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Transferability, Calibration, and Decision Thresholds of Polygenic Risk Scores for Coronary Artery Disease in Latinx Populations: Bench-To-Population Perspectives

Kirenyo Nalubega F.

Faculty of Medicine Kampala International University Uganda

ABSTRACT

Polygenic risk scores (PRSs) for coronary artery disease (CAD) hold promise for improving early risk stratification and preventive cardiology, yet their clinical translation across diverse populations remains challenging. This paper examines the transferability, calibration, and clinical decision thresholds of CAD PRSs in Latinx populations from a bench-to-population perspective. It synthesizes current evidence on how differences in genetic ancestry, linkage disequilibrium structure, allele frequencies, and environmental context influence predictive performance when PRSs derived largely from European genome-wide association studies are applied to Latinx cohorts. The analysis highlights that while some multi-ancestry models retain a partial predictive signal, performance often declines, with systematic miscalibration leading to over- or underestimation of absolute risk. Recalibration methods, including adjustment of intercepts and slopes and incorporation of population-specific prevalence data, can improve clinical reliability. Determining appropriate decision thresholds is also critical, as differences in disease prevalence and baseline risk necessitate ancestry-informed cut-points to balance sensitivity and specificity in screening and prevention strategies. The paper further discusses methodological, ethical, and implementation considerations, including dataset representation, governance of genomic data, and integration with traditional cardiovascular risk assessment tools. Strengthening Latinx representation in genomic research and developing ancestry-aware modelling strategies are essential to ensure equitable clinical utility. Ultimately, optimizing PRS transferability, calibration, and threshold selection could enable more accurate and inclusive genomic risk prediction for CAD in Latinx populations.

Keywords: Polygenic risk scores**,** Coronary artery disease**,** Latinx populations**,** Genetic risk prediction, Calibration and transferability.

INTRODUCTION

Polygenic risk scores (PRSs) exhibit considerable variability in predictive performance between populations, complicating their clinical translation [1]. Initial evaluation of a multi-ancestry PRS for coronary artery disease (CAD) in the UK Biobank indicated effective transfer from European to Latinx cohorts, yet subsequent assessments revealed performance deterioration across two Latinx populations [2]. These observations underscore the necessity of accounting for population-specific differences when extending PRSs beyond their training data [3]. Coronary artery disease poses a major burden on cardiovascular health, characterized by the accumulation of atherosclerotic plaques in coronary arteries [4]. The importance of maximizing the transferability and calibration of a multi-ancestry PRS for CAD among Latinx populations extends beyond the advancement of PRS methodology; it aims to improve health outcomes for a population recognized as one of the fastest-growing worldwide, while addressing the underrepresentation of Latinx individuals in biomedical research[5].

Background on Polygenic Risk Scores and Coronary Artery Disease

Coronary artery disease (CAD) represents a leading global cause of mortality, with an estimated 18.6 million deaths attributable to the disease in 2019. CAD results from complex pathophysiological processes at the cellular

and molecular levels, influenced by environmental, lifestyle, pathophysiological, and genetic risk factors [6]. The cumulative genetic contribution of thousands of common variants across the genome accounts for ~20% of the variance in CAD liability, resulting in considerable inter-individual variation in disease susceptibility. Polygenic risk scores (PRSs) aggregate the effects of thousands of genetic variants influencing disease risk and serve as an efficient way to quantify an individual's genetic predisposition to CAD [7]. PRS are constructed by assigning a risk weight to each of several genetic variants based on their population-specific effect sizes derived from genome-wide association studies (GWAS). The weight assigned to each variant reflects its observed association strength with CAD, typically as estimated by odds ratios from disease case-control cohorts [8]. This weight is then applied to the genotype at that locus, either by adding the risk weight directly (when using non-imputed genotypes) or by incorporating an appropriate imputation weight depending on the reference panel utilized for imputation (for imputed datasets). Polygenicity captures the contribution of thousands of variants influencing CAD liability on a genome-wide scale, accounting for linkage disequilibrium, a key factor in disease manifestation that accelerates progress toward understanding CAD mechanisms and improving prevention, detection, and treatment [9, 10]. Recent large-scale trans-ancestry and ancestry-specific meta-GWAS analyses have identified thousands of genome-wide significant variants and millions of additional candidate loci influencing CAD [11]. The growing polygenic architecture and increasing number of available non-Latinx and Latinx reference samples from non-Latinx ALFRED databases and gnomAD release data, respectively, have spurred considerable interest in the development of multi-ancestry CAD-related PRS [12]. Large-scale cohort analyses have demonstrated consistently greater prediction performance and sharper stratification for lifestyle diseases, including CAD, in Latinx individuals compared to European and Asian cohorts, with marked inter-population variability in the degree of polygenicity and emergent population-specific risk alleles. The potential impact of developing more precise, more readily interpretable, and more widely applicable Latinx-optimized CAD-related PRS remains substantial [13].

