	**Test Name** 8 Cardiovascular Genomic Risk Stratification (PRS-Based)	Estimates inherited susceptibility to common cardiovascular diseases, including CAD and cardiometabolic diseases, to support preventive and therapeutic decision-making.
	Polygenic Risk Score (PRS) Genome-wide polygenic susceptibility assessment • **Coronary artery disease** • Atrial fibrillation • Type 2 diabetes • Lipoprotein(a) levels • Hypercholesterolemia • Hypertension • Thoracic aortic aneurysm risk • Venous thromboembolism	

Clinical Intent: Risk prediction and preventive management in asymptomatic individuals for population risk stratification and primary prevention; not intended for evaluation of suspected monogenic cardiovascular disorders.

Technology: Blended Genome-Exome™ sequencing (95.5× WES + genome-wide lcWGS) enabling genome-wide polygenic risk assessment, performed in a CAP/CLIA-certified laboratory in collaboration with the Broad Institute (MIT/Harvard). PRS models validated across multi-ancestry populations and calibrated for Middle Eastern ancestry. ***Clinical Output:*** Integrates PRS with clinical and lifestyle factors to generate an integrated genomic report, personalized prevention plan, and virtual genetic counseling via the Avigena OmniInsight™ platform. Supports preventive therapy decisions, screening intensity selection, and lifestyle intervention planning.

Clinical Indications

Population	Description
General prevention assessment (adults ⩾18 yrs)	Individuals seeking personalized cardiovascular risk assessment.
Borderline or intermediate cardiovascular risk (adults 40–79 yrs)	Statin decision support and refinement beyond traditional calculators
Early prevention (youth 10–18 yrs)	Family history of cardiometabolic disease or presence of obesity, dyslipidemia, or diabetes — for lifetime risk stratification and preventive counseling.

Logistics

- Sample Collection: Non-invasive saliva collection kit
- Turnaround Time: 6–8 weeks
- Laboratory: CAP/CLIA-certified laboratory in collaboration with the Broad Institute (MIT/Harvard)
- CPTCodes: 81599, 96040

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Avigena OmniInsight™ (AVI-OI™) - Comprehensive Cardio Diseases

Genomics-guided comprehensive cardiac risk prediction and prevention

Avigena OmniInsight™ – Comprehensive Cardio Diseases (AVI-OI-CCVD™) IPC: AVI-OI-CCVD-02	**Test Name**	**Description**
	Comprehensive Cardio Genomic Risk Evaluation (PRS + Monogenic Analysis)	Combines polygenic risk stratification with targeted monogenic analysis when Mendelian inherited cardiac disease is suspected
	Polygenic Risk Score (PRS)	**Included Genes**
	Genome-wide polygenic susceptibility assessment Polygenic risk assessment for CAD, lipids, hypertension, and arrhythmia. • **Coronary Artery Disease** • Lipids andlipoprotein(a) • Atrial Fibrillation • Hypertension	*Familial Hypercholesterolemia:* LDLR, APOB, PCSK9, LDLRAP1 *Aortopathy:* FBN1, TGFBR1, TGFBR2, SMAD3, ACTA2, MYH11, COL3A1 *Cardiomyopathy:* MYH7, MYBPC3, TTN, LMNA, FLNC, RBM20, BAG3 *Channelopathy:* KCNQ1, KCNH2, SCN5A, RYR2, CALM1-3

Clinical Intent: Risk prediction and preventive management, with evaluation for mendelian inherited cardiac disorders when clinically indicated. Not a substitute for specialist-directed cardiogenetic evaluation when a specific inherited syndrome is strongly suspected.

Technology: Blended Genome-Exome™ sequencing (95.5× WES + genome-wide lcWGS) enables simultaneous analysis of rare monogenic variants and genome-wide polygenic variation. Polygenic risk scores are derived from hundreds to millions of SNPs using ancestry-specific models (e.g., CAD ~10^3–10^6 variants; atrial fibrillation ~4×10^5; hypertension ~2×10^5; lipid traits ~10^2–10^5), performed in a CAP/CLIA-certified laboratory in collaboration with the Broad Institute (MIT/Harvard).

Clinical Output: Integrates PRS, monogenic variants, clinical and lifestyle factors to generate an integrated genomic report, personalized prevention roadmap, and virtual genetic counseling via the Avigena OmniInsight™ platform. Supports early preventive therapy decisions (lipid-lowering, imaging, and specialist referral).

