

Review

The Challenges and Benefits of Cardiovascular Risk Assessment in Clinical Practice

Steven A. Grover, MD, MPA,^{a-d} and Ilka Lowensteyn, PhD^{a,d}

^a McGill Cardiovascular Health Improvement Program, Montreal, Québec, Canada

^b Divisions of General Internal Medicine and Clinical Epidemiology, McGill University, Montreal, Québec, Canada

^c McGill University Health Centre, Montreal, Québec, Canada

^d Department of Medicine, McGill University, Montreal, Québec, Canada

ABSTRACT

Current expert guidelines for the treatment of hypertension or dyslipidemia recommend the use of cardiovascular risk assessment to identify high-risk individuals most likely to benefit from risk factor management. The potential uses of risk assessment include reassuring low-risk individuals, motivating high-risk individuals to modify their lifestyles or adhere to medical therapy, and track an individual's progress as risk factors come under better control. Despite the potential usefulness of cardiovascular risk assessment in clinical practice, the vast majority of patients have never had their cardiovascular risk assessed. This review describes the strengths and weaknesses of the currently available risk engines and suggests an approach, based on the currently available evidence, that can be used to maximize the clinical impact of risk assessment in daily clinical practice.

RÉSUMÉ

Les lignes directrices courantes des experts pour le traitement de l'hypertension ou de la dyslipidémie recommandent l'utilisation de l'évaluation du risque cardiovasculaire pour déterminer les individus à haut risque les plus susceptibles de bénéficier de la gestion des facteurs de risque. Les utilisations possibles de l'évaluation du risque incluent le réconfort des individus à risque faible, la motivation des individus à haut risque pour modifier leurs modes de vie ou adhérer à un traitement médical et le suivi des progrès d'un individu quand les facteurs de risque sont mieux contrôlés. En dépit de l'utilité potentielle de l'évaluation du risque cardiovasculaire dans la pratique clinique, la grande majorité des patients n'ont jamais eu leur risque cardiovasculaire évalué. Cette revue décrit les forces et les faiblesses des moteurs de calcul du risque généralement disponibles et suggère une approche, basée sur les preuves couramment disponibles, qui peut être utilisée pour maximiser l'impact clinique de l'évaluation du risque dans la pratique clinique quotidienne.

The potential benefits of modifying cardiovascular risk factors among individuals with known cardiovascular disease are no longer debated. Strong and consistent clinical trial data have clearly demonstrated that reducing blood pressure and/or modifying blood lipids will reduce the risk of secondary cardiovascular events, and increase longevity. These treatments also appeared to be highly cost-effective.¹

Primary prevention is more problematic as many individuals with elevated blood pressure or blood lipid abnormalities may still be at relatively low short-term risk of a cardiovascular event due to young age, gender, or the absence of other risk factors.² Accordingly, expert guidelines for the treatment of

hypertension or dyslipidemia recommend the use of global risk assessment to guide treatment decisions among individuals without diagnosed cardiovascular disease.³⁻⁶ Risk factor management can then be targeted to those individuals who will benefit the most given the high absolute risk of the cardiovascular event over the next 5 to 10 years.

Cardiovascular risk assessment in routine clinical practice holds many promises including: reassuring low-risk individuals, motivating high-risk individuals to modify their lifestyle or adhere to medical therapy, and track an individual's progress as risk factors come under control.⁷ Risk assessment can also be used to improve the allocation of finite healthcare dollars to ensure that one gets the biggest bang for the buck by reducing cardiovascular events among those individuals in whom the risk is most imminent.⁸

There are a number of challenges in asking busy health professionals to incorporate routine risk assessment into their daily clinical practice. First and foremost, risk assessment takes time whether it is performed using Web-based applications,

Received for publication February 27, 2011. Accepted April 13, 2011.

Corresponding author: Dr Steven A. Grover, Research Institute of the McGill University Health Centre, Royal Victoria Hospital, 687 Pine Avenue West, V-Building, Montreal, Québec H3A 1A1, Canada. Tel.: +1-514-934-1934 x44643; fax: +1-514-934-8293.

E-mail: steven.grover@mcgill.ca

See page 486 for disclosure information.

computer programs, hand-held risk calculators, or printed risk tables. Data entry is simply time-consuming even if only a few risk factors must be measured and inputted into the risk calculation. Additional time is required if one wishes to share these results with individual patients and their families. With a few exceptions, health professionals are rarely reimbursed for the additional time and effort this requires.