Population Genetics and Latinx Diversity

Latinx populations exhibit notable genetic diversity and ancestry-specific polygenic architectures. An individual's genetic ancestry and population stratification impact the distribution of polygenic scores, yet Latinx populations are frequently overlooked in polygenic studies [14]. A genetic and polygenic understanding of Latinx populations requires attention to their intercontinental admixture, continent-specific selections, and northern versus southern contrasts [1]. When transmitting polygenic risk scores to Latinx populations, several considerations apply. First, let us define polygenic risk scores and coronary artery disease, outlining their construction, underlying statistical modelling assumptions, predictive performance metrics (area under the curve, calibration plots, net reclassification improvement), and summarizing existing evidence from varying non-Latinx populations across polygenic risk-score estimation and evaluation studies [15].

Transferability of Polygenic Risk Scores across Populations

Polygenic risk scores (PRS) derive from allele counts and effect sizes associated with trait-relevant variants identified by genome-wide association studies (GWAS) [16]. Incorporated within a linear regression framework, PRS operate under multiple statistical assumptions; each of these elements varies across populations and influences transferability when developing PRS with discovery cohorts characterized by genetic variants and effect sizes identified predominantly in European ancestry samples [17]. In the context of CAD, diverse studies support the cross-population, non-Latinx to Latinx transferability of PRS developed using European discovery cohorts. Indeed, GWAS variances modeled in European ancestries are observed to explain more than half of the CAD heritability across Latinx populations [4].

Calibration of Risk Scores in Latinx Cohorts

Clinical stratification for Coronary Artery Disease (CAD) remains a global challenge and threat [1]. Bench-to-population translational work has aimed at evaluating transferability, calibration, and decision thresholds of polygenic risk scores in Latinx populations and cohorts to assess how CAD risk estimates based on genetic data might be adapted and integrated in practice according to local differences across epidemiology, genomics, and methodology [18]. Calibration-in-the-large was examined on Latinx cohorts, revealing that unadjusted scores yielded greatly overestimated absolute risk values [3]. Variations across regions and sites indicated incorporation of site-specific adjustment of intercept and slope in the original risk equation: recalibration of calibration-in-the-large therefore stands as the most straightforward solution to restore relevant absolute risk estimates, enabling use of unmodified algorithms and familiar, extensive, practice-ready datasets [19].

Determining Clinical Decision Thresholds for Latinx Populations

An accessible approach to determining clinical decision thresholds for Latinx populations is to derive absolute risk cut-points that optimize the balance between sensitivity and specificity at a polygenic risk-score (PRS) level [2]. Absolute risk thresholds can be estimated in cohorts with the scoring scheme applied if the target population prevalence is known [3]. This information can be obtained from alternatives to Latinx-specific cohorts: publicly

accessible estimates are available for the United States 1 and the Latin American and Caribbean NCD Alliance (2019)[20]. Given the lower prevalence of coronary artery disease (CAD) in Latinx populations, higher absolute-risk PRS thresholds are anticipated. Furthermore, even larger absolute-risk cut-points are required for the PRS-based identification of Latinx individuals, equivalent to those identified among European-descent individuals. Population-specific Clinical Decision Points recommended for different ancestries call for distinct thresholds within the PRS framework [21]. The evolving intersection of polygenic-risk models and existing guidelines shows promise for improving systematic investment in health equity and stratified intervention by linking research findings to ongoing initiatives [5].

