

# Health impact of “reduced yield” cigarettes: a critical assessment of the epidemiological evidence

Michael J Thun, David M Burns

## Abstract

**Cigarettes with lower machine measured “tar” and nicotine yields have been marketed as “safer” than high tar products over the last four decades, but there is conflicting evidence about the impact of these products on the disease burden caused by smoking. This paper critically examines the epidemiological evidence relevant to the health consequences of “reduced yield” cigarettes. Some epidemiological studies have found attenuated risk of lung cancer but not other diseases, among people who smoke “reduced yield” cigarettes compared to smokers of unfiltered, high yield products. These studies probably overestimate the magnitude of any association with lung cancer by over adjusting for the number of cigarettes smoked per day (one aspect of compensatory smoking), and by not fully considering other differences between smokers of “high yield” and “low yield” cigarettes. Selected cohort studies in the USA and UK show that lung cancer risk continued to increase among older smokers from the 1950s to the 1980s, despite the widespread adoption of lower yield cigarettes. The change to filter tip products did not prevent a progressive increase in lung cancer risk among male smokers who began smoking during and after the second world war compared to the first world war era smokers. National trends in vital statistics data show declining lung cancer death rates in young adults, especially males, in many countries, but the extent to which this is attributable to “reduced yield” cigarettes remains unclear. No studies have adequately assessed whether health claims used to market “reduced yield” cigarettes delay cessation among smokers who might otherwise quit, or increase initiation among non-smokers. There is no convincing evidence that past changes in cigarette design have resulted in an important health benefit to either smokers or the whole population. Tobacco control policies should not allow changes in cigarette design to subvert or distract from interventions proven to reduce the prevalence, intensity, and duration of smoking.**

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Cigarettes with lower machine measured “tar” and nicotine yields have been marketed as “safer” than high tar products over the last four decades,<sup>1,2</sup> but there is limited and conflicting evidence about the net impact of these products on the disease burden caused by smoking. The principal uncertainties are the extent to which compensatory smoking behaviours induced by “reduced yield” cigarettes may offset any putative reductions in the toxicity or carcinogenicity of these products, and whether health claims used to promote “Light” cigarettes may delay cessation among smokers who might otherwise quit, or increase initiation among non-smokers. Epidemiological studies have generally focused narrowly on the intrinsic toxicity and carcinogenicity of “reduced yield” products, rather than on their indirect effects on smoking behaviour or the number of people smoking.

This paper critically examines the epidemiological evidence relevant to the health consequences of “reduced yield” cigarettes. It begins by briefly reviewing the historical development of products with lower tar and nicotine yields, as measured by the standard Federal Trade Commission (FTC) protocol of machine smoking.<sup>3</sup> It then considers the evidence that smokers “compensate” for reductions in machine measured tar and nicotine by increasing the number of cigarettes smoked per day as well as by increasing the puff volume and number of puffs per cigarette. It discusses the strengths and limitations of several epidemiological approaches that have been used to assess the health effects of “reduced yield” cigarettes. These include analytic (cohort and case-control) studies that compare the risks to smokers who use different types of cigarettes; selected cohort studies that illustrate how the lung cancer risk to smokers has increased over time; and analyses of trends in national death rates from lung cancer by age in relation to age specific smoking patterns.

## Historical development of “less hazardous” cigarettes

The postulate that cigarettes with lower tar delivery might be less hazardous emerged from the early research findings on tobacco related diseases. Epidemiological studies repeatedly demonstrated increased lung cancer risk in smokers beginning in the 1950s<sup>4-7</sup>; experiments showed that painting cigarette smoke condensate on the backs of mice produced skin tumours.<sup>8</sup> By 1967, independent scientists and public health authorities recommended to the US Congress that cigarettes with lower

American Cancer Society, Atlanta, Georgia, USA  
M J Thun

Department of Medicine, University of California, San Diego, California, USA  
D M Burns

Correspondence to:  
Michael J Thun MD,  
American Cancer Society,  
1599 Clifton Road, Atlanta,  
GA 30329-4251, USA  
mthun@cancer.org

particulate yield be developed and marketed to smokers who could not quit.<sup>9</sup>

The tobacco industry responded to health concerns about cigarette use first by adding filters to some brands of cigarettes beginning in the 1950s, and then by offering cigarettes that delivered progressively less "tar" as measured by machine smoking.<sup>3-10</sup> Much of the reduction in "tar" (total particulate matter minus nicotine and water) was achieved by the addition of ventilation holes around the filter to dilute the smoke with entrained air. A method of machine smoking that was developed in the 1930s<sup>11</sup> became codified in the Federal Trade Commission (FTC) annual ratings of cigarettes.<sup>3</sup> This protocol specifies fixed smoking parameters for the machine: 35 ml puff volume, 2 second puff duration, 1 puff per minute frequency, and a fixed butt length to which the cigarette is smoked.<sup>12</sup> Brands that yield approximately 1–6 mg of tar per cigarette by the FTC method are referred to as "Ultra-light"; those with approximately 7–15 mg tar as "Light", and those yielding more than 15 mg tar as "Regular" or "Full flavoured".<sup>13</sup> Before the mid 1950s, unfiltered cigarettes typically yielded 25–30 mg tar by the FTC method.

The FTC ratings do not take into account variations in tar and nicotine yield that can be obtained by smokers seeking to maintain a particular intake of nicotine.<sup>3 14-16</sup> Smokers who use "reduced yield" products can increase the amount of nicotine and tar extracted from each cigarette by taking more puffs per cigarette, obstructing the ventilation holes around the filter, and inhaling a larger puff volume more deeply into the lungs.<sup>17</sup> Smokers can also compensate for reduced yield by smoking more cigarettes per day.<sup>17</sup> Internal documents from the tobacco industry express scepticism about the efficacy of filter tip and "lower yield" products in reducing the exposure of smokers, even

during the years when these products were first heavily marketed to assuage the health concerns of smokers.<sup>1 2</sup> A memo from Helmut Wakeham of Philip Morris (dated 24 March 1961) states, "As we know, all too often the smoker who switches to a hi-fi cigarette winds up smoking more units in order to provide himself with the same delivery which he had before".<sup>18</sup>

### Compensatory smoking

Many experimental studies document that smokers who are switched to cigarettes with lower nicotine yield than their usual brand are able to maintain higher plasma concentrations of nicotine metabolites than would be expected from the FTC ratings. Compensation has been demonstrated experimentally in both short term<sup>19-23</sup> and long term studies.<sup>24-29</sup> What is less certain is the extent to which smokers compensate by increasing the number of cigarettes smoked per day as opposed to other behavioural changes that extract more nicotine from each cigarette. This distinction is important with respect to the epidemiological studies, since most of these studies adjust for the number of "reduced yield" cigarettes smoked per day, and may thereby over-control for one aspect of compensatory smoking.<sup>30</sup>

We assessed the relation between daily cigarette consumption and the FTC rating of nicotine yield in several additional large studies, the American Cancer Society Cancer Prevention Study I (CPS-I) cohort, and the 1990 and 1996 California Tobacco Surveys (CTS).<sup>30</sup> In the CPS-I cohort, questionnaires were administered periodically during the follow up to document changes in smoking behaviour or cigarette brand. Based on these data, fig 1 shows the mean change in cigarettes smoked per day in relation to the change in nicotine yield among 169 610 white male smokers who changed brands between enrollment in 1959 and the end of follow up in 1972.<sup>27</sup> Each milligram decrease in machine measured tar yield was associated, on average, with an increase of 2.31 cigarettes smoked per day. This prospective analysis controlled for age, cigarettes smoked per day before the switch, and tar and nicotine yields of the cigarette smoked before the switch. A similar relation was seen in the 1990 and 1996 CTS for cigarette brands with less than 0.95 mg nicotine yield (fig 2). This analysis was restricted to adult smokers, age 25–64 years, who smoked at least five cigarettes daily during the year before the survey and had not attempted to quit smoking in the previous 12 months. The intent was to limit these analyses to persons with relatively stable smoking patterns<sup>31 32</sup> and less likelihood of having changed their smoking because of illness. These studies suggest a small but demonstrable increase in the number of cigarettes smoked per day among smokers of lower yield cigarettes. Previous reports<sup>14 28 29 33 34</sup> have been less consistent, perhaps because of smaller sample sizes and the inclusion of people with less stable smoking patterns.

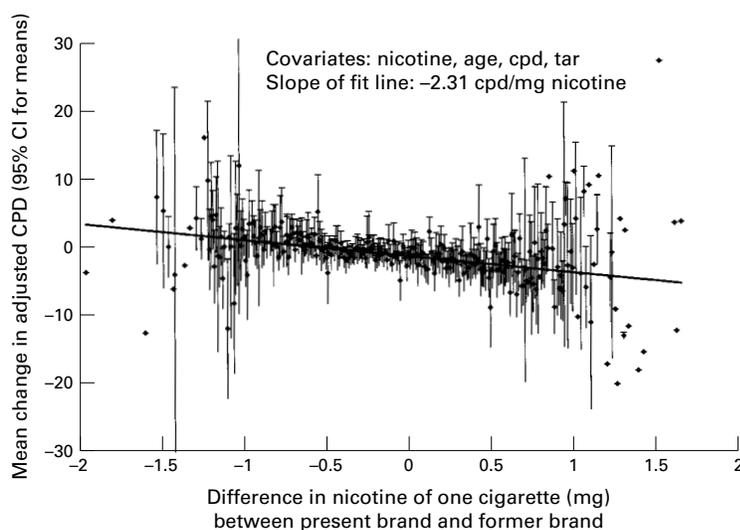


Figure 1 Mean change in adjusted cigarettes smoked per day (CPD) reported for subjects changing brand smoked versus change in machine measured nicotine yield per cigarette: white male smokers ( $n = 169610$ ), American Cancer Society Cancer Prevention Study I (CPS-I) study, followed 1960 to 1972. Each milligram decrease in machine measured tar yield among CPS-I smokers who changed brands between enrollment in 1959 and end of follow up in 1972 was associated with an increase of 2.31 cigarettes smoked per day. Based on 169610 white male smokers.<sup>30</sup>

