CJC Open 8 (2026) 690–699

Original Article

Initial Cardiovascular Disease Risk Assessment in Nonclinical Settings: Framingham-Simplified Model Predictive Performance in a Canadian Cohort

Jacquecin Garçon, MPH,[a,b,‡] Mathilde Lavigne-Robichaud, PhD,[b,‡] Alain Milot, MD,[b,c] Denis Talbot, PhD,[a,b] Mahée Gilbert-Ouimet, PhD,[b,d] Chantal Brisson, PhD,[a,b,e] Clermont E. Dionne, PhD,[a,b] and Xavier Trudel, PhD[a,b]

[a] *Department of Social and Preventive Medicine, Faculty of Medicine, Université Laval, Québec, Québec, Canada*

[b] *Population Health and Optimal Health Practices Research Program, CHU de Québec-Université Laval Research Center, Québec, Québec, Canada*

[c] *Department of Medicine, Faculty of Medicine, Université Laval, Québec, Québec, Canada*

[d] *Department of Health Sciences, Université du Québec à Rimouski (UQAR), Lévis, Québec, Canada*

[e] *VITAM, Centre de recherche en santé durable, Québec, Québec, Canada*

ABSTRACT

Background: The Framingham risk model is recommended for estimating 10-year cardiovascular disease (CVD) risk in Canada. Its simplified version (Framingham-SP) replaces lipids with body mass index, eliminating the need for laboratory testing. The predictive performance of the Framingham-SP in Canada is unknown. This external validation study assessed the discrimination and calibration of the Framingham-SP model in a prospective cohort from Quebec, Canada.

Methods: The study sample included 3692 women and 3671 men free of CVD at baseline (1999-2001; follow-up until 2011). Incident CVD events were identified using medico-administrative databases. Multiple imputation was used to handle missing data. Discrimination was assessed using C-statistics. Calibration was evaluated using calibration-in-the-large, absolute difference, and discordance.

Results: Over the 10-year follow-up, 211 CVD events occurred among women and 453 occurred among men. For women, the predicted 10-year CVD risk was 4.07% (95% confidence interval [CI]: 3.94-4.20), and the observed risk was 5.72% (95% CI: 5.01-6.51). The C-statistic

RÉSUMÉ

Contexte : Le modèle de risque de Framingham est recommandé au Canada pour estimer le risque de maladie cardiovasculaire (MCV) à 10 ans. Sa version simplifiée (Framingham-SP) remplace les taux lipidiques par l'indice de masse corporelle, éliminant ainsi le besoin de tests en laboratoire. La performance prédictive du modèle Framingham-SP au Canada est inconnue. Cette étude de validation externe a évalué la discrimination et la calibration du modèle Framingham-SP dans une cohorte prospective du Québec, au Canada.

Méthodologie : L'échantillon de l'étude comprenait 3 692 femmes et 3 671 hommes sans MCV au départ (1999-2001 ; suivi jusqu'en 2011). Les cas incidents de MCV ont été identifiés dans les bases de données médico-administratives. Une imputation multiple a été utilisée pour traiter les données manquantes. La discrimination a été évaluée à l'aide de la statistique C (indice de concordance). La calibration du modèle a été évaluée à l'aide de la calibration globale, de la différence absolue et de la discordance.

Résultats : Au cours du suivi de 10 ans, 211 cas incidents de MCV sont survenus chez les femmes et 453 chez les hommes. Chez les femmes, le

Cardiovascular diseases (CVDs) are the leading cause of mortality and morbidity worldwide, responsible for an estimated 17.9 million deaths annually and accounting for 393 million disability-adjusted life years (DALYs) globally.[1] In Canada, CVDs remain a major public health concern, contributing to nearly 1 in 4 deaths and generating substantial healthcare expenditures, exceeding $21 billion annually.[2,3] Early identification of individuals who have an elevated risk for CVD is essential to prevent disease development through timely lifestyle changes and pharmacologic interventions.

Risk prediction models are central to primary prevention strategies, enabling the identification of individuals at risk

Keywords: risk prediction; cardiovascular disease; Framingham, non-laboratory based; cohort study, nonclinical settings; external validation study

‡J. Garçon and M. Lavigne-Robichaud share first authorship.

Corresponding authors:
Xavier Trudel
E-mail: xavier.trudel@crchudequebec.ulaval.ca
Alain Milot
E-mail: alain.milot@fmed.ulaval.ca
LinkedIn: www.linkedin.com/in/mathilde-lavigne-robichaud-phd

See page 698 for disclosure information.

Received for publication October 10, 2025. Accepted January 29, 2026.

https://doi.org/10.1016/j.cjco.2026.01.014

2589-790X/© 2026 The Authors. Published by Elsevier Inc. on behalf of the Canadian Cardiovascular Society. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

was 0.72 (95% CI: 0.68-0.76). For men, the predicted risk was 11.73% (95% CI: 11.44-12.02), and the observed risk was 12.34% (95% CI: 11.32-13.44). The C-statistic was 0.72 (95% CI: 0.69-0.74). Overall, calibration was lower among women, particularly those in intermediate and high predicted risk categories.

Conclusions: The Framingham-SP model demonstrated acceptable predictive performance in this Canadian cohort. Its simplified format may help support preliminary CVD risk assessment and lifestyle counselling, including in nonclinical settings, while remaining complementary to guideline-based clinical evaluation. Recalibration may improve performance among high-risk women.

risque prédit de MCV à 10 ans était de 4,07 % (intervalle de confiance [IC] à 95 % : 3,94-4,20) et le risque observé était de 5,72 % (IC à 95 % : 5,01-6,51). La statistique C était de 0,72 (IC à 95 % : 0,68-0,76). Chez les hommes, le risque prédit était de 11,73 % (IC à 95 % : 11,44-12,02) et le risque observé était de 12,34 % (IC à 95 % : 11,32-13,44). La statistique C était de 0,72 (IC à 95 % : 0,69-0,74). Dans l'ensemble, la calibration était plus faible chez les femmes, en particulier chez celles appartenant aux catégories de risque prédit intermédiaire et élevé.

Conclusions : Le modèle Framingham-SP a démontré une performance prédictive acceptable dans cette cohorte canadienne. Son format simplifié pourrait faciliter l'évaluation préliminaire du risque de MCV et guider les conseils sur le mode de vie, y compris dans des contextes non cliniques, tout en restant complémentaire à une évaluation clinique basée sur les lignes directrices. Une recalibration du modèle pourrait améliorer sa performance chez les femmes à haut risque.

before CVDs occur. The 10-year Framingham Risk Score (FRS)[4] is recommended by Canadian national guidelines for estimating overall cardiovascular risk.[5-7] The FRS estimates risk based on age, sex, systolic blood pressure, smoking status, diabetes, and lipid levels and supports decision-making regarding the management of dyslipidemia, hypertension, and diabetes.[4]

A simplified version of the FRS, the Framingham-SP model, replaces lipid levels with body mass index (BMI), while maintaining comparable predictive performance.[8] By eliminating the need for laboratory tests, the Framingham-SP provides a practical alternative for routine risk assessment in nonclinical settings. The Framingham-SP can be used to support initial screening by nonphysician professionals (eg, physiotherapists, kinesiologists, fitness trainers), to facilitate immediate lifestyle counselling in workplace and community settings, and encourage individual assessment through online calculators and mobile apps. The Framingham-SP also can facilitate broader applications in population health research and public health surveillance.[8,9] Finally, this model also can be valuable as a pragmatic triage and counselling tool, supporting risk-modulated referral to primary care for confirmatory laboratory testing and pharmacotherapy when indicated. Although Canada has strong laboratory capacity, access to preventive care remains uneven. In 2023, nearly 17% of Canadian adults, close to 7 million people, reported not having a regular family doctor, nurse practitioner, or other primary care provider.[10,11] In this context, a simplified risk assessment is relevant to promote equitable access to cardiovascular risk evaluation.

