Global Cardiovascular Disease Risk Scores:
A Systematic Review of the Literature
and
Study of the Effect of Risk Scores on Physician Adherence to
Guidelines for the Primary Prevention of Cardiovascular
Disease

Eric Michael Crespo, M.D.

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Stacey Sheridan, M.D., MPH
Advisor

Russell Harris, M.D., MPH
Second Reader
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Introduction

Several major guidelines and organizations now advocate routine assessment of cardiovascular risk using Framingham-derived coronary heart disease (CHD) risk scores.\(^1\)\(^-\)\(^3\) Yet there is relatively little data on whether the routine use of such assessments translates into improved clinical outcomes. In particular, little is known about how knowledge of a patient’s calculated 10-year CHD risk affects a physician’s perception of the patient’s risk and whether that in turn translates into improved adherence to published guidelines for the primary prevention of CHD.

This paper is organized into three major sections: First, a thorough review of the theoretical benefits of global CHD risk calculation is presented. Following this is a systematic review of the literature supporting the efficacy of incorporating Framingham-derived global risk scores into routine clinical practice. Lastly we present the methods and preliminary results of a study designed to determine whether physicians make better CHD prevention decisions when they interpret a patient’s risk factor information in the context of a calculated 10-year risk of CHD than when they use the risk factor information alone.
Background:

Cardiovascular Disease Prevention and the Rationale for Global CHD Risk Calculation
The Burden of Suffering Associated with Cardiovascular Disease in the U.S.

Almost one million Americans die of cardiovascular disease (CVD) each year, which is more than cancer, chronic obstructive lung disease, accidents, diabetes, and influenza/pneumonia combined. The major forms of CVD, heart disease and stroke, account for one of every five and one of every 15 deaths respectively, and together they account for more than one of every three deaths in the U.S each year. This is true regardless of gender or ethnic group. Despite a 26% decline in the death rate from CVD over the last decade, the actual number of CVD deaths has declined by only approximately 9.4% because of the increasing prevalence of CVD and its risk factors in our society.\textsuperscript{4,5}

Sixty-four million Americans currently have some form of CVD, and each year they are joined by 1.9 million more (700,000 new myocardial infarctions [MI], 500,000 recurrent MIs, 500,000 new strokes, and 200,000 recurrent strokes)\textsuperscript{4}. These numbers are expected to increase over the next decade given the growing number of older Americans and the high prevalence of CVD risk factors. Although the incidence and prevalence of CVD clearly increase with age, this is not simply a problem of the elderly. In fact, approximately 16.8\% of all heart disease deaths occur in people under 65 years old.\textsuperscript{4,6}

Along with the staggering human toll of CVD, the economic impact is significant. CVD is the #1 disease category listed on hospital discharges (6.2 million/year),
and it accounts for 71 million physician office visits, 5.6 million outpatient department visits, and 4.2 million emergency department visits each year.\textsuperscript{4, 7, 8} In 2004, the direct and indirect costs due to CVD are estimated at $368.4 billion.\textsuperscript{4}

**Opportunities for Prevention**

Cardiovascular disease and its risk factors are among the most common conditions encountered by primary care clinicians,\textsuperscript{7} and an increasing number of people now have multiple risk factors\textsuperscript{4, 9, 10} Table 1 summarizes the prevalence of the various CVD risk factors and puts these numbers into the perspective of the average clinician’s patient panel. The numbers vary depending on the location and nature of practices, but the overall message is clear: every clinician sees many patients who either already have CVD or are at risk for developing it.

**Table 1: Prevalence of CVD and Its Risk Factors Among U.S. Adults\textsuperscript{\textdagger}**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Prevalence in U.S. (%)</th>
<th># of Patients in an average Primary Care Practice Panel With Condition (Based on 1490 patients/PCP)***</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiovascular Disease\textsuperscript{\textdagger}</strong></td>
<td>22.6</td>
<td>337</td>
</tr>
<tr>
<td>Coronary Heart Disease</td>
<td>6.4</td>
<td>95</td>
</tr>
<tr>
<td>Stroke</td>
<td>2.0</td>
<td>30</td>
</tr>
<tr>
<td><strong>Risk Factors</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>One or more risk factors\textsuperscript{*}</td>
<td>64.0</td>
<td>954</td>
</tr>
<tr>
<td>Hypertension (≥140/90)</td>
<td>32.8</td>
<td>489</td>
</tr>
<tr>
<td>Pre-Hypertension (120-139/80-89)**</td>
<td>22.0</td>
<td>328</td>
</tr>
<tr>
<td>LDL-C ≥130</td>
<td>45.8</td>
<td>682</td>
</tr>
<tr>
<td>HDL-C 40</td>
<td>26.4</td>
<td>393</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>8.4</td>
<td>125</td>
</tr>
<tr>
<td>Impaired Glucose Tolerance</td>
<td>7.1</td>
<td>106</td>
</tr>
<tr>
<td>Metabolic Syndrome</td>
<td>23.7</td>
<td>353</td>
</tr>
<tr>
<td>Overweight/Obese (BMI ≥ 25)</td>
<td>64.5</td>
<td>961</td>
</tr>
<tr>
<td>Obese (BMI ≥ 30)</td>
<td>30.5</td>
<td>454</td>
</tr>
<tr>
<td>Physical inactivity\textsuperscript{\textdagger}</td>
<td>54.6</td>
<td>814</td>
</tr>
<tr>
<td>Tobacco</td>
<td>22.8</td>
<td>340</td>
</tr>
</tbody>
</table>

*White and African-American men and women over age 20, except for tobacco use and physical inactivity statistics, which refer to adults ≥18 years.*

Cardiovascular disease is defined as ICD/10 codes I00-99 and Q20-28. Coronary Heart Disease is defined as ICD/10 codes I20-25. Stroke is defined as ICD/10 codes I60-69.

*One or more of: hypertension, elevated cholesterol, diabetes, tobacco use, obesity.*

**JNC 7**

***As of 2000, there were 67.1 Internists and Family Practitioners per 100,000 U.S. population, this translates to 1490 patients per primary care physician. The number of patients was calculated by multiplying the prevalence of the risk factor by 1490.*

Physical inactivity is defined as not meeting one of the following standards: activity of moderate intensity for ≥30 minutes per day on ≥5 days per week or vigorous intensity for ≥20 minutes per day on ≥3 days per week.

Although sobering, these estimates point out the substantial opportunity that exists for preventive interventions to make a difference in the lives of a large number of patients. Much of this work has to be done at a population level, but through consistent application of evidenced-based preventive interventions, clinicians are in a position to help reduce the number of individuals in their communities who will have a new or recurrent CVD event over both the short- and long-term. Achieving this, however, requires not only a familiarity with guidelines, but also an understanding of overarching framework of CVD prevention.
The Continuum of Cardiovascular Disease and Prevention

Prevention can be conceptualized as a continuum with components that correspond to the underlying burden of atherosclerotic disease, and thus to the risk of CVD events in the target patient population (Figure 1).

Primordial prevention targets populations without any underlying atherosclerosis and can be thought of as the prevention of CVD by preventing the development of CVD risk factors in the first place. Interventions in this category include counseling for healthy diet, physical activity, obesity, and smoking prevention. Although individual counseling of patients is desirable, these interventions are most effective when part of a more intensive, multidisciplinary program with a strong community-based component. The goal of these activities is to decrease the prevalence of risk factors across all of society, which in turn translates into a reduction in the number of people developing CVD.

Primary prevention refers to efforts to modify existing risk factors with the aim of delaying or preventing new-onset CVD. These efforts target a heterogeneous group of asymptomatic people without clinical cardiovascular disease who are at varying levels of risk depending on the number, combination, and magnitude of their risk factors. While some patients are at low risk, others have risk that equals that of patients who already have CVD.
Over time, and with the accumulation of risk factors, patients will develop varying degrees of subclinical atherosclerosis. These patients are at considerable risk for experiencing a CVD event (MI, stroke, TIA, etc...) in the short term. In these patients, the goal is to retard the progression of subclinical disease and prevent a first CVD event. Prevention of a first CVD event in patients who have vascular disease but who have not yet suffered a clinical event is termed secondary prevention.

Unlike primordial, primary, and secondary prevention, all of which aim to prevent a first CVD event, the goal of tertiary prevention is to prevent recurrent events in people who have already had a clinical CVD event. These people are at very high risk for another event, and they require aggressive risk reduction strategies.

The framework provides insight into the manner in which the different CVD prevention guidelines fit together. It also emphasizes that each level of prevention is a stepping stone into the next, such that the preventive activities that are begun in each stage are added on to the ones begun previously. The manner in which the different CVD prevention guidelines fit into this framework is shown in Appendix 1.
The Prevention Gap

Despite the availability of numerous guidelines and abundant evidence regarding the efficacy of interventions to prevent CVD, the majority of people with CVD risk factors do not have them under adequate control. The facts are startling: 69% of hypertensive patients do not have their blood pressure under control, and almost half are not even on any treatment. Additionally, only 18% of patients with dyslipidemia have their cholesterol treated to goal levels. The reasons for this are complex and involve the interplay of multiple patient-, physician-, and health care system-related factors.

For physicians, implementing evidenced-based guidelines into practice is challenging. In part, this is due to the sheer number of existing guidelines and the time constraints of everyday practice. Other factors include competing patient demands, issues with patient adherence, misunderstanding about the proper focus of preventive efforts (on the part of both the patient and physician), lack of consistency among different guidelines, and confusion caused by rapidly advancing science. The latter is especially true in the area of CVD prevention where new biomarkers and diagnostic tests are announced frequently.

An additional factor that may play a role in the poor implementation of interventions aimed at preventing CVD is that physicians have difficulty accurately estimating risk. Risk assessment is important because it not only helps identify candidates for preventive interventions, but also because it guides the
intensity and types of interventions that are chosen. Unfortunately, estimating risk is not as straightforward as it may seem, and several studies have shown that physicians vary significantly in their ability to accurately estimate a patient’s risk of CVD. It follows that inaccurate risk estimation may contribute to difficulty in appropriately implementing CVD-preventing interventions.

Integrating Risk Information: The Rationale for Global Cardiovascular Disease Risk Scores

Risk Stratification
Over the last decade several authorities have proposed that risk stratification (i.e. estimation of a patient’s overall risk of CVD) should be the first step in deciding how to proceed with preventive interventions.

The rationale for this recommendation arises from the fact that the balance of benefits and harms for treating CHD risk factors varies across the spectrum of CHD risk. This is because several “traditional” risk factors demonstrate a fairly linear relationship with the incidence of CVD such that the risk of CVD is reduced/increased by a relatively constant proportion across the spectrum of CHD risk. As a corollary, the absolute benefit caused by a change in risk factor level depends on the patient’s baseline level of risk.

This linear association and its implications are the underpinning of what most physicians understand intuitively; that is, as the risk of CVD increases, the net
benefits of treatment increase as well. The corollary to this is that as baseline risk increases, the benefits of treatment will outweigh potential harms by a greater margin since the harms of a particular treatment are generally independent of CVD risk and therefore tend to remain at a relatively fixed level (Figure 2)\(^\text{37}\). With this in mind, it becomes clear that estimation of baseline risk is central to balancing the benefits and harms of any intervention.\(^\text{38}\)

![Figure 2: The relationship between CVD risk and the Benefits and Harms of Treatment](image)

Benefit increases as CVD risk increases, but harm is constant because it is generally independent of CVD risk. When CVD risk exceeds a certain threshold, the benefits of treatment outweigh the harms. (Adapted from: Glasziou P, Irwig M. An evidence based approach to individualising treatment. BMJ. 1995;311:1356-1359.)

The clinical utility of these observations can be illustrated using the example of aspirin. **Table 2** shows that when aspirin is used for primary prevention, a greater
number of events are prevented as the baseline risk for coronary heart disease increases. In contrast, the risk of gastrointestinal bleeding from aspirin remains constant across all CHD risk levels.\textsuperscript{3} The number of coronary heart disease events prevented begins to exceed the number of adverse events at a 5-year risk of 3%. Below this threshold level aspirin therapy has the potential to cause more harm than benefit, and thus should be avoided.

[Table 2 is located at the end of the paper]

Framingham Risk Scores
It is possible to arrive at a reasonably good estimate of CVD risk by screening for the traditional CVD risk factors and then calculating a patient's global 10-year coronary heart disease (CHD) risk score. This score, which can be easily calculated using a number of readily available tools,\textsuperscript{39} is based on data collected from the Framingham Heart Study and the Framingham Offspring Study. In 1998 a simplified and updated algorithm was published that integrated the categorical approaches that are a part of blood pressure and cholesterol guidelines.\textsuperscript{36}

The risk estimate is derived from a prediction model that was developed using data from 5345 subjects (47% male) who were free of clinically apparent coronary heart disease (CHD) and were between the ages of 30 and 74 years old at the time of enrollment in the study. The patients were followed for 12 years in order to collect end-point information. Multivariable and logistic regression were used to develop models for the 10-year incidence of CHD events, and the \(\beta\)-
coefficients from the model were used to develop a point system for summing the effects of various risk factors. Table 3 lists the risk factors included in the original model. It is notable that obesity, triglycerides, physical activity, and family history, which are all established risk factors, are not included in the model. It is not clear whether the inclusion of these in the model would provide significant additional information because these risk factors are at least partially collinear with the risk factors already included in the model, and therefore the independent contribution of these risk factors would is obscured.25

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>Definition Used in Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking</td>
<td>Regular smoking during the past 12 months</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Patient on therapy for diabetes, or fasting blood glucose &gt;140 mg/dl</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>Categorized by JNC-V definitions</td>
</tr>
<tr>
<td>Total or LDL-Cholesterol</td>
<td>mg/dl</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>mg/dl</td>
</tr>
<tr>
<td>Age</td>
<td>years</td>
</tr>
</tbody>
</table>

The Framingham model can be used to predict a patient’s 10-year risk of “Total CHD” (angina, recognized and unrecognized MI, unstable angina, and CHD death) or “Hard CHD” (MI and CHD death only). Appendix 2 presents a simple point-based method for calculating a Framingham risk score. Although absolute risk estimates are used most often, relative risk can also be calculated. The reference group is a “low risk” cohort, defined as non-diabetic non-smokers with BP<120/80 mmHg, total cholesterol <200 mg/dl (LDL-C < 130 mg/dl), and HDL-
C \geq 45 \text{ mg/dl (men) or } \geq 55 \text{ mg/dl (women). This definition of "low risk" is based on data from the Multiple Risk Factor Intervention Trial (MRFIT).}^{40,41}

The model adds a degree of perspective because it reveals that different risk factors impart a relatively comparable degree of risk -- generally on the order of a RR 1.2-2.0 (Table 4). The similar influences of the different risk factors is not widely appreciated.\textsuperscript{18,36} Additionally, the model highlights the importance of age as a risk factor: even in the absence of other risk factors, absolute risk for CHD rises progressively with age (Table 5). The higher baseline risk associated with older age points out the substantial opportunity for prevention in the elderly.\textsuperscript{18}

\textit{[Tables 4 and 5 are located at the end of the paper]}

**Clinical Relevance of Risk Scores**
Risk scores help clinicians classify patients as being either at low-, intermediate-, or high-risk of coronary heart disease events over the next 10 years (short-term risk). Definitions for the cut-offs between levels of risk, along with the general implications of each level, can be found in Table 6.