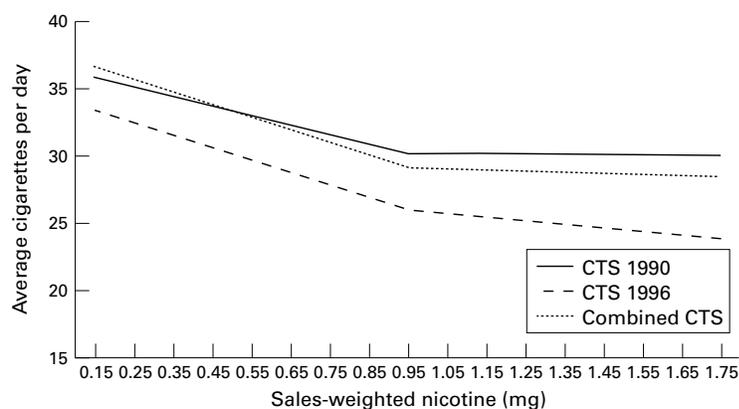


Figure 2 Piecewise linear regression of cigarettes smoked per day by sales-weighted nicotine yield of the brand smoked (California data), illustrating the inverse relation between cigarettes smoked per day and machine measured tar yield below approximately 1 mg. Based on the 1990 and 1996 California Tobacco Surveys.<sup>30</sup>

### Epidemiological studies

#### COHORT AND CASE-CONTROL STUDIES

##### COMPARING DIFFERENT TYPES OF CIGARETTES

Over 50 epidemiological studies have compared disease risks among smokers who use different types of cigarettes.<sup>35-89</sup> These studies, discussed elsewhere,<sup>30</sup> typically measure the occurrence of lung cancer,<sup>35-78</sup> coronary heart disease,<sup>39-42 44 45 51 56 64 67 79-82</sup> and respiratory diseases caused by smoking<sup>39 42 45 67 83-89</sup> in smokers who use filter tip products compared to those who use unfiltered, "high yield" products. Most of these studies report lower lung cancer risk among smokers who use "reduced yield" products, relative to those who smoke unfiltered, "higher yield" cigarettes. They do not consistently report lower risk of coronary heart disease, total stroke, or chronic obstructive pulmonary disease (COPD).

The studies that compare different types of cigarettes have at least two limitations that complicate their interpretation. First, they focus mostly on differences in the intrinsic pathogenicity of "reduced yield" cigarettes compared to unfiltered, higher yield products without adequately considering the indirect adverse effects that lower yield cigarettes may have on smoking behaviour. For example, the studies compare risk among smokers who use different types of cigarettes but do not assess whether some smokers have deferred quitting because of health claims about "reduced yield" products. Lung cancer risk increases exponentially with longer duration of smoking.<sup>90</sup> Factors that delay cessation may outweigh any putative reductions in tar yield, increasing rather than decreasing the lung cancer risk for an individual smoker. Furthermore, health claims used to market "Light" cigarettes could worsen the population burden of disease by trivialising the actual hazards of smoking, thus promoting initiation or resumption of smoking.<sup>1 2</sup>

Secondly, the published cohort and case-control studies may overestimate the magnitude of any attenuation in lung cancer risk by inappropriately controlling for the number of cigarettes smoked per day and by under controlling for other factors that could

reduce risk. In adjusting for the number of cigarettes smoked per day, the epidemiological studies assume that smokers who switch to "reduced yield" cigarettes do not compensate for the lower yield by increasing their daily cigarette consumption. Some adjustment for cigarettes per day is needed to assess the intrinsic carcinogenicity of the cigarette, but this adjustment is inappropriate if it obscures an adverse effect of compensatory smoking. Furthermore, smokers who are able to switch to cigarette brands with lower nicotine yield "reduced yield" products may have other characteristics that attenuate their lung cancer risk, relative to smokers who cannot switch. Their change in brands may reflect comparatively less dependence on nicotine and other addictive components of smoking. Smokers who switch may have smoked less intensively in the past and be more likely to quit during the follow up. Epidemiological studies have not historically measured nicotine addiction or related parameters such as puff volume, puffs per cigarette, or depth of inhalation. Prospective studies that have assessed smoking behaviour only at the time of enrolment cannot control for differences in cessation rates during follow up. Thus, the relatively lower risk of lung cancer among smokers who switch may result, not from switching per se, but rather from behavioural differences related to addiction. Some or all of what has been interpreted as efficacy may actually reflect selection bias or residual confounding.

#### COHORT STUDIES OF LUNG CANCER RISK IN DIFFERENT GENERATIONS OF SMOKERS

Several major cohort studies of smoking and disease have bridged the period when the greatest reduction occurred in the tar ratings of cigarettes. The British Doctors' Study examined lung cancer rates in relation to smoking behaviour among British physicians over a 40 year period.<sup>91</sup> Age standardised incidence rates among smokers were compared between the intervals 1951-1971 and 1971-1991. A similar comparison was made in the USA where the American Cancer Society (ACS) conducted two large cohort studies of comparable design begun 23 years apart: Cancer Prevention Study I (CPS-I), begun in 1959, and CPS-II, begun in 1982.<sup>92 93</sup>

In both the British Doctors' Study<sup>91 94</sup> and the two ACS cohorts<sup>92 93</sup> lung cancer risk increased among smokers from the 1950s to the 1980s. This increase occurred despite a dramatic decrease in the machine measured tar level of cigarettes in both countries during this time period.<sup>94</sup> Among the British doctors, the age standardised lung cancer incidence increased by 19%, from 264 per 100 000 to 314 per 100 000 from the first to the second 20 year period.<sup>94</sup> In the ACS studies, an even larger increase occurred in the age standardised lung cancer death rate among both male and female smokers from CPS-I to CPS-II, while this rate remained essentially constant in lifelong non-smokers (fig 3).<sup>95</sup> The age standardised rate increased from 187 to 341 (deaths per 100 000 person years) among

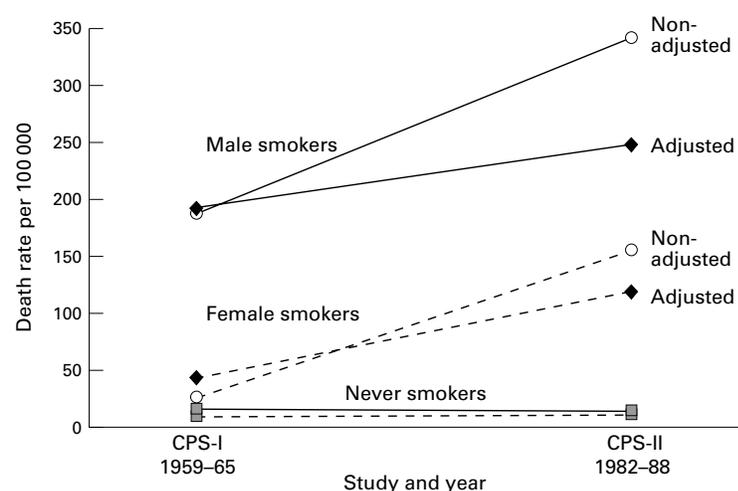


Figure 3 Death rates from all lung cancers by smoking status, CPS-I and CPS-II (adjusted for current amount and duration of smoking). The "non-adjusted" points indicate the age standardised death rate from lung cancer among male and female current cigarette smokers and lifelong non-smokers in CPS-I (1959–1965) and CPS-II (1982–1988). The "adjusted" values signify the results adjusted for age, cigarettes smoked per day, and years of smoking as reported at enrolment into the study.

male current cigarette smokers and from 26 to 155 among female smokers from CPS-I to CPS-II. The slope of the increase was reduced, but not eliminated, when the rates were adjusted for differences in the number of cigarettes smoked per day and duration of smoking, as reported at the time of enrolment. The ACS analyses are restricted to the first six years of follow up to enhance comparability.

These cohort studies indicate that the absolute risk of lung cancer continued to increase rather than decrease among older smokers despite the widespread adoption of "reduced yield" cigarettes. The substitution of filter tip products among smokers in CPS-II, for the unfiltered, very high yield cigarettes used extensively by smokers in CPS-I did not prevent a sustained increase in lung cancer among female smokers over age 45 and male smokers over age 50. Only among younger male smokers, ages 40–45, were lung cancer death rates lower in CPS-II than in CPS-I.<sup>95</sup> These studies help to put into perspective the relative attenuation in lung cancer risk seen in the earlier cohort and case-control studies that compare one type of cigarette to another. Whatever the relative impact of "reduced yield" products, their absolute impact was clearly inadequate to prevent a major increase in lung cancer risk among older smokers.

There are at least three possible explanations why lung cancer risk increased among smokers despite a dramatic decline in the machine measure tar yield of the cigarettes being smoked. One is that smokers who became addicted during and after the first world war presumably smoked less intensively as adolescents and young adults than did smokers who initiated smoking during and shortly after the second world war.<sup>94–95</sup> Manufactured cigarettes were less available and relatively more expensive after the first than the second world war. Consequently, earlier generations of smokers who consumed predominantly unfiltered, high yield products may have been

spared the full consequences of early life smoking that affected later generations. While the CPS-I and CPS-II analyses adjusted for the usual number of cigarettes smoked per day, as reported at the time of enrolment in the studies, this might not reflect large differences in early life smoking. Adverse changes in smoking behaviour may have overwhelmed any putative change in tar yield.