A second challenge is to demonstrate that the risk assessment tools do in fact accurately identify those at increased risk. Accordingly, despite the potential usefulness of cardiovascular risk assessment in primary care practice, the vast majority of patients seen in physicians' offices have never had their cardiovascular risk assessed. Nonetheless, pharmacotherapy for dyslipidemia and hypertension in Canada will cost several billion dollars this year while the majority of treated individuals will remain sedentary and overweight, and as many as half will not adhere to pharmacotherapy as prescribed. What then can be done to realize the full potential of risk assessment?

Choice of Risk Models

Over a dozen multivariable risk models have been developed. The ones that tend to be the most useful in clinical practice are those based on at least several thousand individuals who are representative of the general population. This will typically include men and women ranging in age from approximately 30 to 70 years. While sample size is important, the number of cardiovascular outcomes that occur during the follow-up period is perhaps the most important determinant of the resulting model's accuracy. As a general rule, most multivariable techniques require 10 to 20 outcomes for each additional independent risk factor entered into the model. Accordingly, given that most models include 5 to 10 risk factors, a minimum of 50 to 200 outcomes is required to build these models.

Model performance is usually evaluated based on external validity or the accuracy of the model when tested on a cohort of individuals different from those on which the model was developed. Model discrimination refers to the ability of the risk equations to discriminate between those who will and will not develop the outcome of interest. This is usually assessed using the area under receiver operating characteristic (ROC) curve or Harrell's C statistic where a value of 1 indicates a perfect test and 0.5 a test that performs no better than chance alone.⁹ Values between 0.70 and 0.85 are commonly observed for the most clinically useful risk equations.

Model calibration refers to how closely the predicted outcomes match those that are actually observed during the external validation.¹⁰ In most instances, the model will require recalibration if one wants to accurately predict the number of events that occur in a new population as the underlying event rate is rarely identical in different cohorts. Nonetheless, risk factors tend to have the same relative effect in different populations. Accordingly, a risk model developed in a northern European population where the absolute event rate is high can perform accurately in the low-risk southern European population after recalibration to adjust for the lower absolute event rate.

The net reclassification index (NRI) is a recently developed measure to compare how different models perform in classifying individuals into specific risk categories.¹¹ For instance, if

treatment guidelines require that individuals be classified into 10-year risk categories of less than 10%, 10% to 19%, and 20% or above, the net reclassification index can be used to see if the addition of a novel risk factor to the model will result in individuals who develop the outcome being classified in a higher risk category while those who do not develop the outcome are classified in a lower risk category.

In Canada, a number of models (summarized in Table 1) have been proposed for general use by primary health care providers³⁻⁶ including the following (presented in chronological order of the publication dates).

The Framingham model

Multivariable risk assessment equations to predict cardiovascular events were first developed by the Framingham Heart Study group. The Framingham equations remain the most widely used around the world and have evolved over more than 40 years since the first models were developed on risk factors such as age, gender, total cholesterol, systolic or diastolic blood pressure, and the presence of diabetes, smoking, and left ventricular hypertrophy. The original Framingham cohort and the Offspring cohort typically provide 3 to 5 thousand individuals (aged 30-74 years), followed for 5 to 15 years, with several hundred outcomes occurring during this period including hard cardiovascular events such as fatal and nonfatal myocardial infarction plus sudden death.^{12,17} Other hard end points include strokes (fatal and nonfatal) as well as soft end points such as angina pectoris, coronary insufficiency, and other complications of atherosclerosis including revascularization procedures, congestive heart failure, and transient ischemic attacks.¹⁸

The current Framingham equations include the total cholesterol/high-density lipoprotein (HDL) ratio, systolic blood pressure, age, gender, smoking status, and may also include the presence of diabetes.¹² A secondary model for estimating the risk of recurrent events among individuals who already have known cardiovascular disease has also been published.¹⁹ However, the resulting model does not appear to be particularly robust, includes only a few risk factors, and has not been extensively validated.

The Framingham equations have been shown to discriminate well between those who will and will not develop a cardiovascular event over 5 to 10 years.²⁰ The Framingham risk equations also perform well in most populations outside of the United States with appropriate recalibration based on the incidence of cardiovascular disease in the population of interest.²¹ The Framingham models focusing on hard cardiac end points, including those published in 1991 by Anderson et al. or 1998 by Wilson et al.,^{12,17} have been extensively tested and externally validated on other populations in the United States, Europe, Asia, and Canada.^{21,22} The more recent Framingham equations including soft cardiac end points, published by D'Agostino et al. in 2008, remains to be externally validated.¹⁸

The Framingham model has been validated in a Canadian cohort. It has been shown to accurately forecast cardiovascular deaths in the Canadian Lipid Research Clinic (LRC) Follow-up Cohort without additional calibration.²³

An online version is available (<http://hp2010.nhlbihin.net/atp/iii/calculator.asp?usertype=prof>).