\textit{[Tables 6 is located at the end of the paper]}

Although generally accepted, the cut-offs between risk levels have some uncertainty about them,\textsuperscript{25,42} and the they must be viewed as guides rather than as absolute indications for any particular therapy. This uncertainty exists because definitions of risk must take into account more than just the balance of the
potential harms and benefits of a particular treatment. Issues of cost-effectiveness and individual patient preferences also must play a role. The distinctions become particularly hazy among patients that fall into the intermediate risk category. Although most U.S. authorities agree that those with a 10-year CHD risk of >20% can be considered to be at high short-term risk and therefore candidates for more aggressive risk reduction, there is less agreement on the boundary between low- and intermediate risk, with some arguing that 5% to 6% is a more appropriate cut-off for the "low risk" designation because it limits the category to patients who are less likely to have any of the traditional CHD risk factors other than age.

The issue is further complicated because low short-term risk does not necessarily imply a low long-term risk. For example, a 50 year old man with a 10-year risk of 8% has an approximately 24% risk of having a CHD event over the next 30 years. Depending on the patient's general state of health this may be turn out to be significant.

**Limitations**
There are several important limitations to Framingham-derived global risk scores:

- They are meant only for primary prevention and are invalid for patients who already have CHD -- these patients are already at high risk and require aggressive risk reduction.
- Scores do not apply to severe forms of any particular risk factor, and they probably underestimate risk in those situations.
- Measurements were made several years ago and it is possible that the absolute level of risk associated with a particular risk factor in the population has changed over time.
- The study population was relatively homogeneous, and estimates may not apply to different populations.
- The magnitude of risk reduction achieved by modifying each risk factor may not equal (in reverse) the increment of risk conferred by that risk factor.
- Scores are average values and they may vary among individuals depending on other risk factors not included in equation that might modify overall risk.

Validity
Despite these limitations, global CHD risk scores have been demonstrated to be valid among both white and black Americans, and they perform acceptably in Hispanic Americans.\textsuperscript{25, 44} People of South Asian origin appear to have about twice the absolute risk of whites when living in America, while East Asian Americans may have a lower absolute risk than other ethnic groups in the U.S. However, while absolute risks may be different, the relative risk conferred by risk factors is probably similar for all ethnic groups.\textsuperscript{23}
Other Factors –

CHD Equivalents
Although diabetes is included in the scoring system, its presence has now been elevated to a CHD equivalent, and therefore diabetic patients require more aggressive risk reduction than is indicated by their risk score. Although the elevation of diabetes to a CHD equivalent is a useful clinical decision tool because it reminds physicians that over the long-term, diabetics are at higher risk of developing cardiovascular disease, it can also be somewhat inaccurate because not all diabetics have a short-term CHD risk of >20%. This is particularly true of younger patients.

The distinction is not evident, however, when using newer risk scoring tools (such as those available through the National Cholesterol Education Program) because these tools preclude calculation of a numerical risk once “diabetes” has been noted. In these instances, the answer “CHD equivalent” is returned instead of a numerical estimate of risk. Given that diabetics generally are at higher risk, the distinction is somewhat academic for most patients, but it is worth noting.

On the other hand, peripheral vascular disease and cerebrovascular disease are true CHD equivalents. Considering them as such underscores the fact that atherosclerosis is a systemic disease that can affect the entire vasculature. Patients with these disorders are automatically included in the high risk category.

The Metabolic Syndrome
Approximately 44% of American adults over 50 years old have the metabolic syndrome (defined by the National Cholesterol Education Program [NCEP] as three or more of the following: (1) abdominal obesity (waist circumference >40 inches for males, >35 inches for females), (2) systolic blood pressure ≥ 130 mmHg or diastolic blood pressure ≥ 85 or on anti-hypertensive medication, (3) fasting triglycerides of ≥ 150 mg/dl, (4) HDL-C <40 mg/dl [males] or < 50 mg/dl [females], (5) impaired fasting glucose [110-125 mg/dl]). The metabolic syndrome is associated with an increased risk of CHD (OR 2.07, 95% CI 1.66-2.59), and the importance of its detection is highlighted in the most recent NCEP guidelines.2, 45 Although some components of the metabolic syndrome are included among the major risk factors used in Framingham calculations, not all elements are accounted for, and the true risk for patients with the metabolic syndrome may be underestimated by Framingham global risk scores.2, 18, 45-47

This concern has been called into question, however, by an analysis of data from NHANES III, which suggests that Framingham scores may indeed be accurate in patients with metabolic syndrome. In this study, the metabolic syndrome was a significant predictor of CHD in univariate analysis. However, in a multivariable model that included waist circumference, triglycerides, HDL-C, blood pressure, impaired fasting glucose, diabetes, and metabolic syndrome, only HDL-C, blood pressure, and diabetes were significant.45 This result suggests that the metabolic syndrome is highly correlated with the traditional risk factors, and thus Framingham scores may indeed be valid in this population.
**Novel Risk Factors and New Screening Tests**

Over the last decade a variety of new risk factors and screening tests for CVD have been proposed (*Table 7*), and some, such as C-reactive protein (CRP), have received wide attention in both the medical and lay press. Many of these tests show significant promise for the early detection of CVD, and it has been proposed that they may be useful in identifying low- and intermediate-risk patients who need more aggressive risk reduction.48-53 However, it is not clear whether any of these tests provide clinically useful information that cannot be derived from screening for the traditional risk factors – especially in patients deemed to be at either low- or high- risk for CVD.54-60

*Table 7 is located at the end of the paper*

This uncertainty exists even with the most highly touted novel marker, CRP; despite over 22 published studies, there is still much debate about the extent to which it can add to the prediction of CVD risk over and above the traditional risk factors. Most notably, a recently published large cohort study and updated meta-analysis concluded that although CRP is indeed a statistically significant predictor of CHD, the strength of the association is markedly attenuated when adjustments are made for traditional risk factors.61 This study followed a recent systematic review that also examined the evidence for CRP and concluded that the optimal use for CRP in routine screening and risk stratification remains to be determined.55
Similar situations exist with all of the other novel risk factors and new screening tests that have been proposed. Specifically, no controlled trials of screening for and treating any of the novel risk factors (or using the new screening tests) have been conducted to determine whether integration of such tests in clinical practice makes a difference in patient outcomes. Therefore, although it is likely that some of these risk factors and screening tests will eventually play a key role in prevention, the most appropriate way to use them is still unclear. Cost effectiveness also remains to be determined. Until these issues are resolved, the greatest clinical benefit can probably be derived by continued focus on screening for and treating traditional CVD risk factors, which, as noted above, still remain woefully under-treated in the U.S.

Is There Evidence Supporting the Use of Global Risk Scores?

As noted above, the use of global risk scores has several potential benefits:

- Improved physician estimation of a patient's CVD risk
- Improved identification of high-risk patients who require immediate attention\(^{25}\)
- Improved balancing of potential benefits and harms of preventive interventions
- Ability to modify the intensity of preventive interventions depending on the baseline estimate of risk\(^{25,62}\)
Motivation for both physicians and patients to overcome the clinical inertia that often prevents an effective intervention from being initiated.\(^{63, 64}\)

- Improved patient understanding of the rationale for treatment
- Improved patient understanding of the individual’s risk of CVD\(^{33}\)
- Improved patient adherence to prescribed risk-reducing interventions\(^{65}\)
- Improved cost-effectiveness of risk-reducing interventions\(^{66}\)

These proposed benefits address a number of factors that are perceived as being responsible for the current under-utilization of interventions that reduce the risk of CVD. Underlying all of them is the assumption that the routine use of Framingham global CHD risk scores will lead to improved clinical outcomes.

In many ways this makes sense. It is clear that some form of risk estimation is a requisite part of good preventive medicine, and it is also clear that both physicians and patients have difficulty accurately estimating risk. It follows from these observations that a tool that corrects misperceptions and raises awareness of risk should be useful. To some extent this appears to be the logic behind the various recommendations for the use routine use of global risk scores.

Although this may in fact be the case, there is relatively little data on whether the routine use of Framingham-derived global risk calculations actually translates into improved clinical outcomes. In an era of evidence-based medicine this is a
seeming paradox: widespread support of a screening test without any evidence of benefit. Even more interesting is the fact that this is done with the goal of increasing the use of other evidence-based interventions. This situation likely stems from four factors. First, as described above, the risk scores themselves are valid predictors of CHD risk. Second, their use is associated with a very low cost in terms of both time and money. Third, there is an assumption that no possible harm can arise from using them, and lastly, and perhaps most importantly, there is such an enormous underutilization of appropriate CVD risk-reducing interventions that any tool that is perceived to be a useful remedy to the situation is welcomed.

The goal of this paper is not to refute this line of reasoning. Rather, we hope to shed light on the issue of whether there is evidence that the routine use of global risk scores is in fact associated with clinical benefits. Two approaches are used. First, the result of a systematic review of the literature in this area is presented. Since it was expected that there would be a paucity of studies addressing this issue, the systematic review was performed concurrent with a small study that attempts to test the hypothesis that physicians make better CHD prevention decisions when they interpret risk factor information in the context of a global risk score than when they view the risk factor information only. The methods and preliminary results of this study are presented following the systematic review.
Is the Routine Use of Global Coronary Heart Disease Risk Scores Associated with Clinical Benefits?

A Systematic Review of the Literature
Methods

Questions to be reviewed:
The primary question addressed by this review is: Does routine calculation of a global CHD risk score by physicians (as opposed to either simple risk factor counting or no formal assessment of risk) lead to clinical benefits? For the purposes of this review, clinical benefits are broadly defined as either (1) improved physician adherence with evidence-based guidelines for the primary prevention of CVD, (2) increased appropriate prescribing of risk reducing therapies, (3) improved control of patient CVD risk factors (i.e. blood pressure, cholesterol), (4) a reduction in CVD events, or (5) increased patient adherence with therapies targeted at the primary prevention of CVD.

Given that any screening test may be associated with harm, a secondary question is whether there are any harms associated with screening using global risk scores.

Search Strategy
We searched MEDLINE (1966- April 2004) and the Cochrane database using MeSH terms and four distinct search strategies. This broad search strategy was used in order to increase the likelihood of finding all pertinent studies. The search was limited to studies in humans and to the English language literature.

- **Search strategy 1**: “Cardiovascular Diseases/pc [Prevention & Control]” AND “Risk Assessment”

- **Search strategy 2**: (“Heart Diseases” OR “Vascular Diseases”) AND “Risk Assessment” AND (“Feasibility Studies” OR Cross-Sectional
Studies” OR “Follow-up Studies” OR “Longitudinal Studies” OR “Prospective Studies” OR “Cross-Over Studies” OR “Intervention Studies” OR “Cohort Studies” OR “Epidemiologic Studies” OR “Case-Control Studies” OR “Retrospective Studies” OR “Multicenter Studies” OR “Evaluation Studies”)

- Search strategy 3: “Cardiovascular Diseases/pc [Prevention & Control]” AND (“Framingham.mp” OR “Global Risk.mp” OR “Global Risk Score.mp” OR “Coronary Risk.mp” OR “Coronary Risk Score.mp” OR Cardiovascular Risk.mp” OR “Cardiovascular Risk Score.mp”) AND (“Feasibility Studies” OR Cross-Sectional Studies” OR “Follow-up Studies” OR “Longitudinal Studies” OR “Prospective Studies” OR “Cross-Over Studies” OR “Intervention Studies” OR “Cohort Studies” OR “Epidemiologic Studies” OR “Case-Control Studies” OR “Retrospective Studies” OR “Multicenter Studies” OR “Evaluation Studies”)

- Search strategy 4: “Cardiovascular risk score$.mp” OR “Coronary risk score$.mp”

**Study Inclusion Criteria:**
We included studies of any design as long as they met the following criteria: (1) study population consisted of asymptomatic adults ≥ 18 years old with no prior history of CVD; (2) Global CHD risk calculation was specified as the primary study intervention (multi-factorial interventions were acceptable as long as the calculation of global CHD risk was the most prominent part of the intervention);
(3) There was clear documentation of the calculation of a global CHD risk score by a physician or other health care provider as part of an individual patient encounter (Questionnaire-based studies were considered acceptable as long as they were designed to simulate clinical encounters with patients); and (4) One or more of the following endpoints was used: (i) rates of prescribing for aspirin, anti-hypertensive medication, or lipid-lowering medication; (ii) change in patient blood pressure or cholesterol levels; (iii) physician compliance with guidelines for CVD prevention; (iv) rate of CVD events (defined here as new onset stroke/transient ischemic attack, myocardial infarction, acute coronary syndrome, stable angina, peripheral vascular disease, carotid artery disease, or cardiac death); (v) patient adherence with therapy; or (vi) harms of CHD risk calculation.

We considered Framingham-derived estimates preferable, however other scoring systems were acceptable as long as they presented risk in a comparable fashion (i.e. as an absolute risk estimate or in terms of risk categories – low, intermediate, high). Additionally, in order to be included, studies had to provide enough information so that it was possible to determine the method of global risk calculation as well as the manner in which the risk assessment was used in the clinical encounter.

Study Exclusion Criteria:
We excluded studies for the following reasons: (1) they involved children or adolescents; (2) risk calculation was performed primarily by patients; (3) risk
calculation was performed outside of a clinical setting; or (4) the study primarily addressed secondary prevention of CVD.

**Data Extraction Strategy**
A single reviewer determined which studies were eligible for inclusion in the review and then independently abstracted the data from the studies into tables for analysis. A second reviewer checked these tables for accuracy and disagreements were resolved by discussion between the reviewers.

**Assessment of Study Quality**
We assessed study quality using criteria proposed by the U.S. Preventive Services Task Force. Under these criteria both research design and internal/external validity are taken into account when assessing the quality of an individual study. Each study is given a grade based on the traditional hierarchy of evidence (I [randomized controlled trial], II-1 [controlled trial without randomization], II-2 [cohort or case-control study], II-3 [multiple time-series with or without intervention, or dramatic results from an uncontrolled experiment], III [expert opinion, case reports, descriptive studies]).

Additionally, grades are also assigned for internal and external validity based on a three-category rating system (good, fair, and poor). The study grade for internal validity is based on fulfillment of the following criteria: (1) comparable comparison; (2) equal, valid, and reliable measurement; (3) clear definition of the intervention; and (4) consideration of all important outcomes. Studies that meet all of these criteria are graded as “good”. Studies that meet most of these criteria
are graded as “fair”, and studies that meet few or none of these criteria, or those that have a fatal flaw, are graded as “poor”. Similarly, the study grade for external validity is based on the fulfillment of the following criteria: (1) study population reflects the type and spectrum of patients that are likely to be seen by a general practitioner, and (2) study procedures reflect actual clinical practice. Grades of “good,” “fair,” or “poor” are assigned in a similar fashion as for internal validity.

Once the quality of each individual study has been assessed, the body of evidence as a whole is taken into account. At this level the internal and external validity of all the studies are considered in the aggregate, and the consistency and coherence of the evidence are each assigned a grade of “good,” “fair,” or “poor”.

Results

Search Results: We summarize the results of our May 2004 literature review in Tables 8 and 9. Overall, we reviewed 5254 citations.