Two alternative explanations for the higher lung cancer risk among smokers in the second world war generation, compared to the first world war generation, involve a shift in the demographics of smokers and/or an increase, rather than decrease in the carcinogenicity of "reduced yield" cigarettes. Contemporary smokers are less educated and affluent, and have less healthy dietary patterns than previous generations of cigarette smokers.<sup>96–99</sup> Persons who continue to smoke despite repeated health warnings may also be more addicted and smoke more intensively.<sup>95</sup> Neither of these explanations would suggest that design changes in contemporary cigarettes have compensated adequately for the increased vulnerability of smokers or for adverse changes in smoking behaviour.

#### National trends in lung cancer mortality at specific ages

Several analyses<sup>100–102</sup> have examined trends in national lung cancer death rates by age in relation to age specific smoking patterns. Studies based on national trends are called "ecological" because they lack data on individual behaviours and outcomes, and cannot separate disease occurrence in current smokers from events in former or never smokers. However, the trends in national rates do reflect accurately the extent of progress towards reducing disease occurrence in the overall population.

One approach is to compare the trend in lung cancer death rates for a particular age group with the trends in smoking prevalence at younger ages. This approach does not integrate all of the relevant parameters of smoking behaviour, nor does it consider the time lag that occurs between the initiation of smoking and the onset of lung cancer, but it does allow a visual comparison of trends in age specific prevalence with trends in lung cancer in adjoining birth cohorts. Figure 4 shows the trends in lung cancer death rates for men and women, ages 35–39 in the USA and UK between 1965 and 1997, compared to the trend in cigarette smoking prevalence at ages 25–34 over the same time period. Among US men, the decrease in lung cancer mortality essentially parallels the decrease in cigarette smoking prevalence (fig 4A), whereas for men in the UK, the proportionate decrease in lung cancer mortality is much steeper than the decrease in smoking prevalence (fig 4B). Among US women, the lung cancer death at ages 35–39 changed very little over this time period despite a 40% decline in smoking prevalence (fig 4C). Among women of corresponding ages in the UK, both lung cancer and smoking prevalence decreased by 38%

over the interval (fig 4D). The temporal trends in lung cancer mortality in women are more difficult to interpret than those in men, because of the much larger recent changes in the ages when women initiated smoking and increases in the intensity of adolescent smoking.

Reductions in the tar yield of cigarettes are one possible explanation for the very rapid decline in lung cancer death rates at younger ages among men in the UK<sup>94</sup> and the more gradual decreases elsewhere in Europe, North America, and Australia<sup>100</sup> in recent decades. However, design changes in cigarettes are not the only potential explanation. The particularly high rates of lung cancer that prevailed among men in the UK until the 1950s and that are seen presently in some Eastern European countries may also reflect the exacerbating effects of diet, or reductions in occupational exposures or air pollution from coal burning that could potentiate the risks from smoking. Other potentially important parameters that have not been directly considered in these temporal and geographic comparisons include differences in cigarette composition across countries, and variations in the age of initiation, intensity of smoking at various ages, and age at cessation, both within and across countries. Thus, the analyses based only on national trends cannot convincingly separate the contribution of “reduced yield” cigarettes from other factors that might affect lung cancer risk.

Furthermore, it should be noted that, while the decline in lung cancer death rates at younger ages is encouraging, there is no certainty that these trends will be sustained into the future. Recent vital statistics data suggest that reductions in lung cancer death rates among young adults in the USA have slowed and possibly reversed, as a result of adverse changes in smoking behaviour among the young.<sup>102</sup> To the extent that health claims about “reduced yield” cigarettes defer cessation, this could also interfere with the progression of favourable trends into older ages where most lung cancers occur.

#### Other comprehensive reviews

Recent reviews by the Institute of Medicine<sup>1</sup> and the National Cancer Institute<sup>2</sup> have examined the evidence for a reduction in disease risks associated with the use of low yield cigarettes. Their conclusions reinforce the cautions raised by the UK Royal College of Physicians.<sup>103</sup> The Institute of Medicine report on *The scientific base for tobacco harm reduction* stated: “There have been many efforts in the past to develop less harmful cigarettes, none of which has proved to be successful.”<sup>1</sup> The National Cancer Institute review concluded: “Epidemiological and other scientific evidence, including patterns of mortality from smoking caused diseases, does not indicate a benefit to public health from changes in cigarette design and manufacturing over the last 50 years.”<sup>2</sup>

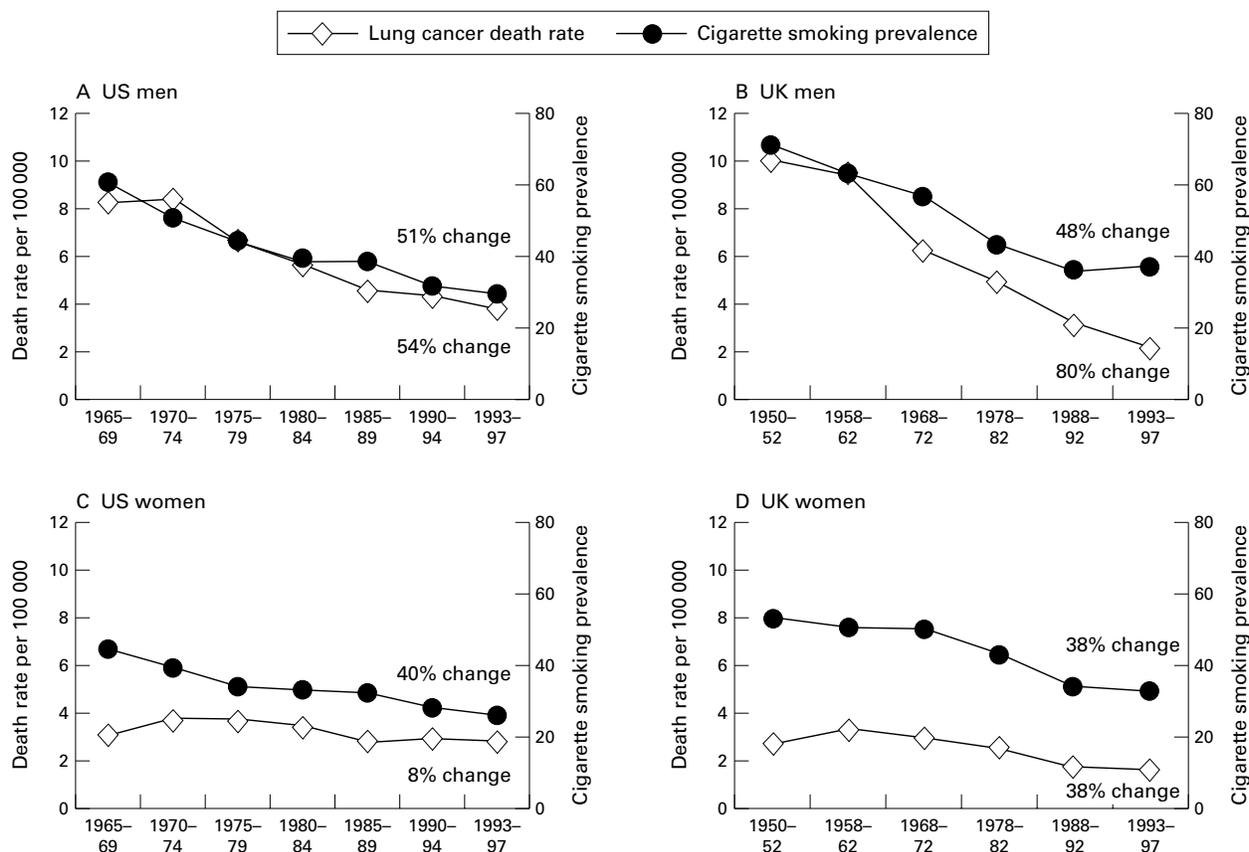


Figure 4 Lung cancer death rates at ages 35–39 and cigarette smoking prevalence at ages 25–34. The graphs indicate the temporal trend in lung cancer death rates at ages 35–39 and in cigarette smoking prevalence at ages 25–34 among men in the USA (A) and UK (B) and among women in the USA (C) and UK (D).

## Conclusions

In summary, there is no convincing evidence that changes in cigarette design between the 1950s and the mid 1980s have resulted in an important decrease in the disease burden caused by cigarette use for either smokers as a group or for the whole population. While many epidemiological studies have found attenuated risk of lung cancer among people who smoke these products, the extent to which these studies may overestimate the magnitude of the lung cancer association remains unclear. No studies have adequately assessed whether health claims used to market “reduced yield” products delay cessation among smokers who might otherwise quit or increase initiation or relapse among non-smokers. The widespread shift from unfiltered, high yield cigarettes to filter tip, lower yield products that occurred in the USA and UK since the 1950s did not prevent continuing increases in lung cancer risk among older smokers in large cohort studies. While one can postulate that lung cancer rates among older smokers might have risen even further in the absence of “reduced yield” cigarettes, other explanations are also possible. The temporal decrease in lung cancer risk at younger ages has been encouraging, but may be short lived if health claims about lower yield cigarettes are allowed to discourage or delay genuine cessation efforts by smokers. Furthermore, the extent to which changes in age specific lung cancer death rates reflect modifications in cigarette design versus changes in smoking behaviour including initiation and cessation has yet to be proven. There is no consistent evidence that “reduced yield” cigarettes have attenuated the risk of other smoking attributable diseases besides lung cancer.

A central challenge in tobacco policy is to prevent the misuse of unproven health claims to promote novel products. Alternative nicotine delivery devices may ultimately help to mitigate the harm caused by smoking to the approximately 47 million Americans who continue to smoke.<sup>1</sup> However, the evidence base required to market these products should correspond to the evidence required for any new drug delivery device. Furthermore, the publicity and marketing of these products must not distract attention away from interventions proven to reduce the prevalence, intensity, and duration of smoking.