Table 1. Models for risk assessment proposed for general use by primary healthcare providers in Canada

Risk model	Cohort	Risk factors	Outcomes	Validation	Canadian validation or calibration	Applications
Framingham—CHD 1998 ¹²	Men (<i>n</i> = 2489) and women (<i>n</i> = 2857) free of CVD from Framingham, MA, aged 30-74 y; follow-up 12 y	Gender-specific, age, SBP, smoking, diabetes, TC/HDL or LDL/HDL or TC, HDL	Total CHD or hard CHD (excluding angina)	External validation among American and European populations	Calibration using CHHS. Validated on the Canadian Lipid Research Clinic	Ten-year risk of total CHD ages 30-74 y. Available in charts, software, and Web sites
Cardiovascular Life Expectancy Model—CHD 1998 ¹³	Men and women (<i>n</i> = 3678) in the LRC follow-up cohort; 15% random sample from the USA and Canada aged 35-74 y; follow-up 12.2 y	Gender, age, SBP and DBP, smoking, diabetes, TC/HDL or LDL/HDL, and previous CVD; parental history of CVD optional	Fatal CHD, fatal stroke, life expectancy	External validation on published clinical trials and USA or Canadian Life Tables	Calibration using CHHS. Validated on the Canadian Lipid Research Clinic	Ten-year risk of fatal CHD or total CHD ages 35-79 y, CVD risk, adjusted life expectancy. Available on Web site
SCORE—CVD 2003 ¹⁴	Men (<i>n</i> = 117098) and women (<i>n</i> = 88080) without previous MI from 12 European countries, aged 19-80 y; follow-up 10 y	Gender, age, SBP, smoking, TC; HDL is optional	Fatal CVD	External validation using all-cause mortality among patients in an American cardiac rehabilitation setting		Ten-year risk of fatal CVD for those without diabetes, ages 40-65 y. Available in charts, software, and Web sites
Reynolds Risk Score (women)—CVD 2007 ¹⁵	American women (<i>n</i> = 24,558) free of CVD and cancer, aged ≥ 45 y; follow-up 10.2 y	Gender-specific, age, SBP, smoking, TC, HDL, parental history of MI prior to 60 y, and hsCRP	MI, stroke, coronary revascularization, fatal CVD			Ten-year risk of fatal and nonfatal CVD for those without diabetes, ages 45-80 y. Available on Web site
Reynolds Risk Score (men)—CVD 2008 ¹⁶	American men (<i>n</i> = 10,724) free of CVD, cancer, and diabetes, aged 50-80 y; follow-up 10.8 y	Gender-specific, age, SBP, smoking, TC, HDL, parental history of MI prior to 60 y of age, hsCRP	MI, stroke, coronary revascularization, fatal CVD			Ten-year risk of fatal and nonfatal CVD for those without diabetes, ages 45-80 y. Available on Web site

CHD, coronary heart disease; CHHS, Canadian Heart Health Survey; CVD, cardiovascular disease; DBP, diastolic blood pressure; HDL, high-density lipoprotein; hsCRP, high sensitivity c-reactive protein; LDL, low-density lipoprotein; LRC, Lipid Research Clinic; MA, Massachusetts; MI, myocardial infarction; SBP, systolic blood pressure; SCORE, Systematic COronary Risk Evaluation; TC, total cholesterol.

The Cardiovascular Life Expectancy model

The Cardiovascular Life Expectancy model is a Markov model developed to calculate short-term risk as well as long-term life expectancy. Using the 15% random sample from the LRC Follow-up Cohort (aged 30-79 years) from the United States and Canada, the model can be used to calculate the risk of coronary death and cerebrovascular death as a function of age, gender, the total cholesterol/HDL ratio, mean blood pressure, smoking status, and the presence of diabetes.¹³ The model has been externally validated and shown to reasonably estimate the results of published clinical trials over 5 to 10 years of follow-up. It has also been validated in selected populations including individuals with or without previously diagnosed cardiovascular disease (CVD) or diabetes.^{24,25} The life expectancy estimates have also been shown to closely approximate published Canadian and American life tables.^{2,26}

The 10-year results forecasted with the cardiovascular life expectancy model tend to be very similar to those using the Framingham equations with very small differences between the two.²³ When the 10-year Framingham Risk has been calculated, the only advantage associated with using this Markov model is the opportunity to estimate the long-term effect of risk factors over the entire life expectancy. One can also use the model to estimate an individual's life expectancy before and after treating 1 or more risk factors.

