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Comparing Non-Laboratory-Based and Laboratory-Based Cardiovascular Risk Predictions: Systematic Review and Meta-Analysis

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ABSTRACT

Introduction: Cardiovascular disease (CVD) remains the leading cause of global morbidity and mortality. This study assesses the agreement between non-laboratory-based and laboratory-based CVD risk equations across diverse settings.

Methods: PubMed, Scopus, Web of Science, ProQuest Dissertations and Theses Global, and Google Scholar were systematically searched for studies published up to March 4, 2025. The protocol was registered with PROSPERO (CRD42021291936). Studies comparing laboratory-based and non-laboratory-based CVD risk equations were included, excluding those with participants who had CVD at baseline. A meta-analysis was conducted using mixed-effects meta-regression. The agreements for each study item (unit of analysis) were measured using the Spearman correlation coefficient and Kappa statistics.

Results: A total of 33 studies, including 243,587 participants and nine CVD risk equations, were identified. The pooled Spearman correlation between the non-laboratory-based and laboratory-based equations was 0.954 (95% CI: 0.928–0.971, $I^2 = 100\%$, $p < 0.0001$), and the pooled kappa was 0.64 (95% CI: 0.61–0.67, $I^2 = 99\%$, $p < 0.0001$). Correlation was higher in studies conducted before 2000 (0.974; 95% CI: 0.970–0.978) compared to those conducted after 2000 (0.953; 95% CI: 0.948–0.958; $p < 0.0001$). Studies from high-income settings had higher correlations (0.967; 95% CI: 0.963–0.970) than those from low-income settings (0.945; 95% CI: 0.933–0.955). Equations predicting fatal outcomes had higher correlations (0.979; 95% CI: 0.977–0.982) than those predicting both fatal and non-fatal outcomes (0.942; 95% CI: 0.937–0.947).

Conclusion: Non-laboratory-based CVD risk equations demonstrate strong correlation and substantial agreement with laboratory-based equations. Non-laboratory-based CVD risk equations show strong concordance with laboratory-based equations in many settings; however, strong correlation and substantial agreement do not necessarily indicate predictive equivalence, and their interchangeability and implementation should be considered context-specific and require external validation and recalibration.

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1 | Introduction

Cardiovascular disease (CVD) remains the leading cause of global morbidity and mortality [1]. Most primary CVD events are preventable through lifestyle modification and pharmacotherapy, including blood pressure and lipid-lowering treatments targeted at high-risk individuals. Estimating 10-year individual CVD risk using multivariable prediction equations aligns with international prevention guidelines [2–4].

Traditional risk equations depend on laboratory tests like lipid profiles, which can be costly or inaccessible in low-resource settings. Consequently, non-laboratory-based risk equations have been developed using proxy measures such as body mass index (BMI) to substitute for cholesterol levels and diabetes status in risk estimation [5].

Although some studies have compared laboratory-based and non-laboratory-based risk equations in specific populations, a comprehensive analysis across diverse settings and different CVD risk equations is lacking. The overall agreement between these approaches and the factors influencing it remains unclear.

Therefore, this systematic review and meta-analysis aim to quantify the correlation and agreement between laboratory-based and non-laboratory-based CVD risk equations, and to assess whether the correlations vary by data recency, outcome type (fatal vs. non-fatal), sex, income level, and risk equation type.

2 | Methods

This systematic review and meta-analysis protocol was registered with PROSPERO (CRD42021291936). Ethics approval was not required for this systematic review and meta-analysis of published studies. The study was conducted in accordance with the registered PROSPERO protocol, and no deviations were made. Reporting follows the transparent reporting of multivariable prediction models for individual prognosis or diagnosis checklist for systematic reviews and meta-analyses (TRIPOD-SRMA), provided in Supporting Information S1: Material A [6].

2.1 | Search Strategy

A systematic search was conducted in five databases for studies published up to March 4, 2025: PubMed, Scopus, Web of Science, ProQuest Dissertations & Theses Global, and Google Scholar. Searches used combinations of terms such as "laboratory-based," "non-laboratory-based," and "cardiovascular risk equations." Full search terms and strategies are provided in Supporting Information S1: Material B.