This paper summarises material from a chapter on disease risks associated with light cigarettes from the upcoming National Cancer Institute Monograph 13: *Risks associated with smoking cigarettes with low machine-measured yields of tar and nicotine*.<sup>30</sup> The authors wish to thank the many colleagues who contributed to our thinking on this topic, including Sir Richard Peto, Drs Gary Giovino, Theodore Holford, Scott Leischow, Jay Lubin, Jonathan Samet, and Robert Tarone. We thank Michael Pleasant for preparation of the manuscript.

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- 1 Institute of Medicine. Clearing the smoke: the science base for tobacco harm reduction. In: Stratton K, Shetty P, Wallace R, Bondurant S, eds. *Committee to assess the science base for tobacco harm reduction, board on health promotion and disease prevention*. Institute of Medicine, National Academy of Sciences, National Academy Press, 2001.
- 2 National Cancer Institute. Risks associated with smoking cigarettes with low machine-measured yields of tar and nicotine. In: Burns DM, Benowitz NL, eds. *Smoking and*

- Tobacco Control Monograph, No 13*. US DHHS, Public Health Service, National Institutes of Health, National Cancer Institute, 2001 (in press).
- 3 National Cancer Institute. *The FTC cigarette test method for determining tar, nicotine, and carbon monoxide yields of US cigarettes*. Smoking and Tobacco Control Monograph No. 7. Bethesda, Maryland: US Department of Health and Human Services, Public Health Service, National Institutes of Health, 1996 (NIH Publication No. 96-4028.)
- 4 Wynder EL, Graham EA. Tobacco smoking as a possible etiologic factor in bronchiogenic carcinoma. A study of six hundred and eighty-four proved cases. *JAMA* 1950; 143:329-36.
- 5 Doll R, Hill AB. A study of the aetiology of carcinoma of the lung. *BMJ* 1952;2:1271-86.
- 6 Doll R, Hill AB. The mortality of doctors in relation to their smoking habits: a preliminary report. *BMJ* 1954; 1(4877):1451-5.
- 7 Hammond EC, Horn D. Smoking and death rates—a report on 44 months of follow-up of 187,783 men. II. Death rates by cause. *JAMA* 1958;166:1294-308.
- 8 Wynder EL, Graham EA, Croninger AB. Experimental production of carcinoma with cigarette tar. *Cancer Res* 1953;13:855-64.
- 9 US Congress. *Hearings before the Consumer Subcommittee of the Committee on Commerce*. Senate, 90th Congress, 23, 24 August 1967, p 7.
- 10 US Department of Health and Human Services. *The health consequences of smoking: the changing cigarette. A report of the Surgeon General, 1981*. Rockville, Maryland: Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health, 1981. (DHHS Publication No (PHS) 81-50156.)
- 11 Bradford JA, Harlan WR, Hanmer HR. Nature of cigarette smoke. Technique of experimental smoking. *Industrial and Engineering Chemistry* 1936;28:836-9.
- 12 Peeler CE. Cigarette testing and the Federal Trade Commission: a historical overview. *The FTC cigarette test method for determining tar, nicotine, and carbon monoxide yields of US cigarettes*. Smoking and Tobacco Control Monograph No. 7. Bethesda, Maryland: US Department of Health and Human Services, Public Health Service, National Institutes of Health, 1996:1-8 (NIH Publication No. 96-4028.)
- 13 Kozlowski LT, O'Connor RJ, Sweeney CT. Cigarette design. In: *Risks associated with smoking cigarettes with low machine-measured tar and nicotine yields*. NCI Smoking and Tobacco Control Monograph No 13. Bethesda, Maryland: National Cancer Institute (in press).
- 14 Jarvis MJ, Boreham R, Primatesta P, et al. Nicotine yield from machine-smoked cigarettes and nicotine intakes in smokers: evidence from a representative population survey. *J Natl Cancer Inst* 2001;93:134-8.
- 15 Kozlowski LT. Perceiving the risks of low-yield ventilated-filter cigarettes: the problem of hole-blocking. In: Covello V, Flamm WG, Rodericks J, Tardiff R, eds. *Proceedings of the International Workshop on the Analysis of Actual vs. Perceived Risks*. New York: Plenum, 1983:175-82.
- 16 Kozlowski LT, Mehta NY, Sweeney CT, et al. Filter ventilation and nicotine content of tobacco in cigarettes from Canada, the United Kingdom, and the United States. *Tobacco Control* 1998;7:369-75.
- 17 Benowitz NL. Compensatory smoking of low-yield cigarettes. In: *Risks associated with smoking cigarettes with low machine-measured tar and nicotine yields*. NCI Smoking and Tobacco Control Monograph No 13. Bethesda, Maryland: National Cancer Institute (in press).
- 18 Wakeham H. Trends in tar and nicotine deliveries over the last 5 years. Memo to Mr Hugh Cullman. 24 March 1961. Bates number 1000861953.
- 19 Russell MA, Wilson C, Patel UA, et al. Plasma nicotine levels after smoking cigarettes with high, medium, and low nicotine yields. *BMJ* 1975;2:414-6.
- 20 Benowitz NL, Jacob P, III. Nicotine and carbon monoxide intake from high- and low yield cigarettes. *Clin Pharmacol Ther* 1984;36:265-70.
- 21 Benowitz NL, Jacob P, III. Nicotine renal excretion rate influences nicotine intake during cigarette smoking. *J Pharmacol Exp Ther* 1985;234:153-5.
- 22 West RJ, Russell MA, Jarvis MJ, Feyerabend C. Does switching to an ultra-low nicotine cigarette induce nicotine withdrawal effects? *Psychopharmacology (Berl)* 1984; 84:120-3.
- 23 Zacny JP, Stitzer ML. Cigarette brand switching: effects on smoke exposure and smoking behavior. *J Pharmacol Exp Ther* 1988;246:619-27.
- 24 Russell MAH, Sutton SR, Iyer R, et al. Long-term switching to low-tar low-nicotine cigarettes. *Br J Addict* 1982; 77:145-58.
- 25 Robinson JC, Young JC, Rickett WS, et al. A comparative study of the amount of smoke absorbed from low yield (“less hazardous”) cigarettes. Part 2: Invasive measures. *Br J Addict* 1983;78:79-87.
- 26 Gori GB, Lynch CJ. Smoker intake from cigarettes in the 1-mg Federal Trade Commission tar class. *Regul Toxicol Pharmacol* 1983;3:110-20.
- 27 Peach H, Hayard DM, Ellard DR, et al. Phlegm production and lung function among cigarette smokers changing tar groups during the 1970s. *J Epidemiol Community Health* 1986;40:110-6.
- 28 Guyatt AR, Kirkham AJT, Mariner DC, et al. Long-term effects of switching to cigarettes with lower tar and nicotine yields. *Psychopharmacology (Berl)* 1989;99:80-6.