Clinical Indications

Population	Description
Adults (18–79 yrs)	Assessment of inherited mendelian cardiac risk and preventive risk stratification, including coronary artery disease.
Situations suggesting monogenic cardiovascular disease (comprehensive genomic evaluation is appropriate).	Order with LDL ⩾190 mg/dL in adults or ⩾160 mg/dL in children; Suspected familial hypercholesterolemia; Family history of sudden cardiac death; Myocardial infarction at a young age (30s–40s) in patient or first-degree relative
Pediatric group (10–18 yrs)	Early risk assessment in the presence of strong family history or inherited predisposition.

Logistics

- Sample Collection: Non-invasive saliva collection kit.
- Turnaround Time: 6-8 weeks
- Laboratory: CAP/CLIA-certified laboratory in collaboration with the Broad Institute
- (MIT/Harvard) CPT Codes: 81599, 96040

Cardiovascular Test Selection Guide

	CVD — 8 Common Cardiovascular & Metabolic Genomic Risk Test	**CCARD** — Comprehensive Cardiac Genomic Risk Evaluation
Type of assessment	Population preventive genomic assessment	Preventive genomic assessment + monogenic evaluation
Genetic analysis	Polygenic risk only (PRS)	Polygenic risk (PRS) + targeted monogenic variants
Coverage	Broader cardiometabolic trait coverage	Fewer traits, more focused evaluation
When to use	No clinical features suggest a monogenic condition	Phenotype raises suspicion of a monogenic condition; single gene mutation

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THE WEIGHT OF ILLNESS IS HEAVY.
PREDICTING AND PREVENTING IT IS PRICELESS.

OmniInsight™ uses advanced genomic intelligence, combining PRS + monogenic variants + clinical and lifestyle data to predict heart disease and cancer risk years before it appears.

One saliva test. Your inherited risk. Personalized prevention.

Polygenic Risk Scores (PRS), a breakthrough genomic tool shaping the future of public health is the new foundation of modern preventive medicine.
IT'S TIME TO SHIFT FROM TREATING DISEASE TO PREVENTING IT.

OmniInsight™ for Breast Cancer (OI-BC™)
(PRS + Monogenic)

A Breakthrough Saliva Genetic Test for Predicting Breast Cancer Risk
Polygenic Risk Score (PRS) +
Monogenic Genes: (BRCA1, BRCA2, ATM, BARD1, CDH1, CHEK2, NF1, PALB2, PTEN, RAD51C,

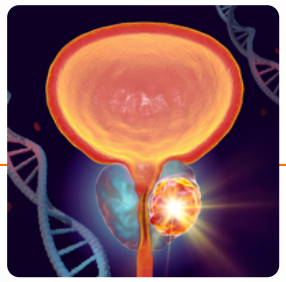
PRS Cardiovascular (CV-PRS)

A Breakthrough Saliva Genetic Test for Predicting 8 Common CV Diseases
Polygenic Risk Score (PRS): (CAD, Diabetes, Hypertension; Hypercholesterolemia; Elevated Lipoprotein (a): Atrial fibrillation; Thoracic Aortic Aneurysm and Venous blood clots)

OmniInsight™ for coronary artery disease (OI-CAD™) (PRS + Monogenic)

A Breakthrough Saliva Genetic Test for Predicting CAD Risk
Polygenic Risk Score (PRS) +
Monogenic Genes (FH): (LDLR, APOB,

OmniInsight™ for Prostate Cancer (OI-PC™) (PRS + Monogenic)



A Breakthrough Saliva Genetic Test for Predicting Prostate Cancer Risk
Polygenic Risk Score (PRS) +
Monogenic Genes: (BRCA1, BRCA2, ATM, PALB2, CHEK2, HOXB13, MLH1, MSH2, MSH6, PMS2

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Adults with high coronary artery disease (CAD) polygenic risk scores but low clinical risk demonstrated that about half had previously undetected subclinical plaque despite favorable cardiovascular health.

