Spotlight

I looked into your eyes …

*Tien Yin Wong, Ronald Klein, David J Couper, Lawton S Cooper, Eyal Shahar, Larry D Hubbard, Marion R Wofford, A Richey Sharrett. Retinal microvascular abnormalities and incident stroke: the Atherosclerosis Risk in Communities Study. Lancet 2001;***358:***1134–40.*

The neuro-epithelium of the retina and its blood-supply are 'externalized' compartments of the brain. Cerebral and retinal vessels share common anatomy and physiology. Therefore, inspection of fundus colour photographs appears a promising way of non-invasively assessing the status of the intracerebral microvasculature. This study was conducted during the third round of examinations in the large prospective ARIC study (n = 10 358) to investigate whether alterations of retinal vessels show associations with the risk of incident stroke occurring over a median follow-up of 3.5 years (110 cases). Single 45° retinal photographs were taken of a randomly selected eye and were submitted to a standardized grading and classification of fundal abnormalities such as vascular narrowing, nicking, haemorrhages, microaneurysms, exudates and others. In addition, a semi-quantitative method was used to estimate generalized arteriolar narrowing from photos converted to digital images by high-resolution scanning. A relative measure, the arteriolar-to-venule ratio (AVR) was derived from measurement of all vessels coursing through a defined area of the eye. The absolute risk of incident stroke over 3 years ranged from below 1% to well over 3 to 5% depending on retinal vessel abnormalities. The multivariate relative hazards adjusting for 6-year average blood pressure, smoking, diabetes and other established stroke risk factors were significantly raised in individuals showing any type of retinovascular alteration. For example, the HRR was 2.58 (95% CI : 1.59–4.20) for any retinopathy, 3.11 (1.71–5.65) for microaneurysms and 3.08 (1.42–6.68) for soft exudates. The relative risk of stroke increased also with decreasing AVR. The associations were similar for ischaemic strokes specifically, and for individuals with hypertension and with diabetes. The authors discuss the role of retinal microvascular abnormalities as markers of hypertension, inflammation, and endothelial dysfunction. They emphasize the potential of this method as a tool for the non-invasive assessment of the status of the intracerebral microcirculation. Some limitations of the study need, however, to be considered. The small number of total stroke cases prohibited analyses for haemorrhagic strokes. The possibility of residual confounding due to hypertension as a major determinant of retinovascular alterations was not adequately addressed and selection bias related to concurrent ocular conditions rendering retinal photographs ungradable may have distorted the results. However, these limitations are unlikely to account for the consistency and strength of the associations observed in this study. Why not look a bit deeper into your patients' eyes?

HW HENSE, Münster

Smoking: a choice for coping

*Stead M, MacAskill S, MacKintosh A.-M, Reece J, Eadie D. 'It's as if you're locked in': qualitative explanations for area effects on smoking in disadvantaged communities. Health & Place 2001;***7:***333–343.*

Most epidemiological studies which consider smoking designate it as an independent variable or view it as something that needs to be 'controlled' for along with a host of other factors. However, a different slant on smoking views this key health behaviour as an outcome in its own right. To what extent is smoking a result of environmental or individual disadvantage; and if smoking is a lifestyle 'choice', do some people have more choices than others?

This paper by Stead and colleagues contributes to a growing body of literature which considers to what extent health behaviours and outcomes are the result of individual factors and whether there are independent effects derived from living in a disadvantaged community. It reports the results of qualitative focus groups conducted with smokers and non-smokers in three Glasgow communities with high levels of deprivation. A broad range of smoking-related issues were discussed. The results indicate that smoking is used as a means of coping with living in a stressful, disadvantaged area, where unlike many other things, tobacco is in abundant supply. Powerful community norms reinforced smoking as a behaviour, discouraging and undermining cessation. Socializing was strongly bound up with drinking, drug-taking and especially smoking. Respondents reported strong normative pressure to smoke, with non-smokers saying they often needed a legitimate reason, such as asthma, for not smoking—as one smoker said 'it's ma only pleasure'.

MARY SHAW, Bristol

Smoking: a genetic condition?