Methodological Considerations for Translational Research

Evaluating polygenic risk scores (PRS) for coronary artery disease (CAD) in Latinx populations is essential to reduce health disparities in genomics [1]. Latinx individuals continue to be underrepresented in risk-prediction research despite their global demographic significance and the socio-economic burden of CAD in Latin America, where the condition is the leading cause of morbidity and mortality [2]. Studies in this area may enhance risk-stratification improvement through the inclusion of genetic information, thus bolstering prevention and treatment activities. Bench-to-population translational research aims to assess the transferability, calibration, and clinical decision thresholds of existing PRS for CAD in Latinx individuals by leveraging existing genetic and phenotypic data [2]. The anticipated impact is substantial, as the absolute 10-year risk for CAD in Latinx individuals recorded in several countries exceeds the action threshold established by American Heart Association guidelines. PRS are summaries of an individual's genetic predisposition to a given trait, constructed by aggregating the effects of numerous genetic variants associated with the trait or disease [22]. CAD, predominantly characterized by atheroma formation within the arteries, causes coronary disease, angina, myocardial infarction, and sudden cardiac death. The variants investigated in large meta-analyses, the Genetic Epidemiology Research on Adult Health and Aging (GERA) cohort, and the CanCOGeN dataset have yielded transferable PRS, corroborated by independent studies in Latinx individuals seeking a preventive approach against CAD. PRS utility hinges on the approximation of allele frequencies, linkage disequilibrium patterns, and effect-size distributions in the target population relative to discovery populations [23]. The consequent likelihood of effective cross-population transfer and the emergence of distinct variants in Latinx-of-Latin-American-ancestry populations reliant on European and East-Asian discovery samples underscores the need for extensive empirical evaluation. Latinx populations present exceptional diversity, characterized by distinct ancestral admixture, varying regional representation, and diverse imputation strategies. Genetic variation across Latinx individuals follows five predominant components of ancestry (indigenous Amerindian, Afrodescendant, Middle-Eastern, South-European, and East-Asian), and distributions differ by region; nearly one-quarter of Mexican–Americans and Puerto-Ricans derive greater than 50% of genetic ancestryfrom Amerindian sources. Subcontinental-level structuring exists, with Mexican populations forming two distinct clusters (northwestern and southeastern), and cohorts as far apart as Argentina and the United States have been evaluated [1]. The prominent continental component linked to Puerto-Rican cohorts is Afrodescendant ancestry, likely reflecting the predominant West-African admixture of the Iberian invaders. Patterns of ancestry and demographic histories exert substantial influence on allele-frequency spectra, linkage disequilibrium decay, and imputation quality. Cross-population transfer of PRS can be hindered by dissimilar frequency distributions of associated variants, with successful adjustment reliant on at least partial overlap in effect sizes for shared risk- and protective-alleles [24]. Empirical evidence from non-Latinx discovery-to-Latinx target migration has demonstrated variable performance across distinct PRS, with GERA- and CanCOGeN-derived CAD models recording the best transfer outcomes [2]. Several models constructed with non-Latinx cohorts have exhibited favourable effects on reclassification and are readily accessible for prospective analysis in Latinx populations, reinforcing the necessity of comprehensive appraisal. Detailed assessments of the calibration of long-term absolute risk predictions for CAD provided by multi-ancestry PRS have not been undertaken in Latinx populations, which rank among the highest global targets for translational research [3]. Nevertheless, calibration-in-the-large analyses of GERA- and CanCOGeN-derived PRS have been performed; the observed long-term (10-year) risk estimates estimated from the GERA cohort closely matched the overall CAD incidence trends reported for white individuals in North America and were subsequently consistent with cohort-specific survey data on the frequency of CAD endpoints. Calibration-in-the-large formal tests have been simplified to an intercept evaluation following model adjustment to convert cumulative incidence to long-term absolute risks, omitting cohort-representative coefficients [25]. Calibration plots revealing the alignment of predicted and observed outcomes further corroborate the compatibility of original GERA estimates with CAD onset projections in Latinx cohorts. Clinical guidelines often recommend intervention for individuals above a specific risk-threshold, motivating the establishment of fixed decision thresholds to accompany PRS across diverse populations [7]. Recommended options to derive absolute-risk cut-points include seeking a population–specific prevalence ratio matching that of the discovery cohort and minimizing the distance from the ideal operating point for a given sensitivity, specificity, or likelihood ratio. Non-Latinx discovery cohorts remain widely employed in PRS research, encouraging the

pursuit of such cut-points conditioned on independent Latinx-case-control sampling and prevalence estimates [26].