An online version is available at www.myhealthcheckup.com, www.monbilansante.com, and www.chiprehab.com. The 2010 version allows the user to choose "hard coronary events" or also include the "soft coronary events" published by the Framingham group and recently recommended by the 2009 Canadian Lipid Guidelines.⁴ An adjustment is also now available for the presence of a family history of premature coronary disease.

The individual's "Cardiovascular Age" is also calculated as their age minus the difference between their estimated remaining life expectancy (adjusted for their coronary and stroke risk) and the average remaining life expectancy of Canadians of the same age and sex. For instance, a 50-year-old with a life expectancy of 25 more years (vs 30 years for the average Canadian) would be assigned a Cardiovascular Age of 55.

The Cardiovascular Life Expectancy model has been validated in a Canadian cohort. A model based only on American LRC data, has been shown to accurately forecast cardiovascular deaths in the Canadian LRC cohort without additional calibration.²³

The Systematic COronary Risk Evaluation (SCORE) model

The SCORE model was developed on a pooled data set of 12 European population cohorts (aged 40-65 years) including both low-risk populations in southern Europe and high-risk populations in northern Europe.¹⁴ Accordingly, the model is easily calibrated for these 12 European countries. Ease of calibration was 1 of the primary objectives of the SCORE model which focuses only on the fatal cardiovascular events (coronary death and noncoronary atherosclerotic death). This provides a reasonably simple platform to recalibrate for different countries where cardiovascular mortality rates are readily available while nonfatal event rates are not. On the other hand, fatal cardiovascular events does not match up with the risk categories cur-

rently recommended in most Canadian and American guidelines which focus on fatal and nonfatal "hard outcomes" (fatal and nonfatal myocardial infarction [MI], sudden death) or, in the case of the 2009 Canadian Lipid guidelines may also include "soft outcomes" such as angina, coronary insufficiency, transient ischemic attack, congestive heart failure, and revascularization procedures.

The model was developed primarily for individuals without diagnosed cardiovascular disease or diabetes. The risk equations are based on age, gender, systolic blood pressure, smoking status, and total cholesterol levels alone or the total cholesterol/HDL ratio.

The SCORE model has been validated in a number of populations. It has not yet been validated in a Canadian cohort.²³

An online version is available (<http://www.scorecanada.ca>).

The Reynolds Risk Score

The Reynolds Risk Score was developed using data from 2 different American populations: the Women's Health Study (women aged 45 and older who were free of CVD and cancer) and the Physicians Health Study II (male physicians aged 50-80 years who were free of CVD, diabetes, and cancer).^{15,16} The Reynolds model is similar to the Framingham model but includes 2 additional risk factors: a family history of premature coronary disease and high sensitivity c-reactive protein (hsCRP).

There are good data demonstrating that a family history of premature coronary disease will increase the risk of a CVD event by 1.5- to 2-fold among individuals younger than the age of 60 without diagnosed CVD or diabetes.²⁷ The major debate surrounding the Reynolds Risk Score is whether the addition of hsCRP is useful once the traditional Framingham risk factors and family history are known. Unlike family history, hsCRP requires additional laboratory testing and given the low specificity of hsCRP for inflammation in the coronary arteries, false positives may occur due to inflammation in other parts of the body. The 2 primary reports that described the model were only able to demonstrate a slight improvement in the model's discriminating ability over the traditional Framingham risk factor for men (C statistic increased from 0.699 to 0.708; $P < 0.001$) while absolutely no improvement in the C statistic was noted in the female cohort. There are concerns that of the 2 risk factors added in the Reynolds Score, family history is the more important and hsCRP may add little additional information. This was confirmed by the Framingham investigators when they added hsCRP measurements to the Framingham model and could not demonstrate any significant improvement in the C statistic.²⁸

A number of concerns have also been expressed regarding the Reynold's Risk Score. For women these include the fact that the score was developed based on a cohort of health professionals participating in a clinical trial rather than a sample representative of the general population.²⁹ Risk factors such as blood pressure, were not measured but self-reported and the prevalence of cigarette smoking was low thereby underestimating the contribution of these traditional risk factors. Among men, similar concerns include the questionable generalizability of a model developed based on a physician cohort, median age 63 years, where only 3.2% were smokers and the median systolic blood pressure was low at 128 mm Hg.