*Osler M, Holst C, Prescott E, Sorensen TIA. Influence of genes and family environment on adult smoking behavior assessed in and adoptive study. Genetic Epidemiology 2001;***21:***193–200.*

The factors that determine smoking behaviour are not fully understood and although environmental factors are likely to be important twin studies have suggested that genetic factors may also play a role. A review of over 17 500 monozygotic (MZ) and dizygotic (DZ) twin pairs from 14 different studies concluded that genetic, family-environmental, and individual-specific environmental factors accounted for 56%, 24%, 20%, respectively, of the liability to smoke (*Nicotine Tobacco Res* 1999; **1(suppl):**s51–7). However, comparing levels of concordance between MZ and DZ twin pairs may not readily distinguish between genetic and environmental influences in areas of behaviour. MZ twins spend more time together and share peer groups more closely than do DZ twins (*Psychol Med* 1998;**28:** 625–33).

Osler *et al.* have assessed the possibility of a genetic influence on smoking behaviour in an adoption study. The smoking behaviour amongst adult adoptees (aged 35–76) from the Danish Adoption Register was compared with smoking behaviour amongst adoptive and biological parents during the

time the adoptees were school children (data obtained from self-report by the parents or by their children) and with contemporary smoking behaviour amongst the adoptees' biological full and half sibs. Subjects were recruited from a sub-sample (N = 840) of the adoption register who had participated in a study of obesity.

Ever smoking (but not current smoking) amongst the subjects was weakly associated with smoking (when the subject was a school child) amongst adoptive and biological fathers but not amongst adoptive mothers. Ever smoking and current smoking were associated with smoking (when the subject was a school child) amongst the biological mothers: odds ratio (95% confidence interval) 1.42 (0.91, 2.20) for ever smoking and 1.36 (0.91, 2.06) for current smoking. Smoking behaviour amongst the subjects was strongly associated with contemporary smoking behaviour amongst their full biological sibs with weaker associations amongst their half sibs.

The association between biological maternal smoking and adoptee smoking may reflect genetic factors or the effects of exposure to maternal smoking during pregnancy and in early infancy. The authors of this paper suggest that their finding of a weak association between adoptees' and maternal half-siblings' smoking behaviour 'speaks against the importance of pre-adoptive pre- or post-natal influences'. However, the magnitude of these associations were similar to those for paternal half-sibs. The authors conclude that their results suggest that there is a genetic influence on smoking within the same generation. The absence of comparisons between adoptees and their adoptive sibs is a major weakness of this study. Smoking behaviour is strongly associated with social class and in countries, such as the UK and US, where recent decades have seen big drops in smoking prevalence smoking is now almost the preserve of the most deprived groups in society. Tackling inequalities may be a more important public health input into reducing smoking behaviour and improving health than looking to genetic interventions for behaviour modification.

DEBBIE LAWLOR, Bristol

Sudden cardiac death on the decline in the US

*Zhi-Jie Zheng, Janet B Croft, Wayne H Giles, George A Mensah. Sudden cardiac death in the United States, 1989 to 1998. Circulation 2001;***104:***2158–2163.*

Sudden cardiac death (SCD) is the most common lethal manifestation of heart disease, and in many cases it is the descent's first and only symptom. The high incidence combined with the low success rate of resuscitation make SCD a major problem in clinical medicine and public health. This study investigated the most recent epidemiological features and secular trends of SCD in the US. SCD was identified from US official mortality data compiled by the National Center for Health Statistics using a definition of any cardiac death occurring out of hospital, in the emergency room or dead on arrival. Of the 719 456 cardiac deaths in 1998 occurring among those aged over 35 years, 456 076 (63.3%) were defined as SCD. The overall proportion was very similar for men (62.9%) and women (63.8%) but with higher proportions for men at younger ages.

Relative changes from 1989 to 1998 indicated that SCD declined in all age groups except for women between 35 to 44 years who showed a 21.1% rise in SCD rate. Generally, age-adjusted SCD rates declined more strongly in men (–11.7%) than in women (–5.8%). This decline was observed in all race groups except in American Indian/Alaska Native women who experienced declining rates until 1996 with an increase thereafter. Within the overall 10 year decline in rates of SCD there was a decline in the proportion of that occurred in the hospital or the emergency room and an increase in the proportion that occurred outside of hospital.

