The Effect of Giving Global Coronary Risk Information to Adults

A Systematic Review

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Background: Global coronary heart disease (CHD) risk estimation (ie, a quantitative estimate of a patient’s chances of CHD calculated by combining risk factors in an empirical equation) is recommended as a starting point for primary prevention efforts in all US adults. Whether it improves outcomes is currently unknown.

Methods: To assess the effect of providing global CHD risk information to adults, we performed a systematic evidence review. We searched MEDLINE for the years 1980 to 2008, Psych Info, CINAHL, and the Cochrane Database and included English-language articles that met prespecified inclusion criteria. Two reviewers independently reviewed titles, abstracts, and articles for inclusion and assessed study quality.

Results: We identified 20 articles, reporting on 18 unique fair or good quality studies (including 14 randomized controlled studies). These showed that global CHD risk information alone or with accompanying education increased the accuracy of perceived risk and probably increased intent to start therapy. Studies with repeated risk information or risk information and repeated doses of counseling showed small significant reductions in predicted CHD risk (absolute differences, −0.2% to −2% over 10 years in studies using risk estimates derived from Framingham equations). Studies providing global risk information at only 1 point in time seemed ineffective.

Conclusions: Global CHD risk information seems to improve the accuracy of risk perception and may increase intent to initiate CHD prevention among individuals at moderate to high risk. The effect of global risk presentation on more distal outcomes is less clear and seems to be related to the intensity of accompanying interventions.

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undertook a systematic review to assess outcomes we considered to be in the pathway of potential impact: accuracy of risk perception, intent to start therapy, adherence to therapy, and reduction in CHD risk factors and predicted CHD risk.

**METHODS**

The final work plans for this review were developed in conjunction with a specially appointed work group comprising individuals from the 6 member institutions of the Centers for Disease Control and Prevention (CDC) Cardiovascular Health Intervention Research and Translation Network (CHIRTN).

**DATA SOURCES AND SEARCHES**

In collaboration with a medical librarian, we searched MEDLINE (1980 to December 18, 2008) using the MESH headings “cardiovascular disease” (major topic headings “epidemiology” and “prevention and control”), “patient education as a topic,” “communication,” “physician-patient relations,” “consumer participation,” “patient participation,” “community health services,” “community health centers,” and “community medicine.”

We then searched PsycINFO, CINAHL, and the Cochrane Database using the keywords “cardiovascular,” “risk assessment,” “communication,” “patient relations,” or “education.” Each search was limited to studies in humans and to the English language. We also hand searched the bibliographies of all included articles, searched our files, and queried content experts. Because of the disparate indexing of relevant articles, we also performed related articles searches of all included MEDLINE articles.

**STUDY SELECTION**

We included studies of any design that were conducted in community or practice settings, included adults with no history of cardiovascular disease (CVD), specified global CHD risk presentation as the primary intervention or part of a multipart intervention, clearly documented the presentation of global CHD risk directly to individuals, and measured an outcome of interest. Outcomes of interest for this study were accuracy of risk perception; intent to start aspirin, antihypertensive medication, lipid-lowering medication, smoking cessation therapies, diet, or exercise; adherence to therapy; or change in predicted global CVD risk or CVD event rates. We were secondarily interested in changes in patient blood pressure (BP), cholesterol levels, aspirin use, smoking cessation, and diet or exercise but reported these results separately in an online appendix available at http://www.archinternmed.com.

We excluded studies that were unrelated to global CHD risk, answered the wrong question about CHD risk (eg, they were related to secondary prevention; the development and validation of risk scores; the accuracy of systems to calculate CHD risk; the acceptability of decision aids including CHD risk; the cost-effectiveness of programs using CHD risk; or the understanding of various risk formats for treatment benefit information), used risk scores for an alternate purpose (eg, as part of the end points of a study but not as information for individuals), did not have a quantitative experimental design (eg, qualitative studies), focused on physician knowledge or actions in response to global CHD risk scores, only had an outcome that was not of interest (eg, attitudes about CHD prevention), used CHD risk in combination with other health risks presented to individuals (eg, as part of traditional health risk appraisal tools), presented hypothetical rather than actual risks, or were determined to be of poor quality (see the next subsection).