The predictive performance of the Framingham-SP model in Canada is unknown. The lack of validation in Canada may limit confidence in its applicability and the ability of guideline developers to endorse its use.[6] This study, therefore, aimed to evaluate the 10-year predictive performance of the Framingham-SP model in a Canadian prospective cohort.

Material and Methods

The PROspective Quebec (PROQ) cohort

Participants were recruited into the PROQ cohort between 1991 and 1993, including 9188 individuals aged 18-65 years, representing 75% of all workers in participating organizations who were invited and agreed to enroll in the study.[12] Medico-administrative data were available for 96% of the cohort, enabling the identification of CVD events that occurred from January 1, 1991 onward. Informed consent was obtained from all participants, and the study was approved by the ethics committee of the Centre de Recherche du CHU de Québec—Université Laval. Data can be made available from the corresponding author upon reasonable request.

Study reporting

The research reported in this paper is an external validation study and adhered to the TRIPOD+AI (**T**ransparent **R**eporting of a Multivariable Prediction Model for **I**ndividual **P**rognosis **o**r **D**iagnosis) guidelines.[13]

Study population

At the first in-person follow-up visit of the PROQ cohort (1999-2001), 8120 of the 9188 original participants attended the visit (88%), corresponding to 90% of those eligible for follow-up (ie, still alive at the time of the visit; 117 participants had died between recruitment and this follow-up visit).[12] This visit was considered the analytical baseline for assessing CVD risk factors. Prevalent CVD cases (n = 651) were excluded using 2 complementary sources. Before January 1, 1991, history of CVD was based on self-report at cohort entry. From January 1, 1991 onward, prevalent cases were ascertained through validated linkage with Quebec's medico-administrative databases. Additional exclusions were applied for age (< 30 years, n = 28; ≥ 75 years, n = 5), participants who had moved out of the province between baseline and follow-up (n = 5), and absence of administrative data linkage (n = 68). In total, 7363 participants (3692 women and 3671 men) were included in the analytic sample (Fig. 1).

Cardiovascular disease

Incidence of CVD was defined as follows: the first occurrence of coronary heart disease, stroke or transient ischemic attack, congestive heart failure, or peripheral artery disease; or cardiovascular death (Table 1). Events were ascertained using validated algorithms[14-17] applied to

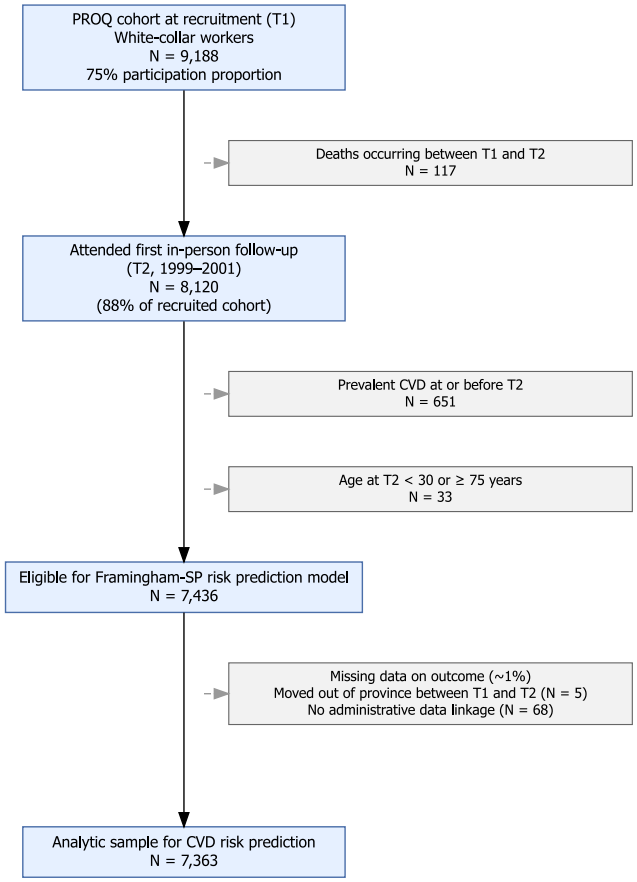


Figure 1. Flow of participants from the original PROspective Quebec **(**PROQ) cohort to the analytic sample. Flow diagram shows participant selection for external validation of the Framingham-simplified version (SP) cardiovascular disease (CVD) risk prediction model in the PROQ cohort. At recruitment (T1), 9188 white-collar workers were enrolled. At the first in-person follow-up (T2: 1999–2001), 8120 participants attended (88% of the recruited cohort; 90% of those eligible, excluding 117 deaths between T1 and T2). Participants with prevalent CVD at or before T2 were excluded (n = 651), based on self-reported history for events before January 1, 1991, and validated linkage with Quebec medico-administrative databases thereafter. Additional exclusions were age at T2 < 30 or ≥ 75 years (n = 33), relocation out of province (n = 5), and lack of administrative data linkage (n = 68). The final analytic sample included 7363 participants. Outcome missingness was < 1%; missing predictor data were handled using multiple imputation.

Quebec's medico-administrative databases,[18] as well as the death registry database.[19] Diagnostic and procedure codes from hospitalization and outpatient records in the Régie de l'assurance maladie du Québec (RAMQ) administrative database were used to identify CVD events. Specifically, diagnostic codes included International Classification of Diseases (ICD), 9th revision − Clinical Modification (ICD-9-CM) and ICD-, 10th revision—Canada (ICD 10-CA), and procedure codes were drawn from the Canadian Classification of Diagnostic, Therapeutic, and Surgical Procedures (CCP) or the Canadian Classification of Health Interventions (CCI). Full case definitions and coding algorithms are provided in Supplemental Appendix S1 and Supplemental Table S1. CVD events were then identified over a 10-year follow-up period (2001-2011).

Cardiovascular risk factors

Cardiovascular risk factors were measured using standardized procedures during the first in-person visit (1999-2001), the analytical baseline. Sex and age were collected via a self-administered questionnaire. Height and weight were measured by trained research nurses according to standardized protocols, and BMI was subsequently calculated as weight in kilograms divided by height in meters squared (kg/m^2). Systolic blood pressure (SBP) was measured following the American Heart Association protocol.[20] Three readings were taken on both arms, spaced 1-2 minutes apart, after participants had been seated for 5 minutes, and the average of these readings was used. Antihypertensive medication use was self-reported as a binary variable (yes/no). Smoking status was self-reported and categorized as either nonsmoker or current smoker. Diabetes was defined by self-reported use of insulin or oral hypoglycemic agents, or by a prior diagnosis of diabetes.