<table>
<thead>
<tr>
<th>Search Strategy</th>
<th>Initial Number of Articles Identified</th>
<th>Number Remaining After Review of Article Titles</th>
<th>Number Remaining After Review of Abstracts</th>
<th>Number Remaining After Review of Articles</th>
<th>Number Included in Systematic Review</th>
</tr>
</thead>
<tbody>
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<td>3</td>
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<td>1299</td>
<td>58</td>
<td>6</td>
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</tr>
</tbody>
</table>
3955 (75%) were clearly not relevant to the questions of interest. An additional 1241 articles were excluded after careful review of the title and abstract. Detailed review of the remaining 58 articles excluded 52 articles. The most common reasons for exclusion were that the cited articles did not have an experimental design (i.e. they were reviews, editorials, and letters) (11 articles) or that they related to the development and validation of different risk scoring systems (23 articles). A number of studies were also excluded because they either used risk scores as part of the eligibility criteria (for example, to limit a study population to high risk patients) or as an endpoint, but not as an intervention (18 articles). Six studies met our inclusion criteria and were included in our review.

Of the six studies, four addressed whether routine calculation of a global risk score improves clinical outcomes (Table 10). The remaining two addressed the question of whether there are harms associated with risk calculation (Table 11). No studies directly addressed the effect of risk scores on patient adherence. All included studies were published after 1996.

[Tables 10 and 11 are located at the end of the paper]
Of the four studies that addressed whether routine use of global risk scores is associated with clinical benefit, three were randomized controlled trials and one was a cohort study. Three took place in actual clinical settings, whereas a fourth study queried physicians using hypothetical patient scenarios. Three studies (those by Ramachandran, Lowensteyn, and Montgomery) took place in a general practice population, but the fourth (a study by Hall) was conducted in a Diabetes referral clinic. All four studies used some form of a Framingham-derived risk score. In two studies, risk scores were provided to the physician; in the other two, physicians were only given the tools for risk calculation. The number of patients and, more importantly, physicians included in each study varied widely, with the study by Hall being the smallest (6 physicians, 323 patients) and the study by Lowensteyn being the largest (253 physicians, 958 patients). As can be seen in Table 10, the primary endpoints of the four studies varied widely.

Of the two studies that address the potential harms of using global risk scores, one is a non-blinded randomized controlled trial and one is a cohort study. Both studies used risk scores derived from epidemiologic databases other than Framingham (Northwick Park Heart Study; British Regional Heart Study and Dundee risk score). However, in both instances the risk scores were presented in a categorical format (low, intermediate, high) similar to Framingham scores. Both included a relatively large number of patients, took place in a general practice population, and used questionnaires to assess their endpoints (Table 11).
As shown in Table 12, study quality for both studies on the benefits and harms of global risk calculation ranged from poor to fair. Three of the four studies addressing the clinical benefits of global risk scores received a rating of “I-fair” and one was rated as “II-2-poor”. The two studies addressing harm were evenly split between the same ratings. Across all of the studies a major limitation in determining both the internal and external validity of each study was a significant lack of information about not only the study procedures, but also the baseline characteristics of both the physicians and the patients. This lack of information creates difficulty in assessing for important issues such as the adequacy of randomization, confounding, and selection bias. Additionally, among the randomized trials, blinding was only attempted in the studies by Hall and Lowenstein, and in both instances it is unclear if it was successful. These issues are explored further in the discussions of each study.

[Table 12 is located at the end of the paper]

**Does routine calculation of a global risk score improve clinical outcomes?**

Of the four studies assessing the benefit of global CHD risk calculation, the study by Hall and colleagues\(^7\) provides the most direct evidence that risk calculation is associated with a change in physician prescribing habits. The only other study that addressed this outcome (Montgomery) examined as a secondary outcome.\(^7\)
In the Hall study, six diabetologists saw 323 consecutive patients who were free of CVD and were alternately randomized to either an experimental or control group. For patients in the experimental group, physicians received 5-year CHD risk scores (New Zealand risk score) on the front of the chart. They received no such information for control patients. The primary outcomes were a documented change in treatment of diabetes, prescription rates of lipid lowering or antihypertensive drugs, or referral to a dietician. Overall, documentation of a risk score did not have an effect on physician prescribing habits; however, Physicians were more likely to prescribe a lipid lowering or anti-hypertensive drug to patients with a 5-year CHD risk >20% (52% of the patients (p<0.02).

Several issues limit the usefulness of this study and result in it receiving a rating of “I-fair”. Foremost among these is that almost no baseline data about patients or physicians was provided. This lack of information precludes knowing whether randomization was successful and leaves open the possibility of significant confounding. Adding to this is the fact that it is unclear whether the attempt at blinding the physicians and patients was successful. Furthermore, the endpoint is somewhat difficult to interpret because the only significant result came from subgroup analysis and no information was given as to the appropriateness of the changes in therapy. Although it can be argued that in high-risk patients increased prescribing of lipid-lowering and anti-hypertensive drugs is often the correct thing to do, from a decision-making standpoint, it would have been more informative to
determine whether risk scores led the physicians to make more appropriate (i.e. guideline-directed) decisions.

The generalizability of the study is also questionable because of the small sample size and the type of patients included. Only six physicians from a single practice were included in the study, and it is difficult to know whether these physicians are truly representative most primary care clinicians. Furthermore, the population studied is not necessarily representative of the general primary care population. These patients all had diabetes, and by U.S. standards 87% of the patients would be considered as being at high risk (10-year CHD risk >20%). Despite this, the types of decisions being made by the physicians are reflective of typical primary care decisions. Similar endpoints were examined in the study by Montgomery and colleagues.71

In this study, patients’ global risk and physicians’ prescribing habits were examined in 27 general practices in the UK (comprised of 74 physicians and 11 nurses) after physicians and nurses were randomized to receive one of three interventions: (1) a computer-based clinical decision support system plus a CHD risk chart (2) CHD risk chart alone (New Zealand risk chart), or (3) usual care. Both the computer-based system and the risk chart calculated identical estimates of 5-year CHD risk, the only difference being that the computer-based system presented the risk numerically and the chart presented it pictorially. From within each practice, 30 treated hypertensive patients were randomly selected for
invitation to participate in the study and a total of 614 patients attended a baseline screening visit and were enrolled in the study. Follow-up was completed at 12 months.

After adjustment for practice computer system and for baseline CHD risk, a similar proportion of patients in all three groups were still at high risk (5-year CHD risk ≥10%) after one year. Therefore it appeared that providing physicians with the tools to calculate global CHD risk did not improve patients CHD risk beyond usual care. Interestingly, however, analysis of secondary endpoints revealed that risk calculation was associated with lower mean systolic blood pressure at 12 months compared to usual care group (-4.6 mmHg). Patients in the chart only group were also more than two times as likely to be prescribed cardiovascular drugs than were patients in the other groups (p<0.01). Although these results did not adequately account for multiple comparisons and were attenuated in the computer group, they suggest that global risk calculation may affect physician prescribing and intermediate risk factor outcomes, but not enough to move an appreciable number of people across the high-risk cut-off.

As with the studies by Hall, this study also received a quality rating of "I-fair". The primary reason for this rating is that the study was not blinded and that too few details were given regarding the study protocol. The former issue is particularly important because it leaves open significant room for confounding and bias on the part of both the physician and the patient. It is also problematic
that >25% of the patients already had CVD at baseline. Since global risk scores are meant only for primary prevention, their use in this population is questionable.

The limited provision of data also limits the external validity of this study. No information is given as to the types of drugs (lipid lowering, antihypertensive, anti-anginal...) that were prescribed, and limited details are provided about exactly how the risk chart or computer system was used (by physician or nurse; before, during or after patient visit...). This lack of treatment and protocol details makes study interpretation difficult. Additionally, as with all the studies included in this review, these were generally high risk patients: the mean 5-year CHD risk at baseline was 18.5% (37% 10-year risk). Despite this, the overall control of risk factors at 12 months was surprisingly poor. For example, the mean systolic blood pressure in all three groups was still >150 mm Hg. This suggests that these study patients were not being treated to a similar standard as would currently be expected.

Similar to the studies by Hall and Montgomery, the study by Lowensteyn also focused on physician calculation of global risk scores for patients. In this study, 253 Canadian physicians were recruited from among 445 attendees at a continuing medical education meeting on CVD risk assessment. The physicians were randomized to either a profile group or a control group, and they were all instructed to enroll patients between the ages of 30-74 who were free of CVD and "in whom they thought a risk profile would be clinically useful." When a patient
was enrolled in the study, the physician and patient completed different parts of the enrollment form (data on risk factors, etc...) which was then mailed to the coordinating center. Profile physicians scheduled patients to come back in 2 weeks for their risk profile results; control physicians did not. Control patients only received their risk profile if they returned for a follow-up visit in 3-6 months. At the 2 week follow-up, the profile group patients were told their 8-year CHD risk. Follow-up after that was at the discretion of the physicians and patients.

The main hypothesis of the study was that being labeled as high risk at the initial visit would encourage physicians and patients to have a follow-up visit. Therefore, the main outcome measure was the likelihood of a high vs. low risk patient being seen at the 3-month follow-up. The difference in likelihood of follow-up between the two groups was considered to represent the effect of the risk profile. Secondary outcomes included changes in specific risk factor values between baseline and the 3-month follow-up.

Overall, the likelihood of physicians reassessing high vs. low risk patients was significantly greater in the profile group, which suggested to the authors that calculation of a risk profile leads to increased follow-up of high risk patients. Additionally, after adjustment for baseline differences, the profile group patients demonstrated significantly greater reductions in lipid values (total cholesterol -0.49 vs. -0.09 mmol/L) and calculated 8-yr CHD risk (-1.8% vs. -0.3%). There was not a significant difference for other risk factors.
The conclusions of this study must be viewed with criticism. First, although 253 physicians were randomized into the study, only 51% of physicians (57% profile; 39% control) actually enrolled patients in the study. The method of enrollment also raises the question of selection bias because physicians were instructed to enroll patients "in whom they thought a risk profile would be clinically useful." Combined with the broad inclusion criteria (age and freedom from CVD), these instructions allowed significant leeway in deciding which patients to recruit into the study. Since no data is given on patients who were screened but not enrolled it is impossible to rule out selection bias. This is true despite the fact that baseline patient characteristics were similar in both groups.

More worrisome however, is the exceedingly high attrition rate among study patients. Although 958 patients were initially enrolled, only 50.6% of control and 25.8% of profile group patients actually came back for follow-up. The disparity between groups appears to due to the study design which encouraged control patients to come back for follow-up because the results of the risk profile were withheld until that visit. This "encouragement" confounds the normal patient and physician motivation for scheduling (and for actually returning for) follow-up.

The external validity of this study is hampered by the awkward study design, which does not mimic clinical practice. In fact, it is somewhat difficult to draw a parallel between the study scenario (patient provides risk factor information;
information is mailed to an outside agency; risk score is mailed back two weeks later) and what actually happens in the real-world (risk score is calculated and acted upon during a single clinic visit).

In contrast to these studies, the final study addressing the clinical utility of global risk scores suggests that risk calculation may not be associated with a discernible benefit. This study, however, has important differences: it is a cohort study, which allows greater potential for confounding; it uses hypothetical patient scenarios to address the effects of risk calculation on clinical outcomes; and it has several flaws, which result in a poor quality rating.

In this small study Ramachandran and colleagues mailed questionnaires to 200 randomly selected general practitioners (GPs) in the UK. Each questionnaire consisted of 20 patient case scenarios in which CVD risk factor information was given, and the GPs were asked to indicate what method they used to determine the patient's risk of CVD and whether they felt that lipid lowering therapy was indicated. The primary outcome was the proportion of correct responses based on the UK guidelines at that time which stated that lipid-lowering therapy is indicated in patients with a 10-year CHD risk >30%. GPs were reminded of this guideline in the cover letter that accompanied the questionnaire.

Only 61 physicians (30.5%) responded. Of these, 14 (26%) calculated a 10-year CHD risk score (Sheffield Table), 26 (48%) solely used clinical judgment to
gauge risk, and the remaining 21 (26%) used some combination of methods. Overall 62.1% of decisions were in accordance with the guideline; however, there was no difference in the appropriateness of decision-making between GPs who used clinical perception as the sole means of risk assessment and those who used the Sheffield table either on its own or in conjunction with other methods (p=0.21).

While interesting, this study received a quality rating of “II-2-poor” for a variety of reasons. Foremost among these are the extremely low response rate and the fact that physicians were not randomized as to the method of risk determination used. Both of these introduce the possibility of significant confounding. Additionally, the low response rate suggests that the study likely does not have enough power to detect a significant difference in the primary endpoint. This is difficult to determine because no sample size calculations were reported. Lastly, since no information is given on physician characteristics it is impossible to determine if the physicians included in the study reflect the average general practitioner.

When these four studies are examined in aggregate the quality of the evidence supporting a clinical benefit to the routine use of global risk scores is poor to fair. Given that the no meaningful conclusions can be drawn from Ramachandran study, the overall evidence quality is pulled towards fair. However, this is counterbalanced by the fact that of the remaining three studies, only the small
study by Hall directly addresses the question of interest. While the studies by Lowensteyn and Montgomery both provide some evidence of a benefit associated with the use of global risk scores, neither addresses the routine use of risk scores as they would be expected to be used in practice. Additionally the lack of treatment details in all three studies creates difficulty in establishing a fully credible linkage between the use of risk scores and the modest clinical benefits seen in each study. Additional difficulty in drawing a conclusion is introduced because the Hall study, which represents the most direct evidence, only included six physicians. In terms of physician sample size this is significantly outweighed by the Lowensteyn and Montgomery studies.

**Are there harms associated with screening using global CHD risk scores?**

Two studies directly address whether CVD screening using global risk scores is associated with adverse psychological outcomes. Both studies suggest that there is not any significant harm. However, both are marked by several flaws which limit their validity.

Connelly\(^70\) performed a prospective cohort study of 5772 men in which all participants underwent CHD screening and were informed of their level of CHD risk based on an algorithm derived from the Northwick Park Heart Study. The men were not told their risk level at the initial screening; rather they were later sent a letter that informed them of their personal risk for CHD (categorized as low, moderate, or high). Men deemed to be at “high” risk were seen back for a follow-up appointment during which they were able to discuss the results. The
letters sent to men deemed to be at “moderate” risk included general advice about the particular risk factor that was elevated; however no follow-up appointment was offered. The “low” risk men were simply sent a letter stating that they did not have any special risk for CHD.

Psychological symptoms in all of the men were assessed at the baseline screening, at the time that they were informed of their risk level, and again at three months. Overall, men who were labeled as being at either “high” or “low” risk showed a decrease in their psychological symptoms after labeling. Interestingly, the men who were labeled as “moderate” risk actually had an increase in psychological symptoms. Taken together, this result appears to suggest the intuitive conclusion that if information about elevated risk is not accompanied by appropriate support, there may some degree of increased anxiety among patients.

This study received a quality rating of “II-2-fair” based on several factors. The primary internal validity issues were that the study was uncontrolled and not blinded. Additionally, not enough details were provided about the risk score and what went on at the screening and follow-up visits. When coupled with a 25% drop-out rate, these issues raise questions about the strength of the evidence provided by the study that even the large sample size does not completely outweigh. The generalizability of the study is hampered by a lack of details about the interventions and by the nature and clinical significance of the “psychological symptoms” experienced by the patients.
Despite being a randomized controlled trial, the study addressing harms by Marteau and colleagues is fraught with such a substantial omission of information that it is very difficult to use in drawing conclusions. This study randomized approximately 3000 couples to a screening or a control group. The screening group couples underwent psychological and CHD screening, including an estimate of their global CHD risk. They were then counseled on ways to reduce CHD risk, and they were offered follow-up at a frequency commensurate with their level of risk (more frequent follow-up for higher risk patients). Control patients did not undergo any screening until the one year mark, at which time they underwent the same procedure as the screening group. The primary outcome was a comparison of the perceptions of health, the risk of suffering a heart attack, and the ability to reduce that risk in the intervention and the control groups at 1 year.