**DATA EXTRACTION AND QUALITY ASSESSMENT**

Two of us (S.L.S. and A.J.V.) independently reviewed titles, abstracts, and, if necessary, full articles to determine inclusion. Disagreements were resolved by discussion. One of us (D.L.) abstracted study information into tables; 2 of us (S.L.S. and A.J.V.) then checked this information for accuracy. Two reviewers independently assessed study quality (L.E.S. and C.I.L. for the study by Sheridan et al11 and S.L.S. and A.J.V. for all others).

After assessment of potential quality grading schemes for quantitative study designs,12-14 the CHIRTN Work Group decided on criteria adapted from the US Preventive Services Task Force and used in previous reviews.2,15-17 For each study, reviewers graded prespecified criterion (Table 1) as good, fair, or poor. We converted ratings for each item into numeric values (good = 2, fair = 1, poor = 0) and a composite rating for each study. We weighted each item equally, excluding items judged to be not applicable based on study design. We based final quality grades on averaged scores, with 1.5 or higher considered to denote good quality, 1.0 to 1.49 considered to denote fair quality, and less than 1.0 considered to denote considerable methodological limitations.

**DATA SYNTHESIS AND ANALYSIS**

After data abstraction, CHIRTN Work Group members met for discussion about results. They compared and contrasted relevant features of studies, giving priority to study design and size, and continued iterative review until reaching consensus about key messages and conclusions. No meta-analyses were performed owing to the heterogeneity of study populations, interventions, and outcomes.

**RESULTS**

**SEARCH RESULTS**

Overall, we identified 20 articles, reporting on 18 unique studies, for inclusion in our review (Figure). We identified 9 through our primary literature search and 11 through hand searching and searches of related articles.

**STUDY CHARACTERISTICS**

Included studies had a wide variety of designs (Table 1). Three24,30,31 were cluster randomized controlled trials (cRCTs), 11 were traditional randomized controlled trials (RCTs), 3 were quasi-experimental studies,10,23,27 and 1 was an open-label follow-up of an RCT.34 Fourteen took place in clinical settings and 4 in community settings.18,22,28,33 Of studies in clinical settings, 9 took place in general practice settings (with 1 recruiting from population registries) and 5 took place in specialty clinics or risk factor treatment centers.19,24,25,27,32 Of those in community settings, 2 took place in work sites and 2 in individuals’ homes.18,33

Ten studies gave participants a Framingham or a Framingham-derived risk score,11,19,21,23,25,26,31,34-36 six studies used other risk scores (Dun- dee risk score,22 United Kingdom Prospective Diabetes Study risk score,24 Take Heart,31 Cardiovascular...
Table 1. Summary of Included Articles