JACC VOL. ■, NO. ■, 2026
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Polygenic Risk Based Detection and Treatment of Subclinical Coronary Atherosclerosis in the PROACT Clinical Trials

Roukoz Abou-Karam, MD,[a,b] Min Seo Kim, MD,[a,b] So Mi Jemma Cho, PhD,[a,b] Fouad Bitar, MD,[a,b] Shoshana Gady, BS,[a,b] Fangzhou Cheng, BS,[a,b] Abigail Grace Thompson, BA,[a,b] Elizabeth W. Karlson, MD,[c] Pradeep Natarajan, MD, MMSc,[a,b] Patrick T. Ellinor, MD, PhD,[a,b] Borek Foldyna, MD,[d] Musie S. Ghebremichael, PhD,[e,f] Steven J. Atlas, MD, MPH,[g] Paul M Ridker, MD, MPH,[h,*] Michael T. Lu, MD, MPH,[d,*] Akl C. Fahed, MD, MPH[a,b,*]

ABSTRACT

BACKGROUND Coronary artery disease (CAD) polygenic risk scores (PRS) may identify individuals at elevated genetic risk "flying under the radar" in contemporary practice. The aims of the PROACT (Polygenic Risk Based Detection and Treatment of Subclinical Coronary Atherosclerosis) trials are to prospectively identify these individuals, quantify subclinical coronary plaque, and slow its progression with pharmacologic interventions.

OBJECTIVES The aim of this study is to report interim feasibility and implementation findings from PROACT, a genotype-first, biobank-enabled trial, characterizing eligibility yield, callback engagement, and subclinical coronary atherosclerosis on coronary computed tomographic angiography among individuals with high CAD PRS.

METHODS Within a hospital-based biobank, adults 40 to 75 years of age with high CAD PRS, without cardiovascular disease, and not on lipid-lowering therapy were invited. The authors characterize 2,495 eligible individuals with high CAD PRS, report on the feasibility and early operational outcomes of a genotype-first callback strategy for a clinical trial in the first 1,314 invited, and describe plaque prevalence by age and sex in the first 204 participants using coronary computed tomographic angiography.

RESULTS Among 64,092 genotyped participants, 2,495 (3.9%) were eligible and had high CAD PRS despite low clinical risk (median 10-year pooled cohort equations risk for atherosclerotic cardiovascular disease 3%; Q1-Q3: 1%-8%). Recruitment showed high engagement: among 1,314 invited individuals, 283 (21.5%) opted in, and 204 (15.5%) completed baseline imaging. Compared with participants who did not opt in, those who opted in had higher specialty care engagement and lived closer to the study site. Analysis of the first 204 participants enrolled by January 31, 2025 (mean age 56.3 ± 8.5 years, 69% women), showed that despite the low clinical risk and favorable cardiovascular health (mean Life's Essential 8 score 73.3 ± 11.5 vs the U.S. average of ~65), one-half the participants (102 of 204) had subclinical plaque. Subclinical plaque prevalence was 76.2% in men and 38.3% in women and was high across age groups.

CONCLUSIONS These exploratory findings highlight the feasibility of implementing genotype-first recruitment for prevention trials and reveal a large proportion of "silent" high-genetic risk individuals with subclinical plaque for whom pharmacotherapy could be beneficial but who remain undetected by standard clinical assessments. (Polygenic Risk Based Detection of Subclinical Coronary Atherosclerosis and Change in Cardiovascular Health [PROACT 1], NCT05819814; Polygenic Risk Based Detection of Subclinical Coronary Atherosclerosis and Intervention With Statin and Colchicine [PROACT 2], NCT05850091) (JACC. 2026;■:■-■) © 2026 by the American College of Cardiology Foundation.

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This NEJM 2026 review shows that CAD polygenic risk scores (PRS) identify a high-risk group, often missed by traditional calculators and meaningfully improve risk prediction and provide absolute benefit is greatest in high-PRS individuals.

The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

The Inherited Basis of Coronary Artery Disease

Heribert Schunkert, M.D.,[1,2] Pradeep Natarajan, M.D.,[3-5] and Nilesh J. Samani, M.D.[6-9]

N ENGL J MED 394;6 NEJM.ORG FEBRUARY 5, 2026

SUMMARY

Investigations of the genetic basis of coronary artery disease have led to advances in mechanistic insights, therapeutics, prevention, and risk prediction. Indeed, most contemporary medicines for coronary artery disease target pathways that promote atherosclerosis due to underpinning genetic mechanisms. Monogenic causes of coronary artery disease occur in approximately 1 out of 250 people and mostly result in massively elevated lipid levels. At the population level, hundreds of common variants with small effect sizes have even greater influence. They can be combined in polygenic risk scores that depict genetic risk in a person relative to the average in the general population. The risk among persons in the highest 5% is 3 to 5 times that among persons with an average score; relative risk derived from the polygenic risk score can be used to multiply the absolute risk derived from a clinical risk score. Key questions remain regarding the clinical value, cost-effectiveness, and implementation strategies required to integrate coronary artery disease polygenic risk scores into clinical practice.