Overall, the study results suggest that participation in a screening program including calculation of a CHD risk score was not associated with adverse concerns about health, but that it may be associated with a sense of less control over one's own personal risk.

Exceedingly few details were provided regarding several key issues, including the screening and enrollment process, randomization, attempts at blinding, baseline patient characteristics, content of follow-up visits, and treatments that were undertaken. Additionally, the clinical relevance of the questionnaire measures is unclear. Taken as a whole, these omissions make it impossible to rule-out a
variety of sources of bias and confounding. Therefore the study received a quality rating of "I-poor".

In aggregate, there is poor to fair evidence that global risk scores are not associated with significant psychological harms. The large sample size of the Connelly study bolsters the "fair" rating, however given that it is only a single study it is difficult to draw any firm conclusions.

Discussion

Despite calls for widespread use of global CHD risk scores as a clinical screening tool there is a paucity of literature supporting the numerous theoretical benefits associated with such screening. The majority of the literature on global risk scores currently relates to the development and validation of different risk scoring systems. This review highlights the fact that not much research has been done to elucidate the actual clinical benefits that can be expected if clinicians adopt global CHD risk scores into their everyday practice.

Based on the available studies reviewed in this paper, there is fair to poor evidence that the routine use of global CHD risk scores may be associated with an increase in the prescribing of cardiovascular drugs to high risk patients and possibly with improved control of blood pressure and cholesterol levels. However, even excluding the one poor quality study, the small number of studies with disparate outcomes and the relatively small number of physicians included in
these studies precludes definitive conclusion about the effects of global risk
calculation on physician prescribing habits and any subsequent reduction in
patient risk factors and global CHD risk.

Indirect support for the clinical benefit of global CHD risk scores comes from two
studies which were not included because they did not meet all of the inclusion
criteria. Both studies examined whether global CHD risk estimation, in
conjunction with a broader screening and/or behavioral intervention is associated
with clinically apparent changes in CHD risk factors. Both found some evidence
of benefit.

In 1985, Lovibond and colleagues\textsuperscript{74} performed a small trial in which 75 patients
deemed to be at high risk of CHD were randomized to one of three behavioral
interventions: (1) maximal behavioral treatment, (2) extended behavioral
treatment, or (3) basic behavioral treatment. All three interventions involved
attendance at both group and individual counseling sessions for 6 months and all
were designed so as to attempt simultaneous correction of multiple CHD risk
factors through intensive multi-faceted lifestyle modification. As a part of this
larger program, groups 1 and 2 were also provided with information about their 5-
year risk of CHD. Group 3 was not.

After 12 months all three interventions were found to be associated with
significant beneficial changes in weight, blood pressure, aerobic capacity,
cholesterol, smoking status, and overall calculated CHD risk. The groups that received information about global CHD risk appeared to have a slightly greater benefit, thus suggesting that provision of global CHD risk information was a beneficial component of the overall program.

In a different study, Engberg and colleagues\textsuperscript{75} investigated the effect of a general health screening on the cardiovascular risk profile of a randomly selected population in a small rural county in Denmark. In this randomized controlled trial, 1507 residents of the county who were between the ages of 30 and 49 years were randomized to one of three arms: (1) a control group that received no health screenings, (2) an intervention group that received two health screenings, and (3) an intervention group that received two screenings and a 45-minute follow-up consultation annually for five years. The health screenings were performed by a laboratory assistant (not a physician) and included calculation of a CVD risk score. Each person received personal written feedback from his/her doctor within a few weeks after the screening session. If values for CVD risk factors fell outside the normal range advice about lifestyle modification was given. If CVD risk was calculated as "high" the letter suggested that the person should follow-up with the doctor. Educational pamphlets were also sent to all participants. The health discussion was a 45-minute consultation with the physician at the end of which goals were set for lifestyle modification.
After five years, there was a small but statistically significant difference in the mean CVD risk score, body mass index, and cholesterol levels in the intervention groups as compared to the control group. The benefit appeared to be greater among smokers and overweight patients. Additionally, there was also a lower prevalence of patients deemed to be at "high" risk in the intervention groups. This again appears to suggest a modest benefit to global CHD risk scores when used in conjunction with a larger screening and behavior modification program.

Although both of these studies are of interest, they were not included in the formal review because the risk score itself was not the primary intervention and because they both had a greater focus on patient use of risk information rather than on physician use of that information. Additionally, their direct applicability to everyday clinical practice is questionable. In both cases it is impossible to tease out the effect of global CHD risk scoring from the rest of the intensive intervention, and neither study addresses whether routine calculation of global risk scores by physicians is clinically beneficial. Therefore, at best, these studies provide a modicum of additional indirect support for the idea that global risk scores can be helpful.

An issue which was not addressed by included studies is whether patients were provided with their global risk scores and whether this altered patient behavior. Indirect evidence is also limited, with the only study that marginally addresses this issue being by another Montgomery and colleagues.76
In this small study they found that when CHD risk scores were incorporated as part of an intensive decision-analysis session, newly diagnosed hypertensive patients reported less decisional conflict about whether to start an antihypertensive drug than did patients who only received an informational video and leaflet. The patients who were told their risk score and who underwent intensive decision-analysis counseling reported less decisional conflict about starting anti-hypertensive treatment. Unfortunately, as with the studies by Lovibond and Engberg, the study design makes it is impossible to distinguish the separate effect of CHD risk scoring apart from the overall intervention. Additionally, at three months there was no difference in the number of patients who actually wound up taking medication, which suggests that the reduced decisional conflict did not translate into a clinically meaningful difference.

Given that only one of the two studies addressing the psychological harms associated with global CHD risk screening was judged to be of at least fair quality, it is not possible to come to any final conclusion about the balance of benefits and harms associated with risk calculation. The study by Connelly does suggest that there are no discernable psychological harms when global CHD risk screening is accompanied by appropriate information and support. As noted, however, the quality of this study and the fact that it is only a single, albeit large, study limits the conclusions that can be drawn.
One interesting facet to the question of harm is that in order for psychological harm to be plausibly related to the use of global risk scores, patients must be told of their calculated risk. Of the studies assessing clinical benefits, only the Lowensteyn study specifically stated that patients were told their risk score. The other studies do not provide enough information to determine if patients were told or not. The Lovibond and Engberg studies also informed patients of risk, however no harm-related endpoints were collected. The curious lack of attention to possible harms associated with the use of global risk scores suggests that the investigators in all of these studies assumed that no possible harm could arise. As we have shown, there is very little evidence to either support or refute this belief.

Overall, the paucity of evidence makes it is impossible to reach any broad conclusions about the true clinical benefits or harms associated with the use of global CHD risk scores. The evidence that does exist appears to point in the direction of a modest benefit without any discernible harm. Importantly, there are no studies that demonstrate a benefit in terms of hard outcomes. Despite this, the increase in prescribing of cardiovascular drugs suggests that the routine use of global risk scores may lead to a tangible clinical benefit. Although an indiscriminant increase in prescribing would not be helpful, increased prescribing of anti-hypertensive and lipid-lowering drugs is often indicated in patients at intermediate to high risk of CVD. As previously reviewed, there is currently a gross underutilization of such evidence-based therapies, and if global risk scores
can be shown to improve this situation it would be an important reason to use them.

Furthermore, the small improvements in lipid levels and blood pressure seen in two of the studies may be important. On a population level these changes, which likely reflect the increased use of risk-modifying medications, have the potential to benefit a significant number of patients.

As noted in the introduction, risk stratification is requisite for achieving a favorable balance between the potential benefits and harms of therapies aimed at reducing CVD risk. Given that both clinicians and patients have difficult estimating risk, as well as the theoretical benefits and the fact that global risk scores are simple, readily available in the clinic, non-invasive, inexpensive, and well-validated it is important that the medical community better understand the their use.

This review highlights the fact that very little is actually known about the best manner in which to incorporate global CHD risk scoring into everyday practice. Further research is needed to better elucidate the true benefits, best target population, and most appropriate setting for the use of global risk scores. Additionally, it is important to delineate the mechanism of any benefits that are proven. Only with a thorough understanding how risk scores affect clinical decision-making can we hope to appropriately integrate them into practice.
Future studies will ideally address these issues in a more direct manner than has been used in the studies described here. Prospective trials in which global risk is calculated in a systematic and standardized fashion are most likely to provide useful information. Although it is unlikely that any trials will be large enough to address hard endpoints, smaller studies addressing issues of physician decision-making and adherence with guidelines, patient adherence with therapies, and control of blood pressure, lipid levels, and other risk factors are quite feasible and would provide much useful information. Importantly, any future studies must also carefully collect data on treatments that are undertaken (or foregone) on the basis of risk score information, as this data is critical to having a full understanding of the dynamics of global risk score information in practice.

Lastly, the issue of harm cannot be ignored. Although significant harm seems unlikely, the current evidence is insufficient to reach that conclusion. Aside from the potential for psychological harm associated with labeling, there is also a possibility that use of risk scores could be associated with an increase in inappropriate prescribing or even with under utilization of therapies if the patient and clinician are falsely reassured. Any future research should carefully document these issues. Clinicians should also be aware that the meager evidence that is currently available suggests that CVD risk score should be accompanied by an appropriate amount of explanation and support. What is “appropriate” likely depends on the level of risk and remains to be determined in future studies.
This analysis has several limitations. Although a relatively exhaustive literature search was attempted, there remains the possibility that relevant literature may not have been located. The questions posed by this review lend themselves to a variety of research designs by investigators in different disciplines, and as found in this search, no one search strategy was able to identify all of the relevant articles. Although it was felt that the current search likely identified the most relevant articles and that a more extensive search would likely provide a very low yield, it is possible that some studies may have been missed. A similar problem is posed by the limitation of the search to the English language literature and by the fact that no attempts were made to contact experts in this field outside of the University of North Carolina to query them about literature that they might be aware of. Finally, there is no way to assess for publication bias.

Global CHD risk scores represent a potentially useful addition the clinician’s armamentarium. Given the burden of CVD in our society and the significant amount of work that needs to be done to improve our CVD prevention efforts, global risk scores may well be an important additional screening tool. If future research continues to bear out their usefulness, their ease of use and low opportunity cost will cause them to be counted as a significant addition to preventive medicine in the U.S.
The Effect of Global CHD Risk Scores on Physician Decisions Regarding the Primary Prevention of Cardiovascular Disease:

Preliminary Results
Background

Physicians often do not accurately estimate a patient’s risk for future cardiovascular events.\textsuperscript{26-32} This is potentially problematic because the assessment of global coronary heart disease (CHD) risk has been integrated into multiple guidelines for the primary prevention of CHD\textsuperscript{1-3}. Estimations of risk can be improved by the use of risk calculations based on data from the Framingham cohort,\textsuperscript{18,25,36} and these calculations are relatively simple to perform using either hand-scored sheets or readily available calculators for personal digital assistants (PDAs) and computers\textsuperscript{39}. Despite the theoretical benefit of risk calculation, however, there is relatively little data on whether the routine use of such calculations actually translates into improved clinical outcomes. In particular, there is little known about how knowledge of a patient’s calculated 10-year risk affects a physician’s perception of a patient’s risk and whether that in turn translates into improved adherence with published guidelines for the primary prevention of CHD.

The purpose of this study is to determine whether clinicians make better CHD prevention decisions when they interpret risk factor information in the context of a calculated global risk score than when they view risk factor information alone. The methods and preliminary results of this study are briefly presented here.

Methods

\textbf{Study Design:} A multi-center, randomized, single-blinded study.
**Setting:** Two academic medical centers in North Carolina: The University of North Carolina (UNC) and Duke University Medical Center (DUMC).

**Subjects:** Convenience sample of Internal Medicine, Medicine/Pediatrics, and Family Medicine residents who were recruited into the study during one of their regularly scheduled educational conferences.

**Inclusion Criteria:** Participants had to be Internal Medicine, Medicine/Pediatrics, or Family Medicine residents at UNC or DUMC.

**Exclusion Criteria:** (1) Failure to consent to participate in the study; (2) Non-resident status (medical student, attending physician, or physician extender)

**Intervention:** The study intervention consisted of a questionnaire containing ten clinical case scenarios and several additional questions pertaining to the participant's attitude towards CHD risk scores. The patient scenarios were crafted to explore physician's knowledge about preventive guidelines for CHD, including the recommended thresholds for prescription of aspirin, hypertension medications, and cholesterol lowering medications. Additionally, the order of the scenarios was varied among three different sequences in order to avoid bias from any ordering effects.
Participants were randomly assigned into two groups: Group 1, the intervention group, was given the calculated Framingham risk score for each patient scenario. Group 2, the control group, was not given the risk score, nor were they allowed to calculate the score on their own. A computerized program was used to generate the randomization scheme.

Table 13 lists the ten cases that were presented on the questionnaires along with the rationale for including the case and whether guidelines indicated that aspirin, lipid-lowering therapy, or anti-hypertensive therapy is indicated by current guidelines. Appendix 3 shows an example of how the cases were presented. For each case, subjects were asked to estimate the patient’s short-term CHD risk using traditional risk categories (low, intermediate, high). Group 1 was given a numeric risk estimate that could be used as a basis for selecting a categorical risk level, and group 2 had to both select a risk category and make a numerical estimate of the patient’s risk. Following this, subjects were asked whether the patient met criteria for the metabolic syndrome, and then they were asked whether aspirin, lipid-lowering therapy, or anti-hypertensive therapy were indicated for the patient. They were also asked if any additional diagnostic tests were indicated.

[Table 13 is located at the end of the paper]

Assessment: As noted, participants were asked to state their recommendations for preventive interventions in each scenario. Questions formatted as Likert-type
scales were used to assess their level of comfort with the guidelines for CHD prevention and their general impressions regarding the utility of Framingham-based global risk scores. Post-graduate year and type of residency program were the only demographic information collected.

**Blinding:** The investigators were blinded to group assignment until after the questionnaire had been completed. Subjects were blinded to study design and they were not told that some questionnaires include risk scores and others did not.

**Endpoints:**

**Primary Endpoint:**

The pre-specified primary endpoint was a comparison of the mean number of correct preventive recommendations in each group. “Correct” answers are those that are in compliance with the recommendations set forth in the JNC-VII, NCEP-ATPIII, and USPSTF guidelines. For each participant an overall questionnaire score was calculated using the following formula: Overall score = (number of correct recommendations regarding aspirin, cholesterol medication, and antihypertensive medication/Total number of possible recommendations) x 100. Since each questionnaire had ten cases and subjects were required to recommend for or against preventive intervention in three domains (aspirin chemoprophylaxis, cholesterol-lowering medication, and antihypertensive medication) in each case, a score of 100% indicates that the subject made “correct” recommendations for all 30 questions. Scores for the individual
guideline areas were calculated in the same manner (denominator for these calculations was 10).

**Secondary Endpoints:**

Secondary endpoints included a comparison between Groups 1 and 2 for each of the following:

- The proportion of subjects who correctly make all of the appropriate recommendations for each of the individual cases.

- The proportion of subjects who recommend appropriate lipid-lowering therapy for all patients who need it.

- The proportion of subjects who appropriately recommend aspirin use in all those for whom it is indicated.

- The proportion of subjects who appropriately recommend cessation of tobacco use in all those for whom it is indicated.

- The proportion of subjects who correctly identify \( \leq 130/80 \) mmHg as the goal blood pressure in diabetic patients.