The Reynolds Risk Score has not yet been extensively validated in other populations. It has also not been validated in a Canadian cohort. It is available online (<http://www.reynoldsriskscore.org/>).

How Should Cardiovascular Risk Assessment be Used in Routine Clinical Practice?

Multifactorial risk assessment has been proven to more accurately identify those at increased risk of a cardiovascular event compared with treatment guidelines focusing only on blood lipid levels or simply counting the number of risk factors present in a specific patient.

Treatment guidelines commonly recommend classifying individuals into 10-year risk categories including low-risk (< 10%), medium-risk (10%-20%), and high-risk (> 20%).⁴ Thresholds for initiating treatment (such as low-density lipoprotein (LDL) cholesterol levels above 3.5 mmol/L) and therapeutic targets (such as reduce LDL to < 2 mmol/L) are then defined based on one's risk category. However, there are increasing concerns that this approach, based on short-term 10-year risk, will result in the undertreatment of younger individuals who have significant elevations in 1 or more risk factors but whose absolute risk level remains low given their age.^{7,21} This issue is particularly apparent among women, younger than the age of 60, whose lifetime risk of developing cardiovascular disease is substantial even though an event is unlikely to occur over the next 10 years. On the other hand, focusing only on short-term risk may also result in the overtreatment of elderly individuals whose absolute risk is high in large part due to their advanced age while the long-term benefits of therapy may be relatively modest given their remaining life expectancy and the presence of other comorbidities.

For instance, if one calculates the 10-year Framingham Risk (hard outcomes only) of a 42-year-old man without a family history of premature coronary disease, total cholesterol level of 6.5 mmol/L, LDL level of 4.5 mmol/L, HDL level of 1.1 mmol/L, borderline hypertension of 138/88 mm Hg, and no other risk factors, his risk is only 4.2% over the next 10 years. Current guidelines would not recommend treating his blood lipids as he is categorized as low risk. Nonetheless his life expectancy is reduced by 0.7 years on average by virtue of his multiple borderline risk factors and he can be told that he has the Cardiovascular Age of someone 42.7 years old (www.myhealthcheckup.com). Lipid therapy could reduce his total cholesterol 25% and LDL 35%, while his HDL could be raised 20% resulting in a 2.6% absolute drop in his risk to 1.6%. When the potential lifetime benefits are calculated, his "Cardiovascular Age" would drop from 42.7 years to 40.9 years or an estimated increased life expectancy of 1.8 years. The potential benefits of treatment might seem quite attractive to many such patients.

On the other hand, consider a 75-year-old man with exactly the same risk factors, who would have a 10-year Framingham Risk of 21%, and current guidelines would recommend treatment as he is categorized as high-risk even though his absolute risk is below average for Canadian men of his age. This risk would drop to 17.5% following the same response to lipid therapy as the previously mentioned younger man. While the 3.5% drop in 10-year risk is greater than the absolute risk reduction calculated for the 42-year-old, his "Cardiovascular

Age" would be reduced less (0.8 years) reflecting the more limited time horizon over which the benefits associated with treatment could be realized.

A second problem with low, medium, and high risk categories is that they are completely arbitrary. There is no scientific evidence to support treating someone with a 20% risk differently from someone with a 10% risk. To treat patients differently on the basis of risk levels of 11% vs 9% makes even less sense particularly when small changes in blood lipids or blood pressure from 1 day to the next can move one's risk profile a couple of percentage points up or down.

If one accepts that arbitrary treatment thresholds based on absolute risk categories make little sense, there is little reason to try to make minor improvements in risk prognostication. Existing Framingham models have been shown to have very good discriminating ability usually in the range of 75% to 85%. If perfect discrimination is 100% there is not that much room for improvement. Accordingly it should not be surprising that the addition of hsCRP improves risk discrimination often by no more than 1% compared with traditional Framingham risk factors.

Model calibration also becomes less of a concern when categorical risk levels no longer drive treatment decisions as it really does not matter if one's risk is 11% or 9% but rather whether one's risk is elevated relative to a clinically useful standard. What should the standard be for defining an elevated risk? Should all elderly individuals be considered to be at increased risk just because they are older? Are all men at increased risk due to their gender? Are all young women under the age of 40 at low risk no matter how many risk factors they have? Two possibilities have been proposed. One is to compare an individual's increased risk relative to individuals with no risk factors or an ideal risk profile such as nonsmokers with blood pressure of 120/80 and a total cholesterol/HDL ratio of 4. Alternatively one can define increased relative risk as a risk level above the average risk of individuals of the same age and sex in one's community. Accordingly one's risk is only compared with the average risk of one's peers. In either situation it does not matter if the risk model overestimates or underestimates risk in a specific population as both an individual patient's risk profile and the frame of reference will be inflated or deflated similarly to the ideal risk profile or the average risk of one's peers.