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PRS in Coronary Artery Disease: From Genetic Data to Clinical Action

Introduction:

Coronary artery disease risk begins at conception, yet current prevention strategies rely on mid-life phenotypic markers. Polygenic risk scores (PRS) provide a lifelong, stable marker of risk that identifies individuals decades before clinical disease. Evidence now demonstrates that PRS refines risk classification, modifies treatment benefit, improves adherence, and is cost-effective at scale. PRS is not an additional biomarker—it represents a shift toward time-based, preventive cardiology, moving care from late detection to early intervention.

Topic / Key Finding	Key Insight	Evidence	Clinical Action
	Clinical Problem		
Genetic Heritability of CAD	CAD is 40–60% heritable with genetic risk present from birth and persisting lifelong.	Nature Comm Rev 2024 Nature Reviews Cardiology 2026 NEJM Evidence 2026; JACC 2026 npj (Nature Portfolio), 2026	Early genomic risk detection enables primordial prevention before phenotypic expression.
Limitation of Current Risk Paradigm	Traditional 10-year risk models (e.g., Pooled Cohort Equations) underperform in identifying younger individuals with high lifelong genetic risk. Consequently, prevention strategies detect disease only after biological initiation, whereas PRS identifies susceptibility before pathogenesis begins.	JACC 2020 Nature Reviews Cardiology 2026	PRS complements clinical risk scores, particularly in early or subclinical disease stages. CAC detects disease already present; PRS predicts disease before it forms
	What PRS Measures		
Definition of PRS	PRS aggregates thousands to millions of variants across the genome and can be calibrated across ancestries. Unlike biomarkers, PRS is invariant over time and therefore uniquely suited for lifelong risk stratification	Nature Communications 2023; Nature Med 2023	Aone-time, lifetime genomic test applicable across diverse populations. Therefore, PRS defines an individual's baseline biological risk, not a dynamic biomarker.
Multi-Ancestry and MENA Validation	Multi-ancestry PRS models demonstrate improved calibration across Middle Eastern populations when locally validated.	Nature Comm 2023 Current Protocols 2023	Enables equitable and region-specific implementation.

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PRS in Coronary Artery Disease: From Genetic Data to Clinical Action

AHA Statement on PRS	PRSfunctions as a lifelong, independent risk enhancer complementary to traditional risk assessment tools such as PCE and CAC	Circulation 2022 Circulation 2025 ACC/AHA Guidelines Circulation 2026	PRS can be integrated into preventive cardiology workflows as a risk-modifying tool.

Does PRS Detect Disease?

Predictive Strength of PRS	PRS provides risk discrimination comparable to, and in some analyses exceeding, individual traditional risk factors.	JACC 2018 Atherosclerosis 2023 MED 2024 EJHG, 2025	Enables personalized preventive intensity stratification.
Invisible Hypercholesterolemia	Individuals with high PRS and LDL ≥130 mg/dL may exhibit risk comparable to those with LDL ≥190 mg/dL.	Circulation 2021, MED 2024	Reclassifies moderate LDL levels into high-risk categories when PRS is elevated.
Plaque Vulnerability and Subclinical Disease	High PRS is associated with subclinical coronary plaque burden and markers of vulnerable plaque.	ATVB 2023, JACC CV Imaging 2024, Sci Rep Med 2025 Circulation 2025	PRS refines imaging decisions and intensifies preventive therapy in asymptomatic individuals

Does It Change Outcomes?