Despite the overall positive trends the authors suggest that trends in women are disturbing as more than half of their sudden deaths occurred out of hospital, their overall decline of SCD rates was only half that of men and among young women rates have increased over the last 10 years. The reasons for these differences are unknown. The authors discuss a number of study limitations mostly related to the use of death certificates. Thus, the lack of medical validation and data on the time of onset of disease symptoms may have introduced misclassification bias. They conclude that 'These limitations notwithstanding, this surveillance report is valuable for its provision of the most recently available statistics for SCD for the entire US adult population'.

HW HENSE, Münster

What we ask and what people hear

*Mallinson S. Listening to respondents: a qualitative assessment of the Short-Form 36 Health Status Questionnaire. Social Science and Medicine 2002;***54:***11–21.*

Questions that ask people to describe their own health are central to surveys in epidemiology, policy and allied disciplines. Among social scientists there is increasing interest in the validity of such measures, especially in the light of evidence about the contextual and varied nature of people's views of health and illness (for instance, see Lindenbaum S & Lock M (eds) 1993. Knowledge, Power, and Practice: The Anthropology of Medicine and Everyday Life. Berkeley: University of California Press). Mallinson's paper tackles this issue head-on: 'How can one know if people hear the questions in the manner they were intended without doing some kind of data checks between respondents, or in different situations, or within individuals over time?'. Drawing on sociological and psychological approaches, the study explores the commonly used Short-Form 36 (SF-36) questionnaire through face-to-face interviews with 56 people aged 65 years and over. The physical functioning and general health perceptions dimension of the SF-36 are the focus for analysis of interview transcripts, and findings suggest that people's responses to the SF-36 are neither clear-cut nor consistent. Reasons for this include: technical flaws in question construction; responses reflecting varieties of adaptive changes to chronic illness or disability; and the fact that people interpret and react to these questions in different ways to one another and across time. This leads Mallinson to suggest that 'qualitative validation techniques' should figure more highly as part of procedures for validation of health questionnaires.

The paper reconfirms existing disquiet about the use of subjective health measures. In health-related research this concern is rapidly gaining momentum, possibly partly fuelled by increasing input from social and behavioural scientists. The suggestion

that validation techniques from these areas may help to resolve such unease is a welcome addition to the debate. Mallinson's paper should serve as an interesting and perhaps salutary read for all those who engage with survey material at any level.

RACHEL GOOBERMAN-HILL, Bristol

Dietary saturated fats may not be harmful in the absence of other risk factors

*Glew RH, Williams M, Conn CA, Cadena SM, Crossey M, Okolo SN, VanderJagt DJ. Cardiovascular disease risk factors and diet of Fulani pastoralists of northern Nigeria. Am J Clin Nutr 2001;***74:***730–6.*

A strong association between dietary fat intake (particularly saturated fat), raised serum cholesterol levels, and cardiovascular disease risk has been demonstrated in many studies. Most of these studies have been undertaken on populations from industrialized countries. The study by Glew *et al*. is unique and interesting in that it reports serum lipid profiles, anthropometric measurements and dietary assessments in a group of seminomadic pastoralists. The study was carried out in 4 Fulani hamlets in the Jos Plateau (2200 m above sea level) of Nigeria with a total adult population of 600–650. All of the subjects were Fulani, seminomadic pastoralists whose main occupation was cattle rearing. This population are very physically active, rarely smoke or drink alcohol but have a diet (mainly cattle meat, dairy produce and palm oil) which is high in saturated fat.

Blood samples were spun and frozen on site before being transported for analysis. Dietary assessment was by interviewer administered food frequency questionnaire and 7-day dietary recall. The authors comment that concerns about the validity of dietary recall may not be appropriate in a population that has a very stable diet. Nutrient composition of reported food intake was analysed using specialized computer software applying standardized references for food composition in Africa (from US department of Agriculture database) and Fulani recipes and portion sizes based on direct observation of meals by a dietician.