- The proportion of subjects who correctly categorize each patient’s 10-year CHD risk on a scale of low-intermediate-high.

- The proportion of subjects who correctly recognize patients with the metabolic syndrome.

- The proportion of subjects that recommend hs-CRP testing in accordance with AHA/CDC recommendations.\(^49\)
- The proportion of subjects who recommend a variety of other risk-stratification tests including coronary electron-beam computed tomography, exercise treadmill testing, and ankle-brachial index.

- An assessment of resident attitudes towards CHD risk scores.

**Statistical Analysis:**

Descriptive statistics were used to describe participant’s baseline characteristics and perceptions of CHD risk scores. For the primary endpoint, the mean number of correct preventive recommendations in each group was compared using a t-test. The proportion of correct responses for the categorical endpoints (as outlined in the secondary endpoints section) in each group was compared using a Chi-square test. An alpha level of 0.05 was considered statistically significant.

**Sample Size Calculation:**

The literature suggests that 35-58% of treatments are prescribed in accordance with guidelines.\textsuperscript{20-23} Therefore it was assumed that the control group would reflect this pattern and would have an adherence with guidelines (as measured by mean questionnaire score) of approximately 45%. We estimated that an increase of $\geq 15\%$ would be clinically meaningful. No data on the standard deviation for questionnaire scores is available; however a small study examining the effect of documented risk scores on physician prescribing practices found a standard deviation (SD) of $\sim 30\%$ for the mean change in level of prescribing of different
medication classes. Accepting a two-sided alpha level of 0.05 and power of 0.80, we estimate that a sample size of 126 (63 per group) is required to detect a difference in the primary endpoint between the groups. (Calculation performed with Stata 8.0 for Windows; Stata Corporation, College Station, TX)
Results

To date 71 subjects have taken part in the study. The baseline characteristics of the subjects are shown in Table 14. The only significant difference between the groups was that the subjects in Group 2 reported a slightly higher comfort level with the guidelines for using aspirin in primary prevention.

Table 14: Baseline Subject Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(Risk Score)</td>
<td>(No Risk Score)</td>
<td>N = 71</td>
</tr>
<tr>
<td></td>
<td>N = 38</td>
<td>N = 33</td>
<td></td>
</tr>
<tr>
<td>Site</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>UNC</td>
<td>12 (31.6)</td>
<td>11 (33.3)</td>
<td>23 (32.4)</td>
</tr>
<tr>
<td>Duke</td>
<td>26 (68.4)</td>
<td>22 (66.7)</td>
<td>48 (67.6)</td>
</tr>
<tr>
<td>Type of Residency (N [%])</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internal Medicine</td>
<td>37 (97.4)</td>
<td>31 (94.0)</td>
<td>68 (95.8)</td>
</tr>
<tr>
<td>Med/Peds</td>
<td>1 (2.6)</td>
<td>2 (6.0)</td>
<td>3 (4.2)</td>
</tr>
<tr>
<td>Family Medicine</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Post-Graduate Year (N [%])</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>16 (42.1)</td>
<td>12 (36.4)</td>
<td>28 (39.4)</td>
</tr>
<tr>
<td>2</td>
<td>9 (23.7)</td>
<td>9 (27.3)</td>
<td>18 (25.4)</td>
</tr>
<tr>
<td>3</td>
<td>11 (29.0)</td>
<td>10 (30.3)</td>
<td>21 (29.6)</td>
</tr>
<tr>
<td>4</td>
<td>2 (5.3)</td>
<td>2 (6.1)</td>
<td>4 (5.6)</td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Level of Comfort with Cholesterol Guidelines (N [%])</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very Comfortable</td>
<td>10 (26.3)</td>
<td>16 (48.5)</td>
<td>26 (36.6)</td>
</tr>
<tr>
<td>Somewhat Comfortable</td>
<td>26 (68.4)</td>
<td>13 (39.4)</td>
<td>39 (54.9)</td>
</tr>
<tr>
<td>Somewhat Uncomfortable</td>
<td>1 (2.6)</td>
<td>4 (12.1)</td>
<td>5 (7.0)</td>
</tr>
<tr>
<td>Very Uncomfortable</td>
<td>1 (2.6)</td>
<td>0</td>
<td>1 (1.4)</td>
</tr>
<tr>
<td>Level of Comfort with Aspirin Guidelines (N [%])</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very Comfortable</td>
<td>8 (21.1)</td>
<td>13 (39.4)</td>
<td>21 (29.6)</td>
</tr>
<tr>
<td>Somewhat Comfortable</td>
<td>25 (65.8)</td>
<td>17 (51.5)</td>
<td>42 (59.2)</td>
</tr>
<tr>
<td>Somewhat Uncomfortable</td>
<td>4 (10.5)</td>
<td>3 (9.1)</td>
<td>7 (9.9)</td>
</tr>
<tr>
<td>Very Uncomfortable</td>
<td>1 (2.6)</td>
<td>0</td>
<td>1 (1.4)</td>
</tr>
<tr>
<td>Level of Comfort with Hypertension Guidelines (N [%])</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very Comfortable</td>
<td>13 (34.2)</td>
<td>16 (48.5)</td>
<td>29 (40.9)</td>
</tr>
<tr>
<td>Somewhat Comfortable</td>
<td>22 (57.9)</td>
<td>14 (42.4)</td>
<td>36 (50.7)</td>
</tr>
<tr>
<td>Somewhat Uncomfortable</td>
<td>2 (5.3)</td>
<td>2 (6.1)</td>
<td>4 (5.6)</td>
</tr>
<tr>
<td>Very Uncomfortable</td>
<td>1 (2.6)</td>
<td>1 (3.0)</td>
<td>2 (2.8)</td>
</tr>
</tbody>
</table>

P ≤ 0.05 for all between group comparisons except for comfort level with aspirin guidelines, for which P = 0.04.
**Risk Estimation**
Subjects in both groups had difficulty estimating the categorical short-term CHD risk of the case patients (Table 15). Overall, the subjects were only correct 56 ± 18% of the time. There was a trend toward more accurate risk assessment in the group that was given a risk score (64 ± 13% versus 47 ± 27%), but the difference did not reach statistical significance (p = 0.10).

[Table 15 is located at the end of the paper]

In addition to difficulty with risk estimation, the subjects were inconsistent in the way that they categorized CHD risk (Table 16). Specifically, the categorical risk level (i.e. low, moderate, high risk) selected by the subjects only correlated with the numeric risk estimate 64 ± 11% of the time. There was no difference between the two groups as to the correct matching of categorical and numeric risk estimates (p = 0.22).

[Table 16 is located at the end of the paper]

**Appropriateness of Decisions**
Overall, subjects made guideline-appropriate decisions about the three risk-reducing interventions 72% of the time (Table 17). There was a trend toward a greater proportion of correct answers in patients who received global CHD risk scores, however this trend did not reach statistical significance. When we examined each intervention separately, the only significant difference between the groups was seen with decisions about aspirin therapy. The subjects who received
global CHD risk scores were significantly more likely to make correct decisions about aspirin therapy than were the subjects who did not receive risk scores (Table 17).

**Table 17: Proportion of Guideline-Appropriate Recommendations**

<table>
<thead>
<tr>
<th>Endpoint</th>
<th>Overall</th>
<th>Group 1 (Risk Score)</th>
<th>Group 2 (No Risk Score)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 71</td>
<td>N = 38</td>
<td>N = 33</td>
<td></td>
</tr>
<tr>
<td>Overall Score*</td>
<td>71.7 (8.8)</td>
<td>73.1 (7.5)</td>
<td>70.2 (10.0)</td>
<td>0.174</td>
</tr>
<tr>
<td>Aspirin Score*</td>
<td>68.2 (15.4)</td>
<td>71.8 (16.0)</td>
<td>63.9 (13.7)</td>
<td>0.030</td>
</tr>
<tr>
<td>Cholesterol Score*</td>
<td>68.5 (12.5)</td>
<td>68.7 (13.0)</td>
<td>68.2 (12.1)</td>
<td>0.867</td>
</tr>
<tr>
<td>Hypertension Score*</td>
<td>78.6 (18.9)</td>
<td>78.7 (18.0)</td>
<td>78.5 (20.2)</td>
<td>0.965</td>
</tr>
</tbody>
</table>

All data are presented as mean (SD).

* Overall score = (# of correct recommendations regarding aspirin, cholesterol medication, and anti-hypertensive medication/Total number of possible recommendations) x 100. Each questionnaire had ten cases and subjects were required to recommend for or against aspirin chemoprophylaxis, cholesterol-lowering medication, and anti-hypertensive medication in each case. A score of 100% indicates that subjects made correct (guideline-directed) recommendations for all 30 questions. Scores for the individual guideline areas were calculated in the same manner (denominator for these calculations was 10).

For all cases save #1 and #2, less than 50% of subjects were able to make correct decisions about all three CHD preventive interventions (i.e. aspirin, blood pressure lowering, cholesterol lowering) simultaneously (Table 18). Overall, provision of a risk score was not associated with an increased likelihood of making correct (p = 0.91).

[Table 18 is located at the end of the paper]

**Tables 19-21** provide a case by case listing of the proportion of subjects who made correct decisions for each CHD preventive intervention. For the majority of cases there was no difference between those who received risk scores and those
who did not. However, significant differences were seen for cases 8 (aspirin and lipid-lowering therapy) and 9 (aspirin).

[Tables 19-21 are located at the end of the paper]

Identification of the Metabolic Syndrome
Three cases met NCEP-ATP III criteria for the diagnosis of the metabolic syndrome. Only 23% of subjects correctly identified these cases (Table 22). There was no difference between groups 1 and 2 \( (p = 0.91) \).

<table>
<thead>
<tr>
<th>Case</th>
<th>Overall</th>
<th>Group 1 (Risk Score)</th>
<th>Group 2 (No Risk Score)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>35.7</td>
<td>39.5</td>
<td>31.3</td>
<td>0.474</td>
</tr>
<tr>
<td>3</td>
<td>8.6</td>
<td>10.8</td>
<td>6.1</td>
<td>0.479</td>
</tr>
<tr>
<td>7</td>
<td>25.7</td>
<td>21.6</td>
<td>30.3</td>
<td>0.407</td>
</tr>
</tbody>
</table>

All values represent the proportion of subjects who correctly identified the metabolic syndrome in the patient’s who met NCEP-ATP III criteria for the metabolic syndrome.

Additional Diagnostic Testing
Only a minority of respondents indicated that additional diagnostic testing was necessary prior to making treatment decisions. Table 23 lists the proportion of subjects that indicated that further testing was necessary for each case.
Table 23: Frequency of Additional Diagnostic Testing

<table>
<thead>
<tr>
<th>Case # (Risk Level)</th>
<th>CRP</th>
<th>EBCT</th>
<th>ETT</th>
<th>ABI</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (intermediate)</td>
<td>8.5</td>
<td>1.4</td>
<td>25.3</td>
<td>8.5</td>
</tr>
<tr>
<td>2 (high)</td>
<td>7.0</td>
<td>0</td>
<td>18.3</td>
<td>5.6</td>
</tr>
<tr>
<td>3 (low)</td>
<td>7.0</td>
<td>0</td>
<td>9.9</td>
<td>0</td>
</tr>
<tr>
<td>4 (high)</td>
<td>8.5</td>
<td>0</td>
<td>15.4</td>
<td>2.8</td>
</tr>
<tr>
<td>5 (low)</td>
<td>2.8</td>
<td>0</td>
<td>7.0</td>
<td>14.1</td>
</tr>
<tr>
<td>6 (intermediate)</td>
<td>12.7</td>
<td>0</td>
<td>11.2</td>
<td>2.8</td>
</tr>
<tr>
<td>7 (low)</td>
<td>7.0</td>
<td>0</td>
<td>2.8</td>
<td>2.8</td>
</tr>
<tr>
<td>8 (intermediate)</td>
<td>7.0</td>
<td>0</td>
<td>15.5</td>
<td>2.8</td>
</tr>
<tr>
<td>9 (low)</td>
<td>11.3</td>
<td>2.8</td>
<td>11.3</td>
<td>4.2</td>
</tr>
<tr>
<td>10 (low)</td>
<td>4.2</td>
<td>1.4</td>
<td>7.0</td>
<td>4.2</td>
</tr>
<tr>
<td><strong>Overall</strong></td>
<td>7.6</td>
<td>0.6</td>
<td>12.4</td>
<td>4.8</td>
</tr>
</tbody>
</table>

Each value represents the proportion of subjects who recommended the diagnostic test for each case (N = 71)

**Attitudes Towards CHD Risk Scores**

Only 21 respondents (30%) correctly identified ≥20% as the cut-off value for high short-term risk. Thirty respondents (44%) thought that the risk cut off was lower, and 18 respondents (26.1%) thought it was higher (10-year risk ≥25-50%). Only 26 (37%) of respondents correctly identified ≤10% as the cut-off value for low short-term risk. Forty-two respondents (60%) thought the cut-off was lower, and two respondents (3%) thought the cut-off was higher.

23% of respondents reported that they never calculate a 10-year CHD risk score when making decisions about CHD prevention. Of the 77% of respondents who reported that they do calculate a risk score, only 1% “always” do so. Most respondents reported calculating a risk score “sometimes” (62%) or “most of the time” (13%).

Of those respondents who do calculate a risk score, the majority (69%) reported that they used a personal digital assistant (PDA) to do so. The rest either used a
computer/website (22%) or a paper chart (9%). When asked whether the calculation of a risk score had ever changed their initial perception of a patient’s CHD risk, 98% of respondents replied “sometimes” (80%) or “often” (18%). The majority (72%) also reported sharing the risk score with the patient.

When asked whether they agreed with the statement “I find CHD risk calculation useful”, 95% of respondents either agreed or strongly agreed. Similarly, 90% of respondents either agreed or strongly agreed that CHD risk calculation can help them to make better decisions about whether or not to recommend therapies to prevent CHD events. However, when asked whether they felt that CHD risk calculation saves time, only 44% of respondents agreed.

Among those respondents who do not routinely calculate a CHD risk score the most common reasons for not doing so are that they already document CHD risk factors and do not feel that calculating a risk score provides additional useful information (13%), it takes too long (21%), lack of convenient access to a risk score calculator (17%), lack of familiarity with risk scores (8%), and a belief that the calculated score is not valid for their patient population (1%).

Discussion

As with previous studies, the participants in this study demonstrated considerable difficulty with CHD risk estimation. In fact, whether or not a global risk score was provided subjects did little better than chance when they attempted to
estimate categorical CHD risk. It is particularly interesting that there was a demonstrable lack of knowledge as to the definitions of low, intermediate, and high risk. When asked about the cut-offs for each of the risk levels, only approximately one third of the subjects were able to correctly identify the traditionally accepted cut-offs. This confusion about risk levels was also apparent in the fact that subjects often did not correctly match their numeric risk estimate with the correct categorical risk level. For example, a subject might state that a given patient was at “high” CHD risk, but then would provide a numeric risk estimate well below 20%. This confusion was also true whether or not a risk score was provided and was independent of the patient’s true level of risk.