Framingham-SP model

The 10-year CVD risk was predicted for each participant using the sex-specific coefficients from the Framingham-SP

Table 1. Definition of cardiovascular disease (CVD) in this study

CVD events were defined as the first occurrence of any major cardiovascular outcome recorded in provincial administrative databases, as follows:

- **Coronary heart disease**
  - o ICD-9: 410−414
  - o ICD-10: I20−I25
  - o Procedure codes (48.02; 48.03; 48.09; 481; 1IJ50; 1IJ57GQ; 1IJ76)
- **Stroke or transient ischemic attack**
  - o ICD-9: 362.3; 430−432; 434-436
  - o ICD-10: I60; I61; I63 (excluding I63.6); I64; H34.0; H34.1; G45 (excluding G45.4)
- **Congestive heart failure**
  - o ICD-9: 428
  - o ICD-10: I50.0; I50.1; I50.9
- **Peripheral arterial disease**
  - o ICD-9: 440.2; 440.21−440.24; 440.9; 443.9I
  - o ICD-10: I70.2; I70.9; I73.9
  - o Procedure codes (1WK93; 9611; 3KG10VA; 3KG20; 3KG30; 3KG40; 275; 276; 287; 5088)

CVD events and deaths were ascertained from hospital discharge, procedure, and mortality databases using these International Classification of Diseases revision 9 (ICD-9) and ICD revision 10 (ICD-10) diagnostic and procedure codes. The complete list of codes and the detailed algorithms are provided in the Supplemental Table S1 and Supplemental Appendix S1, respectively.

model, as published by D'Agostino et al.[8] This version replaces lipid measurements with BMI and includes the following predictors: age; SBP (modelled with separate coefficients for treated and untreated participants); smoking; diabetes; and BMI (Table 2). Further details on the variables included and comparisons between the Framingham-SP and the traditional Framingham models are provided in Supplemental Table S2.

Statistical analysis

All statistical analyses were conducted using multiple imputation by chained equations (MICE) to handle missing predictor data (approximately 5% overall) following recommendations for survival analysis.[21] Imputation models were specified separately for men and women, consistent with the sex-specific structure of the Framingham-SP model. The number of imputed datasets was set to be equal to the percentage of participants with at least one missing value. Each imputed dataset was analyzed separately, and results were pooled using Rubin's rules to obtain overall estimates and 95% confidence intervals (95% CIs), unless otherwise specified.

The time-to-event for each participant began on the day of their in-person visit during the first follow-up (1999-2001). Time was measured from this exact date until the first occurrence of a cardiovascular event, death from noncardiovascular causes, or the end of the 10-year follow-up period. Participants who did not experience a CVD event or who died from noncardiovascular causes were censored at the date of death or at the end of the study period, whichever came first. The cumulative incidence of cardiovascular events was estimated using the Kalbfleisch-Prentice estimator to account for competing risks, defined as noncardiovascular deaths.[22,23] These analyses were stratified by sex.

To evaluate prediction accuracy, the mean predicted 10-year risk was calculated using the Framingham-SP equation. The predicted risk corresponds to the mean predicted 10-year risk across imputed datasets. The observed 10-year risk was derived using the cumulative incidence function that accounts for competing risks, with noncardiovascular deaths treated as competing events. Because the outcome was not imputed, the observed risk was identical across imputed datasets. Its 95% CI was therefore estimated using a conventional binomial approach based on the total number of events and participants, which provides a pragmatic approximation of uncertainty. Predicted and observed risks were also compared across risk categories: low risk (< 10% predicted 10-year CVD risk); intermediate risk (10%-19.9%); and high risk (≥ 20%).

Table 2. Framingham simplified (Framingham-SP) risk prediction model

The **Framingham-Simplified (SP) model** is a sex-specific, BMI−based version of the Framingham 10-year general cardiovascular disease risk score. It uses nonlaboratory predictors, with distinct coefficients according to sex and antihypertensive treatment status.

Predictors measured at T2 (1999−2001):

- **Sex** (women/men); all model coefficients are sex specific.
- **Age** (years, continuous, 30−75 years old)
- **SBP** (mm Hg, continuous)
  - o Different SBP coefficients are applied for individuals treated vs not treated for hypertension.
- **Treatment for hypertension** (yes/no)
- **Current smoking** (yes/no)
- **Diabetes** (yes/no)
- **BMI** (kg/m^2, continuous)

Outcome: 10-year risk of a first major cardiovascular disease event.

BMI, body mass index; SBP, systolic blood pressure.

The Framingham-SP model was evaluated for both calibration and discrimination over the 10-year follow-up period.[8]

Calibration. Calibration, the agreement between predicted and observed absolute risk, was assessed using complementary descriptive, model-based, and graphical approaches. We first calculated relative discordance using the following formula:

$$Discordance = \frac{predicted - observed}{observed}$$

A discordance of 0 indicates perfect calibration; positive values reflect overestimation of risk in the model; and negative values indicate underestimation. In addition, the absolute difference between predicted and observed risks was also calculated to summarize the absolute deviation from the observed event rate. Together, these measures provide an intuitive assessment of calibration accuracy.[24]

Then, calibration-in-the-large, expressed as the predicted-to-observed ratio, was computed to assess systematic over- or underestimation at the population level. Values close to 1 indicate good calibration, whereas values < 1 indicate underestimation, and values > 1 indicate overestimation.[24,25]

The calibration slope at 10 years was estimated using a secondary Cox proportional hazards model, with time since baseline as the time scale and a transformed predicted 10-year survival probability as the sole covariate, as described by McLernon et al.[26] Estimates were pooled across imputations. A calibration slope of 1 indicates perfect calibration; values > 1 suggest under-dispersion of predicted risks, whereas values < 1 indicate over-dispersion.

Finally, calibration was further examined graphically by plotting the pooled observed risks against deciles of pooled

predicted risk, providing a visual comparison of model performance across different levels of predicted risk.

Discrimination. Discrimination was evaluated by calculating Harrell's C-statistic and plotting the receiver operating characteristic (ROC) curve, which visualizes sensitivity vs 1-specificity.[27] The C-statistic reflects the model's ability to differentiate between individuals who experienced a cardiovascular event and those who did not. C-statistics were calculated separately within each imputed dataset and pooled. Standard errors were obtained using an analytical variance estimator, and 95% CIs were derived accordingly. A value of 0.50 indicates discrimination that is no better than chance alone, whereas high values indicate better performance.[28]

To explore potential variation in model performance by age, calibration and discrimination metrics were additionally estimated, by sex, within 2 age strata (30-49 and 50-74 years).