2.2 | Inclusion and Exclusion Criteria

We included primary CVD risk models that were developed in populations free of CVD at baseline and aimed at predicting incident CVD events, rather than models derived from CVD populations predicting subsequent events. Studies were included if they: (a) include participants who are free of CVD events such as myocardial infarction or stroke at baseline; (b) include CVD risk prediction equations that estimate primary CVD risk (i.e., participants free of CVD events at baseline); (c) include participants or studies from all regions of the world, without geographical restrictions; (d) compared the correlation or agreement between laboratory-based and non-laboratory-based CVD risk prediction equations within the same sample; (e) reported correlation coefficients or kappa statistics; (f) were published in English.

Studies were excluded if they: (a) included participants with existing CVD at baseline; (b) were published in a non-English language; (c) were conference abstracts; or (d) were case reports.

2.3 | Screening, Quality Assessment, and Data Extraction

Titles/abstracts and full texts were independently reviewed by two authors (Y.M.A. and S.M.A.) in a two-stage process. We made our full text review criteria wider than inclusion/exclusion criteria to include studies that match in the title; however, they do not have enough information in abstracts that are listed in Supporting Information S1: Materials C. Quality assessment was conducted by two authors (Y.M.A. and K.W.), with disagreements resolved through discussion and consensus. Data were extracted by Y.M.A. and S.M.A. using a pre-defined form.

Studies included in this systematic review and meta-analysis, which assessed the agreement or correlation between non-laboratory-based and laboratory-based equations, were cross-sectional in design. A modified version of the appraisal tool for cross-sectional study design was used to assess the risk of bias in the included studies. A modified version was used, meaning that a few parameters not applicable to this review were excluded; these are listed in Supporting Information S1: Material D [7]. The assessment tool included study objectives, design, population, sampling frame, participant representativeness, risk factors, outcomes, statistical methods, response bias, internal consistency, results, conclusions, limitations, conflicts of interest, and ethical approval.

We extracted study-level data, including sex, study year, sample size, equation types, standard error, chance agreement, percent agreement, and outcome measures such as the Spearman correlation coefficient and Kappa statistic. Country income level was classified according to World Bank criteria [8].

2.4 | Meta-Analysis

We conducted a random-effects meta-analysis to estimate the overall correlation between laboratory-based and non-laboratory-based CVD risk scores using Spearman correlation coefficients and Kappa statistics. Spearman correlation coefficients were first transformed using Fisher's z-transformation to approximate normality, then pooled, and finally back-transformed for interpretation [9, 10]. Pooled estimates are presented in forest plots.

Kappa statistics were analyzed using a random-effects meta-analysis model to estimate the agreement between

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laboratory-based and non-laboratory-based risk equations. The variance of each Kappa estimate was calculated as $V(\kappa) = \frac{(1-\text{po})}{(1-\text{pc})^2 * (n)}$ where percent agreement (po), chance agreement (pc), and sample size of each observation (n) [11]. These estimates were subsequently pooled to generate a summary effect size [9, 10]. To address the dependency among multiple effect sizes, we used cluster-robust variance estimation, with population categories specified as clusters for both correlation and kappa measures. We also assessed whether pooled agreement estimates differed across kappa categories.

2.5 | Subgroup Analysis and Meta-Regression

We conducted subgroup analyses stratified by equation type and mixed-effects meta-regression stratified by sex, outcome type (fatal vs. non-fatal), data recency, and income level to explore sources of heterogeneity. Correlations across groups were compared using multivariable meta-regression. Meta-regression was performed only when data were available from at least 10 studies, as recommended [12].