<table>
<thead>
<tr>
<th>Source</th>
<th>Design</th>
<th>Quality Grade</th>
<th>Setting</th>
<th>Sample Size</th>
<th>Intervention</th>
<th>Intervention Focus</th>
<th>Control</th>
<th>Duration of Follow-up</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avis et al.18, 1989</td>
<td>RCT but main analysis is preintervention</td>
<td>F (1.29)</td>
<td>15 Neighborhoods and towns in Boston, MA, US</td>
<td>732</td>
<td>Risk by 4 different instruments</td>
<td>NA</td>
<td>NA</td>
<td>7-12 wk</td>
<td>Accuracy of risk perception</td>
</tr>
<tr>
<td>Christian et al.18, 2005</td>
<td>Nested RCT</td>
<td>F (1.25)</td>
<td>1 US academic radiology clinic</td>
<td>125</td>
<td>Risk + educ</td>
<td>NA</td>
<td>NA</td>
<td>1 mo</td>
<td>Accuracy of risk perception</td>
</tr>
<tr>
<td>Grover et al.20, 2007 and 2009</td>
<td>RCT</td>
<td>G (1.71)</td>
<td>PCPs in 4 Canadian provinces</td>
<td>3053 patients; 230 physicians</td>
<td>Patient: (risk + educ) × 5</td>
<td>Physician: educ</td>
<td>C</td>
<td>Usual care</td>
<td>12 mo</td>
</tr>
<tr>
<td>Hanlon et al.22, 1995</td>
<td>RCT</td>
<td>F (1.36)</td>
<td>2 Work sites in Glasgow, Scotland</td>
<td>757</td>
<td>Risk + educ + risk factors</td>
<td>C</td>
<td>No interv</td>
<td>5 mo</td>
<td>CHD risk (Dundee risk score), BP, cholesterol, smoking, diet, exercise</td>
</tr>
<tr>
<td>Paterson et al.23, 2002</td>
<td>Quasi-exp</td>
<td>F (1.38)</td>
<td>Family practices in southern ON, Canada</td>
<td>20 MDs; 40 patients</td>
<td>Risk + educ</td>
<td>B, C, D, E, O, S (varied)</td>
<td>NA</td>
<td>Immediate</td>
<td>Accuracy of risk perception, intent to start therapy</td>
</tr>
<tr>
<td>Sheridan et al.24, 2006</td>
<td>RCT</td>
<td>F (1.21)</td>
<td>1 Academic general medicine clinic, US</td>
<td>87</td>
<td>Risk + educ</td>
<td>A, B, C, S (varied)</td>
<td>List of risk factors</td>
<td>Immediate</td>
<td>Intent to start therapy</td>
</tr>
<tr>
<td>Weymiller et al.25, 2007</td>
<td>cRCT</td>
<td>G (1.5)</td>
<td>1 Academic metabolic clinic (focusing on dyslipidemia and DM), US</td>
<td>21 Clinicians; 98 patients</td>
<td>Risk + educ</td>
<td>C</td>
<td>Educ</td>
<td>3 mo</td>
<td>Knowledge, adherence, cholesterol</td>
</tr>
<tr>
<td>Edelman et al.26, 2006</td>
<td>RCT</td>
<td>G (1.64)</td>
<td>CIM, Durham, NC, US</td>
<td>154</td>
<td>Risk + (counseling × 28)</td>
<td>D, E, O (varied)</td>
<td>Risk + educ</td>
<td>5 mo, 10 mo</td>
<td>Intent to start therapy, CHD risk (Framingham5), BP, cholesterol, exercise</td>
</tr>
<tr>
<td>Krones et al.27, 2008</td>
<td>cRCT</td>
<td>F (1.43)</td>
<td>Urban and rural GP clinics in Germany</td>
<td>14 Physician CME groups; 162 practices; 1332 patients</td>
<td>Physician CME groups: educ + counseling</td>
<td>A, B, C, E, S</td>
<td>Physician CME groups: educ (alt)</td>
<td>Patient: usual care</td>
<td>Knowledge, CVD risk (Framingham5)</td>
</tr>
<tr>
<td>Lalonde et al.28, 2004</td>
<td>Quasi-exp</td>
<td>F (1.4)</td>
<td>Hypertension and CHD prevention clinics at a UH in Canada</td>
<td>16</td>
<td>Risk + counseling</td>
<td>B, C, D, E, O, S</td>
<td>NA</td>
<td>2 wk</td>
<td>Accuracy of risk perception, knowledge</td>
</tr>
<tr>
<td>Lovibond et al.29, 1986</td>
<td>RCT</td>
<td>F (1.29)</td>
<td>School of Psychology, University of NSW, Australia</td>
<td>75</td>
<td>Maximal (risk + counseling) × 16</td>
<td>D, E, O, S</td>
<td>Extended: (risk + counseling [alt]) × 16</td>
<td>Basic: educ × 16</td>
<td>6 and 12 mo</td>
</tr>
</tbody>
</table>

(continued)
global CHD risk information on aspirin use or actual CVD event rates.

**STUDY QUALITY**

Six studies were determined to be of good quality and 12 of fair quality.

**THE EFFECT OF GLOBAL RISK PRESENTATION ON OUTCOMES**

**Risk Perception**

We found 1 RCT and 3 quasi-experimental studies addressing the effect of global risk presentation on accuracy of risk perception (Table 2). All including the RCT examined the effect of global risk presentation on accuracy using preintervention to post-intervention analysis.