Results

Baseline characteristics of the study population

This study included 3692 women and 3671 men free of CVD at baseline (1999-2001), with a 10-year follow-up period. The mean age was 45.8 years (standard deviation [SD] = 7.5) in women and 48.6 years (SD = 8.4) in men. The mean BMI was 24.9 kg/m^2 (SD = 4.7) in women and 26.4 kg/m^2 (SD = 3.7) in men. The mean SBP was 115.5 mm Hg (SD = 14.4) in women and 125.3 mm Hg (SD = 14.0) in men. The diabetes prevalence was 2.2% in women and 3.1% in men, and current smoking was reported by 16.0% of women and 12.9% of men. Detailed baseline characteristics are shown in Table 3. Over the 10-year follow-up, 211 CVD events occurred in women and 453 in men. Key findings are summarized in the Central Illustration.

Calibration and discrimination of Framingham-SP for 10-year CVD risk

In women, the mean predicted 10-year CVD risk was 4.07% (95% CI: 3.94%-4.20%), and the observed risk was 5.72% (95% CI: 5.01%-6.51%; Table 4). The discordance was −29%, and the absolute difference between predicted and observed risk was −1.6%. The calibration-in-the-large was 0.71 (95% CI: 0.62-0.82). The calibration slope was 1.07 (95% CI: 0.91-1.23). The C-statistic was 0.72 (95% CI: 0.68-0.76). In men, the predicted 10-year CVD risk was 11.73% (95% CI: 11.44%-12.02%), whereas the observed risk was 12.34% (95% CI: 11.32%-13.44%), yielding a discordance of −5%, and an absolute difference of −0.61%. The calibration-in-the-large was 0.95 (95% CI: 0.87-1.04). The calibration slope was 1.03 (95% CI: 0.91-1.15). The C-statistic was 0.72 (95% CI: 0.70-0.74).

Figure 2 presents the ROC curve derived from a randomly selected imputed dataset ([A] for women and [B] for men). The stability of area under the curve estimates across imputations, along with the corresponding ROC curves for all imputations, is illustrated in Supplemental Figures S1 and S2. The pooled area under the curve was 0.7265 (95% CI: 0.6864-0.7666) among women and 0.7282 (95% CI: 0.7007-0.7558) among men.

Table 3. Descriptive analysis of study sample at first follow-up in 1999-2001, stratified by sex (women n = 3692; men n = 3671)

Baseline characteristics	Women (50.1%)	Men (49.9%)
Age, years	45.8 ± 7.5	48.6 ± 8.4
Education		
High school or less	1511 (40.9)	458 (12.5)
College	1104 (29.9)	942 (25.7)
University	1077 (29.2)	2272 (61.9)
BMI, kg/m^2	24.9 ± 4.7	26.4 ± 3.7
SBP, mm Hg	115.6 ± 14.4	125.3 ± 14.0
Anti-hypertensive medication use	245 (6.6)	292 (7.9)
Diabetes	83 (2.2)	113 (3.1)
Current smoker	591 (16.0)	472 (12.9)

Data are presented as mean ± standard deviation for continuous variables, and number (percentage) for categorical variables. Descriptive statistics were obtained by pooling estimates across all imputed datasets.

BMI, body mass index; SBP, systolic blood pressure.

Graphical calibration plots (Fig. 3) further illustrate the agreement between the predicted and observed risks across deciles of predicted risk. Deviation below the 45-degree line indicates risk underestimation, which was more pronounced among women with higher observed risk levels (Fig. 3A).

Risk stratification based on the Framingham-SP model

In women, the model underestimated CVD risk across the 3 categories, with the greatest discrepancy observed in the intermediate-risk group (13.19% predicted vs 22.82% observed) and the high-risk group (27.98% predicted vs 40.75% observed; Table 5). In men, predicted and observed risks were more closely aligned. However, estimates in the intermediate- and high-risk strata among women should be interpreted cautiously, given the smaller number of observed events and the resulting imprecision in subgroup-specific calibration estimates.

Age-stratified performance of the Framingham-SP model

Across age strata, discrimination was lower than it was in the overall analyses for both women and men

Table 4. Calibration and discrimination of the Framingham-simplified risk score for 10-year cardiovascular disease (CVD) prediction, stratified by sex (women n = 3692; men n = 3671)

Metric	Women (211 events)	Men (453 events)
Predicted risk, % (95% CI)	4.07 (3.94−4.20)	11.73 (11.44−12.02)
Observed risk, % (95% CI)	5.72 (5.01−6.51)	12.34 (11.32−13.44)
Discordance, %	−29	−5
Absolute difference, %	−1.6	−0.61
Calibration-in-the-large	0.71 (0.62−0.82)	0.95 (0.87−1.04)
Calibration slope	1.07 (0.91−1.23)	1.03 (0.91−1.15)
C-statistic (95% CI)	0.72 (0.68−0.76)	0.72 (0.670−0.747)

Analyses used multiple imputations for missing predictors; estimates were pooled using Rubin's rules unless otherwise specified. Predicted risk is the mean Framingham-Simplified 10-year CVD risk across imputations (95% confidence interval [CI] via Rubin's rules). Observed risk is the 10-year cumulative incidence of CVD; because outcomes were not imputed, its 95% CI was estimated using a binomial approach. Discordance was calculated as *[(Predicted-Observed)/Observed]*100*. Absolute difference represents the percentage difference between predicted and observed event rates. Calibration-in-the-large is the ratio of predicted to observed risk. Calibration slope reflects risk dispersion. Discrimination was assessed using Harrell's C, pooled across imputations with 95% CI obtained by bootstrap (1000 replications).