Randomized Controlled Trial Evidence – MI-GENES	PRS disclosure increased statin uptake and significantly improved LDL reduction compared with conventional risk disclosure.	Circulation 2016; Circ Geno Precis Med 2025	Genomic disclosure improves behavioral and therapeutic compliance.
Effect of PRS Disclosure on Outcomes	Clinical implementation studies show that returning PRS results is associated with increased uptake of preventive therapy and improved risk factor control.	JACC: Advances 2022; Circulation: Genomic and Precision Medicine, 2025	Disclosure acts as a behavioral catalyst for preventive adherence.
Therapeutic Benefit Stratification	PRS modifies absolute treatment benefit, not only treatment eligibility. High-PRS individuals derive greater absolute and relative benefit from statins and PCSK9 inhibitors.	Circulation 2017; Clin Pharm Ther 2023; Circulation 2020	Lowers number needed-to- treat (NNT) and enhances cost effectiveness
Lifestyle Modification	Adherence to a favorable lifestyle reduces relative risk by approximately 45–50%, even among individuals with high PRS.	NEJM 2016 European Heart Journal 2024	PRS motivates preventive lifestyle engagement. Genetic risk is actionable—not deterministic

Implications for Clinical Practice

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PRS in Coronary Artery Disease: From Genetic Data to Clinical Action

Risk-Enhancing Factor (Top 20%)	Individuals in the top 20% of the PRS distribution (~2× relative risk) should be considered to have a risk- enhancing factor.	Circulation 2022, JAMA Card 2022 European Journal of Preventive Cardiology 2025 NEJM 2026	Supports statin initiation in borderline or intermediate- risk patients.
Very High Genetic Risk (Top 10%)	Individuals in the top 10% (~3× relative risk) demonstrate risk approaching that observed in monogenic hypercholesterolemia populations.	Nat Genet 2018, Nat Rev Card 2021 NEJM 2026	Consider earlier and more intensive lipid-lowering therapy.

Why Systems Should Adopt

Population Scale Impact	Approximately 8–12% of adults carry PRS conferring risk approaching monogenic FH levels.	Nat Genet 2018, BMJ 2017	Supports population-level genomic risk screening strategies.
Cost-Effectiveness	PRS-guided prevention strategies demonstrate ICERs below $50,000/QALY in modeled analyses, with high probability of cost-effectiveness.	JAHA 2022, Front Public Health 2023 npj Cardiovascular Health, Nature portfolio 2026	Supports payer adoption and reimbursement integration.

Clinical Application of PRS in Practice

Polygenicriskscores(PRS)provideactionable insights across common clinical scenarios:

1. Borderline or Intermediate Risk (10-year ASCVD risk 5–20%)	• Refine risk stratification • High PRS supports earlier initiation of statin therapy
2. Low-Risk Patients with Strong Family History	• Uncovers hidden inherited susceptibility • Supports earlier screening and preventive strategies
3. Patients with LDL-C 130–189 mg/dL	• High PRS may reclassify risk to levels comparable to severe hypercholesterolemia • Supports intensification of lipid-lowering therapy
4. Younger Individuals (<55 years)	• Identifies elevated lifetime risk before phenotypic expression • Enables early lifestyle and preventive interventions
5. Patients Hesitant About Therapy	• Improves risk communication and shared decision-making • Associated with higher adherence to therapy and lifestyle modification
6. High PRS (Top 10–20%)	• Justifies earlier and more intensive preventive strategies • Lowers threshold for pharmacologic intervention
7. Age-Dependent Clinical Utility	• Early life: predictive and preventive tool • Mid-adulthood: risk refinement and treatment intensification

PRS does not replace clinical judgment—it enhances it by revealing inherited risk long before disease manifests.

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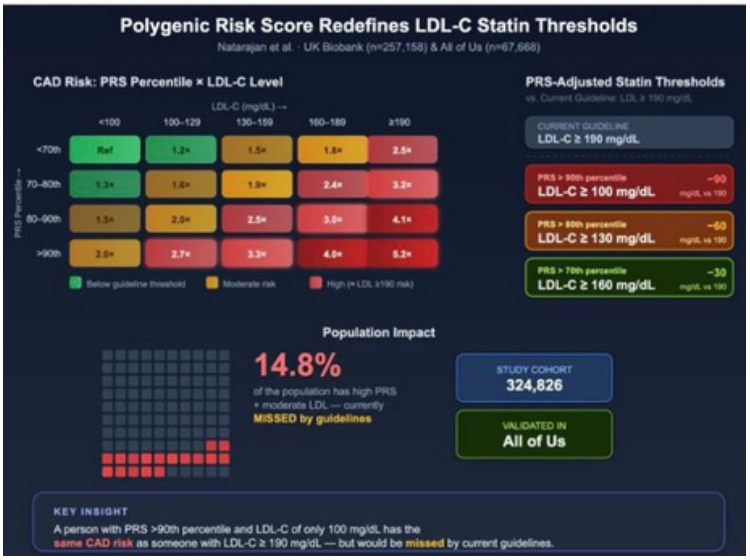
PRS in Coronary Artery Disease: From Genetic Data to Clinical Action