Three studies (n=897 participants) examined the effect of global risk presentation alone or with a modicum of additional education. The largest (n=732 participants), a population-based RCT designed to compare the effects of 4 risk appraisal instruments, demonstrated a 9% increase in the accuracy of designating oneself as lower than average, average, or higher than average risk (significance not reported) at 2- to 3-month follow-up. Two smaller clinic-based studies (n=165 participants) showed similar sized improvements in subjects’ accuracy in reporting their categorical risk. However, 1 (n=40 participants) measured accuracy only indirectly, showing improvements in population perceived risk (absolute difference, 16%; P<.001) and the other (n=125 participants), which stratified risk perception by level of risk, showed improved accuracy only for the low-risk group (n=63 participants; absolute difference, 13%; P=.02), an effect that attenuated over a 1-month follow-up.

A small quasi-experimental study (n=16 participants) examined the effect of global risk information + counseling on the accuracy of risk perception. Consistent with the findings of other studies, this study showed improved accuracy of categorical risk perception (absolute difference, 43%; P=.03). It also showed dramatic improve-

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**Table 1. Summary of Included Articles (continued)**

<table>
<thead>
<tr>
<th>Source</th>
<th>Design</th>
<th>Quality Grade</th>
<th>Setting</th>
<th>Sample Size</th>
<th>Intervention</th>
<th>Intervention Focus</th>
<th>Control</th>
<th>Duration of Follow-up</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Turnbull et al, 2006</td>
<td>cRCT</td>
<td>F (1.36)</td>
<td>67 GP clinics in 2 Australian states</td>
<td>67 practices; 371 patients</td>
<td>Practice/physician: risk + disease management support</td>
<td>B, D, E</td>
<td>Usual care</td>
<td>6 and 12 mo</td>
<td>CHD risk (Take Heart), BP</td>
</tr>
<tr>
<td>Williams et al, 2006</td>
<td>RCT</td>
<td>G (1.64)</td>
<td>Tobacco treatment center, US</td>
<td>1006</td>
<td>Risk + (counseling &gt; 2-6)</td>
<td>C, D, S</td>
<td>Educ</td>
<td>18 mo</td>
<td>Cholesterol, smoking, diet</td>
</tr>
<tr>
<td>Wister et al, 2007</td>
<td>RCT</td>
<td>F (1.43)</td>
<td>Fraser Health Region, BC, Canada</td>
<td>315 Primary prevention groups</td>
<td>Patient: risk + (counseling &gt; 3-7)</td>
<td>D, E, O, S</td>
<td>Usual care</td>
<td>1 y</td>
<td>CHD risk (Framingham), BP, cholesterol, smoking, diet</td>
</tr>
<tr>
<td>Family Heart Study Group, 1994</td>
<td>RCT</td>
<td>F (1.14)</td>
<td>26 GPs in 13 towns in UK</td>
<td>14 Practices; 12,472 participants</td>
<td>Risk + (counseling &gt; 2-6)</td>
<td>D, E, O, S</td>
<td>Usual care</td>
<td>1 y</td>
<td>CHD risk (Dundee), BP, cholesterol, smoking</td>
</tr>
<tr>
<td>Emmett et al, 2005</td>
<td>Observational follow-up of RCT</td>
<td>G (1.71)</td>
<td>21 GPs in SW England</td>
<td>216</td>
<td>Risk + decision analysis, educ, risk + decision analysis + educ</td>
<td>B</td>
<td>No intervention (usual care)</td>
<td>3 y</td>
<td>CVD risk (Framingham), BP</td>
</tr>
<tr>
<td>Montgomery et al, 2003</td>
<td>RCT, factorial</td>
<td>G (1.57)</td>
<td>21 GPs in SW England</td>
<td>217</td>
<td>Risk + decision analysis, educ, risk + decision analysis + educ</td>
<td>B</td>
<td>No intervention (usual care)</td>
<td>Immediate 3 mo for many outcomes</td>
<td>Knowledge, intent to start therapy</td>
</tr>
</tbody>
</table>

Abbreviations: alt, alternative; BP, blood pressure; CHD, coronary heart disease; CIM, Center for Integrative Medicine; CME, continuing medical education; cRCT, cluster randomized controlled trial; CVD, cardiovascular disease; DM, diabetes mellitus; educ, education; F, fair; G, good; GP, general practice; NA, not applicable; NSW, New South Wales; ON, Ontario; PCP, primary care practice; postinterv, postintervention; preinterv, preintervention; quasi-exp, quasi-experimental; RCT, randomized controlled trial; SW, southwest; UH, university hospital; UK, United Kingdom; US, United States.