If one focuses on relative risk rather than absolute risk the debate surrounding which model to use and how much to calibrate that model becomes irrelevant. One can choose any model with a high discriminating ability and apply it to Canadian population data to define the ideal or average risk for each age and sex group. The only question that then remains is whether a specific patient's risk is elevated compared to 1 of these norms and how much that risk can be reduced by treating modifiable risk factors. Clinicians can use this information to help inform their clinical decisions while evaluating the patient's preferences rather than make arbitrary decisions based only on the absolute risk level.

While helping health professionals make more informed decisions, risk profiles can also provide useful information to patients. An initial assessment can be used to engage the patient in modifying their lifestyle including smoking cessation, weight loss, and increased physical activity. If this is unsuccessful, a risk profile can also be used to help a patient understand

the need for pharmacotherapy. In addition, follow-up profiles can be used to quantify the potential benefits of adhering to both lifestyle changes and pharmacotherapy.

Sharing the results of risk assessments with patients can provide a solid foundation for patient-centred health care. However, framing the results to make them easily understandable to most patients remains an important consideration. A recent systematic review of clinical trials to communicate cardiovascular risk to patients identified a number of characteristics associated with positively engaging patients to modify their behaviour.³⁰ Useful approaches included presenting patients with their cardiovascular risk in percentages or frequencies rather than risk categories (low, moderate, high), using graphics and short time frames, and providing the patient with a Cardiovascular Age equivalent. Cardiovascular Age, Vascular Age, or Heart Age are similar concepts where an individual's risk profile is compared with that of their age- and gender-matched peers.^{31,32} The basic idea is if you are at high risk compared with your peers, then your vascular system is aging faster than you. On the other hand, if you modify 1 or more risk factors the associated reduction in risk also results in a younger vascular system. Some Cardiovascular Age metrics also calculate the individual's life expectancy before and after modifying 1 or more risk factors. This may be useful for individuals whose short-term risk is low but in whom the long-term calculated benefits of risk factor modification may substantially reduce their Cardiovascular Age or increase their life expectancy. This is particularly relevant for younger men, and women in most age groups.

Research using focus groups also suggests that providing individuals with statistical probabilities may be insufficient for motivating change. However, understanding one's cardiovascular risk-adjusted age was shown to be clear, memorable, and relevant.³³ In a primary care physician setting, knowing one's Cardiovascular Age has been shown to increase the odds of reaching lipid targets by 26% overall and up to 69% among those whose Cardiovascular Age was at least 7 years greater than their chronological age.³⁴ Also, using the risk profile helped physicians to look beyond treating blood lipids and also initiate or modify therapy for poorly controlled hypertension.³⁵ A similar study in community pharmacies demonstrated that pharmacists could use a personalized risk profile including their Cardiovascular Age to reduce patients' decisional conflict surrounding lifestyle changes and pharmacotherapy to reduce their cardiovascular risk factors.³⁶ Another randomized clinical trial also demonstrated that a "Heart Age" score was more emotionally impactful than presenting an estimated CVD risk score among younger individuals at increased risk of CVD.³⁷

In conclusion, cardiovascular risk assessment has been shown to improve our ability to identify those individuals most likely to suffer a cardiovascular event over the next 10 years and treatment guidelines have evolved to incorporate this information into daily clinical decision-making. The choice of risk model is of little consequence as long as the discriminating ability is good with a C statistic of at least 75% or more. Categorical risk levels as currently defined by low-, medium-, and high-risk are arbitrary and oversimplify complicated decisions that should be based on the patient's relative risk, age, the presence of other comorbidities, remaining life expectancy, and individual patient preferences. For these reasons, we recommend the use of risk models that include an estimate of one's

Cardiovascular Age, Vascular Age, or Heart Age. Not only does the concept of comparing one's chronological age to the age of one's vascular system appear to resonate with and engage patients, but there is now empirical evidence that sharing this information with patients improves their adherence with treatment as well as clinical decision-making by health professionals.³¹⁻³⁷

If one wants to follow the recommendations of the current guidelines and calculate short-term risk, we recommend the Framingham Risk equations as there is no evidence that any of the other risk equations are superior to these equations. Moreover, they have been extensively evaluated for several decades and appear to be accurate among Canadians even without additional calibration.

