

group has to include only one member, a health economist, whose role is to promote the social viewpoint. The health economists are often relatively junior, new to the disease area, and struggling with a lack of economic evidence. For cost effectiveness to underpin NICE guidelines in these circumstances is particularly challenging.

Recommendations made within a clinical guideline are graded according to the strength of the evidence on which they are based. The highest grades are afforded to recommendations based on meta-analysis of randomised controlled trials and the lowest grade to recommendations based on expert opinion, including the view of the development group. This classification also has the effect of reducing the impact of cost effectiveness considerations: health economic evidence is often sparse in established clinical areas and, where it does exist, is of variable quality. Rarely is economic evidence based entirely on clinical trials: most economic analyses require additional data sources or assumptions. Members of the guideline development group, who may wish to downplay economic evidence, can use the grading system to this end by claiming that clinical evidence is of a higher grade. Qualitative evaluation has identified exactly this tendency in the Netherlands.⁸

We applaud the efforts of NICE and the guideline development groups to consider cost effectiveness. However, the absence of evidence on the cost effectiveness of guideline recommendations is not an adequate rationale for issuing guidelines as though they had no implications for resources. One solution might be for NICE to delineate clearly the individual viewpoints of patients and society and allocate expertise to tasks that are appropriate in the light of this distinction. In this scenario, collaborating centres would be commissioned to produce wholly clinical guidelines, at arm's length from NICE. This work would provide a crucially important foundation for subsequent cost effectiveness assessment undertaken by specialist academic units. Clinical guidelines that carry the NICE stamp of approval—and its associated weight in the NHS—should be produced by guideline appraisal com-

mittees, analogous to NICE technology appraisal committees, based on consideration of the best available evidence on clinical and cost effectiveness. A membership that includes expertise in a broad range of clinical specialties, health economics, public health, and statistics, together with representatives of NHS organisations, can be expected to make better recommendations that truly reflect the societal viewpoint that NICE must reflect.

Such an approach would promote consistency between the appraisal and guidelines functions of NICE, make the basis for recommendations transparent, and avoid accusations that NICE guidelines are wish lists created by panels of clinical experts that threaten the efficient and equitable use of scarce NHS resources.⁹

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Competing interests: School of Health and Related Research receives funding from NICE for work relating both to clinical guidelines and to technology appraisals.

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Smoking and blindness

Strong evidence for the link, but public awareness lags

While most people and many patients attending eye clinics recognise many adverse health hazards of tobacco smoking, they remain largely unaware of its link with blindness. Although smoking is associated with several eye diseases, including nuclear cataract^{w1} w2 and thyroid eye disease,^{w3} the most common cause of smoking related blindness is age related macular degeneration, which results in severe irreversible loss of central vision. Current treatment options are of only partial benefit to selected patients. Identifying modifiable risk factors to inform efforts for prevention is a priority.

A risk factor is generally judged to be a cause of disease if certain causality criteria are fulfilled.^{w4} Applying commonly used criteria^{w4} to available evidence provides strong evidence of a causal link between tobacco smoking and age related macular degenera-

tion. The strength of association is confirmed in a pooled analysis of data from three cross sectional studies, totalling 12 468 participants, in which current smokers had a significant threefold to fourfold increased age adjusted risk of age related macular degeneration compared with never smokers.¹ By way of comparison, although the relative risks associated with smoking for lung cancer and chronic obstructive pulmonary disease are in excess of 20, the relative risk for ischaemic heart disease in men is only 1.6.^{w5} Consistency of effect is demonstrated as smoking was the strongest environmental risk factor for age related macular degeneration across these three different

study populations in Australia, North America, and Europe.²⁻⁴ A temporal relation between exposure and outcome was established through long term follow up in these cohorts.⁵⁻⁷ A dose-response relation between exposure to smoking and age related macular degeneration is demonstrated as the risk of early disease increases with number of pack years.⁶⁻⁷ Finally, this causal association is biologically plausible, as age related macular degeneration may reflect accumulated oxidative damage in the retina and smoking is known to impede the protective effects of antioxidants and to reduce macular pigment density.⁸

Owen et al estimated 214 000 UK residents to have visual impairment (best visual acuity 6/18-3/60 Snellen) and 71 000 individuals to be blind (better eye visual acuity <3/60 Snellen) because of age related macular degeneration.⁹ We estimate that 53 900 United Kingdom residents older than 69 years may have visual impairment because of age related macular degeneration attributable to smoking of whom 17 800 are blind (see table and methods on bmj.com).^{1-9 w6 w9}

Randomised controlled trials examining whether smoking cessation interventions reduce incidence or progression of smoking related diseases are problematic. Observational studies show a protective effect of smoking cessation on the development of age related macular degeneration, as former smokers have an only slightly increased risk of age related macular degeneration compared with never smokers.¹ The reversibility of this association in smokers with age related macular degeneration in one eye has important implications for prevention of late macular involvement in the second eye. In addition, continuing smoking is associated with poorer outcome after photocoagulation with argon laser.¹⁰ Continued smoking could perhaps also adversely affect the long term response to newer treatments such as photodynamic therapy.

Robust evidence indicates that smoking cessation support results in higher abstinence rates.^{w8} Guidelines recommend that smokers are referred to professional smoking cessation services and should generally be offered nicotine replacement therapy.^{w8} Many diabetes, cardiac, and respiratory NHS clinics now incorporate smoking cessation support into their services and ophthalmology or optometry services could follow likewise. The acceptability of this intervention among eye care personnel in the United States is high, but time and knowledge constraints may hinder implementation.¹¹

Primary smoking prevention is perhaps even more important. In New Zealand, publicity about smoking and blindness resulted in increased telephone calls to the national Quitline^{w9} and a television campaign incorporating the (slightly modified) Australian eye advertisement (www.quitnow.info.au/script/eye.html) was considered more successful than other advertisements relating smoking to stroke and heart disease (N Wilson, personal communication, 2003). A sustained public health campaign in the United Kingdom is warranted to increase awareness of the ocular hazards associated with smoking, "North West Action on Smoking and Health" (www.nwash.co.uk) has launched a leaflet describing these risks alongside user friendly advice on smoking cessation. The Royal College of Ophthalmologists supports this initiative. More novel, varied, and specific pack warnings of the impact of smoking on health,^{w10} including eyesight, might help as

primary prevention efforts. Warnings targeted at specific concerns may be more effective than current general statements—"Smoking is a major cause of blindness" has been suggested.¹² The finding that smokers develop age related macular degeneration around 10 years earlier than non-smokers⁵ could also be a potent message in public awareness campaigns.

Tobacco smoking is the prime modifiable risk factor for age related macular degeneration. Evidence indicates that more than a quarter of all cases of age related macular degeneration with blindness or visual impairment are attributable to current or past exposure to smoking. Patients, health professionals, and the public will benefit from greater awareness of this causal association. Smoking cessation advice should be introduced and evaluated. Similarly, research examining the behaviour of smokers as a result of acquired knowledge about smoking and the risk of visual impairment or blindness could usefully inform public health campaigns. Policy initiatives based on these concepts are now clearly needed.

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We are grateful to P McElduff, lecturer in statistics, Evidence for Population Health Unit, University of Manchester for statistical help.

The Retinal Research Endowment Fund, Bolton Hospital NHS Trust supported this work.

Competing interests: RE is the chair (unpaid) of North West Action on Smoking and Health, and the Faculty of Public Health's representative to the Royal College of Physicians Tobacco Group.

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