EDITORIAL

The value of risk scores

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Efforts to improve cardiovascular risk scoring should not be limited to broadening the biomarkers but should also include the individual's personal circumstances and socioeconomic status

According to the Nobel laureate Nils Bohr, predictions are difficult—especially about the future. What holds true for weather forecasts and financial markets applies to medical science and in particular to cardiovascular risk prediction.

However, during the last decade the perception has evolved that the intensity of measures taken to tackle risk factors should be adjusted by the severity of risk. The joint European societies and other organisations adopted this concept in their guidelines. But if clinical decision making should be based upon a quantifiable risk, doctors must be provided with robust tools that allow a prediction. The most widely used method to assess the “global risk” is based on equations from the Framingham heart study and includes classical risk factors.

In this issue of *Heart*, a systematic review by Brindle and colleagues assesses the predictive power of the Framingham risk score, the extent to which it is used, and the impact of calculating these scores on practice patterns of physicians.¹ In brief, this thorough study reveals an extremely variable performance of the risk scores with under-prediction of the risk in a high risk population and over-prediction in a lower risk population. Further, there is no clear evidence that risk assessment improved primary prevention or health outcomes. The regrettably poor predictive value of the risk scores raises the question whether the scores should be abolished or improved and how improvement could be achieved.

POTENTIAL VALUE OF RISK SCORES

Coronary heart disease follows a very variable course. More than a quarter of patients with myocardial infarction or sudden death have been asymptomatic. This highlights the need to identify individuals at-risk before an initial event for appropriate risk-modifying primary and secondary prevention. From a societal perspective the quantification of risk could inform clinical decision making and would allow a more efficient allocation of scarce resources to those at highest risk. Likewise over-treatment in low-risk populations could be reduced.

On an individual level communication of accurate prognostic information offers the patient an opportunity to make fully informed

choices about the medical care. Often these decisions mean a trade-off between quality of life and quantity of life. Inaccurate predictions may alter the patient's choice of treatment. Both the level of risk based upon a single patient's characteristics and her or his value-laden views and preferences on the initiation of treatment are necessary to tailor advice and individualise treatment. Consequently, the development of tools that enable us to predict as accurately as possible could further improve cardiovascular disease prevention by medical means.

CURRENT LIMITATIONS OF RISK SCORES

Risk scores are only of broad clinical value if they work when applied in a population other than the one from which they were derived. However, the portability of the currently recommended risk scoring methods derived from the Framingham study is limited. The overall absolute coronary risk assigned to individuals in the United Kingdom and most other European populations has been systematically and significantly overestimated.^{2,3} Furthermore, the accuracy of the Framingham risk score is better than any single risk factor or clinical acumen alone, but the predictive power leaves room for improvement. Ideally, a suitable risk score should consider the most relevant causal factors to quantify accurately the risk of disease.

WHAT CAUSES DISEASE?

Robert Koch (1843–1910) postulated a mono-causal origin of communicable diseases. His concept was soon to be extended and the perception prevailed that the interaction of inherited and environmental factors causes disease. Epigenetics and complex concepts like socioeconomic status, job control and social hierarchy further added to a more comprehensive understanding of causes of disease well beyond classical behavioural, chemico-physical, or infectious risk factors.^{4–6}

HOW CAN SCORES BE REFINED?

First, to increase the external validity of the prediction, data from populations with different absolute cardiovascular risk level should be pooled to derive common risk functions. This approach is being pursued by the SCORE study group.⁷ Further, regional recalibration methods seem attractive. Second, methodological improvement of prediction rules—for example, neural network techniques instead of conventional logistic regression—seems promising. Third, in spite of rather disappointing previous efforts, improvement of the accuracy by inclusion of additional risk factors should be investigated. Currently, several major risk factors are

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not considered in the Framingham or European risk calculations. These include independent risk factors for coronary heart disease like family history of premature myocardial infarction and ethnicity. Obesity, the metabolic syndrome, insulin resistance, lack of exercise, markers of inflammation, and psychological stress, which emerged as another major factor, are missing.⁸⁻⁹

Another very important risk factor complex comprises renal diseases. Of note, renovascular disease and microalbuminuria add to the risk prediction independent of traditional cardiovascular risk factors.¹⁰⁻¹¹ In addition, a reverse correlation of glomerular filtration rate and incidence of cardiovascular disease has been shown even for mildly reduced renal function. In turn, the devastating cardiovascular effect of end-stage renal disease can be stopped by restoration of renal function through transplantation.¹²⁻¹⁴

LOW SOCIOECONOMIC STATUS

Finally, there is ample evidence to support the idea that lower socioeconomic status—usually expressed as education, occupation, income or combinations of these—increasingly confers heightened cardiovascular risk and mortality. In accord with this concept, impaired functional capacity and abnormal heart rate recovery is strongly associated with low socioeconomic status and explains a major proportion of the correlation between socioeconomic status and mortality.¹⁵ In the United States less educated individuals were found to have 9.2 less potential life-years than those who were better educated, and ischaemic heart disease was the major contributor (0.84 years per person) to this striking educational disparity.¹⁶ The mediating pathways remain poorly elucidated and to prove causality is further complicated by the fact that individual health is both an outcome of socioeconomic status and also a determinant. It is unlikely that access to high quality medical treatment is a key factor that explains the excess mortality and morbidity rates among the less affluent; this is because even countries with publicly funded comprehensive medical coverage and strong emphasis on egalitarian healthcare policies face this gradient.¹⁷

Some authors suggest that the “wealth–health gradient” in cardiovascular mortality may be partially mediated by known risk factor pathways—but the causes of the uneven distribution of these risk factors by socioeconomic status remain vague.¹⁸⁻¹⁹

The most widely used risk scores do not consider socioeconomic status or any surrogate marker. Thus, in sharp contrast to the overall overestimation of risk in many populations a serious underestimation of cardiovascular risk for less affluent or deprived groups within these populations is no surprise. Recent examples include the Scottish heart health extended cohort (SHHEC) study and another prospective study by Brindle from the West of Scotland.³⁻²⁰ The authors of these studies warn that the systematic underestimation of risk in socially deprived individuals could be misleading and may exacerbate the social gradients of disease via relative undertreatment of the most needy.

CONCLUSION

Efforts to improve risk scores should not be limited to methodological issues and calibration. Further causal factors of diseases need to be considered. These are not limited to biomarkers and behavioural points but include socioeconomic status. The recognition that an individual's circumstances and

status interact with biological risk factors offers opportunities for refined risk prediction and prevention.

In tune with Rudolf Virchow's remark that medicine was “a social science, and politics nothing but medicine on a grand scale”, the measures required are not confined to the health care systems and allocation of preventive medical treatment—concerted political action is also needed. At a societal level measures should tackle risk factors with high population attributable risk percentage (smoking and obesity) and seek more equitable distribution of public and private resources.

In the best case, refined risk scores will gain acceptance and help to individually target resources for rigorous management of known risk factors supported by public health measures that address the social gradient of health.

Conflict of interest: none

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