Since an understanding of the definitions of the different levels of short-term CHD risk is a prerequisite for proper interpretation of a global CHD risk score, it is not surprising that a greater difference was not seen between the groups in terms of the guideline-appropriateness of participant decisions. The fact that participants who received global risk scores did just as poorly at estimating categorical level of risk as participants who did not receive risk scores, suggests that much of the information provided by the global risk score was not apparent to the participants. In theory, if participants understood the definitions of the risk categories, 100% of the subjects who received global CHD risk information should have made correct categorical risk estimates.
This apparent lack of understanding of global risk score information creates difficulty in interpreting the preliminary results of this study. Although the use of global CHD risk scores appears to be associated with better decisions about aspirin chemoprophylaxis and a trend towards overall increased appropriateness of decisions about interventions to prevent CHD, the magnitude of this benefit is likely masked by the participant's seeming inability to correctly interpret the risk score information.

Of the three preventive interventions explored in this study, the guidelines for aspirin therapy are the most dependent on CHD risk assessment, and therefore it is not surprising that the effect of risk score calculation is most prominent for this domain. However, the current NCEP-ATP III guidelines stipulate that risk calculation should be a part of the evaluation of all patients with ≥2 risk factors, and therefore the global risk score should have been useful in seven of the ten cases. In particular, in both cases #2 and #4 the global risk score suggests that a lower LDL-C goal should be targeted. Despite this, there was no effect apparent in the group that was provided with a risk score. This, along with the fact that subjects often recommended aspirin in cases where it was clearly not indicated by the risk score (cases #5, 7, 9, and 10) also suggests a general lack of familiarity or understanding of current guidelines for the primary prevention of CHD. This is apparent despite the fact that the majority of participants indicated that they felt "somewhat" or "very" comfortable with these guidelines. Given the general lack of understanding of how to interpret risk score information, it is somewhat
surprising that 77% of respondents reported calculating a risk score on at least some occasions, and that >90% of those who do so find CHD risk calculation useful.

This lack of full familiarity with guidelines is also apparent in the poor identification of the metabolic syndrome and in the way that additional diagnostic tests were ordered. Although three of the cases met the NCEP-ATP III criteria for the metabolic syndrome, the majority of subjects were not able to identify these cases. Additionally, although only a minority of subjects requested additional diagnostic testing, the circumstances in which the tests were ordered were often inconsistent. For example, although the AHA/CDC guideline only suggests hs-CRP testing for individuals at intermediate risk, subjects were equally likely to order it for high and low risk patients.

Given that only 71 physicians have taken part so far, no firm conclusions can yet be drawn from this data. Despite this, the trends that have been briefly discussed above do suggest that future studies that aim to examine the usefulness of global risk scores may benefit from providing participants with greater background information about current guidelines and the interpretation of risk scores. As with any tool, it is difficult to study the potential benefits of global risk scores if the participants in the study do not fully grasp how to use them.
Additional Figures
Figure 1:

The Continuum of Cardiovascular Disease Risk and Prevention

No Risk Factors → Accumulation of Risk Factors → Risk For CVD Events

Low → High

No atherosclerosis

Primordial Prevention → Primary Prevention → Secondary Prevention → Tertiary Prevention

Healthy Diet
Physical Activity
Tobacco-use Prevention

Aspirin for those with >6% 10-year CHD risk
Tobacco-use cessation
Screening and Treatment for:
- Hypertension
- Hyperlipidemia
- Diabetes
- Obesity

As risk increases, more aggressive treatment is indicated

Continue throughout Patient's Lifetime
Table 2: Estimates of benefits and harms of aspirin therapy given for 5 years to 1,000 individuals with various levels of baseline risk for coronary heart disease

<table>
<thead>
<tr>
<th>Benefits and harms</th>
<th>Baseline risk for coronary heart disease over 5 years†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1%</td>
</tr>
<tr>
<td>Total mortality</td>
<td>No effect</td>
</tr>
<tr>
<td>CHD events†</td>
<td>1-4 avoided</td>
</tr>
<tr>
<td>Hemorrhagic strokes**</td>
<td>0-2 caused</td>
</tr>
<tr>
<td>Major gastrointestinal bleeding events+++</td>
<td>2-4 caused</td>
</tr>
</tbody>
</table>

Table 4: Relative Risk of CHD Events Associated with the Various CHD risk factors

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Men RR</th>
<th>95% CI</th>
<th>Women RR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>1.05</td>
<td>1.04-1.06</td>
<td>1.04</td>
<td>1.03-1.06</td>
</tr>
<tr>
<td>BP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>normal</td>
<td>1</td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>High nl</td>
<td>1.32</td>
<td>0.98-1.78</td>
<td>1.34</td>
<td>0.88-2.05</td>
</tr>
<tr>
<td>Stage 1</td>
<td>1.73</td>
<td>1.32-2.26</td>
<td>1.75</td>
<td>1.21-2.54</td>
</tr>
<tr>
<td>Stage 2</td>
<td>1.92</td>
<td>1.42-2.59</td>
<td>2.19</td>
<td>1.46-3.27</td>
</tr>
<tr>
<td>Smoker</td>
<td>1.71</td>
<td>1.39-2.10</td>
<td>1.49</td>
<td>1.13-1.97</td>
</tr>
<tr>
<td>Diabetic</td>
<td>1.47</td>
<td>1.04-2.08</td>
<td>1.80</td>
<td>1.18-2.74</td>
</tr>
<tr>
<td>LDL-C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;130</td>
<td>1</td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>130-159</td>
<td>1.19</td>
<td>0.91-1.54</td>
<td>1.24</td>
<td>0.84-1.81</td>
</tr>
<tr>
<td>&gt;160</td>
<td>1.74</td>
<td>1.36-2.24</td>
<td>1.68</td>
<td>1.17-2.40</td>
</tr>
<tr>
<td>HDL-C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;35</td>
<td>1.46</td>
<td>1.15-1.85</td>
<td>2.08</td>
<td>1.33-325</td>
</tr>
<tr>
<td>35-59</td>
<td>1</td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>&gt;60</td>
<td>0.61</td>
<td>0.41-0.91</td>
<td>0.64</td>
<td>0.47-0.87</td>
</tr>
</tbody>
</table>


Table 5: Total CHD Risk by Age in Patients Without Other Risk Factors*

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age</th>
<th>30-34</th>
<th>35-39</th>
<th>40-44</th>
<th>45-49</th>
<th>50-54</th>
<th>55-59</th>
<th>60-64</th>
<th>65-69</th>
<th>70-74</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td></td>
<td>2%</td>
<td>3%</td>
<td>3%</td>
<td>4%</td>
<td>5%</td>
<td>7%</td>
<td>8%</td>
<td>10%</td>
<td>13%</td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td>&lt;2%</td>
<td>&lt;2%</td>
<td>2%</td>
<td>3%</td>
<td>5%</td>
<td>7%</td>
<td>8%</td>
<td>8%</td>
<td>8%</td>
</tr>
</tbody>
</table>

*10-year risk of total CHD for a non-smoking person without diabetes and with blood pressure <120/80 mmHg, total cholesterol <200 mg/dl, HDL cholesterol ≥55mg/dl. Adapted from Grundy et al. Assessment of cardiovascular risk by use of multiple-risk-factor assessment equations: a statement for healthcare professionals from the American Heart Association and the American College of Cardiology. Circulation. 1999;100(13):1481-1492.
<table>
<thead>
<tr>
<th>10-Year CHD Risk*</th>
<th>Clinical Implication</th>
<th>Preventive Strategy</th>
<th>Interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;6-10%</td>
<td>No definitive treatment necessary.</td>
<td>Encourage a healthy lifestyle.</td>
<td>- Therapeutic lifestyle changes</td>
</tr>
<tr>
<td>Low short-term risk.</td>
<td></td>
<td>Raise awareness that risk factors imply higher long-term risk of CHD</td>
<td>- Consider aspirin if risk is ≥6%.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Treat hypertension to keep blood pressure &lt;140/90.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Treat lipids per NCEP-ATP III guidelines</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Smoking cessation.</td>
</tr>
<tr>
<td>10-20%</td>
<td>These patients may need further risk stratification to guide therapy.</td>
<td>Patient-centered approach.</td>
<td>- Therapeutic lifestyle changes</td>
</tr>
<tr>
<td>Intermediate short-term risk.</td>
<td></td>
<td>Since is not clear whether more aggressive treatment is beneficial, decisions about treatment need to reflect the patient’s values.</td>
<td>- Daily aspirin.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Treat hypertension to keep blood pressure &lt;140/90.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Treat lipids per NCEP-ATP III guidelines</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Smoking cessation.</td>
</tr>
<tr>
<td>≥20%</td>
<td>This level of risk is the same as someone who already has known CHD.</td>
<td>Prompt and aggressive risk factor reduction</td>
<td>- Therapeutic lifestyle changes</td>
</tr>
<tr>
<td>High short-term risk (CHD equivalent)</td>
<td></td>
<td></td>
<td>- Daily aspirin.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Treat lipids with goal of LDL-C &lt; 100mg/dl.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Tight control of blood pressure. If diabetic, target is &lt;130/80.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Smoking cessation.</td>
</tr>
</tbody>
</table>

See text for discussion.
Table 7: Proposed Novel Risk Markers and Screening Tests for CVD

<table>
<thead>
<tr>
<th>Inflammatory Markers</th>
<th>Other Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>C-reactive protein</td>
<td>Homocysteine</td>
</tr>
<tr>
<td>Interleukin-6 (IL-6)</td>
<td>Brain natriuretic peptide (BNP)</td>
</tr>
<tr>
<td>Serum amyloid A</td>
<td>Lipoprotein-associated phospholipase A(2)</td>
</tr>
<tr>
<td>Soluble CD-40 ligand</td>
<td>Microalbuminuria</td>
</tr>
<tr>
<td>Leukocyte count</td>
<td>PAI-1 genotype</td>
</tr>
<tr>
<td>Vascular and cellular adhesion molecules</td>
<td>Angiotensin-converting enzyme genotype</td>
</tr>
<tr>
<td></td>
<td>ApoE genotype</td>
</tr>
<tr>
<td></td>
<td>Infectious agents: <em>Cytomegalovirus</em>, <em>Chlamydia</em></td>
</tr>
<tr>
<td></td>
<td><em>pneumonia</em>, <em>Helicobacter pylori</em>, <em>Herpes simplex virus</em></td>
</tr>
<tr>
<td></td>
<td>Psychosocial factors</td>
</tr>
<tr>
<td>Platelet-Related Factors</td>
<td></td>
</tr>
<tr>
<td>Platelet aggregation</td>
<td></td>
</tr>
<tr>
<td>Platelet activity</td>
<td></td>
</tr>
<tr>
<td>Platelet size and volume</td>
<td></td>
</tr>
<tr>
<td>Lipid-Related Factors</td>
<td></td>
</tr>
<tr>
<td>Small dense LDL</td>
<td></td>
</tr>
<tr>
<td>Lipoprotein (a)</td>
<td></td>
</tr>
<tr>
<td>Remnant lipoproteins</td>
<td></td>
</tr>
<tr>
<td>Apolipoproteins A and B</td>
<td></td>
</tr>
<tr>
<td>HDL subtypes</td>
<td></td>
</tr>
<tr>
<td>Oxidized LDL</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Screening Tests</td>
</tr>
<tr>
<td></td>
<td>Ankle-brachial index (ABI)</td>
</tr>
<tr>
<td></td>
<td>Carotid B-mode ultrasound to measure intima-media</td>
</tr>
<tr>
<td></td>
<td>thickness</td>
</tr>
<tr>
<td></td>
<td>Coronary calcium scoring with electron-beam computed</td>
</tr>
<tr>
<td></td>
<td>tomography (EBCT)</td>
</tr>
<tr>
<td></td>
<td>Brachial artery reactivity testing</td>
</tr>
<tr>
<td></td>
<td>Cardiovascular magnetic resonance imaging (CMR)</td>
</tr>
<tr>
<td></td>
<td>Arterial elasticity testing</td>
</tr>
</tbody>
</table>
### Table 10: Characteristics of Studies Addressing Clinical Benefits of Global Risk Scores

<table>
<thead>
<tr>
<th>Study/Research Objective</th>
<th>Design &amp; Setting</th>
<th>Calculated Patient Level of CHD Risk</th>
<th>Intervention</th>
<th>Endpoints</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hall et al.²³ 2003</td>
<td>RCT Diabetes Clinic, UK</td>
<td>13% Low 35% Mod 52% High</td>
<td>Patients alternately allocated to the experimental or control group. Experimental group patients had their risk score clearly documented on the front of the chart. Control patients did not.</td>
<td>(1) Change in treatment of DM; (2) Prescription of lipid-lowering or antihypertensive drugs, (3) Referral to a dietician</td>
<td>Overall there were no differences in the main outcomes. However, among the high risk patients (&gt;20% 5 year risk) those in the experimental group were more likely to be prescribed CV drugs (p&lt;0.02)</td>
</tr>
<tr>
<td>Ramachandran et al.²⁰ 2000</td>
<td>Cohort study General Practices, UK</td>
<td>Mean 10-year risk: 28.9%</td>
<td>Postal Questionnaires containing 20 patient case scenarios. GPs were asked to indicate whether lipid-lowering medication was indicated, and they were asked what method they used in assessing the patients CHD risk.</td>
<td>Proportion of correct responses based on UK guidelines (i.e. therapy indicated for those with a 10-year CHD risk ≥30%).</td>
<td>There was no difference in the appropriateness of decision-making between GPs who used clinical perception as the sole means of risk assessment and those who calculated CHD risk (p=0.21).</td>
</tr>
<tr>
<td>Lowenstein et al.²² 1998</td>
<td>RCT General practices, Canada</td>
<td>63% “high” risk</td>
<td>Physicians were randomized to either the profile or control group. Profile group received computerized risk report.</td>
<td>(1) The likelihood of returning for follow-up at 3 months, (2) Change in risk factors over 3 months</td>
<td>(1) The likelihood of high-risk patients returning for follow-up was significantly greater in the profile group. (2) Profile group patients had significantly greater reductions in lipid values and calculated 8-yr CHD risk. No difference for other risk factors.</td>
</tr>
</tbody>
</table>

Continued on next page
<table>
<thead>
<tr>
<th>Study/Research Objective</th>
<th>Design &amp; Setting</th>
<th>Calculated Patient Level of CHD Risk$^1$</th>
<th>Intervention</th>
<th>Endpoints</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Montgomery et al.\textsuperscript{71} (2000)</td>
<td>RCT General practices, UK</td>
<td>Mean 5-year CHD risk: 18.5%</td>
<td>Practices were randomized to one of 3 arms: (1) computer-based clinical decision support system &amp; CHD risk chart, (2) CHD risk chart alone, (3) usual care</td>
<td>(1) % of patients in each group with a 5-yr CHD risk $\geq 10%$, (2) Blood pressure, (3) Prescribing of CVD drugs.</td>
<td>(1) No difference between groups as to the proportion of patients still at high-risk at 1 year. (2) Risk calculation was associated with lower mean systolic blood pressure at 12 months and with an increased likelihood of prescription of CV drugs ($p&lt;0.01$).</td>
</tr>
</tbody>
</table>

$^1$ As defined by the study authors.
### Table 11: Characteristics Studies Addressing Harms of Global Risk Scores

<table>
<thead>
<tr>
<th>Study/Research Objective</th>
<th>Design &amp; Setting</th>
<th>Calculated Patient Level of CHD Risk</th>
<th>Intervention</th>
<th>Endpoints</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Connelly et al. 1998</td>
<td>Cohort General practices; UK</td>
<td>81.5% Low/Mod risk; 18.5% High risk</td>
<td>Health screening including CHD risk calculation</td>
<td>Scores on the health questionnaire at 3 months follow-up.</td>
<td>Men who were labeled as either high or low risk showed a decrease in their psychological symptoms after labeling. However, men who were labeled &quot;moderate&quot; risk actually had an increase psychological symptoms to &quot;case&quot; levels.</td>
</tr>
<tr>
<td>N = 5772 patients</td>
<td>To identify the psychological impact of labeling men as having above average risk for CHD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| Martea et al. 1996 | RCT General practices; UK | Not given | Participants randomized to a CHD screening group or a control group. | Perceptions of health, the risk of suffering a heart attack, and the ability to reduce that risk at 1 year. | No difference between groups. |
| N = 2984 married couples | To determine the whether a population-based intervention program to reduce CVD raises concerns about health or undermines a belief in the ability to reduce risk | | | |

N reflects number of participants enrolled, which is not necessarily the same as the number who completed follow-up. RCT = Randomized Controlled Trial. 