2.6 | Interpreting the Magnitude of Spearman Correlation Coefficients and Kappa Statistics

The strength of Spearman correlation coefficients is commonly interpreted as 0.00–0.39 (low), 0.40–0.69 (moderate), 0.70–0.89 (strong), and 0.90–1.00 (very strong) [13–15]. For Kappa statistics, the widely used Landis and Koch scale defines agreement as: ≤ 0 (poor), 0.01–0.20 (slight), 0.21–0.40 (fair), 0.41–0.60 (moderate), 0.61–0.80 (substantial), and 0.81–1.00 (almost perfect) [16]. Other similar classification systems have been proposed [17–19]. Kappa values are affected by the number of categories; as the number of categories increases, the potential for disagreement also increases [20].

2.7 | Heterogeneity and Sensitivity Analysis

Heterogeneity was assessed using the I^2 statistic and Cochran's Q test. To explore potential sources of heterogeneity, subgroup analyses, meta-regression, and sensitivity analyses were conducted. First, the meta-analysis was repeated with tau-squared (τ^2) fixed. We conducted random-effect meta-analysis; however, for sensitivity analysis, we also fixed the τ^2 value at 0.2 and 0.1 to assess how much the pooled estimate is affected by changing τ^2. Second, a leave-one-out analysis was performed by sequentially excluding each study to assess its influence on heterogeneity and the overall pooled estimate.

2.8 | Publication Bias and Small Study Effects

Small study effects were assessed using funnel plots and Egger's test. The Egger test was conducted for pooled estimates that included 10 or more studies. All analyses were conducted using R software (version 4.3.0). We used the "metafor," "metacor," and "meta" R packages.

3 | Results

3.1 | Search Results

Thirty-three studies met the inclusion criteria, with a combined sample of 243,587 participants (Supporting Information S1: Material C). Four studies reported Spearman correlation coefficients, producing 204 data points [21–24], and 21 studies reported Kappa statistics, contributing 58 data points [25–45].

3.2 | Characteristics of Included Studies

Twenty-two correlation measures were compared between the non-laboratory-based Harvard NHANES (National Health and Nutrition Examination Survey) equation and three laboratory-based equations: the D'Agostino Framingham, SCORE (Systematic Coronary Risk Evaluation) high, and SCORE low [46–61]. Fifteen correlation measures were compared between the non-laboratory-based Harvard NHANES equation and the Anderson–Framingham equation [46–55]. Fourteen correlation measures were used to compare the non-laboratory-based Harvard NHANES equation with the CUORE (Cardiovascular Epidemiology of the Italian Population) equation [47–55, 61, 62]. Seven correlation measures were used to compare the non-laboratory-based Harvard NHANES equation with the Pooled Cohort Equation (PCE) [56–61]. Overall, the correlation measures included 4 studies, 22 distinct populations, and 204 effect measures.

Eight studies were population-based surveys or cross-sectional studies [46–53, 55, 56]. Four studies were baseline surveys for cohort studies [57, 59, 60, 63], and two were baseline surveys for interventional studies [54, 58]. Data contributing to the baseline surveys in studies reporting correlation coefficients were collected between 1982 and 2013. Overall, the risk of bias across the included studies was low; details are available in Supporting Information S1: Material D.

Three non-laboratory-based equations and their corresponding laboratory-based counterparts were identified from studies reporting Kappa agreement outcomes: WHO-2019, D'Agostino Framingham, and the Ueda Globorisk extension [64–66]. Equation formulations, including study populations, input variables, and 10-year predicted outcomes, are detailed in Supporting Information S1: Table E. In all studies, risk scores were calculated using both laboratory-based and non-laboratory-based equations. Agreement between scores was assessed using Spearman's correlation coefficients and Kappa statistics.

Of the 29 studies assessed, 30 distinct populations and 58 effect measures were identified; Kappa statistics were used in three comparisons: two risk categories (cut-off < 20% vs. ≥ 20%; low risk < 10% vs. increased risk ≥ 10%), and low versus high risk. Seventeen studies compared three categories (low < 10%, moderate or intermediate 10%–20%, and high > 20%; or 0%–4.9%, 5%–9.9%, and 10%–19.9%). Six studies compared four categories: low (< 10%), moderate (10%–20%), high (20%–30%), and very high (≥ 30%), or 0%–4.9%, 5%–9.9%, 10%–19.9%, and 20%–29.9%. Three studies compared five categories (low < 10%, moderate 10%–20%, high 20%–30%, very high 30%–40%, and extremely high ≥ 40%; or very low, low, moderate, high, and very high). The population surveys of the included studies, in

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which Kappa statistics were measured, were conducted between 1990 and 2021. Six of the included studies were conducted in Africa [28, 30, 31, 33, 37, 67], 14 in Asia [26, 27, 29, 38, 39, 45, 68–74], and nine in America [25, 32, 34–36, 40–42, 75].