All healthy adults from the 4 hamlets were invited to participate and 121 did so. Blood results were obtained on 42 men mean age 34 years (range 18–94) and 79 women mean age 32 years (range 15–77) and complete dietary details on 22 men and 73 women. Total energy intake in both men and women was low—mean daily intake 6980 kJ (SD 1106) for men (70% of mean daily intake recommended for a healthy adult male American) and 6213 kJ (SD 892) for women (75% of mean daily intake recommended for a healthy adult female American). The proportion of energy derived from fat was high—mean 48% (SD 0.08) for men and mean 47% (SD 0.07) for women with over half of this being saturated fat in both sexes; in absolute terms this would be similar to or higher than that of developed populations. Despite this total serum cholesterol levels were low—3.50 mmol/l in both men and women—as were mean low-density lipoprotein cholesterol levels—1.84 mmol/l in men and 1.88 mmol/l in women (the values for low density lipoprotein cholesterol are below the 5th percentile of values for African Americans). Mean body mass index was low in both sexes (20.0 kg/m^2 in both men and women) and percentage body fat was particularly low in men (12.7% in men and 28.5% in women).

The authors conclude that 'the low energy intake, active lifestyle, and rural existence of these seminomadic people override whatever negative effects their diets high in fat and saturated fatty acids might have on their risk of cardiovascular disease'. Whilst these conclusions perhaps overstate the case, given the small numbers and cross-sectional design with no measure of cardiovascular disease, the authors are right to point out the value and feasibility of studies of populations with very different risk factor profiles from those traditionally studied in epidemiological research.

DEBBIE LAWLOR, Bristol

Do medical students benefit from cadaver dissection?

*Jones LS, Paulman LE, Thadani R, Terracio L. Medical student dissection of cadavers improves performance on practical exams but not on the NBME anatomy subject exam. Med Educ Online [serial online] 2001;***6:***2. http://www.med-ed-online.org*

Medical students at the University of South Carolina are required to learn anatomy from a cadaver in their first year. As part of the learning process each student has to dissect one third of the cadaver; the remaining two thirds are learnt from observation of cadavers dissected by fellow students. Assignment to which third of the body the student dissects is done alphabetically on last name. In this study the performance for each student in practical anatomy exams and in the National Board of Medical Education (NBME) anatomy subject exam was assessed as a function of the regions of the body each student actually dissected in order to determine the effect of dissection. In the practical exams the proportion of correct answers on dissected regions was 88.6% compared with 84.9% for regions that had been dissected by someone else ($p < 0.01$). Equivalent results for the NBME exam were 69.1% compared with 67.6% (difference described as non-significant). The NBME exam is a written paper whereas the practical exams involve identification of anatomical structures on a cadaver. The authors conclude that their results indicate that dissection has different effects in the two different exams and that the value of self-dissection should be based on the most important outcome. They imply that the NBME anatomy exam is a better reflection of student learning and subsequent performance in later, clinical rotations, whereas the practical exams are a better measure of student understanding and learning of the three-dimensional concepts taught in gross anatomy. Given the high level of correct answers in both exams and the small difference in both between 'dissectors' and 'non-dissectors' it is hard to be convinced that self-dissection has any important benefit in either exam. A more important question concerns the need (at all) for cadaver based learning in medical education and the impact of this form of learning on future doctor performance.

DEBBIE LAWLOR, Bristol

Cigarettes, prostates and Beagles

*Hickey K, Do KA, Green A. Smoking and prostate cancer. Epidemiol Rev. 2001;***23:***115–25.*