- 29 Frost C, Fullerton FM, Stephen AM, *et al.* The tar reduction study: Randomized trial of the effect of cigarette tar yield reduction on compensatory smoking. *Thorax* 1995;50:1038-43.
- 30 Burns DM, Major JM, Shanks *et al.* Smoking lower yield cigarettes and disease risks. In: *Risks associated with smoking cigarettes with low machine-measured tar and nicotine yields. NCI Smoking and Tobacco Control Monograph No 13.* Bethesda, Maryland: National Cancer Institute (in press).
- 31 Benowitz NL, Henningfield JE. Establishing a nicotine threshold for addiction. *N Engl J Med* 1994;331:123-5.
- 32 Shiffman S. Tobacco "chippers" — individual differences in tobacco dependence. *Psychopharmacology (Berl)* 1989; 97:539-47.
- 33 Lynch CJ, Benowitz NL. Spontaneous brand switching: consequences for nicotine and carbon monoxide exposure. *Am J Public Health* 1987;77:1191-4.
- 34 Peach H, Hayward DM, Shah D. A double-blind randomized controlled trial of the effect of a low- versus a middle tar cigarette on respiratory symptoms—A feasibility study. *IARC Scientific Publications* 1986;74:251-63.
- 35 Bross ID, Gibson R. Risks of lung cancer in smokers who switch to filter cigarettes. *Am J Public Health* 1968; 58:1396-403.
- 36 Bross ID. Effect of filter cigarettes on lung cancer risk. *Toward a less harmful cigarette. National Cancer Institute Monograph 28.* US Department of Health, Education and Welfare, National Cancer Institute, 1968.
- 37 Hammond EC, Garfinkel L, Seidman H, *et al.* Some recent findings concerning cigarette smoking. *Cold Spring Harbor Conferences on Cell Proliferation, Volume 4. Origins of human cancer. Book A. Incidence of cancer in humans.* Cold Spring Harbor, New York: Cold Spring Harbor Laboratory Press, 1977;101-12.
- 38 Hammond EC, Garfinkel L, Seidman H, *et al.* "Tar" and nicotine content of cigarette smoke in relation to death rates. *Environ Res* 1976;12:263-74.
- 39 Lee PN, Garfinkel L. Mortality and type of cigarette smoked. *J Epidemiol Community Health* 1981;35:16-22.
- 40 Higenbottam T, Shipley MJ, Rose G. Cigarettes, lung cancer, and coronary heart disease: the effects of inhalation and tar yield. *J Epidemiol Community Health* 1982;36:113-7.
- 41 Hawthorne VM., Fry JS. Smoking and health: the association between smoking behavior, total mortality, and cardiorespiratory disease in West Central Scotland. *J Epidemiol Community Health* 1978;32:260-6.
- 42 Todd GF, Hunt BM, Lambert PM. Four cardiorespiratory symptoms as predictors of mortality. *J Epidemiol Community Health* 1978;32:267-74.
- 43 Engeland A, Haldorsen T, Andersen A, *et al.* The impact of smoking habits on lung cancer risk: 28 years' observation of 26,000 Norwegian men and women. *Cancer Causes and Control* 1966;7:366-76.
- 44 Borland C, Chamberlain A, Higenbottam T, *et al.* Carbon monoxide yield of cigarettes and its relation to cardiorespiratory disease. *BMJ* 1983;287:1583-6.
- 45 Tang JL, Morris JK, Wald NJ, *et al.* Mortality in relation to tar yield of cigarettes: a prospective study of four cohorts. *BMJ* 1995;311:1530-3.
- 46 Wynder EL, Mabuchi K, Beattie EJ Jr. The epidemiology of lung cancer: recent trends. *JAMA* 1970;213:2221-8.
- 47 Wynder EL, Stellman SD. Impact of long-term filter cigarette usage on lung and larynx cancer risk: a case-control study. *J Natl Cancer Inst* 1979;62:471-7.
- 48 Augustine A, Harris RE, Wynder EL. Compensation as a risk factor for lung cancer in smokers who switch from nonfilter to filter cigarettes. *Am J Public Health* 1989; 79:188-91.
- 49 Kabat GC. Aspects of the epidemiology of lung cancer in smokers and nonsmokers in the United States. *Lung Cancer* 1996;15:1-20.
- 50 Rimington, J. The effect of filters on the incidence of lung cancer in cigarette smokers. *Environ Res* 1981;24:162-6.
- 51 Kuller LH. Cigarette smoking and mortality. MRFIT research group. *Prev Med* 1991;20:638-54.
- 52 Lubin JH, Blot WJ, Berrino F, *et al.* Patterns of lung cancer risk according to type of cigarette smoked. *Int J Cancer* 1984;33:569-76.
- 53 Lubin JH. Modifying risk of developing lung cancer by changing habits of cigarette smoking. *BMJ* 1984; 288:1953-6; 289:921 [letter—response].
- 54 Benhamou S, Benhamou E, Tirmarche M, *et al.* Lung cancer and use of cigarettes: a French case-control study. *J Natl Cancer Inst* 1985;74:1169-75.
- 55 Buffler PA, Contant CF, Pickle LW, *et al.* Environmental associations with lung cancer in Texas coastal counties. *Annu Clin Conf on Cancer* 1986;28:27-34.
- 56 Benhamou E, Benhamou S, Flamant R. Lung cancer and women: results of a French case-control study. *Br J Cancer* 1987;55:91-5.
- 57 Benhamou E, Benhamou S, Auquier A, *et al.* Changes in patterns of cigarette smoking and lung cancer risk: results of a case-control study. *Br J Cancer* 1989;60:601-4.
- 58 Benhamou S, Benhamou E, Auquier A, *et al.* Differential effects of tar content, type of tobacco and use of a filter on lung cancer risk in male cigarette smokers. *Int J Epidemiol* 1994;23:437-43.
- 59 Vutuc C, Kunze M. Lung cancer risk in women in relation to tar yields of cigarettes. *Prev Med* 1982;11:713-6.
- 60 Vutuc C, Kunze V. Tar yields of cigarettes and male lung cancer risk. *J Natl Cancer Inst* 1983;71:435-7.
- 61 Benhamou E, Benhamou S. Black (air-cured) and blond (flue-cured) tobacco and cancer risk. VI: lung cancer. *Eur J Cancer* 1993;29A:1778-80.
- 62 Lange P, Nyboe J, Appleyard M, *et al.* Relationship of the type of tobacco and inhalation pattern to pulmonary and total mortality. *Eur Respir J* 1992;5:1111-7.
- 63 Gillis CR, Hole DJ, Boyle P. Cigarette smoking and male lung cancer in an area of very high incidence. I: report of a case-control study in the West of Scotland. *J Epidemiol Community Health* 1988;42:38-43.
- 64 Alderson MR, Lee PN, Wang R. Risks of lung cancer, chronic bronchitis, ischaemic heart disease, and stroke in relation to type of cigarette smoked. *J Epidemiol Community Health* 1985;39:286-93.
- 65 Wynder EL, Kabat GC. The effect of low-yield cigarette smoking on lung cancer risk. *Cancer* 1988;62:1223-30.
- 66 Stellman SD, Muscat JE, Thompson S, *et al.* Risk of squamous cell carcinoma and adenocarcinoma of the lung in relation to lifetime filter cigarette smoking. *Cancer* 1997;80:382-8.
- 67 Petitti DB, Friedman GD. Cardiovascular and other diseases in smokers of low-yield cigarettes. *J Chron Dis* 1985;38:581-8.
- 68 Sidney S, Tekawa IS, Friedman GD. A prospective study of cigarette tar yield and lung cancer. *Cancer Causes and Control* 1993;4:3-10.
- 69 Wilcox HB, Schoenberg JB, Mason TJ, *et al.* Smoking and lung cancer: risk as a function of cigarette tar content. *Prev Med* 1988;17:263-72.
- 70 Pathak DR, Samet JM, Humble CG, *et al.* Determinants of lung cancer risk in cigarette smokers in New Mexico. *J Natl Cancer Inst* 1986;76:597-604.
- 71 Kaufman DW, Palmer JR, Rosenberg L, *et al.* Tar content of cigarettes in relation to lung cancer. *Am J Epidemiol* 1989; 129:703-11.
- 72 Khuder SA, Dayal HH, Mutgi AB, *et al.* Effect of cigarette smoking on major histological types of lung cancer in men. *Lung Cancer* 1998;22:15-21.
- 73 Armadans-Gil L, Vaque-Rafart J, Rossello J, *et al.* Cigarette smoking and male lung cancer risk with special regard to type of tobacco. *Int J Epidemiol* 1999;28:614-9.
- 74 Pezzotto SM, Mahuad R, Bay ML, *et al.* Variation in smoking-related lung cancer risk factors by cell type among men in Argentina: a case-control study. *Cancer Causes and Control* 1993;4:231-7.
- 75 De Stefani E. Mate drinking and risk of lung cancer in males: a case-control study from Uruguay. *Cancer Epidemiology, Biomarkers and Prevention* 1996;5:515-9.
- 76 Agudo A, Barnadas A, Pallares C, *et al.* Lung cancer and cigarette smoking in women: a case-control study in Barcelona (Spain). *Int J Cancer* 1994;59:165-9.
- 77 Matos E, Vilensky M, Boffetta P, *et al.* Lung cancer and smoking: a case-control study in Buenos Aires, Argentina. *Lung Cancer* 1998;21:155-63.
- 78 Jöckel KH, Ahrens W, Wichmann HE, *et al.* Occupational and environmental hazards associated with lung cancer. *Int J Epidemiol* 1992;21:202-13.
- 79 Palmer J, Rosenberg L, Shapiro S. Low yield cigarettes and the risk of nonfatal myocardial infarction in women. *New Engl J Med* 1989;320:1569-73.
- 80 Negri E. Tar yield of cigarettes and risk of acute myocardial infarction. *BMJ* 1993;306:1567-9.
- 81 Powell JT, Edwards RJ, Worrell PC. Risk factors associated with the development of peripheral arterial disease in smokers: a case control study. *Atherosclerosis* 1997;129:41-8.
- 82 Parish S, Collins R, Peto R. Cigarette smoking, tar yields, and non-fatal myocardial infarction: 14,000 cases and 32,000 controls in the United Kingdom. The International Studies of Infarct Survival (ISIS) Collaborations. *BMJ* 1995;311:471-7.
- 83 Sparrow D, Lee PN, Todd GF. The relationship of tar content to decline in pulmonary function in cigarette smokers. *Am Rev Respir Dis* 1983;127:56-8.
- 84 Dean G, *et al.* Factors related to respiratory and cardiovascular symptoms in the United Kingdom. *J Epidemiol Community Health* 1978;32:86-96.
- 85 Lange P, Nyboe J, Appleyard M. Relationship of the type of tobacco and inhalation pattern to pulmonary and total mortality. *Eur Respir J* 1992;5:1111-7.
- 86 Alderson MR, Lee PN, Wang R. Risks of lung cancer, chronic bronchitis, ischaemic heart disease, and stroke in relation to type of cigarette smoked. *J Epidemiol Community Health* 1985;39:286-93.
- 87 Kryanowski M, Sherrill DL, Paoletti P. Relationship of respiratory symptoms and pulmonary function to tar, nicotine, and carbon monoxide yield of cigarettes. *Am Rev Respir Dis* 1991;143:306-11.
- 88 Brown CA, Crombie IK, Smith WC. Cigarette tar content and symptoms of chronic bronchitis: results of the Scottish Heart Health Study. *J Epidemiol Community Health* 1991;45:287-90.
- 89 Withey CH, Papacosta AO, Swan AV. Respiratory effects of lowering tar and nicotine levels of cigarettes smoked by young male middle tar smokers. II. Results of a randomized controlled trial. *J Epidemiol Community Health* 1992;46:281-5.
- 90 Doll R, Peto R. Cigarette smoking and bronchial carcinoma: dose and time relationships among regular smokers and life-long non-smokers. *J Epidemiol Community Health* 1978;32:303-13.
- 91 Doll R, Peto R, Wheatley K, *et al.* Mortality in relation to smoking: 40 years' observations on male British doctors. *BMJ* 1994;309:901-11.