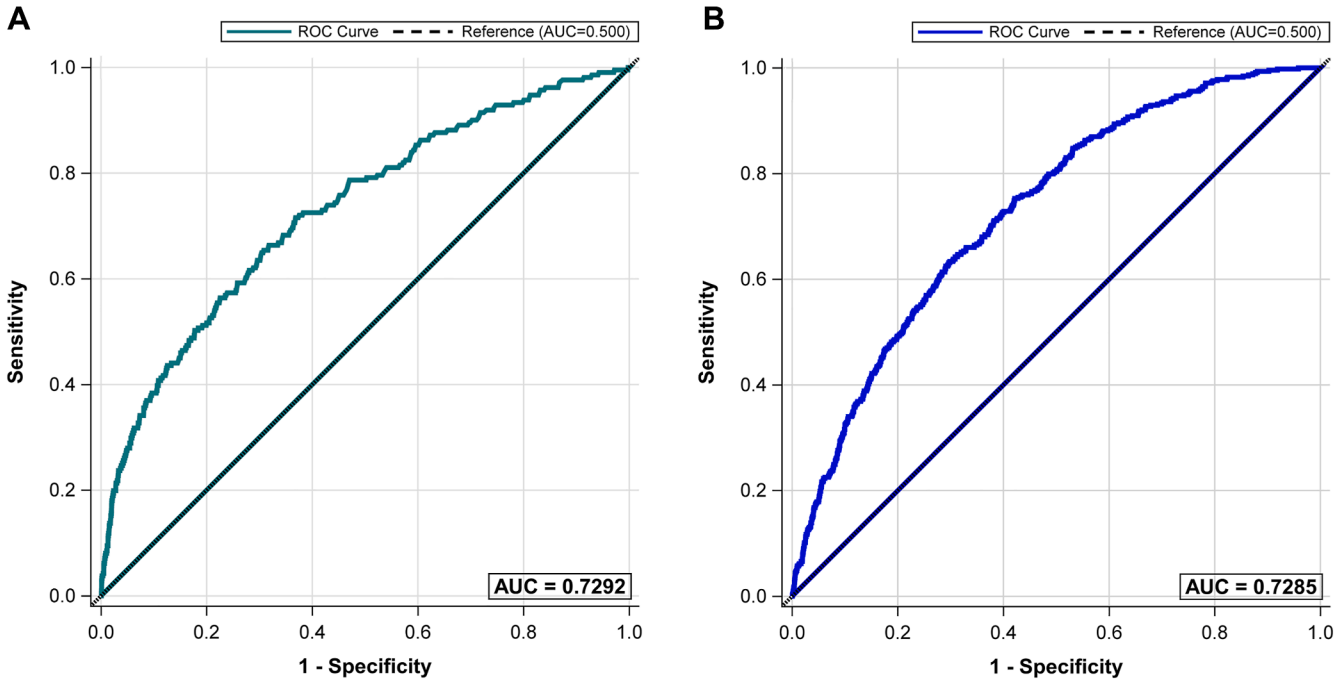


Figure 2. Receiver operating characteristic (ROC) curves for cardiovascular disease (CVD) risk prediction models (women and men). ROC curves illustrating the discriminatory performance of CVD risk prediction models for (**A**) women and (**B**) men based on one randomly selected imputed dataset. The ROC curve plots sensitivity (true positive rate) against 1-specificity (false positive rate). The area under the curve (AUC) quantifies the model's ability to discriminate individuals with vs without CVD events over the 10-year period. In this context, the AUC is equivalent to Harrell's C-statistic obtained from the Cox proportional hazards models. The **diagonal line** represents random classification (AUC = 0.5).

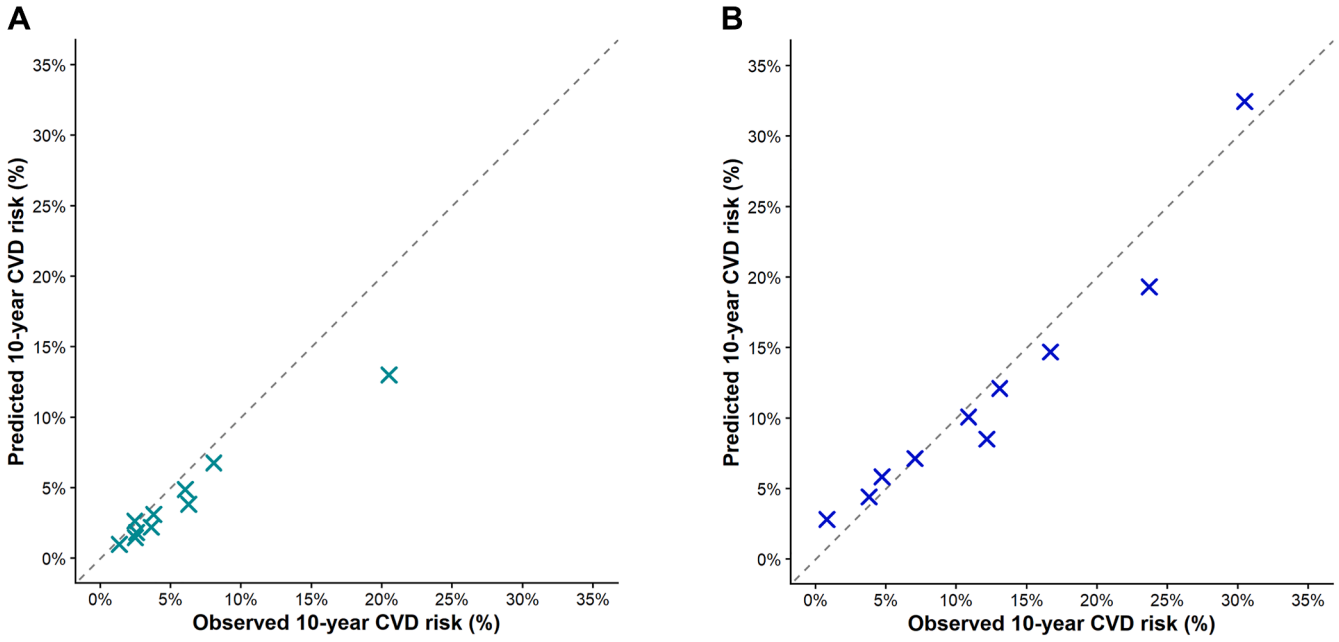
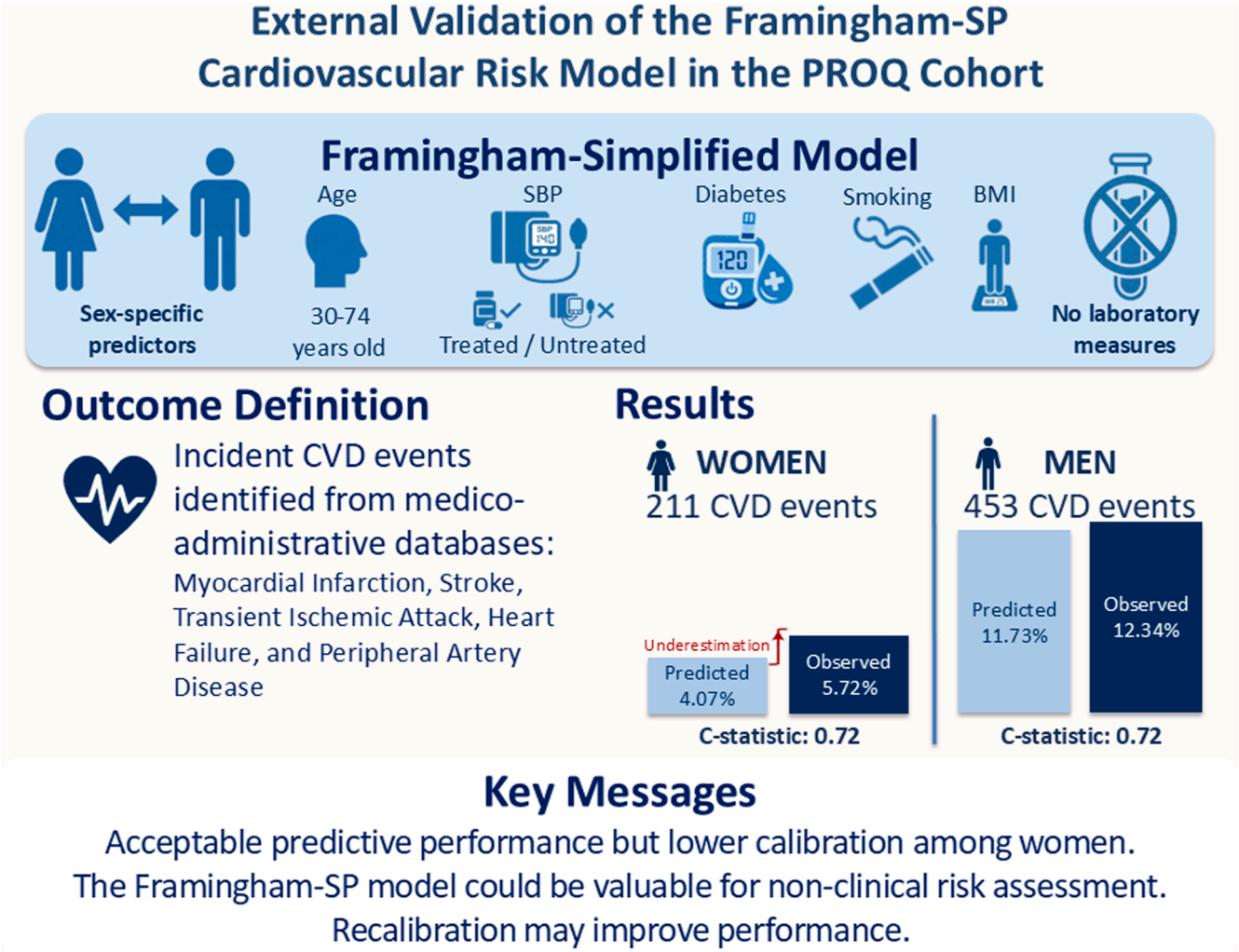