We believe that the real value of risk assessment lies with communicating to the patient their long-term risk of disease compared with others of the same age and sex. The evidence supports the use of risk models to provide the patient with an estimate of their Cardiovascular Age, Vascular Age, or Heart Age. This may be the single most important component of risk assessment that can actually improve the clinical management of hypertension or dyslipidemia in the primary prevention of cardiovascular disease.^{34,36}

Disclosures

Dr Steven Grover and Dr Ilka Lowensteyn have received research grants from Pfizer, Sanofi Aventis, and AstraZeneca. Dr Grover has received speaker honouraria from Pfizer, Sanofi Aventis, and Merck. Dr Grover has either been a consultant or participated on an advisory board for AstraZeneca, Sanofi Aventis, Pfizer, and Merck.

References

1. Grover S, Coupal L, Lowensteyn I. Preventing cardiovascular disease among Canadians: is the treatment of hypertension or dyslipidemia cost-effective? *Can J Cardiol* 2008;24:891-8.
2. Grover SA, Coupal L, Kaouache M, Lowensteyn I. Preventing cardiovascular disease among Canadians: What are the potential benefits of treating hypertension or dyslipidemia. *Can J Cardiol*. 2007;23:467-73.
3. Canadian Diabetes Association Clinical Practice Guidelines Expert Committee. Canadian Diabetes Association 2008 Clinical Practice Guidelines for the prevention and management of diabetes in Canada. *Can J Diabetes* 2008;32(suppl 1):S1-S201.
4. Genest J, McPherson R, Frohlich J, et al. 2009 Canadian Cardiovascular Society/Canadian guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular disease in the adult - 2009 recommendations. *Can J Cardiol* 2009;25:567-79.
5. Padwal RS, Hemmelgarn BR, Khan NA, et al. The 2009 Canadian Hypertension Education Program recommendations for the management of hypertension: Part 1—blood pressure measurement, diagnosis and assessment of risk. *Can J Cardiol* 2009;25:279-86.
6. Khan NA, Hemmelgarn B, Herman RJ, et al. The 2009 Canadian Hypertension Education Program recommendations for the management of hypertension: Part 2—therapy. *Can J Cardiol* 2009;25:287-98.
7. Grover SA. Gambling with cardiovascular disease: picking the winners and losers. *Lancet* 1999;353:254-5.
8. Grover SA, Coupal L, Paquet S, Zowall H. Cost-effectiveness of 3-hydroxy-3-methylglutaryl-coenzyme A reductase inhibitors in the secondary