American Journal of Preventive Cardiology (2026), doi: https://bit.ly/3MQhkZO



ACC/AHA Guidelines on PRS 2026
PRS is recognized as a risk-enhancing factor for ASCVD in the new guidelines.
It refines risk assessment, particularly in borderline or intermediate-risk patients.
High PRS identifies individuals at ~2× increased risk, comparable to other enhancers.
It is especially valuable in younger individuals (<55 years).
PRS uncovers hidden inherited risk often missed by traditional calculators.
High PRS helps identify those who may benefit more from lipid-lowering therapy.
PRS should be used alongside traditional risk factors, not as a replacement.

Conclusion:
PRS defines an individual's baseline biological risk present from birth—it is not a dynamic biomarker.
Polygenic risk scoring redefines cardiovascular prevention by shifting risk assessment from age-dependent estimates to biologically grounded, lifelong susceptibility.
By identifying inherited risk decades before disease onset, PRS enables earlier intervention, more precise treatment decisions, and improved patient engagement.
Its ability to refine risk, personalize therapy, and enhance cost-effectiveness positions PRS not as an incremental tool, but as a foundational component of modern preventive cardiology.
As clinical guidelines increasingly recognize its value, the integration of PRS into routine care represents a critical step toward a more proactive, personalized, and effective healthcare system.

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Leading U.S. Centers Advancing Clinical Use of Polygenic Risk Scores (PRS)

Academic medical center	Location (USA)	PRS delivery model	Main PRS focus / use
Mass General Brigham/Harvard	Boston,MA	Clinical test via LMM+ translational programs	Cardiovascular PRS(clinical); other common diseases (research/implementation)
Massachusetts General Hospital/ Harvard	Boston,MA	Preventive genomics clinics	Cardiovascular and common disease PRS
Brigham and Women's Hospital	Boston,MA	Preventive genomics & clinical genetics programs	Cardiometabolic and common disease PRS
Harvard /Mayo Clinic	Rochester, MN	Genome-Informed Risk Assessment)	Cardiovascular, cancer, diabetes
Northwestern Medicine	Chicago, IL	Research-to-care implementation (eMERGE site)	Cardiometabolic and common diseases
Vanderbilt University Medical Center	Nashvill TN	Translational implementation (precision medicine programs)	Cardiovascular, metabolic and cancer PRS
University of Chicago Medicine	Chicago, IL	Clinical genomics programs	Cancer PRS (especially breast cancer)
Baylor College of Medicine	Houston, TX	Clinical genomics framework with PRS partners	Multi-ancestry PRS for common diseases
Penn Medicine	Philadelphia, PA	Biobank-driven research-to-care programs	Cancer and cardiometabolic PRS
Cleveland Clinic	Cleveland, OH	Genomic medicine programs	Cardiovascular and common diseases PRS

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Shipment Logistics Instructions

A. Shipment Address (via DHL or other courier services)

Broad Clinical Labs Receiving Lab 132 27 Blue Sky Drive Burlington,
MA 01803, USA Attention: Nick Argiro Phone: +1 617-714-8952

B. Sample Labeling Requirements

Product: DNA Genotek saliva collection kits (ORAgene)

All samples must include **two unique identifiers** clearly visible on the collection tube:

1. Barcode (Primary Identifier):
The barcode is pre-printed onthetube andmustremain clearly visible.
Do not cover or obscure the barcode.

2. Patient Identifier (Secondary Identifier):

A label including **the patient's full name and date of birth must be affixed to the tube.**

Ensure that the label does not cover or obscure the barcode or kit code.

Samples may be rejected if:

- The barcode is covered or unreadable
- Patient identifiers are missing or incomplete

C. Required Documentation (to be shared electronically)

Please submit the following documents via email, WhatsApp, or the Clinic Portal:

- Barcode (as printed on the saliva collection
- tube) Patient full name and date of birth
- Signed Consent Forms Completed Patient
- Intake Forms DHL shipment details and tracking information

Handling Instructions:

Store samples at room temperature. Avoid exposure to extreme heat.
Forassistance, pleasecontact:

• Email: info@avigena.com • WhatsApp (Avigena Boston): +1 (617) 412-5580