- 92 Thun MJ, Heath CW Jr. Changes in mortality from smoking in two American Cancer Society prospective studies since 1959. *Prev Med* 1997;26:422-6.
- 93 Thun M, Myers D, Day-Lally C, *et al.* Trends in tobacco smoking and mortality from cigarette use in cancer prevention studies I (1959 through 1965) and II (1982 through 1988). In: Burns D, Garfinkel L, Samet J, eds. *Changes in cigarette-related disease risks and their implication for prevention and control. Smoking and Tobacco Control Monograph No. 8.* Bethesda, Maryland: Department of Health and Human Services, Public Health Service, National Institutes of Health, 1997;305-82. (NIH Publication No. 97-4213.)
- 94 Peto R, Darby S, Deo H, *et al.* Smoking, smoking cessation, and lung cancer in the U.K. since 1950: combination of national statistics with two case-control studies. *BMJ* 2000;321:323-9.
- 95 Thun MJ, Heath CW Jr. Changes in mortality from smoking in two American Cancer Society prospective studies since 1959. *Prev Med* 1997;26:422-6.
- 96 Subar AF, Harlan LC, Mattson ME. Food and nutrient intake differences between smokers and non-smokers in the US. *Am J Public Health* 1990;80:1323-9.
- 97 Zondervan KT, Ocke MC, Smit HA, *et al.* Do dietary and supplementary intakes of antioxidants differ with smoking status? *Int J Epidemiol* 1996;25:70-9.
- 98 Midgette AS, Baron JA, Rohan TE. Do cigarette smokers have diets that increase their risks of coronary heart disease and cancer? *Am J Epidemiol* 1993;137:521-9.
- 99 Cade JE, Margetts BM. Relationship between diet and smoking—is the diet of smokers different? *J Epidemiol Community Health* 1991;45:270-2.
- 100 Blizzard L, Dwyer T. Declining lung cancer mortality of young Australian women despite increased smoking is linked to reduced cigarette "tar" yields. *Br J Cancer* 2001;84:392-6.
- 101 Mannino DM, Ford E, Giovino GA, *et al.* Lung cancer mortality rates in birth cohorts in the United States from 1960 to 1994. *Lung Cancer* 2001;31(2-3):91-9.
- 102 Jemal A, Chu KC, Tarone RE. Recent trends in lung cancer mortality in the United States. *J Natl Cancer Inst* 2001;93:277-83.
- 103 Tobacco Advisory Group (of the Royal College of Physicians). *Nicotine addiction in Britain.* London: Royal College of Physicians of London, 2000.



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## LETTERS

### Tobacco use among school personnel in Bihar, India

Tobacco use often starts in adolescent years when school personnel form important role models, potentially influencing tobacco use. To plan effective interventions, it is essential to have information on the extent and the type of tobacco use among school personnel, their attitudes towards tobacco control, and the existence of school health polices about tobacco.<sup>1</sup>

Tobacco use among 13-15 year old students is being studied worldwide through the Global Youth Tobacco Survey (GYTS)<sup>2</sup> which incorporates the Global School Personnel Survey (GSPS). The objectives of GSPS are: (1) obtain baseline information on tobacco use; (2) evaluate the existence, implementation, and enforcement of tobacco control policies in schools; (3) understand the knowledge and attitudes towards tobacco control policies; (4) assess training and material requirements for implementing tobacco prevention and control interventions; and (5) verify some information obtained from the GYTS. The GSPS was piloted in the state of Bihar, India and this report presents the results from the first pilot of GSPS.

GSPS is a cross sectional survey that employs a cluster sample design to produce a representative sample of school personnel drawn from the same schools that were selected for GYTS. For GYTS, schools were sampled with probability of selection proportional to the school enrolment size in grades 8-10 (corresponding to ages 13-15 years). A total of 50 schools out of 9905 listed for Bihar state were sampled. All school personnel (including all non-teaching staff) in the selected schools were eligible to participate. In India, education is a state responsibility and almost all schools were part of Bihar state educational system.

Bihar GSPS was conducted in the months of September and October 2000 using the same survey personnel who had conducted GYTS. The questionnaire contained 46 multiple choice questions. Survey procedures allowed for anonymous and voluntary participation. School personnel completed the self administered questionnaire during the break hours, recording their responses directly on a sheet which could subsequently be optically read by machine. The data file obtained was analysed using Epi Info. This software took the sampling weights into account for producing unbiased estimates of proportions and confidence intervals.

All selected schools participated in the survey (response rate 100%). Selected schools reported having a total of 697 eligible personnel, out of which 637 returned the completed questionnaire. The main reason for non-response was absence from school on the day of the survey. The school personnel response rate was 91.4%.

Out of 637 participating school personnel, 73% were men. Some 22.5% were less than 40 years old and 38.6% were 50 years or older. Very few were more than 60 as mandatory retirement age is 60 years. Women were

**Table 1** Prevalence of tobacco use among school personnel in Bihar by sex—Bihar GSPS 2000

	Male (%)	Female (%)	Total (%)
Total number	502	128	630
Any tobacco	77.6 (7.8)	77.0 (14.3)	77.4 (7.7)
Smokeless	58.7 (6.3)	53.4 (16.1)	57.3 (7.5)
Smoking	47.4 (8.7)	31.0 (8.9)	43.0 (7.1)
Cigarette	40.5 (5.9)	26.9 (9.9)	36.8 (5.0)
Others*	17.4 (4.6)	4.3 (4.7)	13.9 (3.5)

Figures in parentheses denotes confidence intervals ( $\pm$ CI).

\*Mostly bidi

somewhat younger than men. The majority of school personnel (83.5%) were teachers and there were only two health personnel.

Table 1 shows tobacco use prevalence among school personnel. Some 77.4% reported using tobacco in one form or the other. The prevalence was almost identical among men (77.6%) and women (77.0%). There was little difference in smokeless tobacco use among men (58.7%) and women (53.4%). Although smoking among women in India is generally proscribed, prevalence of smoking among women in this sample was quite high (31%). Most of it was cigarette smoking (26.9%). Among men, overall smoking prevalence was 47.4%, and cigarette smoking 40.5%. It should be noted that prevalence of cigarette smoking and other smoking habits do not add up to the prevalence of smoking, and prevalence of smoking and smokeless tobacco use do not add up to prevalence of tobacco use. This is because many individuals reported using tobacco in multiple forms.

Almost all school personnel (91%) agreed that tobacco was addictive, and 85% admitted that it had serious health consequences. While 92% of never users of tobacco believed that environmental tobacco smoke (ETS) was harmful to people who were repeatedly exposed to it, 83% of current tobacco users agreed with that statement. While 83% of never tobacco users complained that ETS was a nuisance, only slightly fewer (77%) current tobacco users did so.

Except for two people, everyone replied that there was no policy on tobacco use either for students or personnel. Even though tobacco use among school personnel was high, a vast majority was concerned about youth tobacco use (84.7%). A large proportion (90.4%) wanted a policy prohibiting tobacco use by students and, surprisingly, even more wanted a policy prohibiting tobacco use among school personnel (93.9%).

Another striking finding was that 80% thought that tobacco companies deliberately encourage youth to use tobacco. Some 88.3% wanted tobacco companies not to sponsor sports events and 95% wanted a complete ban on tobacco advertisements. Surprisingly, even though a majority were tobacco users, 78.4% agreed with the need to increase prices of tobacco products, with no difference between users and non-users.

The GSPS study findings reveal an alarming picture of very high tobacco use among school personnel, and a total absence of any tobacco control policy in schools administered by the

state government in Bihar. The results dispel the myth of smoking as taboo among middle class women in India in so far as self administered, anonymous questionnaires revealed 31% of female school personnel reported current smoking and 26.9% reported smoking cigarettes. This social change is likely to be due to several factors such as female emancipation and role modelling from western media. The role of marketing strategies by cigarette companies however, cannot be underestimated. Almost all cigarette advertising imagery includes women, and a cigarette brand specially targeted at women with the name "Ms" is available on the market. This kind of cigarette smoking is still practised away from public view—unlike hukka (hubble bubble) and cheroot smoking by rural women—but clearly it may not remain so for long.

The findings, however, do present an encouraging picture of widespread and near total support towards the formulation and implementation of effective tobacco control policy measures.

**D N Sinha**

School of Preventive Oncology, Patna, India

**P C Gupta**

**M S Pednekar**

Tata Institute of Fundamental Research, Mumbai, India

**J T Jones**

Department of Non-communicable Diseases and Health Promotion, World Health Organization, Geneva, Switzerland

**C W Warren**

Office on Smoking and Health, Centers for Disease Control and Prevention, Bethesda, Maryland, USA

Correspondence to: Prakash C. Gupta, Tata Institute of Fundamental Research, Homi Bhabha Road, Colaba, Mumbai 400 005, India; [pcgupta@tifr.res.in](mailto:pcgupta@tifr.res.in)

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### References

- 1 World Health Organization. *WHO Information series on school health; tobacco use prevention: an important entry point for the development of health-promoting schools*. Geneva: WHO, 1998:13-14.

2 Warren CW, Riley L, Asma S, *et al.* Tobacco use by youth: a surveillance report from the GYTS project. *Bull WHO* 2000;78:868–74.

## Exposure to environmental tobacco smoke in public places in Barcelona, Spain

Exposure to environmental tobacco smoke (ETS) has adverse health effects for both children and adults.<sup>1–3</sup> Southern European countries have not had the same level of ETS control measures as other western countries. The purpose of this study was to assess current ETS exposure in several locations in Barcelona, Spain.

We collected airborne nicotine with 31 diffusion monitors containing sodium bisulfate coated filters.<sup>4–5</sup> Between September 1999 and March 2000 different locations were chosen from among the following 18 sites in Barcelona: five underground (subway) stations ( $n = 5$ , one measurement in each station); two restaurants ( $n = 3$ , one of the restaurants, located in one of the two teaching hospitals referred to below, had measurements taken from smoking and non-smoking areas); two large stores ( $n = 4$ , two measurements in each store); two teaching hospitals ( $n = 4$ , two measurements from newborns inpatients and paediatrics outpatients departments from one hospital, and two from emergency rooms and radiography emergency departments from the other hospital); one medical school ( $n = 5$ ), one official language school ( $n = 2$ ); one secondary school ( $n = 1$ ); one general practice ( $n = 2$ ); one public health centre ( $n = 1$ ); and three households ( $n = 4$ , one smoker's home and two non-smoker's households). Nicotine concentrations for the three field blanks all corresponded to airborne concentrations of less than  $0.02 \mu\text{g}/\text{m}^3$ .