a Each study grade was based on a composite score (described in the subsection title “Data Extraction and Quality Assessment” in the “Methods” section) accounting for (1) creation and maintenance of comparable study groups; (2) valid and reliable measurement applied equally to both study groups; (3) provision of a clear definition of the intervention; and (4) performance of appropriate statistical analysis, including appropriate control for confounding, intention-to-treat analysis, and accounting for multiple comparisons and cluster randomized design if necessary.

b Unless otherwise specified, numbers indicate the numbers of participants (either patients or community-dwelling adults).

c Intervention focus: A, aspirin; B, BP medication; C, cholesterol medication; D, diet; E, exercise; O, other; S, smoking cessation; varied, focus of intervention varied depending on risk factors or negotiation with patient.

d Framingham-derived risk estimation.

e Two other experimental groups were planned (risk + education and risk factors + education), but results were not reported.
In aggregate, these studies suggest that global risk information, alone or with accompanying education or counseling, increases the accuracy of perceived risk. Although none of these studies included a “no risk presentation” control and effect sizes varied across studies, the best available evidence suggests that the expected improvement in individuals’ ability to correctly categorize their risk is approximately 10%. Preliminary data suggest that the effect may be larger if accuracy is measured by individuals’ ability to report a more precise quantitative estimate of their risk.

**Intent to Start Therapy**

We found 3 RCTs and 1 quasi-experimental study (n = 498 participants) addressing the effect of global risk presentation on intent to start therapy (Table 3). None examined the effect of global risk presentation alone.

Two studies examined the effect of global risk in combination with...
CHD education on intent to start therapy. The larger of these 2 studies, a small (n=87 participants) RCT\(^{11}\) that compared global risk + education with a list of risk factors provided in a clinical setting, showed that the combination of global risk information and education tended to increase intent to start therapy (absolute difference in proportion of individuals with a specific plan for CHD risk reduction who discussed it with their physician, 13%; 95% confidence interval [CI], −7 to 34%; regardless of whether they discussed it with their physician, 16%; 95% CI, −1% to 33%). However, results did not achieve statistical significance, likely owing to the small sample size. The second study, a small quasi-experimental study (n=40 participants),\(^{23}\) provided only posttest intentions for therapy, limiting conclusions about the intervention’s effect.

One study examined the effect of global risk presentation in combination with counseling on intent to start therapy. This good-quality RCT (n=154 participants),\(^{25}\) which compared the effects of risk presentation + repeated counseling with risk presentation and education alone, showed a statistically greater effect when risk was combined with counseling (absolute difference, 27%; P= .02). Notably, within-group increases in intent to start therapy in the risk + education alone group were similar in magnitude to those reported in studies of risk + education alone above (19% increase in exercise plans for risk and education alone).

One study\(^{35}\) examined the effect of global risk presentation in combination with the results of decision analysis on intent to start therapy. This factorial RCT (n=217 participants) showed increased certainty in the decision of whether to start hypertension therapy, but a trend toward less overall intent (relative risk [RR] for yes vs uncertain, 1.19; 95% CI, 0.59-2.40; RR for no vs uncertain, 3.15; 95% CI, 0.91-10.98).

In summary, global risk presentation in combination with education increases intent to start therapy by approximately 15 to 20 absolute percentage points, with larger effect sizes when risk is combined with counseling instead of education alone. There are too few studies of global risk information combined with recommendations from decision analysis to determine the potential impact of this approach in clinical practice.

**Adherence to Therapy**

We found 1 cRCT (n=98 participants) addressing the effect of global risk presentation on adherence. This study\(^{24}\) showed a small, nonstatistically significant increase in adherence to decisions to take statins (adjusted odds ratio [OR], 1.9; 95% CI, 0.4-9.8) among those randomized to risk + education compared with those randomized to education alone. The study, however, provided its intervention at only 1 time point, analyzed adherence as a secondary outcome, and seemed to lack statistical power for this analysis. Of note, this study did report that risk + education reduced the number of...
participants missing a dose of statins in the past week compared with education alone (unadjusted OR, 3.4; 95% CI, 1.5-7.5). However, the lack of adjustment for baseline differences in CHD risk confounds interpretation of this result.