3.3 | Meta-Analysis

3.3.1 | *Correlation Coefficients*

The overall pooled correlation coefficient between the laboratory-based and non-laboratory-based Harvard NHANES equations was 0.954 (95% CI: 0.928–0.971, $I^2 = 100\%$, $p < 0.0001$). All six comparisons contributing to this estimate identified high correlations with the Harvard NHANES equation: D'Agostino Framingham (0.922; 95% CI: 0.921–0.924, $I^2 = 96.5\%$, $p < 0.0001$), Anderson–Framingham (0.936; 95% CI: 0.935–0.938, $I^2 = 95.6\%$, $p < 0.0001$), SCORE-high (0.976; 95% CI: 0.975–0.976, $I^2 = 98.5\%$, $p < 0.0001$), SCORE-low (0.977; 95% CI: 0.976–0.977, $I^2 = 98.5\%$, $p < 0.0001$), CUORE (0.962; 95% CI: 0.960–0.963, $I^2 = 94\%$, $p < 0.0001$), and PCE (0.917; 95% CI: 0.915–0.919, $I^2 = 97.3\%$, $p < 0.0001$) (Figure 1).

Substantial heterogeneity and a wide prediction interval were observed in the pooled analysis, with an I^2 value of 100% (Q test, $p < 0.0001$) and a prediction interval of 0.76–0.99. Effect estimates for subgroups were presented using tables (Supporting Information S1: Material F). The funnel plot and Egger's test indicated no evidence of publication bias ($p = 0.30$; Supporting Information S1: Material G). Sensitivity analysis revealed that no single study had a significant effect on the pooled estimates (Supporting Information S1: Material H).

The correlation between laboratory-based and non-laboratory-based equations was higher for equations predicting fatal outcomes only (0.979; 95% CI: 0.977–0.982) compared with those including both fatal and non-fatal outcomes (0.942; 95% CI: 0.937–0.947; $p < 0.0001$). Older studies (conducted before 2000) had significantly higher correlations (0.974; 95% CI: 0.970–0.978) compared with more recent studies (after 2000; 0.953; 95% CI: 0.948–0.958; $p < 0.0001$). Correlation coefficients were higher for studies from high-income settings (0.967; 95% CI: 0.963–0.970) than low-income settings (0.945; 95% CI: 0.933–0.955; $p < 0.0001$) (Supporting Information S1: Material F). However, there were no statistically significant differences in correlation coefficients by sex or sample size.

3.3.2 | *Kappa Measures of Agreement*

The overall pooled Kappa statistic (κ) between the laboratory-based and non-laboratory-based equations was 0.64 (95% CI: 0.61–0.67, $I^2 = 99\%$, $p < 0.0001$). Subgroup estimates were $\kappa = 0.66$ (95% CI: 0.60–0.72, $I^2 = 99\%$, $p < 0.0001$) for the WHO-2019, $\kappa = 0.62$ (95% CI: 0.57–0.67, $I^2 = 98.9\%$, $p < 0.0001$) for the D'Agostino-Framingham, and $\kappa = 0.65$ (95% CI: 0.60–0.71, $I^2 = 97.9\%$, $p < 0.0001$) for the Ueda Globorisk extension. Substantial heterogeneity was observed across studies, with $I^2 = 99\%$ (Q test, $p < 0.0001$) and a prediction interval of 0.39–0.89 (Figure 2). The funnel plot and Egger's test showed no evidence of publication bias ($p = 0.49$; Supporting Information S1: Material G). The robust omnibus test of moderators was not statistically significant, indicating that agreement estimates did not differ significantly across kappa categories. The pooled agreement estimates were approximately 0.641, 0.631, 0.684, and 0.658 for kappa categories 2, 3, 4, and 5, respectively. Individual pairwise comparisons between kappa categories were also not statistically significant. Sensitivity analysis suggested that individual studies had minimal effect on the pooled estimate and heterogeneity (Supporting Information S1: Material H).