Monitors were left exposed for periods ranging from 7–13 days, since a minimum period of seven days was required to have a valid measure with passive monitors. One trained investigator completed a standard form with data concerning the date and time, placement and removal, exposure area, ventilation and distribution patterns, and distance from the person smoking nearby. The highest air nicotine concentration was found in restaurants, showing a mean of  $12.4 \mu\text{g}/\text{m}^3$  ( $10.6$ – $15.0 \mu\text{g}/\text{m}^3$ ). The air nicotine concentrations in a secondary school and in a smoker's household were  $9.5 \mu\text{g}/\text{m}^3$  and  $7.9 \mu\text{g}/\text{m}^3$ , respectively. In department stores, the average air nicotine concentration was  $2.8 \mu\text{g}/\text{m}^3$  (range  $0.4$ – $6.2 \mu\text{g}/\text{m}^3$ ). ETS exposure in the language school showed a mean nicotine concentration of  $2.3 \mu\text{g}/\text{m}^3$  (range  $1.7$ – $3.0 \mu\text{g}/\text{m}^3$ ). Other results are presented in table 1.

Although these results need to be interpreted within the limitation of having only 31 measurements and a non-random sample, this is the first attempt to obtain an objective measure of ETS exposure in public places in Barcelona. The data may also provide at least an initial insight into the situation in other southern European countries where measurements of ETS exposure are not common. Restaurants showed high concentrations, including two measurements obtained from hospital canteens where the average nicotine concentrations showed no significant difference between smoking and non-smoking areas ( $15.0$  and  $11.5 \mu\text{g}/\text{m}^3$ , respectively). This may reflect a lack of compliance or a weak physical separation between the two areas, and is especially serious since it involves hospitals. Nicotine concentrations in restaurants

**Table 1** Concentrations of nicotine recorded in public places in the city of Barcelona

Locations	Sampling time (days)*	Nicotine concentration ( $\mu\text{g}/\text{m}^3$ )
Underground (subway) stations (mean)		2.2
Platform	7	0.1
Connection 1†	7	3.8
Connection 2	7	2.1
Connection 3	7	4.1
Coach	12	1.0
Restaurants (mean)		12.4
Main dining room (no division)	7	10.6
Hospital A canteen (non-smoking area)	7	11.5
Hospital A canteen (smoking area)	7	15.0
Large stores (mean)		2.8
Store A, floor 1	7	0.7
Store A, floor 2	7	0.4
Store B, information centre	13	6.2
Store B, hall	13	3.9
Medical school (mean)		0.9
Corridor 1	7	2.1
Corridor 2	7	0.0
Classroom	7	0.1
Cafeteria	7	2.0
Hall	7	0.2
Language school (mean)		2.3
Hall 1	7	3.0
Hall 2	7	1.7
Secondary school (mean)		9.5
Teacher's room	7	9.5
Hospitals (mean)		0.7
Hospital B, newborns inpatients	7	0.0
Hospital B, paediatric outpatients	11	0.2
Hospital A‡, emergency department	7	1.0
Hospital A, radiography department (emergencies)	7	1.6
General practice (mean)		1.1
Doctor's room	7	2.0
Stairs	7	0.4
Public health centre (mean)		3.7
Room	12	3.7
Households, non-smokers (mean)		0.0
House A, living room 1	9	0.0
House B, living room 2	8	0.0
House B, bedroom	8	0.0
Households, smokers (mean)		7.9
House C, living room	7	7.9

\*The monitors were left exposed for 24 hours a day.

†All connections where measures were taken from corresponded to different sites.

‡The same hospital where the canteen's measurement were taken from.

were found to be double those found in a smoker's household. Other studies have shown higher concentrations of nicotine in workplaces, including restaurants, as compared to smokers' homes<sup>6–8</sup>. Our measurements are consistent with and even higher than those found in other studies where mean concentrations ranged from  $2$ – $6 \mu\text{g}/\text{m}^3$  in offices and from  $3$ – $8 \mu\text{g}/\text{m}^3$  in restaurants.<sup>8</sup>

Since all areas in our study were sampled 24 hours a day for at least a full week, concentrations were probably much higher during time of occupancy—that is, when non-smokers, especially children, were exposed. The fact that collection of data was made during the winter means that the results may have been less influenced by open windows. The finding of lower concentrations of nicotine in health centres and medical schools, where several local policies are being put in place, is encouraging.

The results of this study are intended to raise awareness of involuntary exposure to ETS and the need to enforce compliance with

legislation. Such legislation already exists in Catalonia, affecting the public transport system, health and education centres, and large department stores, where smoking is not allowed except in designated areas.<sup>9</sup> Smoke-free policies not only protect non-smokers from second hand smoke, they also create an environment that makes it easier for smokers to stop.

**M Jané, M Nebot, X Rojano, L Artazcoz**  
Institut Municipal de Salut Pública, Barcelona, Spain

**J Sunyer**  
Institut Municipal de Investigació Mèdica, Barcelona

**E Fernández**  
Institut Català d'Oncologia, Barcelona

**M Ceraso, J Samet**  
Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland, USA

S K Hammond,

School of Public Health, University of California,  
Berkeley, California, USACorrespondence to: Manel Nebot, Institut Municipal  
de Salut Pública, Plaça Lesseps 1, 08023  
Barcelona, Spain; mnebot@imsb.bcn.es/  
bexsa@readyssoft.es**Acknowledgements**

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**References**

- 1 **US Environmental Protection Agency.** *Respiratory health effects of passive smoking: lung cancer and other disorders.* Washington, DC: Office of Health and Environmental Assessment, 1992. (Publication No EPA/600/6-90/006F.)
- 2 **Kreuzer M, Krauss M, Kreienbrock L, et al.** Environmental tobacco smoke and lung cancer: a case-control study in Germany. *Am J Epidemiol* 2000;**151**:241–50.
- 3 **Hackshaw A, Law M, Wald N.** The accumulated evidence on lung cancer and environmental tobacco smoke. *BMJ* 1997;**315**:980–8.
- 4 **Hammond SK, Leaderer BP.** A diffusion monitor to measure exposure to passive smoking. *Environ Sci Technol* 1987;**21**:494–7.
- 5 **Leaderer BP, Hammond SK.** Evaluation of vapor-phase nicotine and respirable suspended particle mass as markers for Environmental Tobacco Smoke. *Environ Sci Technol* 1991;**25**:770–7.
- 6 **Siegel M.** Involuntary smoking in the restaurant workplace. *JAMA* 1993;**270**:490–3.
- 7 **Chapman S.** Smoking in public places. *BMJ* 1996;**312**:1051–2.
- 8 **Hammond SK.** Exposure of US workers to environmental tobacco smoke. *Environ Health Perspect* 1999;**107**:329–40.
- 9 **Llei 10/1991,** de 10 de maig, de modificació de la llei 20/1985 de prevenció i assistència en materia de substancies que poden generar dependencia. DOGC num.1445, maig 1991 [Law restricting smoking in Catalonia].

### A smoking cessation telephone resource: feasibility and preliminary evidence on the effect on health care provider adherence to smoking cessation guidelines

Physicians have frequent opportunities to intervene with their smoking patients as approximately 70% of smokers see a physician each year.<sup>1</sup> Even brief counselling by a physician significantly improves the rate of smoking cessation according to meta-analyses performed by the Tobacco Use and Dependence Guideline Panel and summarised as “ask, advise, assist, and arrange follow-up” in the Agency for Health Care Policy and Research (AHCPR) guidelines.<sup>2</sup> Despite these evidence based recommendations, physicians identify only about half of current smokers, advise less than half, and assist and arrange follow up with a small minority.<sup>3</sup> There are several explanations for this disparity between physicians’ knowledge and their actual behaviour including inadequate training, resource and time constraints, and lack of information on community cessation resources.

Office systems that screen patients for smoking status increase the rate of smoking

**Table 1** Adherence of health care providers to smoking cessation interventions

Intervention	Baseline (n=54)	Post-implementation (n=111)	Relative risk Post-implementation v baseline (95% CI)
Asked	37 (69%)	71 (64%)	0.9 (0.7 to 1.2)
Advised to quit	29 (55%)*	65 (59%)	1.1 (0.8 to 1.4)
Quit date discussed	5 (9%)	14 (13%)	1.4 (0.5 to 3.6)
Assistance offered	14 (26%)	46 (41%)†	1.6 (1.0 to 2.6)
Follow up arranged	9 (17%)	38 (34%)‡	2.1 (1.1 to 3.9)

\*One subject’s data missing for this item, n=53.

†p=0.052 versus baseline.

‡p<0.02 versus baseline.

CI, confidence interval

cessation interventions by health care providers.<sup>4</sup> We hypothesised that providers would be more likely to adhere to the AHCPR guidelines if they could delegate the time consuming steps of *assistance* and *follow up* to a telephone cessation resource.

This pilot study assessed the feasibility of a central telephone smoking cessation resource that would proactively call smokers who gave their provider consent for referral. We also evaluated whether providers would be then more likely to adhere to the smoking cessation guidelines. In a quasi-experimental pre-test, post-test design, a sample of patients seen for any type of visit with a provider in three participating primary care clinics in Vermont were interviewed at exit from the clinic. Only current smokers were asked about their providers’ adherence to guidelines. The primary outcome measure was the proportion of current smokers who reported being asked, advised, assisted, and having follow up arranged at baseline and four months after implementation of the resource.

Two hundred and nine patients were referred to the resource from the three clinics over the four month duration of resource availability. We estimated that this represented 20% of the total number of smokers seen at the clinics during this time period. We interviewed 54 smokers at baseline and 111 smokers four months after implementation. After the intervention, rates of asking and advising about smoking were not significantly changed from baseline (table 1). The increase in the proportion of smokers who were offered assistance did not reach significance (p = 0.052). There was a significant increase in those who had follow-up arranged (table 1).

