rather than incident cases, where the OR is required. Though in certain circumstances OR is an approximation to RR, they are not the same index. More importantly, the two types of study involve different considerations in their design, and they are subject to different types of biases which need to be taken into account in both design and analysis. Use of the term ‘case-control study’ implies a backwards-looking study, which is not how any of the studies cited were carried out. They are forwards-looking, with ‘exposure’ being defined as ‘ICSI fertilisation’,5 being a gastroenterologist,7 a child having had a minor injury,9 and parental bereavement in childhood.10

It is easy to see how, for some of the examples quoted, the term ‘case-control study’ has been thought appropriate. How better to describe children sustaining minor injuries, or children conceived via ICSI, than as cases? And from that, how natural to describe the comparison group as controls. The danger is that the term ‘case-control study’ will come to mean any epidemiological study where there is a comparison between two groups of people, irrespective of whether they are defined by disease status or exposure status, and irrespective of whether, having defined the two groups, the subsequent direction of the study is forwards or backwards.

Using the same words to describe studies with diametrically opposite study designs is a recipe for confusion at best. Since it seems unlikely that (mis-)use of the term will go away, some clarity would be introduced (however much it might grate with ‘classical’ epidemiologists) if we used the terms ‘retrospective’ and ‘prospective’ in future to qualify our case-control studies. At least we would then know in which direction we were looking.

‘When I use a word,’ Humpty Dumpty said in a rather scornful tone, ‘it means just what I choose it to mean—neither more nor less.’14

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Smoking among high school adolescents in Karachi, Pakistan

From SHAFQUAT ROZI and SAEED AKHTAR

Sirs—Tobacco use is the single most important preventable cause of disease and causes 3.5 million premature deaths worldwide. Tobacco use primarily begins in early adolescence, reportedly before the time of high school graduation.1 Factors that commonly play a role in initiation of smoking among adolescents include social factors, smoking among family members, peers, teachers, psychological relaxation, pleasure, and economic factors.2,3

In this communication we report the prevalence of and factors associated with smoking among adolescents in inner city Karachi, Pakistan. Self-reported smoking status was assessed based on a 30-day prevalence of cigarette smoking (whether or not one had smoked a cigarette in the past 30 days).4 A two-stage cluster sample stratified by school type was used. We recruited 772 male secondary school students from 33 schools (17 public and 16 private) in inner city Karachi. A structured questionnaire was administered by trained data collectors.

Mean (±SD) age (years) of students was 14.8 (±0.1). The mean (±SD) age (years) of smoking initiation was 13.1 (±0.2). The overall prevalence of smoking among school-going male adolescents was 13.7%. Prevalence of smoking among male

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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 OR = 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 in the family (adjusted OR = 1.7; 95% CI: 1.1, 2.8). 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.

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Advance Access publication 22 April 2004
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.1 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.2–3 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%.2 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.4

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.5 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.6,7 Additionally, the SCORE algorithm in its current form cannot be used in many inner city family practices where the majority of the patients live in areas of

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