4 | Discussions

This is the first systematic review and meta-analysis to assess the correlation and agreement between non-laboratory-based and laboratory-based cardiovascular risk prediction equations. Twenty-two study populations were included from correlation

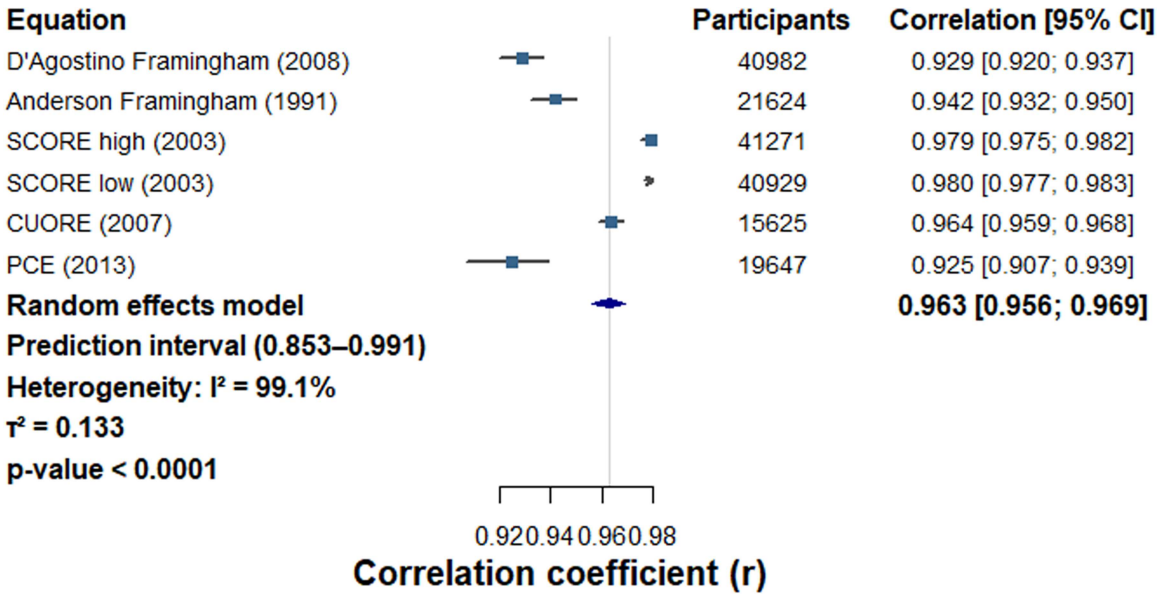


FIGURE 1 | Forest plot on correlations between non-laboratory-based and laboratory-based cardiovascular equations. CI, confidence interval; CUORE, Italian longitudinal study; PCE, pooled cohort equation; SCORE, Systematic Coronary Risk Evaluation.

