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End points for predicting coronary risk must be clarified

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EDITOR—The term “absolute coronary risk” is often used without an explicit definition, resulting in confusing inconsistencies. The 1997 Standing Medical Advisory Committee on statin use and the 1998 Joint British recommendations on coronary heart disease prevention say that among people without established coronary heart disease, those with an absolute risk of non-fatal myocardial infarction or coronary death of 30% or more over 10 years should be identified and treated, and that this threshold should be lowered to 15% as resources allow. 1 2 Yet the Framingham equation they use to calculate the risk of coronary heart disease (in the joint British societies' prediction chart2 and the updated Sheffield table1) actually predicts a very much wider end point: coronary death, clinical non-fatal myocardial infarction, electrocardiographic myocardial infarction, physician assessed angina, and coronary insufficiency.3

We have used data from the British regional heart study to investigate how much difference inclusion of additional events in the end point makes to levels of absolute coronary risk. Among 7301 men aged 40-59 and free of diagnosed coronary heart disease at baseline, the 10 year event rate for an end point that included coronary death, non-fatal diagnosed myocardial infarction, and incident diagnosed angina (ascertained from medical record reviews) was 11.5%, some 50% higher than the event rate for an end point including only coronary death and non-fatal diagnosed myocardial infarction (7.5%). The Framingham end point adds not only stable angina but also coronary insufficiency and electrocardiographic (silent or unrecognised) myocardial infarction, ascertained by biennial screening. Subgroups identified as having a 30% 10 year risk by using the Framingham end point probably have well below a 20% 10 year risk of coronary death or non-fatal clinical myocardial infarction. Similarly, use of a 15% risk threshold based on the Framingham end point would result in treatment of people with a less than a 10% 10 year risk of coronary death or non-fatal clinical myocardial infarction.
Disregard for these differences is most clearly apparent when event rates are compared between studies. Current understanding of the validity of different coronary risk assessment methods is based on an analysis that directly compares Framingham event rates for all coronary heart disease with major clinical coronary event rates from other studies. We should not expect different predictive functions to give the same results if the end points they are predicting are different.

If national policy for statin use and other interventions is to be based on a threshold of absolute rather than relative risk, the end point must be clarified and, if possible, standardised.

References


