Commentary: Cardiovascular risk estimation: important but may be inaccurate

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Manuel and colleagues applied the recommendations from six national guidelines on statin treatment to the same Canadian population and measured each guideline’s impact in terms of number of people recommended for treatment, potential number of deaths from coronary heart disease avoided, and the number needed to treat to avoid one death. They show that markedly different numbers of people are recommended for treatment when different guidelines are followed.

Deciding whether to prescribe statins for a patient for the primary prevention of coronary heart disease would seem to be a relatively straightforward issue in the broader context of decision making in primary care. Over the past 10 years, 14 randomised controlled trials have established the efficacy of statins across a broad range of patient groups. However, as Manuel and colleagues show, integration of evidence into clinical guidelines is inconsistent, particularly with regard to explicit use of Framingham and other multivariable risk functions when estimating the probability of heart disease developing in individual patients.

Some would argue that the results of Manuel and colleagues’ study are not surprising, as the outcome for their study—death from coronary heart disease—was calculated by applying the Framingham or SCORE risk score, an example of internal validity assessment. The guidelines that go furthest in recommending Framingham or SCORE risk score, an example of internal validity assessment, are less explicit—US, Canadian, and European. As the relative benefits from statins are constant irrespective of initial absolute risk and the risks of treatment are small, the approach of explicit absolute risk assessment is justified: higher risk individuals are likely to gain the most in absolute terms.

Equally, uncritical application of absolute risk assessment for primary prevention of coronary heart disease should be discouraged. A systematic review of 27 external validity studies, of the extent to which predicted risk assessments are an accurate reflection of observed risk of heart disease, shows that the performance of the Framingham risk score varies considerably between different countries and populations. Predicted to observed ratios ranged from an underprediction of 0.43 in higher risk populations to overprediction of 2.87 in lower risk populations. Within the United Kingdom, regional differences of risk of heart disease mean that the accuracy of Framingham varies, with overprediction in areas of low incidence of CHD and underestimation in socially deprived areas, where the incidence of heart disease is high. Even if Framingham was consistently accurate, evidence about the benefits of applying absolute risk assessment in the primary prevention of heart disease is scarce; only four randomised controlled trials implementing this approach have been published, with inconclusive results.

In conclusion, the study by Manuel and colleagues contains an important message. Explicit absolute risk assessment is an essential starting point when considering primary preventive treatment for CHD. However, uncritical application of Framingham may mislead patients and health professionals and ongoing studies are needed to ensure CHD risk assessment is as accurate as possible for the group of patients to which it is applied.


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