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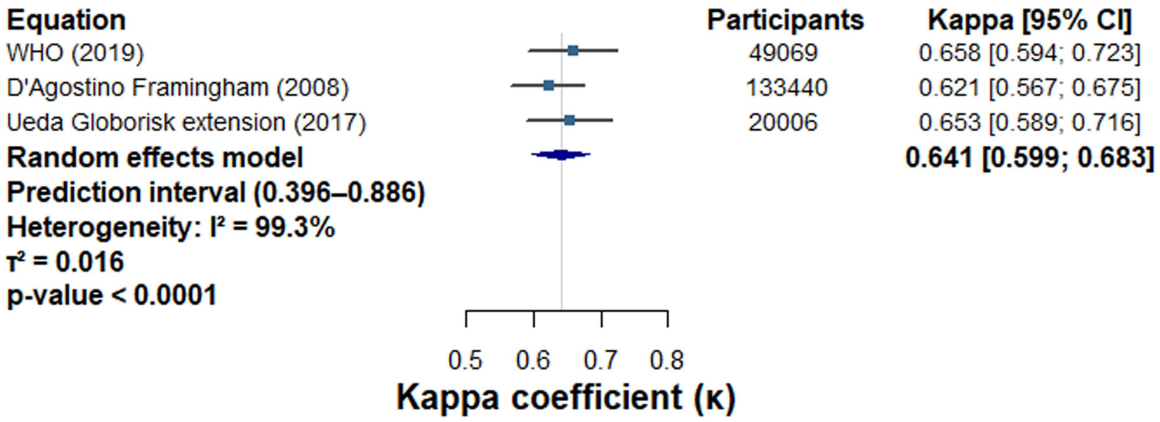


FIGURE 2 | Forest plot on the agreement between non-laboratory-based and laboratory-based cardiovascular risk equations. CI, confidence interval; WHO, World Health Organization.

measure studies (15 Africa [Sub-Saharan]; 3 South Asia; 2 South America; 1 Central Asia; and 1 North America), and 29 study populations were included from kappa statistics measure studies (8 Africa [Sub-Saharan]; 8 South Asia; 6 West Asia; 4 North America; and 3 South America). We found strong correlations between one non-laboratory-based Harvard NHANES equation and six laboratory-based equations, including the D'Agostino-Framingham, Anderson–Framingham, SCORE (high and low risk), CUORE, and the PCE. We also identified substantial agreement between three laboratory-based equations and their corresponding non-laboratory-based versions: WHO-2019, Ueda Globorisk extension, and D'Agostino Framingham.

In this review, some equations are frequently compared for the correlation between non-laboratory-based and laboratory-based equations, and some equations showed a higher correlation than others. Twenty-two correlation measures were used to compare the non-laboratory-based Harvard NHANES equation with three laboratory-based equations: D'Agostino Framingham, SCORE high, and SCORE low. Fifteen correlation measures were used for comparison with the Anderson–Framingham equation, 14 with the CUORE equation, and seven with the Pooled Cohort Equation. Using mixed-effects meta-regression, the type of CVD risk prediction tool was associated with differences in correlations. For example, SCORE high and SCORE low, which predict only fatal outcomes (i.e., death), showed higher correlations than other CVD risk equations that predict both fatal and non-fatal outcomes (such as myocardial infarction and stroke). There is not a statistically significant difference in Cohen's kappa across different types of tools or CVD risk equations.

Strong correlations and substantial agreement between laboratory-based and non-laboratory-based equations across different risk categories and a wide range of risk estimates suggest that non-laboratory-based equations perform well in diverse settings. Although statistically significant differences were observed by data recency, setting, and CVD outcome type, the overall correlations remained high. This systematic review supports the use of non-laboratory-based equations for CVD risk assessment. However, the wide prediction intervals and heterogeneity across studies highlight the need for recalibration and further external validation of these equations within specific regional contexts [76].

Non-laboratory-based equations showed strong concordance with laboratory-based ones. The data required for non-laboratory-based CVD risk prediction can be collected during a single outpatient visit without the need for blood samples. These predictions require only basic medical equipment commonly available in primary health clinics, such as blood pressure apparatus, measuring tapes, and weighing scales. Consequently, non-laboratory-based approaches are crucial for enabling and scaling up CVD risk screening in resource-limited settings. Their use can enhance the uptake of primary CVD prevention services among disadvantaged populations, facilitating broader access to CVD preventive care [77]. Using non-laboratory-based CVD risk equations for primary CVD risk assessment will enhance the accessibility and affordability of primary CVD risk screening. The use of such equations can facilitate the uptake of primary CVD prevention packages. Policymakers must consider the introduction of non-laboratory-based CVD risk equations into the health system for primary CVD risk screening. Future research is warranted to enhance the predictive performance of non-laboratory-based CVD risk equations through external validation, recalibration, and the inclusion of important non-laboratory predictors, such as a history of diabetes.