Ethical, Legal, and Social Implications

Genomic research to advance polygenic risk scores (PRSs) for coronary artery disease (CAD) and related guidelines offers opportunities to improve risk stratification and clinical utility [5]. These enhancements may constitute a substantial step toward realizing the promise of genomic research in Latinx cohorts by addressing the prevailing focus on European ancestry and taking action [27]. Ancestry-aware methods to enrich representation across populations, including Latinx individuals, foster progress at this critical juncture for bench-to-population translation through the establishment of genetically instructive bench markers [5]. The ethical, legal, and social implications of PRSs and genomic information ultimately warrant attention. Providing ancestry-informed risk estimates in non-Latinx cohorts can inadvertently amplify population stratification. Modulation of risk communication to avoid reinforcing stereotypes that harm marginalized communities represents one vector of inquiry with urgency and immediacy [28]. Sorting genomic data, including PRSs, through the ethical-governance framework of either privacy or stewardship enables recognition that the capacity for genomic assemblage remains present regardless of their disposition [2]. Under such conditions, decision-making regarding the retention or dissemination of data promotes explicit articulation of principles, values, interests, and objectives that invariably shape further design considerations in population-appropriate and culturally attuned manners [6]. Non-Latinx populations continue to occupy the majority of national cohorts, reflecting a need to advocate for the generation of Latinx-priority datasets. An emphasis on equitable development encourages galvanization of additional cohorts across diverse Latinx communities that advance equity, development, and governance in genomics [2].

Practical Implications for Clinical Practice and Public Health

Polygenic risk scores yield actionable steps for coronary artery disease prevention and the identification of high-risk individuals among Latinx populations based on a large-scale genome-wide association study [7]. Such scores can augment existing tools based on traditional risk factors and further benefit from integration into electronic health record systems and electronic clinical decision-support systems [4]. To enhance implementation and awareness, clinician education is essential, and population-level health strategies are needed to address key risk factors for coronary artery disease [29]. Coronary artery disease remains the leading cause of death and disability in Latinx communities across the United States and globally; yet most individuals do not receive guideline-recommended preventive interventions. Polygenic risk scores for coronary artery disease, converging with traditional risk factors, now offer the potential to identify others at high risk. The further elucidation of gaps, challenges, and open avenues for inquiry in Latinx populations should encourage additional interest and investment [30].

Gaps, Challenges, and Future Directions

Transferability, Calibration, and Decision Thresholds of Polygenic Risk Scores for Coronary Artery Disease in Latinx Populations: Bench-to-Population Perspectives. Gaps, challenges, and future directions include improving the transferability of polygenic risk scores across diverse populations, addressing differences in linkage disequilibrium and recombination patterns among populations, and incorporating variants from diverse ancestries to enhance discovery and clinical utility[7]. Developing more accurate models of complex effect-size distributions and understanding trans-ancestral genomic architecture are essential. Additionally, establishing guidelines for clinical translation and fostering inclusive research efforts are crucial for equitable advances in genetic risk prediction [31]. Polygenic risk scores for coronary artery disease were constructed in the UK Biobank, and preliminary transferability characterisation was performed. Cross-population transferability of polygenic risk scores among diverse ancestries remains understudied despite increasing uptake of genomics in non-European populations [5]. Population-specific polygenic risk scores exhibit marked improvements in predictive performance. Within-GWAS-regression approaches, even when formally bi-ancestral, do not ensure portability across human populations with markedly different allele-frequency spectra, dominating phylogenetic and geographical distance in shaping polygenic signal [32]. Empirical evidence shows limited transferability of scores based on European-centric cardiometabolic and behavioural traits to Latinx cohorts [2]. Assessment of absolute-scale calibration for these scores in Latinx cohorts reveals statistical miscalibration, notably in the y-intercept; net-reclassification improvement remains negative. Recalibration strategies, such as intercept and slope re-estimation, retain substantial signal, suggesting modifiable quantitative prediction rather than voiceless binary classification [33, 34].

CONCLUSION

Polygenic risk scores offer a potentially transformative approach to identifying individuals at elevated genetic risk for coronary artery disease, but their effective clinical use in Latinx populations depends on careful attention to transferability, calibration, and decision-threshold selection. Evidence indicates that PRSs developed predominantly in European-ancestry cohorts often retain some predictive value in Latinx groups, yet performance

variability and miscalibration can compromise clinical interpretation and decision-making if scores are applied without adjustment. Recalibration strategies such as population-specific adjustment of model intercepts and slopes, incorporation of local disease prevalence, and validation in representative cohorts can substantially improve absolute risk estimation while preserving the predictive signal of existing models. Equally important is the establishment of ancestry-informed clinical thresholds that align genomic risk estimates with preventive guidelines and real-world epidemiological patterns. Future progress requires expanding Latinx participation in genomic studies, refining multi-ancestry modelling approaches, and integrating PRSs with conventional cardiovascular risk factors within clinical decision-support systems. Ethical governance, culturally sensitive risk communication, and equitable access to genomic testing must remain central to implementation to avoid widening existing health disparities. With rigorous validation, inclusive research design, and policy-guided deployment, CAD polygenic risk scores could evolve into a valuable component of precision cardiovascular prevention, supporting earlier intervention and improved population health outcomes in Latinx communities.

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