As defined by the study authors.
### Table 12: Summary of Quality Ratings For Studies Included in the Review

<table>
<thead>
<tr>
<th>Study Design Rating</th>
<th>Internal Validity Rating</th>
<th>Issues with Internal Validity</th>
<th>Study Assessing Benefits</th>
</tr>
</thead>
</table>
| Hall et al.\textsuperscript{73} 2003 | I | Fair | - Inadequate blinding  
- No information on baseline patient characteristics – no way to assess adequacy of randomization or for confounding  
- Lack of treatment details  
- Only 6 physicians included | Fair | - Specialty clinic population.  
- No information on baseline patient characteristics. |
| Lowensteyn et al.\textsuperscript{72} 1998 | I | Fair | - Inadequate blinding  
- Limited baseline patient data  
- Physician groups not similar at baseline  
- Only physicians were randomized; not patients  
- Patients selected by physicians – potential for selection bias  
- Lack of details on patients screened but not enrolled  
- Substantial number of physicians did not enroll patients in study  
- High patient drop-out rate  
- Lack of treatment details | Fair | - Mechanism of obtaining risk score not reflective of actual practice  
- Limited baseline patient data |
| Montgomery et al.\textsuperscript{71} 2000 | I | Fair | - Not blinded  
- Lack of treatment details  
- The manner that risk charts were used was not clearly described | Fair | - Not entirely a primary prevention trial  
- Lack of treatment details  
- The manner that risk charts were used was not clearly described |

Continued on next page
Table 12 (Continued): Summary of Quality Ratings For Studies Included in the Review

<table>
<thead>
<tr>
<th>Study</th>
<th>Study Design Rating</th>
<th>Internal Validity Rating</th>
<th>Issues with Internal Validity</th>
<th>External Validity Rating</th>
<th>Issues with External Validity</th>
</tr>
</thead>
</table>
| Ramachandran et al.\textsuperscript{69}, 2000 | II-2 | Poor | - Lack of control group  
- Non-randomized  
- Not blinded  
- Extremely low response rate  
- Questionnaire not validated | Poor | - Questionnaire-based |
| Connelly et al.\textsuperscript{70}, 1998 | II-2 | Fair | - Lack of control group  
- Not blinded  
- Very little baseline patient information provided  
- Lack of details about nature of risk score  
- Lack of treatment details | Fair | - Very little baseline patient information provided  
- Clinical relevance of psychological endpoints is unclear  
- Lack of treatment details |
| Marteau et al.\textsuperscript{68}, 1996 | I | Poor | - Very limited description of study procedures  
- Not clear if blinded  
- No baseline patient information provided | Poor | - No baseline patient information provided  
- Clinical relevance of psychological endpoints is unclear |

I [randomized controlled trial], II-1 [controlled trial without randomization], II-2 [cohort or case-control study], II-3 [multiple time-series with or without intervention, or dramatic results from an uncontrolled experiment], III [expert opinion, case reports, descriptive studies]. See text for description of criteria for ratings of good, fair, and poor.
**Table 13: Cases Included on Risk Score Study Questionnaire**

<table>
<thead>
<tr>
<th>Case</th>
<th># of NCEP Major Risk Factors</th>
<th>10-year CHD Risk</th>
<th>Aspirin recommended</th>
<th>Lipid-Lowering recommended</th>
<th>Anti-hypertensive recommended</th>
<th>Rationale for Inclusion in Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. 58 year old woman  &lt;br&gt; Type II diabetes  &lt;br&gt; Non-smoker  &lt;br&gt; BP: 134/88  &lt;br&gt; HbA1c: 7%  &lt;br&gt; Waist: 38&quot;  &lt;br&gt; T. Chol: 201 mg/dl  &lt;br&gt; LDL-C: 129 mg/dl  &lt;br&gt; HDL-C: 42 mg/dl  &lt;br&gt; TG: 152 mg/dl</td>
<td>3</td>
<td>15%</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Recognize DM as a CHD equivalent; recognized lower blood pressure goal for diabetics.</td>
</tr>
<tr>
<td>2. 49 year old man  &lt;br&gt; Smoker  &lt;br&gt; BP: 134/88  &lt;br&gt; FBG: 105 mg/dl  &lt;br&gt; Waist: 41&quot;  &lt;br&gt; T. Chol: 242 mg/dl  &lt;br&gt; LDL-C: 178 mg/dl  &lt;br&gt; HDL-C: 34 mg/dl  &lt;br&gt; TG: 155 mg/dl</td>
<td>3</td>
<td>22%</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Recognize someone with multiple risk factors and high risk. Recognize the metabolic syndrome.</td>
</tr>
<tr>
<td>3. 62 year old woman  &lt;br&gt; Non-smoker  &lt;br&gt; On BP medication  &lt;br&gt; BP: 135/82  &lt;br&gt; FBG: 98 mg/dl  &lt;br&gt; Waist: 34&quot;  &lt;br&gt; T. Chol: 245 mg/dl  &lt;br&gt; LDL-C: 166 mg/dl  &lt;br&gt; HDL-C: 48 mg/dl  &lt;br&gt; TG: 155 mg/dl</td>
<td>2</td>
<td>6%</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Recognize someone with multiple risk factors but with low risk. Recognize the metabolic syndrome.</td>
</tr>
</tbody>
</table>

Continued on next page
Table 13 (Continued): Cases Included on Risk Score Study Questionnaire

<table>
<thead>
<tr>
<th>Case Description</th>
<th># of NCEP Major Risk Factors</th>
<th>10-year CHD Risk</th>
<th>Aspirin recommended</th>
<th>Lipid-Lowering recommended</th>
<th>Anti-hypertensive recommended</th>
<th>Rationale for Inclusion in Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>4. 72 year old man</td>
<td>2</td>
<td>24%</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Recognize the risk conferred by older age; especially in men.</td>
</tr>
<tr>
<td>Non-smoker</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BP: 142/82</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>On BP medication</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FBG: 108 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waist: 33”</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T. Chol: 200 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDL-C: 130 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL-C: 42 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TG: 140 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. 51 year old man</td>
<td>1</td>
<td>5%</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Recognize that not all middle-age men are high risk.</td>
</tr>
<tr>
<td>Non-smoker</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BP: 126/78</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FBG: 99 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waist: 33”</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T. Chol: 228 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDL-C: 150 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL-C: 55 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TG: 115 mg/dl</td>
<td></td>
<td></td>
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<td></td>
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<td></td>
</tr>
</tbody>
</table>

Continued on next page
Table 13 (Continued): Cases Included on Risk Score Study Questionnaire

<table>
<thead>
<tr>
<th>Case</th>
<th># of NCEP Major Risk Factors</th>
<th>10-year CHD Risk</th>
<th>Aspirin recommended</th>
<th>Lipid-Lowering recommended</th>
<th>Anti-hypertensive recommended</th>
<th>Rationale for Inclusion in Study</th>
</tr>
</thead>
</table>
| 6. 68 year old woman  
Smoker  
BP: 138/78  
On BP medication  
FBG: 99 mg/dl  
Waist: 32"  
T. Chol: 208 mg/dl  
LDL-C: 128 mg/dl  
HDL-C: 50 mg/dl  
TG: 150 mg/dl | 3 | 10% | Yes | No | No | Recognize someone with multiple risk factors but only intermediate risk. |
| 7. 38 year old woman  
Smoker  
BP: 136/85  
FBG: 108 mg/dl  
Waist: 36"  
T. Chol: 230 mg/dl  
LDL-C: 159 mg/dl  
HDL-C: 42 mg/dl  
TG: 145 mg/dl | 1 | 4% | No | No | No | Recognize someone with a risk factor but with low short-term risk. Recognize the metabolic syndrome. |
| 8. 45 year old man  
Smoker  
BP: 136/84  
FBG: 100 mg/dl  
Waist: 33"  
T. Chol: 228 mg/dl  
LDL-C: 162 mg/dl  
HDL-C: 43 mg/dl  
TG: 115 mg/dl | 2 | 14% | Yes | Yes | No | Recognize someone with multiple risk factors and intermediate risk. |

Continued on next page
**Table 13 (Continued): Cases Included on Risk Score Study Questionnaire**

<table>
<thead>
<tr>
<th>Case</th>
<th># of NCEP Major Risk Factors</th>
<th>10-year CHD Risk</th>
<th>Aspirin recommended</th>
<th>Lipid-Lowering recommended</th>
<th>Anti-hypertensive recommended</th>
<th>Rationale for Inclusion in Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>9.</td>
<td>41 year old woman</td>
<td>2</td>
<td>3%</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td>Smoker</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>BP: 128/77</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>FBG: 105 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Waist: 34&quot;</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>T. Chol: 210 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>LDL-C: 153 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>HDL-C: 42 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TG: 125 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mother had non-fatal MI at age 50</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td>70 year old woman</td>
<td>2</td>
<td>4%</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td>Smoker</td>
<td>but also has a negative risk factor</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>BP: 115/80</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>FBG: 100 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Waist: 31&quot;</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>T. Chol: 245 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>LDL-C: 157 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>HDL-C: 62 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TG: 128 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

BP = Blood pressure; FBG = fasting blood glucose; Waist = waist circumference; T. chol = total cholesterol; TG = triglycerides.
Table 15: Proportion of Subjects Who Made a Correct Assessment of CHD Risk

<table>
<thead>
<tr>
<th>Case</th>
<th>Overall</th>
<th>Group 1 (Risk Score)</th>
<th>Group 2 (No Risk Score)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>41.2</td>
<td>42.1</td>
<td>49.8</td>
<td>0.861</td>
</tr>
<tr>
<td>2</td>
<td>52.1</td>
<td>55.3</td>
<td>48.5</td>
<td>0.569</td>
</tr>
<tr>
<td>3</td>
<td>47.1</td>
<td>59.5</td>
<td>33.3</td>
<td>0.029</td>
</tr>
<tr>
<td>4</td>
<td>34.3</td>
<td>54.0</td>
<td>12.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>5</td>
<td>84.5</td>
<td>81.6</td>
<td>87.9</td>
<td>0.464</td>
</tr>
<tr>
<td>6</td>
<td>77.1</td>
<td>79.0</td>
<td>75.0</td>
<td>0.695</td>
</tr>
<tr>
<td>7</td>
<td>73.2</td>
<td>76.3</td>
<td>69.7</td>
<td>0.530</td>
</tr>
<tr>
<td>8</td>
<td>70.4</td>
<td>73.7</td>
<td>66.7</td>
<td>0.518</td>
</tr>
<tr>
<td>9</td>
<td>31.0</td>
<td>50.0</td>
<td>9.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>10</td>
<td>44.3</td>
<td>70.3</td>
<td>15.2</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

All values represent the proportion of subjects who correctly identified the patient's categorical level of 10-year CHD risk (low - intermediate - high).
Table 16: Proportion of Subjects Who Correctly Correlated the Numeric Risk and the Risk Category

<table>
<thead>
<tr>
<th>Case</th>
<th>Overall</th>
<th>Group 1 (Risk Score)</th>
<th>Group 2 (No Risk Score)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>50.8</td>
<td>63.2</td>
<td>34.5</td>
<td>0.020</td>
</tr>
<tr>
<td>2</td>
<td>57.1</td>
<td>62.2</td>
<td>51.5</td>
<td>0.369</td>
</tr>
<tr>
<td>3</td>
<td>53.0</td>
<td>59.4</td>
<td>45.2</td>
<td>0.239</td>
</tr>
<tr>
<td>4</td>
<td>59.0</td>
<td>54.1</td>
<td>65.5</td>
<td>0.347</td>
</tr>
<tr>
<td>5</td>
<td>87.0</td>
<td>86.8</td>
<td>87.1</td>
<td>0.975</td>
</tr>
<tr>
<td>6</td>
<td>68.7</td>
<td>79.0</td>
<td>55.2</td>
<td>0.038</td>
</tr>
<tr>
<td>7</td>
<td>73.1</td>
<td>76.3</td>
<td>69.0</td>
<td>0.501</td>
</tr>
<tr>
<td>8</td>
<td>71.0</td>
<td>73.7</td>
<td>67.7</td>
<td>0.588</td>
</tr>
<tr>
<td>9</td>
<td>52.1</td>
<td>50.0</td>
<td>54.6</td>
<td>0.702</td>
</tr>
<tr>
<td>10</td>
<td>69.6</td>
<td>70.3</td>
<td>68.8</td>
<td>0.891</td>
</tr>
</tbody>
</table>

All values represent the proportion of subjects who correctly correlated the patient's categorical level of 10-year CHD risk (low – intermediate – high) with the corresponding numeric risk range (<10%, 10-19%, ≥20%). For Group 2 this signifies that the categorical risk level selected by the subject was correctly matched with an appropriate numeric risk. For Group 1, which was provided with a numeric risk score, this signifies that the subject correctly identified the categorical risk level that corresponded with the numeric estimate that was provided.
Table 18: Proportion of Subjects Who Made Correct Recommendations for All Three Preventive Interventions: Case-by-Case

<table>
<thead>
<tr>
<th>Case #</th>
<th>Overall N = 71</th>
<th>Group 1 (Risk Score) N = 38</th>
<th>Group 2 (No Risk Score) N = 33</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (intermediate)</td>
<td>78.9</td>
<td>76.3</td>
<td>81.8</td>
<td>0.571</td>
</tr>
<tr>
<td>2 (high)</td>
<td>50.7</td>
<td>44.7</td>
<td>57.6</td>
<td>0.280</td>
</tr>
<tr>
<td>3 (low)</td>
<td>31.0</td>
<td>21.1</td>
<td>42.4</td>
<td>0.052</td>
</tr>
<tr>
<td>4 (high)</td>
<td>28.2</td>
<td>26.3</td>
<td>30.3</td>
<td>0.710</td>
</tr>
<tr>
<td>5 (low)</td>
<td>45.1</td>
<td>47.4</td>
<td>42.4</td>
<td>0.676</td>
</tr>
<tr>
<td>6 (intermediate)</td>
<td>26.8</td>
<td>26.3</td>
<td>27.3</td>
<td>0.928</td>
</tr>
<tr>
<td>7 (low)</td>
<td>39.4</td>
<td>39.5</td>
<td>39.4</td>
<td>0.995</td>
</tr>
<tr>
<td>8 (intermediate)</td>
<td>32.4</td>
<td>42.1</td>
<td>21.2</td>
<td>0.061</td>
</tr>
<tr>
<td>9 (low)</td>
<td>16.9</td>
<td>26.3</td>
<td>6.1</td>
<td>0.023</td>
</tr>
<tr>
<td>10 (low)</td>
<td>19.7</td>
<td>23.7</td>
<td>15.2</td>
<td>0.367</td>
</tr>
</tbody>
</table>