The integration of non-laboratory-based CVD risk equations into healthcare systems provides essential tools for policy development and public health strategies. These equations use readily available patient information, ensuring broad accessibility and enabling early intervention. By facilitating CVD screening without the need for laboratory tests, they support efficient resource allocation and risk stratification. This, in turn, allows policymakers to design and implement targeted preventive strategies by improving early identification of high-risk and vulnerable groups who lack access to laboratory testing due to cost or access barriers [78, 79]. Involving patients in decision-making regarding the implementation of these tools is crucial for enhancing engagement in preventive care. This approach empowers patients to participate in personalized care plans, increase trust in the patient-provider relationship, and encourages them to take ownership of their health outcomes. By prioritizing patient involvement, healthcare providers can adapt preventive strategies to individual needs and preferences, resulting in improved health outcomes and more effective delivery of preventive care [80].

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Our findings show that studies conducted before 2000 had higher correlations compared to those conducted after 2000, possibly due to differences in baseline CVD risk between the datasets used to derive risk scores and those in which they are applied. Many of the CVD risk equations identified in this review were developed using data collected from the 1960s to the 1990s. In many countries, particularly high-income ones, CVD mortality and event rates have declined over time due to improved prevention and treatment. Moreover, the magnitude of predictors for CVD risk has varied over time. Unless recalibrated, CVD risk equations generally perform less well when applied to populations whose underlying CVD risk differs substantially from that of the derivation dataset. Incidence rates of cardiovascular risk factors have shifted across different time periods. Obesity increased dramatically in the 2010s and 2020s compared to the 1980s and 1990s, while high cholesterol, high blood pressure, and current smoking showed the greatest reductions among overweight and obese groups in recent decades. These changes in risk factor prevalence may be attributed to the increased use of lipid-lowering agents, antihypertensive medications, and overall improved approaches to CVD risk prevention [81–85]. The variation in 10-year CVD risk between populations before and after 2000 reflects changes in both the prevalence of risk factors and the overall management of CVD risk. These variations can be attributed to improvements in risk factor management, particularly in blood pressure control and cholesterol reduction, which have played a major role; significant progress in detecting and treating hypertension, along with the widespread use of statins and other lipid-lowering drugs, has markedly reduced population-level cholesterol levels. Lifestyle changes, including shifts in diet and physical activity, have also influenced overall risk profiles across different time periods. Furthermore, advancements in medical care have improved treatment outcomes, but population ageing has led to a higher absolute number of CVD risk, as older individuals inherently face greater CVD risk. This implies that the equations predicted CVD risk more accurately in populations derived from the same time period than in populations derived from different time periods. Most of the CVD risk equations included in this systematic review and meta-analysis were developed using populations from before 2000 [86, 87].

Almost all equations included in this systematic review and meta-analysis were developed using datasets from high-income settings. Only a few equations, such as WHO-2019 and the Ueda Globorisk Extension, have been recalibrated, at least to some extent, for lower-income regional contexts [64, 66]. Subgroup and meta-regression analyses found that the correlation between laboratory-based and non-laboratory-based equations was higher in high-income settings compared to lower-income settings. This may reflect the underrepresentation of lower-income populations in the cohorts used to develop the most widely applied cardiovascular risk equations. However, our meta-analysis suggests that non-laboratory-based and laboratory-based equations have very similar CVD risk estimates in lower-income groups (correlation coefficient > 0.9). Further development, validation, and recalibration of these equations for specific regional settings are warranted to enhance predictive performance.

This is the first systematic review and meta-analysis to compare the correlation and agreement between non-laboratory-based and laboratory-based cardiovascular risk equations. The synthesis was comprehensive, including nine CVD risk equations without restrictions on the country of development or application. Predefined subgroup and meta-regression analyses were conducted to identify potential sources of variation.