Change in Predicted CHD Risk

We found 9 studies (n=14 752 participants), including 6 RCTs, 2 cRCTs, and 1 observational follow-up of an RCT, addressing the effect of global risk presentation on predicted CHD risk (Table 4). None addressed the effects of global risk presentation alone.

Intervention effects seemed to be related to the intensity of the interventions provided. One good-quality RCT (n=3053 participants) that included repeated presentations of risk with a modicum of education showed a small, statistically significant reduction in predicted CVD risk at 1-year follow-up (absolute difference in 10-year predicted risk: -0.75%, P<.001). In addition, 1 good-quality and 2 fair-quality RCTs, which included repeated doses of counseling (n=12 941 participants), showed small, statistically significant reductions in 10-year global CHD risk (on the order of 0.2%-2% over 10-12 months in the 2 studies using Framingham-based risk calculators). One additional fair-quality RCT (n=75 participants) with repeated risk presentation and counseling showed a trend toward global risk reduction (mean absolute predicted 10-year risk difference, -4%, using the International Coop Study calculator), which did not achieve statistical significance. Good-quality and fair-quality studies with interventions at 1 point in time showed negligible to small effects, regardless of whether global risk was presented with education alone or in combination with counseling.

In summary, studies showed mixed effects of global CHD risk information on predicted CHD risk, which seemed to be related to the intensity of the intervention provided. Studies with repeated global risk presentation or repeated doses of counseling showed small significant reductions in 10-year predicted CHD risk (on the order of 0.2%-2% in studies using Framingham calculators), which would be clinically important (number needed to treat, 50-500) if applied across the population of moderate- to high-risk individuals.

Recent guidelines have suggested providing all adults older than 40 years with their global CHD risk. Our review suggests that providing such information improves accuracy of risk perception and probably increases intent to initiate CHD prevention among adults at moderate to high risk. The effect of global presentation on other outcomes, including the more distal ones, is less clear. Apparent benefits of global risk presentation on predicted CHD risk (and lipid levels as reported in the online appendix) seem to be related more to the intensity of interventions than presentation of global CHD risk. Several fair-to-good-quality studies demonstrate that providing global risk presentation at only 1 time point seems to be ineffective in promoting risk reduction. In addition, only a single study examined and demonstrated the benefit of repeated presentations of global risk (with a minimum of accompanying education) on predicted CHD risk and lipid levels. Current data preclude definitive conclusions on the effects of global CHD risk information on adherence, BP reduction, smoking cessation, and initiation of aspirin, diet, and exercise, although it should be noted that prevention guidelines for many of these outcomes (excluding those related to aspirin and lipids) are not currently risk based and may reduce the likelihood of finding an effect.

An inherent challenge in interpreting this body of work is the diversity of study designs, interventions, and comparators of identified studies. The most direct evidence for the effect of global CHD risk would come from studies comparing global risk alone with usual care, global risk + education with education alone, or global risk + counseling with counseling alone. Unfortunately, few studies made such comparisons and several included no comparator. In addition, no study measured all of the outcomes that might be expected to be affected by global risk presentation. Given these issues, it is reassuring that our findings are consistent with behavioral theory and with recent systematic evidence reviews on the effects of health risk appraisal tools (which are designed to provide information on multiple risks to individuals in workplace settings). All suggest that risk information is not a panacea but rather a tool to be used in conjunction with other tools to promote adherence and risk reduction.

Based on these findings, we believe clinical guidelines should continue to recommend that all individuals be informed of their global CHD risk as a way to improve individuals’ accuracy of risk perception and their intent for therapy. It must be clear, however, that global risk information alone or in combination with usual care is not sufficient to achieve more distal clinical benefits.

Global CHD risk seems most successful in promoting risk reduction when combined with repeated intervention in the form of either counseling or risk feedback. However, many potentially successful combinations of behavior change tools remain untested or undertested. For instance, none of the studies promoting medication use combined CHD risk information with use of effective adherence aids. In addition, few studies included global risk in decision aids, which might be easily disseminated in clinical practice with modest effort. More research is needed to determine the most efficacious and effective adjuncts to CHD risk information.