Figure 3. Observed vs predicted cardiovascular disease (CVD) risk (women and men). Scatter plots comparing observed and predicted CVD risk scores over the 10-year period for (**A**) women and (**B**) men, with estimates pooled across all imputed datasets. Each **cross** represents a risk group. The x-axis shows the observed CVD risk, and the y-axis shows the corresponding mean predicted risk for that group. The **dashed line** represents the line of perfect calibration (y = x). Points closer to this line indicate higher model accuracy, with deviations from this line reflecting under- or over-prediction across different population subgroups.

Table 5. Predicted and observed 10-year cardiovascular disease (CVD) risk by risk category

Risk category	Population	N (%)	Predicted risk (%)	Observed risk (%)
Low risk (< 10%)	W	3452 (93.5)	3.29	4.35
	M	2014 (54.9)	6.08	6.13
Intermediate risk (10%−19%)	W	207 (5.6)	13.19	22.82
	M	1154 (31.4)	13.88	15.13
High risk (≥ 20%)	W	33 (0.9)	27.98	40.75
	M	503 (13.7)	29.43	30.79

Risk categories were defined using the Framingham-simplified 10-year CVD risk score: low (< 10%); intermediate (10%−19.9%); and high (≥ 20%). Analyses used multiple imputation for missing predictors; risk categories and predicted risks were derived within each imputed dataset and averaged across imputations. Predicted risk is the mean 10-year CVD risk within each category, and observed risk is the 10-year cumulative incidence of CVD, estimated conventionally because outcomes were not imputed. M, men; W, women.



Central Illustration. External validation of the Framingham SP (using body mass index) 10-year CVD risk model in the PROQ cohort. The illustration summarizes model discrimination and calibration by sex, highlighting acceptable discrimination in both women and men and underestimation of observed risk in women, particularly at higher predicted-risk levels. BMI, body mass index; CVD, cardiovascular disease; Framingham SP, Framingham simplified; PROQ, Prospective Québec; SBP, systolic blood pressure.

(Supplemental Table S3). In women, risk underestimation increased with age, with the lowest calibration-in-the-large observed among those aged 50-74 years (0.65; 95% CI: 0.54-0.77). In men, age-stratified calibration remained closer to unity across age groups, with only modest underestimation among older men.

Discussion

This study is the first to evaluate the predictive performance of the Framingham-SP model in Canada.[8] In men, the model demonstrated acceptable discrimination (C-statistic 0.72) and average calibration, with calibration-in-the-large close to unity (0.95). In women, discrimination was similar (C-statistic 0.72), but calibration-in-the-large was lower (0.71). Calibration slopes were close to unity in both women and men.

Comparison with previous studies

In the US, Gaziano et al.[29] reported discrimination comparable to our results for the Framingham-SP model, supporting reasonable performance outside the original derivation setting, including in more heterogeneous US populations. Similarly, Pandya et al.[30] found comparable discrimination between the Framingham-SP and the original lipid-based FRS. External validations conducted in European and Asian populations have reported lower C-statistics and a general tendency to overestimate risk.[31-34] This discrepancy with our findings may be attributable to differences in population characteristics, outcome definitions, and the length of the follow-up period.

Comparison within the Canadian context

To our knowledge, the present study is the first to assess the performance of the Framingham-SP in Canada. Two recent studies have assessed the performance of the lipid-based FRS. Ko et al.[35] reported substantial overestimation of risk in an Ontario cohort, and Sud et al.[36] found that recalibration improved performance, although some miscalibrations remained.

Implications

The Framingham-SP model represents a pragmatic and low-cost tool for a timely estimation of 10-year CVD risk. The model enables risk estimation at the most opportune time. Its simplicity makes it well-suited for use both within and outside clinical settings, including in fitness centres, community health programs, and workplace screenings. Moreover, its ease of application facilitates use by not only physicians but also allied health professionals, thereby broadening opportunities for risk communication, lifestyle counselling, and timely referral for further assessment. The Framingham-SP may be considered complementary to guideline-based clinical evaluation, providing an accessible risk-assessment tool to support initial screening and lifestyle counselling in nonclinical settings.[6,7] This usage aligns with Canadian guidelines on the management of risk factors, which promote health behaviour modifications as the "cornerstone of chronic disease prevention."[7]

The present study provides country-specific evidence on the performance of the Framingham-SP model, serving as a reference for its application in Canada and for guiding future validation. For instance, recent European recalibration efforts have demonstrated the value of incorporating socioeconomic factors into CVD risk-prediction models. Contemporary models, including QRISK3,[37] Systematic COronary Risk Evaluation 2 (SCORE2),[38] and the American Heart Association Predicting risk of cardiovascular disease EVENTs (PREVENT)[39] equations, incorporate socioeconomic or regional context and have demonstrated improved predictive performance. Future model adaptations should therefore, examine whether integrating social determinants of health (eg, income, education, neighbourhood deprivation, work environment) enhances the accuracy of risk prediction.

The present study relied on participants from an occupational cohort, and the study period was 2001-2011. Since then, temporal changes have occurred in the prevalence of risk factors.[40-42] Nonetheless, several considerations favour external validity. First, the baseline hazard observed in our cohort is highly consistent with both the original Framingham study and large-scale, contemporary Canadian population-based estimates. Second, CVD incidence is also broadly comparable to that in national data.[43] In addition, the magnitude of associations between risk factors and CVD in the present cohort (Supplemental Table S4) is comparable to those observed in the general population of Ontario,[44] supporting the transportability of the relative risk structure. This concordance suggests that the risk profile of our population is comparable to that observed in the general Canadian population. Nonetheless, further validation across populations and timeframes is required.

