students in public and private schools was 18.3% and 8.1%, respectively—perhaps because of improved information dissemination and greater stress on health education in private schools compared with public schools. In all, 62% of adolescents reported their reason for smoking as enjoyment, while 18% claimed to have been influenced by advertisements to begin smoking. The majority of students (61.3%) were smoking with their friends. In this study adolescents also reported family tobacco use: father 19.8%, mother 27.8%, brother 21.0%, and uncle 27.1%. Multiple logistic regression analysis of factors associated with smoking revealed that after adjustment for age, ethnicity, and place of residence, students in public schools were more likely to be smokers compared with those in private schools (adjusted odds ratio [OR] = 1.6; 95% CI: 1.0, 2.7). Adolescents were more likely to be smokers if their peers were smokers (adjusted OR = 6.2; 95% CI: 3.91, 9.9). Boys who spent most of their leisure time outside their homes were more prone to smoke cigarettes (adjusted 95% CI: 3.9, 95% CI: 1.2, 13.2) as were those who had a smoker in the family (adjusted OR = 1.7; 95% CI: 1.1, 2.8). During adolescence, tobacco use by peers may create a positive image of smoking and create easy access to cigarettes, especially in developing countries where there are no restrictive laws on the sale of cigarette to minors. The findings presented in this study are consistent with other studies conducted on adolescent smoking behaviour, which showed that parents, siblings, and peers are powerful influences for adolescent smoking. Smoking is usually initiated during adolescence and being amenable to behaviour modification it should become a public health priority to educate adolescents and parents regarding hazards of smoking in Pakistan and other developing countries.

References
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Cardiovascular risk assessment—time to look beyond cohort studies
From PETER M BRINDLE1 and TIM A HOLT2

Sirs—In the April issue of the International Journal of Epidemiology, Hans-Werner Hense provides a comprehensive treatise on the current state of cardiovascular risk assessments. He highlights the problems with using risk scores derived from epidemiological data to target preventive treatment at highest risk individuals. His comments add to the growing literature recognizing that the risk assessment methods used in current treatment guidelines do not provide an accurate assessment of an individual’s true risk. Hense identifies some of these potential sources of inaccuracy: the variation of cardiovascular risk between populations, using predictions based on assessment of risk factors at one occasion only, the confusing variety of endpoints used in different risk scoring methods, and the ‘contamination’ of risk predictions by risk-reducing treatments such as blood pressure lowering drugs. Hense also highlights the important, but often unrecognized, implications of basing treatment on different thresholds of risk. For example, when the threshold is ≥30% 10-year risk of coronary heart disease, around 84% of the disease events may occur in the ‘low risk’ group—people who might potentially be reassured by the decision that treatment was not indicated for their level of risk. When the threshold is ≥15%, this false negative rate falls to 25%, but the number identified as being at high risk yet do not have a cardiac event rises from 6% to 45%. Hense is right to say that this information is implicit in the particular thresholds that are chosen, but unfortunately guideline authors or practising clinicians are rarely so explicit. Clinicians might wrongly assume that population screening to identify high risk individuals is supported by evidence of effectiveness and meets the basic requirements of a screening test.

As well as listing the problems with cardiovascular risk assessment, Hense offers some solutions. These include the re-calibration of risk functions to regional event rates, and the pooling of cohort studies to limit the influence of regression dilution bias. He identifies the approach adopted by the SCORE (Systemic Coronary Risk Evaluation) investigators of pooling data from 12 European cohorts, and providing risk assessment charts for high and low risk countries. Unfortunately, the SCORE approach is limited by the use of cardiovascular death as its endpoint and it does not have an indicator variable taking into account treatment effects. The SCORE project represents an impressive collaboration that will have entailed a considerable amount of work to obtain, clean, and pool such a diverse collection of datasets. However, it is not certain that the advantages over the available Framingham scores are sufficient to have justified such effort. A simpler approach might have been to use the published Framingham score that adjusts for hypertension treatment effects, and re-calibrate it for different regions within Europe using a method previously described.

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socio-economic deprivation or are from black and minority ethnic groups. Consequently, the SCORE approach only represents a minor tweaking of the ‘one size fits all’ approach.

To improve the discrimination of risk scores, we need a better understanding of the cardiovascular disease process and Hense suggests including new biochemical and other variables to identify sub-clinical disease. Although the predictive accuracy may be improved, the inclusion of additional variables requiring specialized equipment will cause the acceptability and clinical application of the risk assessment tool to suffer. Collection of basic risk factor information such as high-density lipoprotein cholesterol by primary care teams is at best inconsistent, so inclusion of these ‘new’ risk factors in a clinically valid risk score may not improve population screening in the short or medium term.  

It is unlikely that risk scores derived from cohort studies designed primarily to investigate aetiological rather than prognostic factors related to cardiovascular disease will ever be sufficiently flexible to fully take into account the issues of generalizability, measurement error, treatment effects, and temporal changes in disease incidence. Increasingly large volumes of data related to cardiovascular disease are now routinely collected in standardized form from primary care as a way of monitoring the performance and quality of primary care practitioners. Information technology systems are now sufficiently sophisticated for data held at the practice level to be remotely accessed and analysed by a central system. Risk factor and outcome information collected on millions of individuals will provide an extremely powerful resource for developing risk prediction models. If details of ethnicity were collected, this would remove the problem of a shortage of relevant incidence data within European countries. New, adaptable statistical approaches are required, such as neural networks that are able to modify their predictions or ‘learn’ from new risk factor and outcome information from these routine data sources.  

There is no doubt that using a multi-factorial score to guide the clinician in targeting preventive treatment is preferable to managing patients on the basis of arbitrary levels of single risk factors. The use of various risk scoring methods can play an important part in educating the clinician and the patient about the contribution that lifestyle and physiological measures make to overall risk, and also help with understanding the benefits of preventive intervention. However, the development of more accurate and practical risk scoring methods that are relevant to primary care teams has stalled. A single risk assessment method, such as the Framingham coronary risk score, that is not tailored to the individual situation of the patient, does not provide patients and their doctors with sufficiently accurate information to make major treatment decisions. The pooling of many similar cohort studies to derive a score adapted to national levels of cardiovascular disease offers only a limited advance. Much greater imagination is required to make the most of the huge volumes of regularly updated information on cardiovascular disease that is routinely collected in primary care. The data could be used to devise locally adaptable risk assessment methods that automatically calculate an individual’s risk from the available data. Doctors and nurses perform risk assessments with only moderate accuracy, so this automation would take the process of performing the risk assessment from the time-limited doctor’s consultation and allow the creation of a register of high risk patients. The seamless integration with the consultation, of a continually updated risk score derived from patients in primary care, would overcome the major deficiency of even the most accurate risk scoring tool—that is of being used incorrectly or not being used at all.

References