More research is also needed in several other areas. First, research is needed to determine the effect of global CHD risk information on individuals with low CHD risk. Although global CHD risk would ideally motivate individuals with high risk to accept risk reducing treatments, it would ideally discourage...
Table 4. Evidence for the Impact of CHD Risk Information on Risk

<table>
<thead>
<tr>
<th>Source</th>
<th>Design</th>
<th>Quality Grade (Score)</th>
<th>Setting</th>
<th>Sample Size</th>
<th>Intervention</th>
<th>Control</th>
<th>Follow-up</th>
<th>Outcome</th>
<th>Control</th>
<th>Intervention</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Edelman et al., 2006</td>
<td>RCT</td>
<td>G (1.64)</td>
<td>CIM, Durham, NC, US</td>
<td>154</td>
<td>Risk + (counseling × 28)</td>
<td>Risk + educ</td>
<td>5 mo, 10 mo</td>
<td>Change in risk of total CHD events over 10 y (Framingham)</td>
<td>5 mo: NR</td>
<td>10 mo: −1.3</td>
<td>5 mo: −1.5</td>
</tr>
<tr>
<td>Emmett et al., 2005</td>
<td>Obs</td>
<td>G (1.71)</td>
<td>21 GPs in SW England</td>
<td>216</td>
<td>Risk + DA, Educ, Risk + DA + educ</td>
<td>No interventions (usual care)</td>
<td>3 y</td>
<td>Risk of CVD events over 10 y (Framingham)</td>
<td>No decision</td>
<td>23 Educ: 22</td>
<td>−0.75%</td>
</tr>
<tr>
<td>Grover et al., 2007</td>
<td>RCT</td>
<td>G (1.71)</td>
<td>PCPs in 4 Canadian provinces</td>
<td>3053 Patients; 230 physicians</td>
<td>Risk + educ × 5 Physician: educ</td>
<td>Usual care</td>
<td>12 mo</td>
<td>Change in risk of CVD events over 10 y (Framingham)</td>
<td>−6.63b</td>
<td>−7.37b</td>
<td>−0.75%</td>
</tr>
<tr>
<td>Hanlon et al., 1995</td>
<td>RCT</td>
<td>F (1.38)</td>
<td>2 Worksites in Glasgow, Scotland</td>
<td>757</td>
<td>Risk + educ + risk factors c</td>
<td>No interventions (same site)</td>
<td>5 mo</td>
<td>Change in CHD events over 10 y (Dundee risk score)</td>
<td>0.34</td>
<td>0.53</td>
<td>0.19</td>
</tr>
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<td>Krones et al., 2008</td>
<td>cRCT</td>
<td>F (1.43)</td>
<td>Urban and rural clinics in Germany</td>
<td>14 Physicn CME groups: education; Patients: risk + counseling</td>
<td>Physicn CME groups: educ (alt) Patient: usual care</td>
<td>6 mo</td>
<td>Change in risk of CVD events over 10 y (Framingham)</td>
<td>−3.33</td>
<td>−3.00</td>
<td>0.32</td>
<td></td>
</tr>
<tr>
<td>Lovibond et al., 1986</td>
<td>RCT</td>
<td>F (1.28)</td>
<td>Clinical Section, School of Psychology, University of NSW, Australia</td>
<td>75</td>
<td>Max: (risk + counseling [alt]) × 16</td>
<td>Ext: (risk + counseling [alt]) × 16 Basic: educ × 16</td>
<td>6 and 12 mo</td>
<td>Change in risk of hard CHD events over 10 y (International Coop Study risk score)</td>
<td>Ext: 6 mo</td>
<td>−22b</td>
<td>−16b</td>
</tr>
<tr>
<td>Turnbull et al., 2006</td>
<td>cRCT</td>
<td>F (1.36)</td>
<td>67 GP clinics in 2 Australian states</td>
<td>67 Practices; 371 patients</td>
<td>Practice/physician: risk + disease management support Patient: risk + counseling</td>
<td>Usual care</td>
<td>6 and 12 mo</td>
<td>Change in risk of CHD mortality over 10 y (Take Heart)</td>
<td>6 mo: −12b</td>
<td>−12b</td>
<td>−14b</td>
</tr>
<tr>
<td>Wister et al., 2007</td>
<td>RCT</td>
<td>F (1.43)</td>
<td>Fraser Health Region in BC, Canada</td>
<td>315 Primary prevention groups</td>
<td>Patient: risk + (counseling × 3-7) Physician: risk</td>
<td>Usual care</td>
<td>1 y</td>
<td>Change in risk of total events over 10 y (Framingham)</td>
<td>−1.1</td>
<td>−3.07</td>
<td>−1.97; P = .002</td>
</tr>
<tr>
<td>Family Heart Study Group, 1994</td>
<td>RCT</td>
<td>F (1.14)</td>
<td>26 GPs in 13 towns in Britain</td>
<td>14 Practices: 12472 participants</td>
<td>Risk + (counseling × 2-6) Physician: usual care (all practices)</td>
<td>1 y (same practices) usual care (all practices)</td>
<td>Risk of hard CHD events over 10 y (Dundee risk score)</td>
<td>NR</td>
<td>NR</td>
<td>Men: −35.2; (−28.0 to −42.2)</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: alt, alternative; BC, British Columbia; CIM, Center for Integrative Medicine; CHD, coronary heart disease; CME, continuing medical education; cRCT, cluster randomized controlled trial; CVD, cardiovascular disease; DA, decision analysis; ext, extended; F, fair; G, good; GPs, general practitioners; interv, intervention; max, maximum; metro, metropolitan; NR, not reported; NS, not significant; NSW, New South Wales; obs, observational; PCP, primary care practice; RCT, randomized controlled trial; SW, southwest.