Strengths and limitations

This study has several important strengths. First, it is the first study to evaluate the Framingham-SP model in Canada. The study drew on a prospective cohort with a high follow-up rate (96%) and a high baseline participation rate (75% of eligible workers). The cohort included a similar proportion of men and women, allowing for balanced sex-specific analyses. Missing data were handled using multiple imputation. The use of medico-administrative data within a universal health coverage system allowed the identification of CVD events using previously validated Canadian algorithms. An additional strength is the explicit consideration of competing risks, which provides more accurate estimates of observed CVD incidence.

Limitations should also be acknowledged. First, the cohort consisted predominantly of White participants,[45-48] which may limit the generalizability of results.[41,49] Second, self-reported smoking and diabetes may have led to risk underestimation, although validation studies support the reliability of these measures.[50,51] In addition, this approach is coherent, with real-world usage in nonclinical settings. Third, despite the use of validated algorithms, some CVD events likely were missed, particularly for congestive heart failure and peripheral artery disease. Finally, although results suggest lower calibration among women in predicted intermediate and high-risk categories, these findings must be interpreted with caution, given the small number of women and CVD events in these subgroups, which limits the precision of subgroup-specific estimates. Future studies are required to guide potential sex-specific adaptations.[52] Indeed, sex-specific risk factors have been associated with CVD risk in women (adverse pregnancy outcomes, premature menarche, premature menopause and vasomotor symptoms, polycystic

ovarian syndrome, infertility, and psychosocial factors), prompting the American Heart Association and American College of Cardiology to include *risk-enhancing factors* specific to women in their guidelines on the primary prevention of CVD.[53]

Conclusions

The Framingham-SP model showed acceptable accuracy for predicting 10-year CVD risk in this Canadian cohort. Its simplified format, substituting BMI for cholesterol, makes it especially practical for preliminary risk stratification and risk communication in nonclinical settings. Nonetheless, elevated predicted risk should prompt confirmatory clinical evaluation. Future efforts should focus on recalibration and validation in other Canadian populations. The relevance of considering social and behavioural determinants to improve accuracy also should be determined.

Acknowledgements

This team was supported by the Fonds de recherche du Québec (FRQ) through the research centre grant for the CHU de Québec-Université Laval Research Center (reference: 30641). The authors thank Eric Demers and Caty Blanchette, who assisted with data management, variable coding, and code validation, and were not rewarded for their contributions outside their salaries.

Ethics Statement

The study reported in this paper adhered to the TRIPOD+AI (**T**ransparent **R**eporting of a multivariable prediction model for **I**ndividual **P**rognosis **o**r **D**iagnosis + **a**rtificial **i**ntelligence) guidelines.[13]

Patient Consent

The authors confirm that patient consent forms have been obtained for this article.

Funding Sources

This work was supported by the Canadian Institutes of Health Research (CIHR) [grant number 507249], which did not influence study design; the collection, analysis, and interpretation of data; the writing of the report; or the decision to submit the article for publication.

Disclosures

The authors have no conflicts of interest to disclose.

References

1. Vaduganathan M, Mensah GA, Turco JV, Fuster V, Roth GA. The global burden of cardiovascular diseases and risk: a compass for future health. J Am Coll Cardiol 2022;80:2361-71.
2. Statistics Canada. Table 13-10-0394-01: leading causes of death, total population, by age group. Available from: https://www150.statcan.gc.ca/t1/tbl1/en/tv.action?pid=1310039401. Accessed March 17, 2026.
3. Dai S, Bancej C, Bienek A, et al. Report summary tracking heart disease and stroke in Canada 2009. Health Promotion and Chronic Disease Prevention in Canada 2009;29:192-3. Available from: https://www.canada.ca/content/dam/phac-aspc/migration/phac-aspc/publicat/hpcdp-pspmc/29-4/pdf/cdic-29-4-06-eng.pdf. Accessed March 17, 2026.
4. Sofogianni A, Stalikas N, Antza C, Tziomalos K. Cardiovascular risk prediction models and scores in the era of personalized medicine. J Personal Med 2022;12:1180.
5. Jain R, Stone JA, Agarwal G, et al. Ligne directrice C-CHANGE pour l'harmonisation des lignes directrices nationales de prévention et de prise en charge des maladies cardiovasculaires en contexte de soins primaires au Canada: mise à jour 2022. CMAJ 2023;195:E21-42 [in French].
6. Goupil R, Tsuyuki RT, Santesso N, et al. Hypertension Canada guideline for the diagnosis and treatment of hypertension in adults in primary care. CMAJ 2025;197:E549-64.
7. Pearson GJ, Thanassoulis G, Anderson TJ, et al. 2021 Canadian Cardiovascular Society guidelines for the management of dyslipidemia for the prevention of cardiovascular disease in adults. Can J Cardiol 2021;37:1129-50.
8. D'Agostino RB, Vasan RS, Pencina MJ, et al. General cardiovascular risk profile for use in primary care. Circulation 2008;117:743-53.
9. Kariuki JK, Gona P, Leveille SG, et al. Cost-effectiveness of the non-laboratory based Framingham algorithm in primary prevention of cardiovascular disease: a simulated analysis of a cohort of African American adults. Prev Med 2018;111:415-22.
10. Kiran T, Thelen R, Szymanski K, et al. Public priorities for primary care in Canada: report on insights and actionable recommendations from 5 provincial reference panels. Can Fam Phys 2025;71:396-405.
11. Canadian Institute for Health Information. Better access to primary care key to improving health of Canadians. Available from: https://www.cihi.ca/en/taking-the-pulse-measuring-shared-priorities-for-canadian-health-care-2024/better-access-to-primary-care-key-to-improving-health-of-canadians. Accessed March 17, 2026.
12. Trudel X, Gilbert-Ouimet M, Milot A, et al. Cohort profile: The PROspective Québec (PROQ) Study on Work and Health. Int J Epidemiol 2018;47:693-i.
13. Collins GS, Moons KGM, Dhiman P, et al. TRIPOD+AI statement: updated guidance for reporting clinical prediction models that use regression or machine learning methods. BMJ 2024;385:e078378.
14. Tu K, Mitiku T, Lee DS, Guo H, Tu JV. Validation of physician billing and hospitalization data to identify patients with ischemic heart disease using data from the Electronic Medical Record Administrative data Linked Database (EMRALD). Can J Cardiol 2010;26:e225-8.
15. Tu K, Wang M, Young J, et al. Validity of administrative data for identifying patients who have had a stroke or transient ischemic attack using EMRALD as a reference standard. Can J Cardiol 2013;29:1388-94.
16. Schultz SE, Rothwell DM, Chen Z, Tu K. Identifying cases of congestive heart failure from administrative data: a validation study using primary care patient records. Chronic Dis Inj Can 2013;33:160-6 [Article in English, French].
17. Fan J, Arruda-Olson AM, Leibson CL, et al. Billing code algorithms to identify cases of peripheral artery disease from administrative data. J Am Med Inform Assoc 2013;20:e349-54.
18. Blais C, Jean S, Sirois C, et al. Quebec integrated chronic disease surveillance system (QICDSS), an innovative approach. Chron Dis Inj Can 2014;34:226-35.
19. Blais C, Rochette L. Trends in prevalence, incidence and mortality of diagnosed and silent coronary heart disease in Quebec. Health Promot Chronic Dis Prev Can 2015;35:184-93.

