FEATURED CORRESPONDENCE

Family history of premature coronary heart disease and risk prediction

To the Editor We read with great interest the article by Sivapalaratnam and colleagues.¹ Although the self-reported family history of coronary heart disease (CHD) was an independent risk factor for future CHD events in this study, the addition of family history of CHD to Framingham Risk Score failed to improve the overall risk prediction of future CHD events. Since age is a heavily weighted risk factor incorporated into the contemporary cardiovascular risk scoring models, these models tend to underestimate the risk in younger patients who are otherwise destined to develop CHD.² Hence, it would be important to see the significance of a positive family history of premature CHD in a subgroup of patients who were relatively younger at the time of risk prediction and those who went on to develop CHD at a younger age. It does seem plausible that those who are destined to develop CHD event at a younger age may be better identified by incorporating family history of premature CHD into the risk prediction model. We, therefore, urge the authors to now report reclassification in those subjects who were relatively younger at the time of estimated Framingham Risk Score and among those patients who subsequently developed CHD at a young age.

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The Authors' reply We recently reported in an analysis of the EPIC-Norfolk cohort that a family history of premature coronary heart disease and risk prediction (CHD) was an independent risk factor for future CHD events.¹ However, its use did not improve the classification of people into risk categories based on the Framingham Risk Score. We read with interest the comment by Nadir and Struthers² who urge us to perform two additional analyses. The first request regards people at a relatively young age at the time of risk prediction. We arbitrarily chose the cut-off age of 55 years for men and 65 years for women, limiting the study population to 12 496 people of whom 586 developed CHD during follow-up. In this relatively young population, adding family history to the Framingham Risk Score resulted in 229 people who did not develop CHD being incorrectly reclassified and 9 people who developed CHD being correctly reclassified. The net reclassification improvement was –0.4%, which is slightly better than –2.0% in the entire population, but the net effect of using family history is still negative. Second, Nadir and Struthers requested a reclassification analysis among people who subsequently developed CHD at a young age. This approach is methodologically invalid and clinically irrelevant. The Framingham Risk Score was developed and validated to predict CHD risk during a 10-year follow-up. Therefore, it cannot be used in reclassification analyses to predict only CHD events occurring at a young age, using a variable follow-up depending on the age at the time of risk prediction. Even if one would develop an alternative risk algorithm to predict CHD events occurring only at a young age, the clinical relevance of this approach is questionable. It is well known that the large

majority of western populations are at low 10-year CHD risk but at high lifetime CHD risk.³ Using a risk algorithm to predict CHD events occurring only at a young age would give many people at low short-time risk a false sense of security. In summary, the suggestions by Nadir and Struthers do not provide new insights into the potential role of family history in the prediction of CHD risk.

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