The positive association between smoking and several cancers is well established but whether there is an association with

prostate cancer is less clear. This recent review helps to clarify the situation. A total of 78 studies (prospective and retrospective cohort studies, nested case-control and true case-control studies) were identified, 13 of which were subsequently omitted because it was not possible to calculate the effect estimate or 95% confidence limits. All studies were scored for quality—design, selection, measurement of exposure and disease, appropriate adjustment for confounders and statistical methods. For most prospective cohort studies and all nested case-control studies there was no association between current smoking and incident prostate cancer but for prospective cohort studies which had prostate cancer mortality as an outcome weak positive associations were found. The majority of case-control studies reported null associations. In commenting on the findings the authors highlight the methodological shortcomings of the studies. If smokers have more contact with health services this could account for the positive findings in some of the incidence studies. Smokers are more likely to die than non-smokers and if prostate cancer was recorded as the cause of death then this could explain the mortality findings although this did not occur in the one study which examined this issue. Finally smokers may receive less aggressive treatment than non-smokers—again potentially accounting for the positive association with fatal prostate cancer—and this was not considered in the studies reviewed. A more serious omission was appropriate assessment or analysis of past and present smoking status; failure to include updated smoking details could potentially bias the results towards the null. There was also a concern that, dietary fat, which has been hypothesized as a risk factor for prostate cancer, was infrequently included in analysis. There was no evidence of publication bias.

Despite the deficiencies, some of which could not be addressed in the studies reviewed, the weight of evidence indicates that smoking is not a risk factor for incident prostate cancer. This is consistent with the lack of evidence for the four biologic mechanisms which have been postulated to explain how smoking could cause prostate cancer: toxicity from cadmium in tobacco smoke; an anti-oestrogen effect of smoking acting in tandem with the putative hormone dependency of prostate cancer; a mutation in the tumour suppressor gene p.53; and depressed immune function. The cause of prostate cancer therefore remains elusive. Another interesting detail can be gleaned from this study. The preface to this issue of *Epidemiologic Reviews* states that only humans and dogs have any significant risk of dying of prostate cancer so it is now possible to reassure all those beagles that puffed away in the interests of medical research that while tobacco may have killed them it probably didn't harm their prostates.

PETER McCARRON, Washington DC

Does childhood obesity predict adult health risks?

*Wright CM, Parker L, Lamont D, Craft AW. Implications of childhood obesity for adult health: findings from thousand families cohort study. Br Med J 2001;***323:***1280–4.*

Few epidemiological studies have followed up subjects from birth to the age of 50, and the rare ones which succeed should provide invaluable information. Wright and colleagues followed up a 1947 birth cohort from the North of England and in this study examine whether childhood obesity is associated with increased risk of adult obesity and cardiovascular disease risk factors. Of the 1142 children included at birth, 688 and 628 were measured at age 9 and 13 years. At age 50, height and weight data were available for 529, and 412 attended for clinical examination and blood sampling. The authors found that body mass index (BMI) at age 9 was correlated with BMI at age 50 ($r = 0.24$, $p < 0.0001$) but not with percentage body fat at 50 ($r = 0.10$, $p = 0.07$). After adjusting for adult BMI, BMI at age 9 showed an inverse association with lipids, 2 hour glucose and serum insulin in both sexes, but after adjusting for adult percentage fat, only the inverse associations with triglycerides and total cholesterol in women remained significant. Children who were obese at 13 showed an increased risk of obesity as adults but no excess of adult cardiovascular disease risk factors. Furthermore, thinness in childhood did not protect against adult fatness and was associated with adult cardiovascular disease risk factors. The authors suggest that whole population interventions directed at reducing BMI in childhood may not benefit adult health.

While acknowledging that BMI is not a good measure of fatness or obesity (as muscle mass and body frame also contribute to BMI), no data on percentage body fat at childhood (age 9) and teenage (age 13) were available. Although the authors showed that BMI and percentage fat at age 50 was strongly correlated ($r = 0.63$, $p < 0.01$), they had no data to study such correlation at age 9 or 13. The question of whether childhood percentage fat would show the same inverse relations with risk factors at age 50 as childhood BMI in this cohort remains unanswered.

Although the authors used the term 'childhood obesity' in the title of their paper, what is being studied is 'childhood BMI'. Better measures of childhood obesity and thinness are needed.

These findings support the need for life course epidemiology. But the high attrition rate of over 50% in follow up in this study is not encouraging, and the potential biases could be substantial. It should also be pointed out that morbidity and mortality outcomes were not analysed. This was because the size of the birth cohort is small, and the follow-up relatively 'short': there were only 47 adult deaths.

TH LAM, Hong Kong