20. Frohlich ED. Recommendations for human blood pressure determination by sphygmomanometers. Circulation 1988;77:502A-14A.

21. White IR, Royston P, Wood AM. Multiple imputation using chained equations: issues and guidance for practice. Stat Med 2011;30:377-99.

22. Xia F, Ning J, Huang X. Empirical comparison of the Breslow estimator and the Kalbfleisch Prentice estimator for survival functions. J Biom Biostat 2018;9:392.

23. Prentice RL, Kalbfleisch JD, Peterson AV Jr, et al. The analysis of failure times in the presence of competing risks. Biometrics 1978;34:541-54.

24. Steyerberg EW. Clinical prediction models: a practical approach to development, validation, and updating, Statistics for Biology and Health. New York, NY: Springer, 2009, https://doi.org/10.1007/978-0-387-77244-8.

25. Riley RD, Debray TPA, Collins GS, et al. Minimum sample size for external validation of a clinical prediction model with a binary outcome. Stat Med 2021;40:4230-51.

26. McLernon DJ, Giardiello D, Van Calster B, et al. Assessing performance and clinical usefulness in prediction models with survival outcomes: practical guidance for Cox proportional hazards models. Ann Intern Med 2023;176:105-14.

27. Harrell FE Jr, Lee KL, Mark DB. Multivariable prognostic models: issues in developing models, evaluating assumptions and adequacy, and measuring and reducing errors. Stat Med 1996;15:361-87.

28. Hanley JA, McNeil BJ. The meaning and use of the area under a receiver operating characteristic (ROC) curve. Radiology 1982;143:29-36.

29. Gaziano TA, Young CR, Fitzmaurice G, Atwood S, Gaziano JM. Laboratory-based versus non-laboratory-based method for assessment of cardiovascular disease risk: the NHANES I Follow-up Study cohort. Lancet 2008;371:923-31.

30. Pandya A, Weinstein MC, Gaziano TA. A comparative assessment of non-laboratory-based versus commonly used laboratory-based cardiovascular disease risk scores in the NHANES III population. PLoS ONE 2011;6:e20416.

31. Albarqouni L, Doust JA, Magliano D, et al. External validation and comparison of four cardiovascular risk prediction models with data from the Australian Diabetes, Obesity and Lifestyle Study. Med J Aust 2019;210:161-7.

32. Al-Shamsi S, Govender RD, King J. External validation and clinical usefulness of three commonly used cardiovascular risk prediction scores in an Emirati population: a retrospective longitudinal cohort study. BMJ Open 2020;10:e040680.

33. Sun C, Xu F, Liu X, et al. Comparison of validation and application on various cardiovascular disease mortality risk prediction models in Chinese rural population. Sci Rep 2017;7:43227.

34. Alemu YM, Alemu SM, Bagheri N, Wangdi K, Chateau D. Discrimination and calibration performances of non-laboratory-based and laboratory-based cardiovascular risk predictions: a systematic review. Open Heart 2025;12:e003147.

35. Ko DT, Sivaswamy A, Sud M, et al. Calibration and discrimination of the Framingham Risk Score and the pooled cohort equations. CMAJ 2020;192:E442-9.

36. Sud M, Sivaswamy A, Chu A, et al. Population-based recalibration of the Framingham Risk Score and pooled cohort equations. J Am Coll Cardiol 2022;80:1330-42.

37. Parsons RE, Liu X, Collister JA, et al. Independent external validation of the QRISK3 cardiovascular disease risk prediction model using UK Biobank. Heart 2023;109:1690-7.

38. Chipayo-Gonzales D, Ramakrishna H, Nuñez-Gil IJ. Score2: a new updated algorithm to predict cardiovascular disease risk in Europe. J Cardiothorac Vasc Anesth 2022;36:18-21.

39. Khan SS, Matsushita K, Sang Y, et al. Development and validation of the American Heart Association's PREVENT equations. Circulation 2024;149:430-49.

40. Chen KYA, Eddeen AB, Bennett C, et al. Trends in cardiovascular risk factors in Canada: variation by migration and temporal factors, 2001-2018. CJC Open 2024;6:951-8.

41. Dai H, Younis A, Kong JD, Bragazzi NL, Wu J. Trends and regional variation in prevalence of cardiovascular risk factors and association with socioeconomic status in Canada, 2005-2016. JAMA Network Open 2021;4:e2121443.

42. Chiu M, Maclagan LC, Tu JV, Shah BR. Temporal trends in cardiovascular disease risk factors among White, South Asian, Chinese and Black groups in Ontario, Canada, 2001 to 2012: a population-based study. BMJ Open 2015;5:e007232.

43. Brunham LR, Lonn E, Mehta SR. Dyslipidemia and the current state of cardiovascular disease: epidemiology, risk factors, and effect of lipid lowering. Can J Cardiol 2024;40:S4-12.

44. Ko DT, Chu A, Austin PC, et al. Comparison of cardiovascular risk factors and outcomes among practicing physicians vs the general population in Ontario, Canada. JAMA Netw Open 2019;2:e1915983.

45. Liu R, So L, Mohan S, et al. Cardiovascular risk factors in ethnic populations within Canada: results from national cross-sectional surveys. Open Med 2010;4:e143-53.

46. Anand SS, Yusuf S, Vuksan V, et al. Differences in risk factors, atherosclerosis, and cardiovascular disease between ethnic groups in Canada: the Study of Health Assessment and Risk in Ethnic groups (SHARE). Lancet 2000;356:279-84.

47. Sebastian SA, Sethi Y, Padda I, Johal G. Ethnic disparities in the burden of cardiovascular disease among immigrants in Canada. Curr Probl Cardiol 2024;49:102059.

48. Maple-Brown LJ, Cunningham J, Zinman B, et al. Cardiovascular disease risk profile and microvascular complications of diabetes: comparison of Indigenous cohorts with diabetes in Australia and Canada. Cardiovasc Diabetol 2012;11:30.

49. Tanuseputro P, Manuel DG, Leung M, Nguyen K, Johansen H. Risk factors for cardiovascular disease in Canada. Can J Cardiol 2003;19:1249-59.

50. Wong SL, Shields M, Leatherdale S, Malaison E, Hammond D. Assessment of validity of self-reported smoking status. Health Rep 2012;23:47-53.

51. Schneider AL, Pankow JS, Heiss G, Selvin E. Validity and reliability of self-reported diabetes in the Atherosclerosis Risk in Communities Study. Am J Epidemiol 2012;176:738-43.

52. Nguyen AH, Hurwitz M, Sullivan SA, et al. Update on sex specific risk factors in cardiovascular disease. Front Cardiovasc Med 2024;11:1352675.

53. Arnett DK, Blumenthal RS, Albert MA, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation 2019;140:e596-646.

Supplementary Material

To access the supplementary material accompanying this article, visit *CJC Open* at https://www.cjcopen.ca/ and at https://doi.org/10.1016/j.cjco.2026.01.014