This study-level meta-analysis identified substantial heterogeneity and wide prediction intervals, reflecting the inclusion of studies from diverse settings. The pooled estimates should be interpreted with caution due to extremely high between-study heterogeneity ($I^2 = 99\%–100\%$), indicating substantial variability across included studies. Although laboratory-based and non-laboratory-based risk equations were well correlated, this reflects only comparable performance within the studied populations. Therefore, recalibration and external validation of the equations in specific regional contexts are warranted. The sources of variation contributing to high heterogeneity could be the differences in equation formulation settings. Most of the equations were developed using populations from high-income settings, whereas they were also applied in Low- and Middle-Income Countries (LMIC) settings. Population differences, for example variations in lifestyle such as smoking, prevalence of risk factors like diabetes and hypertension, and demographic factors such as population aging, may also contribute to this heterogeneity. Another potential source of variation is the time in which the equations were developed and applied. Many equations were formulated using data collected before 2000, whereas some of the studies assessing the correlation between non-laboratory-based and laboratory-based equations were conducted after 2000. Consistent with these observations, the meta-regression analysis indicated that correlations were higher in studies conducted before 2000 compared to those conducted after 2000. Studies from high-income settings also showed higher correlations than those from low-income settings, and equations predicting fatal outcomes demonstrated higher correlations than those predicting both fatal and non-fatal outcomes. Overall, the correlations between non-laboratory- and laboratory-based equations are greater than 0.9; however, variations still exist in the strength of these correlations across different populations, time periods, epidemiological trends, study settings, and equation types. Therefore, ongoing external validation, recalibration, and updating of CVD risk equations are needed. While all included studies were published after 2010, some effect measures were derived from surveys conducted before 2000.

The CVD risk equations or risk assessment tools used to compare correlations between studies conducted before and after 2000 were generally the same or used similar predictors and variables. The higher correlations observed in studies conducted before 2000 compared with those conducted after 2000 may therefore reflect differences in populations and epidemiological transitions over time rather than differences in the equations themselves. Most included CVD risk equations were developed using data from the 1960s-1990s, when cardiovascular risk factor distributions and event rates differed substantially from those of more recent populations. Over time, changes in risk factor prevalence, preventive treatments (e.g., statins and antihypertensives), lifestyle patterns, and population ageing have altered baseline cardiovascular risk and reduced comparability across periods. Consequently, CVD risk equations tend to perform more

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consistently in populations similar to their derivation cohorts, which may explain the stronger correlations observed in studies conducted before 2000.

The available evidence on the correlation and agreement between non-laboratory and laboratory-based equations is not evenly distributed across CVD risk equation types, geographic regions, and time periods. Correlation measures predominantly focused on the Harvard NHANES equation, whereas kappa measures were more commonly used for the WHO, Framingham, and Ueda Globorisk equations. In addition, correlation measures were often analyzed in older studies, while kappa measures were more commonly reported in relatively recent studies. This review included only studies published in English, which may limit the representation of non-English-language research and affect the generalizability of the findings.

5 | Conclusions

In conclusion, non-laboratory-based CVD risk equations demonstrate strong correlation and substantial agreement with laboratory-based equations. Non-laboratory-based CVD risk equations show strong concordance with laboratory-based equations in many settings; however, strong correlation and substantial agreement do not necessarily indicate predictive equivalence, and their interchangeability and implementation should be considered context-specific and require external validation and recalibration.

Author Contributions

Yihun Mulugeta Alemu: conceptualization, investigation, methodology, validation, visualization, software, formal analysis, project administration, data curation, writing – original draft, writing – review and editing, resources. **Sisay M. Alemu:** conceptualization, methodology, data curation, validation, investigation, formal analysis, writing – review and editing, visualization. **Nasser Bagheri:** conceptualization, methodology, investigation, validation, writing – review and editing, visualization, formal analysis, supervision. **Kinley Wangdi:** conceptualization, methodology, investigation, validation, supervision, writing – review and editing. **Dan Chateau:** conceptualization, methodology, investigation, validation, writing – review and editing, supervision.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that supports the findings of this study are available in the supporting material of this article.

Transparency Statement

The lead/Corresponding author (Yihun Mulugeta Alemu) affirms that this manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

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