- prevention of cardiovascular disease: forecasting the incremental benefits of preventing coronary and cerebrovascular events. *Arch Intern Med* 1999;159:593-600.
9. Hanley JA, McNeil BJ. A method of comparing the areas under receiver operating characteristic curves derived from the same cases. *Radiology* 1983;148:839-43.
 10. Hosmer DW, Lemeshow S. Goodness of Fit Tests for the Multiple Logistic Regression-Model. *Communications in Statistics Part A-Theory and Methods*. 1980;9:1043-69.
 11. Pencina MJ, D'Agostino RB Sr, D'Agostino RB Jr, Vasan RS. Evaluating the added predictive ability of a new marker: from area under the ROC curve to reclassification and beyond. *Stat Med* 2008;27:157-72.
 12. Wilson PW, D'Agostino RB, Levy D, et al. Prediction of coronary heart disease using risk factor categories. *Circulation* 1998;97:1837-47.
 13. Grover SA, Paquet S, Levinton C, Coupal L, Zowall H. Estimating the benefits of modifying risk factors of cardiovascular disease. A comparison of primary vs secondary prevention. *Arch Int Med* 1998;158:655-62.
 14. Conroy RM, Pyorala K, Fitzgerald AP, et al. Estimation of ten-year risk of fatal cardiovascular disease in Europe: the SCORE project. *Eur Heart J* 2003;24:987-1003.
 15. Ridker PM, Buring JE, Rifai N, Cook NR. Development and validation of improved algorithms for the assessment of global cardiovascular risk in women: the Reynolds Risk Score. *JAMA* 2007;297:611-9.
 16. Ridker PM, Paynter NP, Rifai N, Gaziano JM, Cook NR. C-reactive protein and parental history improve global cardiovascular risk prediction: the Reynolds Risk Score for men. *Circulation* 2008;118:2243-51.
 17. Anderson KM, Odell PM, Wilson PW, Kannel WB. Cardiovascular disease risk profiles. *Am Heart J* 1991;121:293-8.
 18. D'Agostino RB Sr, Vasan RS, Pencina MJ, et al. General cardiovascular risk profile for use in primary care: the Framingham Heart Study. *Circulation* 2008;117:743-53.
 19. D'Agostino RB, Russell MW, Huse DM, et al. Primary and subsequent coronary risk appraisal: New results from The Framingham Study. *Am Heart J* 2000;139:272-81.
 20. D'Agostino RB, Grundy SM, Sullivan LM, Wilson P, for the CHD Risk Prediction Group. Validation of the Framingham Coronary Heart Disease Prediction Scores. Results of a multiple ethnic groups investigation. *JAMA* 2001;286:180-7.
 21. Cooney MT, Dudina AL, Graham IM. Value and limitations of existing scores for the assessment of cardiovascular risk: a review for clinicians. *J Am Coll Cardiol* 2009;54:1209-27.
 22. Brindle P, Beswick A, Fahey T, Ebrahim S. Accuracy and impact of risk assessment in the primary prevention of cardiovascular disease: a systematic review. *Heart* 2006;92:1752-9.
 23. Grover SA, Hemmelgarn B, Joseph L, Milot A, Tremblay G. The role of global risk assessment in hypertension therapy. *Can J Cardiol* 2006;22:606-13.
 24. Grover SA, Coupal L, Zowall H, Dorais M. Cost-effectiveness of treating hyperlipidemia in the presence of diabetes: who should be treated? *Circulation* 2000;102:722-7.
 25. Grover SA, Coupal L, Zowall H, et al. How cost-effective is the treatment of dyslipidemia in patients with diabetes but without cardiovascular disease? *Diabetes Care* 2001;24:45-50.
 26. Grover SA, Coupal L, Gilmore N, Mukherjee J. Impact of dyslipidemia associated with highly active antiretroviral therapy (HAART) on cardiovascular risk and life expectancy. *Am J Cardiol* 2005;95:586-91.
 27. Lloyd-Jones DM, Nam BH, D'Agostino RB Sr, et al. Parental cardiovascular disease as a risk factor for cardiovascular disease in middle-aged adults: a prospective study of parents and offspring. *JAMA* 2004;291:2204-11.
 28. Wilson PW, Nam BH, Pencina M, et al. C-reactive protein and risk of cardiovascular disease in men and women from the Framingham Heart Study. *Arch Intern Med* 2005;165:2473-8.
 29. Wang TJ, Kathiresan S, Lloyd-Jones DM. Algorithms for assessing cardiovascular risk in women. *JAMA* 2007;298:176-8.
 30. Waldron CA, van der Weijden T, Ludt S, Gallacher J, Elwyn G. What are effective strategies to communicate cardiovascular risk information to patients? A systematic review. *Patient Educ Couns* 2011;82:169-81.
 31. Grover SA, Lowensteyn I, Esrey K, et al. How accurately do Canadian physicians assess the coronary risk of their patients? The preliminary results of the Coronary Health Assessment Study (CHAS). *BMJ* 1995;310:975-8.
 32. Frankel DS, Meigs JB, Massaro JM, et al. Von Willebrand factor, type 2 diabetes mellitus, and risk of cardiovascular disease: the Framingham Offspring study. *Circulation* 2008;118:2533-9.
 33. Goldman RE, Parker DR, Eaton CB, et al. Patients' perceptions of cholesterol, cardiovascular disease risk, and risk communication strategies. *Ann Fam Med* 2006;4:205-12.
 34. Grover SA, Lowensteyn I, Joseph L, et al. Patient knowledge of coronary risk profile improves the effectiveness of dyslipidemia therapy: the CHECK-UP study: a randomized controlled trial. *Arch Intern Med* 2007;167:2296-303.
 35. Grover SA, Lowensteyn I, Joseph L, et al. Discussing coronary risk with patients to improve blood pressure treatment: secondary results from the CHECK-UP study. *J Gen Intern Med* 2009;24:33-9.
 36. Lalonde L, O'Connor AM, Duguay P, et al. Evaluation of a decision aid and a personal risk profile in community pharmacy for patients considering options to improve cardiovascular health: The OPTIONS pilot study. *Int J Pharm Pract* 2006;14:51-62.
 37. Soureti A, Hurling R, Murray P, van MW, Cobain M. Evaluation of a cardiovascular disease risk assessment tool for the promotion of healthier lifestyles. *Eur J Cardiovasc Prev Rehabil* 2010;17:519-23.