All values represent the proportion of subjects who made correct (guideline-directed) decisions about aspirin therapy, lipid-lowering therapy, and anti-hypertensive therapy.
Table 19: Proportion of Subjects Who Made Correct Recommendations for Aspirin Therapy: Case-by-Case

<table>
<thead>
<tr>
<th>Case # (Risk Level)</th>
<th>Overall</th>
<th>Group 1 (Risk Score)</th>
<th>Group 2 (No Risk Score)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 71</td>
<td>N = 38</td>
<td>N = 33</td>
<td></td>
</tr>
<tr>
<td>1 (intermediate)</td>
<td>97.2</td>
<td>97.4</td>
<td>97.0</td>
<td>0.919</td>
</tr>
<tr>
<td>2 (high)</td>
<td>87.3</td>
<td>84.2</td>
<td>90.9</td>
<td>0.397</td>
</tr>
<tr>
<td>3 (low)</td>
<td>69.0</td>
<td>66.0</td>
<td>72.7</td>
<td>0.528</td>
</tr>
<tr>
<td>4 (high)</td>
<td>81.7</td>
<td>84.2</td>
<td>78.8</td>
<td>0.556</td>
</tr>
<tr>
<td>5 (low)</td>
<td>59.2</td>
<td>57.9</td>
<td>60.6</td>
<td>0.817</td>
</tr>
<tr>
<td>6 (intermediate)</td>
<td>77.5</td>
<td>79.0</td>
<td>75.8</td>
<td>0.748</td>
</tr>
<tr>
<td>7 (low)</td>
<td>71.8</td>
<td>73.7</td>
<td>69.7</td>
<td>0.710</td>
</tr>
<tr>
<td>8 (intermediate)</td>
<td>70.4</td>
<td>81.6</td>
<td>57.6</td>
<td>0.027</td>
</tr>
<tr>
<td>9 (low)</td>
<td>36.6</td>
<td>55.3</td>
<td>15.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>10 (low)</td>
<td>29.6</td>
<td>36.8</td>
<td>21.1</td>
<td>0.150</td>
</tr>
</tbody>
</table>

All values represent the proportion of subjects who made correct (guideline-directed) decisions about aspirin therapy.
Table 20: Proportion of Subjects Who Made Correct Recommendations for Lipid-Lowering Therapy: Case-by-Case

<table>
<thead>
<tr>
<th>Case # (Risk Level)</th>
<th>Overall N = 71</th>
<th>Group 1 (Risk Score) N = 38</th>
<th>Group 2 (No Risk Score) N = 33</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (intermediate)</td>
<td>94.4</td>
<td>92.1</td>
<td>97.0</td>
<td>0.375</td>
</tr>
<tr>
<td>2 (high)</td>
<td>92.6</td>
<td>94.7</td>
<td>90.9</td>
<td>0.530</td>
</tr>
<tr>
<td>3 (low)</td>
<td>64.8</td>
<td>57.9</td>
<td>72.7</td>
<td>0.192</td>
</tr>
<tr>
<td>4 (high)</td>
<td>35.2</td>
<td>36.8</td>
<td>33.3</td>
<td>0.758</td>
</tr>
<tr>
<td>5 (low)</td>
<td>71.8</td>
<td>71.1</td>
<td>72.7</td>
<td>0.876</td>
</tr>
<tr>
<td>6 (intermediate)</td>
<td>77.5</td>
<td>76.3</td>
<td>78.8</td>
<td>0.804</td>
</tr>
<tr>
<td>7 (low)</td>
<td>53.5</td>
<td>57.9</td>
<td>48.5</td>
<td>0.428</td>
</tr>
<tr>
<td>8 (intermediate)</td>
<td>80.3</td>
<td>89.5</td>
<td>69.7</td>
<td>0.037</td>
</tr>
<tr>
<td>9 (low)</td>
<td>64.8</td>
<td>57.9</td>
<td>72.7</td>
<td>0.192</td>
</tr>
<tr>
<td>10 (low)</td>
<td>49.3</td>
<td>52.6</td>
<td>45.5</td>
<td>0.546</td>
</tr>
</tbody>
</table>

All values represent the proportion of subjects who made correct (guideline-directed) decisions about lipid-lowering therapy.
Table 21: Proportion of Subjects Who Made Correct Recommendations for Antihypertensive Therapy: Case-by-Case

<table>
<thead>
<tr>
<th>Case # (Risk Level)</th>
<th>Overall N = 71</th>
<th>Group 1 (Risk Score) N = 38</th>
<th>Group 2 (No Risk Score) N = 33</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (intermediate)</td>
<td>88.7</td>
<td>86.8</td>
<td>90.9</td>
<td>0.589</td>
</tr>
<tr>
<td>2 (high)</td>
<td>61.9</td>
<td>60.5</td>
<td>63.6</td>
<td>0.788</td>
</tr>
<tr>
<td>3 (low)</td>
<td>74.8</td>
<td>71.1</td>
<td>78.8</td>
<td>0.455</td>
</tr>
<tr>
<td>4 (high)</td>
<td>93.0</td>
<td>97.4</td>
<td>87.9</td>
<td>0.119</td>
</tr>
<tr>
<td>5 (low)</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>-</td>
</tr>
<tr>
<td>6 (intermediate)</td>
<td>43.7</td>
<td>42.1</td>
<td>45.5</td>
<td>0.777</td>
</tr>
<tr>
<td>7 (low)</td>
<td>69.0</td>
<td>71.1</td>
<td>69.0</td>
<td>0.690</td>
</tr>
<tr>
<td>8 (intermediate)</td>
<td>60.6</td>
<td>60.5</td>
<td>60.6</td>
<td>0.995</td>
</tr>
<tr>
<td>9 (low)</td>
<td>97.2</td>
<td>100</td>
<td>93.9</td>
<td>0.124</td>
</tr>
<tr>
<td>10 (low)</td>
<td>97.2</td>
<td>97.4</td>
<td>97.0</td>
<td>0.919</td>
</tr>
</tbody>
</table>

All values represent the proportion of subjects who made correct (guideline-directed) decisions about antihypertensive therapy.
Appendices
## Appendix 1: Recommended Interventions for the Prevention of Cardiovascular Disease:

<table>
<thead>
<tr>
<th>Level of Prevention</th>
<th>Preventive Intervention</th>
<th>USPSTF/TFCPS Guidelines</th>
<th>Other Major Guidelines</th>
</tr>
</thead>
</table>
| **Primordial Prevention** | Physical Activity<sup>79</sup> | - Point-of-decision prompts  
- Community wide educational campaigns about the benefits of physical activity.  
- School-based physical education  
- Social support interventions in community settings  
- Individually-adapted health behavior change programs  
- Creation of or enhanced access to places for physical activity combined with informational outreach activities |  |
|                     | Nutrition                | - TFCPS Reviews are currently in progress<sup>88</sup>  
- USPSTF recommends intensive behavioral dietary counseling for adults with risk factors for CVD. (B)<sup>11</sup> |  |
|                     | Tobacco Prevention<sup>82</sup> | - Increase the price of tobacco products  
- Mass media campaigns  
- Smoking bans and restrictions |  |
| **Primary Prevention** | Screening for Hypertension | - Screen all adults aged 18 and older for HTN (A) | JNC VII<sup>11</sup>:  
- Screen all adults for HTN  
AHA<sup>1</sup>:  
- Screen q2 years beginning at age 20. |
|                     | Screening for Lipid Disorders | - Routinely screen men ≥35 years old and women ≥45 years old. Treat abnormal lipids in those who are at increased risk for CHD. (A)  
- Screen younger adults (men 20-35 years old and women 20-45 years old) if they have other CHD risk factors. (B) | NCEP-ATP III<sup>2</sup>:  
- Screen q5 years beginning at age 20.  
AHA<sup>10</sup>:  
- Screen q5 years beginning at age 20; q2 years if risk factors are present  
ADA<sup>44</sup>:  
- Screen adult diabetics annually. Screen biannually if lipid profiles is low risk. |
|                     | Type II Diabetes<sup>85, 86</sup> | - Screen all adults with HTN or hyperlipidemia for type II DM. (B)  
- The TFCPS strongly recommends a multi-component approach to healthcare delivery, including community-based diabetes self-management education as well as individual case management, for all persons with diabetes | AHA<sup>83</sup>:  
- Screen ALL adults with a FBG q5 years starting at age 20; q2 years if risk factors are present  
ADA<sup>47</sup>:  
- Consider screening q3-year in those ≥45 years old; particularly if BMI ≥25 kg/m<sup>2</sup>.  
Consider screening at a younger if overweight or other CVD risk factors are present. |
|                     | Aspirin                  | - Discuss the potential benefits and harms of aspirin chemoprevention with adults who are have a >6% 10-year risk for CHD (A) | AHA<sup>83</sup>:  
- Use ASA for those with a ≥10% 10-year CHD risk  
ADA<sup>85</sup>:  
- Use ASA for patients with type 2 diabetes who are ≥40 years old or who have CVD risk factors |
|                     | Screening for Obesity<sup>89</sup> | - Screen all adults for obesity (B)  
- Offer INTENSIVE counseling and behavioral interventions to promote sustained weight loss for OBESE adults. (B) | AHA<sup>83</sup>:  
- Record BMI and waist circ. at least q2 years.  
- Initiate a weight-management program for overweight and obese patients |
Smoking Cessation\textsuperscript{7, 82} - Screen all adults and provide tobacco cessation interventions for those who use tobacco products. (A)
- Screen all pregnant women and provide augmented pregnancy-tailed counseling to those who smoke. (A)
- The TFCPS strongly recommends multi-component interventions that include patient support, provider reminder systems and an educational component.
- Reducing patient out-of-pockets costs for effective cessation therapies is recommended by the TFCPS

AHA\textsuperscript{83}:
- Screen for tobacco use at every visit and advise all smokers to quit. Assist by counseling and arrange for follow-up, referral, and pharmacotherapy when appropriate.

Secondary & Tertiary Prevention

Interventions are similar to primary prevention, but treatment is more aggressive because of higher baseline risk for CVD events.

Only USPSTF recommendations with ratings of “A” or “B” are listed.

USPSTF = U.S. Preventive Services Task Force; TFCPS = Task Force on Community Preventive Services; AHA = American Heart Association; ADA = American Diabetes Association; NCEP-ATP III = National Cholesterol Education Program Adult Treatment Panel III; JNC 7 = The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; HTN = hypertension; FBG = fasting blood glucose; ASA = aspirin; BMI = body mass index;

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>The USPSTF strongly recommends that clinicians routinely provide [the service] to eligible patients. (The USPSTF found good evidence that [the service] improves important health outcomes and concludes that benefits substantially outweigh harms.)</td>
</tr>
<tr>
<td>B</td>
<td>The USPSTF recommends that clinicians routinely provide [the service] to eligible patients. (The USPSTF found at least fair evidence that [the service] improves important health outcomes and concludes that benefits outweigh harms.)</td>
</tr>
<tr>
<td>C</td>
<td>The USPSTF makes no recommendation for or against routine provision of [the service]. (The USPSTF found at least fair evidence that [the service] can improve health outcomes but concludes that the balance of benefits and harms is too close to justify a general recommendation.)</td>
</tr>
<tr>
<td>D</td>
<td>The USPSTF recommends against routinely providing [the service] to asymptomatic patients. (The USPSTF found at least fair evidence that [the service] is ineffective or that harms outweigh benefits.)</td>
</tr>
<tr>
<td>I</td>
<td>The USPSTF concludes that the evidence is insufficient to recommend for or against routinely providing [the service]. (Evidence that [the service] is effective is lacking, of poor quality, or conflicting and that the balance of benefits and harms cannot be determined.)</td>
</tr>
</tbody>
</table>
## Appendix 2: Scoring for Global Risk Assessment (Adjusted Framingham Scoring)

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Men</th>
<th>Women</th>
<th>Adding Up the Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;34</td>
<td>-1</td>
<td>-9</td>
<td></td>
</tr>
<tr>
<td>35–39</td>
<td>0</td>
<td>-4</td>
<td></td>
</tr>
<tr>
<td>40–44</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>45–49</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>50–54</td>
<td>3</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>55–59</td>
<td>4</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>60–64</td>
<td>5</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>65–69</td>
<td>6</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>70–74</td>
<td>7</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol, mg/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;160</td>
<td>-3</td>
<td>-2</td>
<td></td>
</tr>
<tr>
<td>169–199</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>200–239</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>240–279</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>≥280</td>
<td>3</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>HDL cholesterol, mg/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;35</td>
<td>2</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>35–44</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>45–49</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>50–59</td>
<td>-1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>≥60</td>
<td>-2</td>
<td>-3</td>
<td></td>
</tr>
<tr>
<td>Blood pressure, mm Hg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;120</td>
<td>0</td>
<td>-3</td>
<td></td>
</tr>
<tr>
<td>121–129</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>130–139</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>140–159</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>&gt;160</td>
<td>3</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Plasma glucose, mg/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;110</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>110–126</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>&gt;126</td>
<td>2</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Smoker</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>

Absolute Risk (Hard CHD), %

<table>
<thead>
<tr>
<th>Risk Points</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Diabetes</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>HDL</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Smoker</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

Adapted from Grundy38
**Case 1:** A 58 year old woman with type II diabetes. She is a life-long non-smoker. She is asymptomatic, and she has no history of CHD. Her calculated 10-year risk of CHD is 15%.

**Blood pressure:** 134/88  
**Hemoglobin A1c:** 7.0%  
**Waist circumference:** 38 inches  

**Lipid panel:**  
- **T. Chol:** 201 mg/dl  
- **LDL-C:** 129 mg/dl  
- **HDL-C:** 42 mg/dl  
- **TG:** 152 mg/dl

1.1: How would you categorize this patient’s risk of developing CHD over the next 10 years?

<table>
<thead>
<tr>
<th>1. Low</th>
<th>2. Intermediate</th>
<th>3. High</th>
</tr>
</thead>
</table>

1.2: Does this patient have the metabolic syndrome?

<table>
<thead>
<tr>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
</table>

Which of the following would you recommend? (Circle all that apply)

| 1.3: Therapeutic lifestyle changes?  
(Intensive patient oriented counseling on physical activity, diet, and, where indicated, smoking cessation) | No | Yes |
|---|---|---|

<table>
<thead>
<tr>
<th>1.4: Daily aspirin?</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>1.5: Lipid lowering medication?</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>1.6: Blood pressure lowering medication?</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
</table>

**Would you recommend any of the following diagnostic tests?**

<table>
<thead>
<tr>
<th>1.7: High sensitivity C-reactive protein (hs-CRP) testing?</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>1.8: Coronary calcium scoring with electron-beam computed tomography (EBCT)?</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>1.9: Exercise Treadmill Testing (ETT)?</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>1.10: Ankle-brachial index (ABI) measurement?</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
</table>


12. Table 102: Number of active physicians (MDs) and physician-to-population ratios by selected specialty, selected years 1970-2000. *U.S. Department of Health and Human Services, Health Resources and Services Administration, Bureau of Health Professions.* Available at: [http://bhpr.hrsa.gov/healthworkforce/reports/factbook02/FB102.htm](http://bhpr.hrsa.gov/healthworkforce/reports/factbook02/FB102.htm).


65. Davidson MH. Strategies to improve Adult Treatment Panel III guideline adherence and patient compliance. *Am J Cardiol.* Mar 7 2002;89(5A):8C-20C; discussion 20C-22C.