a Framingham-derived risk estimation.
bRisk converted to a 10-year risk by research team using the assumption that risk increases linearly over time.
cThree other experimental groups were planned (education, risk + education, and risk factors + education), but results were not reported.
dIntervention also compared with an external control, but results are not reported herein.
eA 12-month follow-up planned but not reported.
fStudy also assessed secondary prevention, but data are not shown here.
individuals at low risk from seeking unnecessary treatments. Second, research is needed to determine the effect of global CHD risk in populations of varying skill with numbers. It is possible that global CHD risk information may have no or adverse effects in individuals with low numeracy skills. Third, research is needed to determine the best presentation of global CHD risk. Although a growing literature suggests that isolated risk estimates are most understandable when presented to individuals in percentage (\(x\%\)) or frequency (\(x/1000\)) formats,\(^{43,44}\) no attention has been given to how to best help individuals make sense of risk information in the context of decisions (ie, how to help individuals integrate CHD risk information with treatment information to decide when they are willing to accept medical treatment). Fourth, research is needed to determine whether giving global CHD risk information to health care providers (eg, physicians, nurse practitioners, physician assistants), or both patients and providers, is more efficacious than offering risk information to patients alone. Only 1 study\(^ {23}\) in our review examined patient-provider interactions following provision of global risk information to patients. This study demonstrates increased patient-provider discussions about CHD risk and presents indirect evidence that giving global risk information to patients helps them solicit appropriate support from their health care provider to lower their CHD risk. However, direct evidence is needed to determine whether patients can indeed effectively use global risk information to solicit appropriate support (including medications) from their provider, or whether better outcomes can be achieved when providers (or both patients and providers) are given global CHD risk information. Finally, research is needed to determine the best setting for risk communication. Although we could identify no appreciable differences in the effectiveness of CHD risk interventions delivered in clinics, communities, and work sites, no studies in our review included head-to-head comparisons of the same intervention delivered in these various settings. Reviews of other multitask factor interventions, however, suggest that clinical settings may be most effective.\(^ {36}\)

Observations aside, we must consider potential limitations of our review. First, although we made rigorous and repeated searches, we may not have identified all relevant literature. Literature on global CHD risk is diversely indexed, making identification of relevant articles difficult. We also excluded non-English articles. Second, although our quality score has been vetted through a rigorous process and used in a number of systematic reviews, there is the potential for misclassification of quality because the score has not been formally validated.

Despite these limitations, our review suggests that global CHD risk information might contribute to improved CHD prevention by increasing the accuracy of CHD risk perception and motivating individuals at moderate to high risk to plan risk reduction. In combination with repeated risk presentation or repeated counseling, it may also reduce predicted CHD risk and improve lipid levels. The effects of global CHD risk information on other outcomes, and in individuals at low risk and with low numeracy skills, need further exploration.

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