Review: Framingham risk scores have variable accuracy in predicting CVD events in different patient populations

Brindle P, Beswick A, Fahey T, Ebrahim S. **Accuracy and impact of risk assessment in the primary prevention of cardiovascular disease: a systematic review.** Heart. 2006;92:1752-9.

Clinical impact ratings: GIM/FP/GP ★★★★★★☆ Hospitalists ★★★★★☆☆ Cardiology ★★★★★★☆

QUESTIONS

In different groups of patients, are Framingham risk assessments accurate for predicting cardiovascular disease (CVD) events? Do risk assessments improve health outcomes?

METHODS

Data sources: MEDLINE, EMBASE/Excerpta Medica, Cochrane Controlled Trials Register (CENTRAL), CINAHL, PsycINFO, ISI Proceedings, British Library's Electronic Table of Contents (ZETOC), bibliographies of relevant studies, and handsearches of key journals (to September 2004).

Study selection and assessment: Studies in any language that compared risk for fatal and nonfatal coronary heart disease (CHD) or CVD outcomes predicted by Framingham risk scores with observed 10-year risk, and randomized controlled trials (RCTs) that assessed the effectiveness of CV risk scores to aid primary prevention in patients predominantly free of symptomatic CVD. Studies of older risk scores not used in clinical practice and studies reporting only fatal outcomes were excluded. 27 risk-assessment studies (n = 71 727, age range 30 to 80 y) met the selection criteria: 8 studies evaluated the Wilson Framingham method, and 19 evaluated the Anderson Framingham method. The year of initiation of study recruitment ranged from 1961 to 1996. 4 RCTs met the inclusion criteria for primary prevention and assessed outcomes related to changes in risk, treatment, and referrals.

Outcomes: Predicted-to-observed ratios of 10-year risk for combined fatal and nonfatal CHD or CVD and the effectiveness of risk scores in primary prevention.

MAIN RESULTS

For CHD outcomes, predicted-to-observed ratios ranged from an underprediction of 0.43 (95% CI 0.27 to 0.67) in a high-risk group to an overprediction of 2.9 (CI 1.9 to 4.3) in a lower-risk group. For CVD outcomes, the trend was similar, with a smaller range than that for CHD outcomes. Underprediction of CHD also occurred in higher-risk patient populations, specifically those with diabetes or a family history of premature CHD. In 4 trials in which physicians were offered or allocated to receive predictions for their patients, compared with no offer or allocation, groups did not differ for any outcome in 3 trials; but in 1 RCT, patients in the risk-score group had lower systolic blood pressure and were more likely to be prescribed CV drugs than those whose physicians did not receive risk scores.

CONCLUSIONS

Framingham risk assessments have variable accuracy in predicting cardiovascular disease events in different patient populations. Risk assessments provided to physicians for individual patients did not consistently improve health outcomes.

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COMMENTARY

It can be argued that the Framingham study has contributed more than any other single research program to the decline in CHD mortality over the past 50 years. As a result, the Framingham risk score has become one of our most venerable truths. All venerable truths, however, can benefit from reexamination.

Brindle and colleagues showed that predictions based on the Framingham risk score deviate predictably, rather than randomly, from the observed, with underestimation in such high-risk populations as patients with diabetes or a family history of CHD and overestimation in most unselected populations. The reasons for these deviations include inadequacies inherent in statistical prediction, changes in population risk over time, and differences in prevalence of unmeasured risk factors. Most clinicians have already accounted for underestimation: Diabetes is now treated as the equivalent of established CHD, and patients with strong family histories receive screening despite otherwise low risk. Overestimation of risk in community samples is, however, potentially problematic as many experts advise clinicians to link treatment of risk factors, particularly cholesterol level, to patients' Framingham risk scores.

It would be a mistake to interpret the findings of this study as showing that the Framingham risk score has no clinical value. The risk score provides a useful framework for clinicians to show patients how factors work together to increase risk for CHD or CVD outcomes. The literature suggests it does a good job of distinguishing high-risk patients from those at low risk.

As Brindle and colleagues point out, the Framingham risk score might be "fixable." The model can be recalibrated as measured risk in population changes over time and might be modified to account for such risk factors as obesity that are increasing in epidemiologic importance. Until epidemiologists provide us with these fixes, clinicians offering advice on primary prevention to individual patients should remember that the Framingham risk score can be a pessimistic predictor. Merely presenting the risk score to patients and clinicians does not seem to be a sufficient intervention for improving preventive services.

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