Our study demonstrates that a smoking cessation proactive telephone resource is feasible and that providers will refer patients to such a resource. The resource had a contact rate of only 52% of referred current smokers, which we attribute to the resource not having evening calling hours, a significant limitation. Implementation of this proactive smoking cessation telephone resource was associated with improved arrangement of follow up. These preliminary data suggest that further studies of the effect of referral resources on adherence of physicians to guidelines are warranted. Because of the non-randomised design of this pilot study, we cannot attribute improvements in provider adherence solely to the availability of the telephone resource, as provider focus groups, surveys, and training also may have increased adherence to the guidelines. Only a randomised study can address this issue.

T W Marcy

National Cancer Institute, Division of Cancer Prevention, Office of Preventive Oncology, Rockville, Maryland; Office of Health Promotion Research, University of Vermont College of Medicine, Burlington, Vermont; Vermont Cancer Center, University of Vermont, Vermont USA  
twmarcy@together.net

L J Solomon

Vermont Cancer Center, and Department of Psychology, University of Vermont

G S Dana, R Secker-Walker

Office of Health Promotion Research, University of Vermont College of Medicine, Burlington, and Vermont Cancer Center, University of Vermont

J M Skelly

Biometry Facility, University of Vermont

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**References**

- 1 **Davis R.** Uniting physicians against smoking: the need for a coordinated national strategy. *JAMA* 1988;**259**:2900–1.
- 2 **Fiore M, Bailey W, Cohen S, et al.** *Treating tobacco use and dependence.* Rockville, Maryland: US Department of Health and Human Services. Public Health Service, 2000.
- 3 **Goldstein M, Niaura R, Willey-Lessne C, et al.** Physicians counseling smokers: a population-based survey of patients’ perceptions of health care provider-delivered smoking cessation interventions. *Arch Intern Med* 1997;**157**:1313–19.
- 4 **Fiore M, Jorenby D, Schensky A, et al.** Smoking status as the new vital sign: effect on assessment and intervention in patients who smoke. *Mayo Clin Proc* 1995;**70**:209–13.

### Ophthalmologists’ and optometrists’ attitudes and behaviours regarding tobacco cessation intervention

Although health care providers can be effective in motivating and helping patients to quit their tobacco use,<sup>1–7</sup> the potential role of eye care professionals has been under recognised. Several chronic ocular diseases are associated with smoking,<sup>8</sup> including formation of cataracts and age related macular degeneration (a leading cause of blindness).<sup>8,9</sup> As a cardiovascular risk factor, smoking may also play a role in the development of anterior ischaemic optic neuropathy.<sup>10</sup> In addition, smoking may increase the risk of ocular disease from other disorders, such as diabetes, the main cause of blindness in persons 20–74 years of age.<sup>11</sup>

**Table 1** Eye care professionals' attitudes, beliefs, and perceived barriers regarding intervention with tobacco using patients

	Ophthalmologists (n=422) (%)	Optometrists (n=629) (%)
<b>Demographics</b>		
Years in practice	23 (SD 11.33)	16 (SD 11.23)
Sex	85% male	72% male
<b>Tobacco related behaviours: "How often do you . . ."</b>		
Ask patients about tobacco use?	71	38
Sometimes advise patients to quit tobacco?	91	81
Regularly advise patients to quit tobacco?	30	16
Provide educational materials on the ocular effects of tobacco use?	5	6
<b>Barriers to intervening with smokers</b>		
Lack of time	83	70
Lack of patient materials	67	79
Lack of training	64	78
Lack of referral resources	63	76
Concerns about effectiveness	63	69
Concerns about patient resistance or loss	61	72
Lack of reimbursement mechanism	57	52
Concerns about office staff resistance	32	40
<b>Attitudes about intervening with smokers</b>		
Believe it is appropriate for them to document patients' tobacco use	81	69
Believe it is appropriate for them to advise patients to quit tobacco	82	71
Interested in learning new ways to help patients quit tobacco	74	80

Before developing a tobacco cessation intervention for eye care professionals, it is essential to assess the current status of tobacco cessation activities in routine eye care. We sent a 12 item questionnaire to all currently licensed ophthalmologists (n = 1209) and a random sample of 1234 optometrists in four western states of the USA (Arizona, California, Oregon, and Washington), assessing demographics and behaviours, attitudes, and barriers regarding intervention with tobacco using patients. The final return rate was 39% for ophthalmologists and 53% for optometrists. Data are presented only for those in current practice (90% of the ophthalmologists and 95% of the optometrists). Since ophthalmologists were significantly less likely to return the survey ( $\chi^2$  (1, n = 2443) = 48.56,  $p < 0.001$ ) than optometrists, we report data for each professional group separately without comparing the two.

As table 1 indicates, both ophthalmologists and optometrists feel it is appropriate to help tobacco using patients with cessation, though few do so regularly and many barriers are perceived. Optometrists employing support staff were more likely to express positive attitudes towards providing tobacco interventions than those who did not ( $t(634) = 2.55$ ,  $p < 0.05$ ), suggesting a correlation between time constraints and attitude toward intervention.

Both ophthalmologists and optometrists cited many barriers to intervening with their tobacco using patients. Lack of time was most commonly cited by ophthalmologists, whereas optometrists were more concerned about lack of patient materials and lack of training. How recently they trained and their sex were related to barriers. Ophthalmologists

and optometrists who had graduated more recently from their programmes perceived fewer barriers to providing cessation services ( $r = 0.18$ ,  $p < 0.01$  for ophthalmologists;  $r = 0.16$ ,  $p < 0.01$  for optometrists). Previous studies<sup>1,2</sup> have shown a reduction in perception of barriers due to receiving education in tobacco cessation intervention.

Surprisingly, female ophthalmologists were less likely to believe they should advise patients to quit ( $t(381) = 2.16$ ,  $p < 0.05$ ), and both female ophthalmologists and optometrists perceived more barriers to doing so ( $t(365) = -2.54$ ,  $p < 0.05$  for ophthalmologists,  $t(586) = -2.93$ ,  $p < 0.01$  for optometrists). This reluctance may be due to female eye care providers' concerns about possible negative patient reactions, or fears of losing patients from their practices.

Although this is a convenience sample, our results suggest the feasibility of brief, office based tobacco cessation interventions for use in eye care settings. An intervention must, however, focus on reducing perceived barriers by training eye care professionals in providing an effective, brief intervention that is readily received by patients, as well as providing resources and materials to practitioners. Our data suggest that cooperative agreements with insurance companies to provide reimbursement to providers would facilitate the adoption of the intervention.

As summarised by the Clinical Practice Guidelines,<sup>2</sup> many types of general and specialist providers have successfully incorporated tobacco cessation activities into their practices. One way to extend the reach of tobacco cessation interventions is to utilise other medical specialists to motivate tobacco users to quit. Ophthalmology and optometry

may provide such an opportunity, given the role of smoking in ocular disease, the fact that most visits are for routine rather than acute care, and the presence of support staff who can help implement an intervention.

**J S Gordon**  
**J A Andrews**  
**E Lichtenstein**  
**H H Severson**  
**L Akers**  
**C Williams**

Oregon Research Institute, 1715 Franklin Boulevard, Eugene, Oregon 97403, USA

Correspondence to: Dr Judith Gordon; judith@ori.org

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### References

- 1 **Andrews JA**, Severson HH, Lichtenstein E, et al. Evaluation of a dental office tobacco cessation program: effects on smokeless tobacco use. *Ann Behav Med* 1999;**21**:48-53.
- 2 **Fiore MC**, Bailey WC, Cohen SJ, et al. *Treating tobacco use and dependence*. Clinical Practice Guideline. Rockville, Maryland: US Department of Health and Human Services. Public Health Service, 2000.
- 3 **Hollis JF**, Lichtenstein E, Vogt TM, et al. Nurse-assisted counseling for smokers in primary care. *Ann Intern Med* 1993;**118**:521-5.
- 4 **Little SJ**, Stevens VJ, Severson HH, et al. An effective smokeless tobacco intervention for dental hygiene patients. *J Dental Hygiene* 1992;**66**:185-90.
- 5 **Severson HH**, Andrews JA, Lichtenstein E, et al. Reducing maternal smoking and relapse: long-term evaluation of a pediatric intervention. *Prev Med* 1997;**26**:120-30.
- 6 **Severson HH**, Andrews JA, Lichtenstein E, et al. Using the hygiene visit to deliver a tobacco cessation program: results of a randomized clinical trial. *J Am Dental Assoc* 1998;**129**:993-9.
- 7 **Wall MA**, Severson HH, Andrews JA, et al. Pediatric office based smoking intervention: impact on maternal smoking and relapse. *Pediatrics* 1995;**96**:622-8.
- 8 **Solberg Y**, Rosner M, Belkin M. The association between cigarette smoking and ocular diseases. *Survey of Ophthalmology* 1998;**42**:535-47.
- 9 **West S**, Munoz B, Emmett EA, et al. Cigarette smoking and risk of nuclear cataracts. *Arch Ophthalmol* 1989;**107**:1166-9.
- 10 **Margulies LJ**. Ocular manifestations of cardiovascular and hematologic disorders. *Curr Opin Ophthalmol* 1995;**6**:97-103.
- 11 **Alexander LJ**, Duenas MR. Eye care for patients with diabetes in the state of Florida: status in 1988. *J Am Optometric Assoc* 1994;**65**:552-8.
- 12 **Gordon JS**, Severson HH. Tobacco cessation through dental office settings. *J Dent Educ* 2001;**65**:354-63.

## CORRECTION

The authors of Health impact of "reduced yield" cigarettes: a critical assessment of the epidemiological evidence (*Tobacco Control* 2001;**10**(suppl 1):i4-i11) would like to correct a statement in figure 1. The legend to figure 1 and the corresponding text on page 15 should say "Each milligram decrease in machine measured nicotine..." rather than "Each milligram decrease in machine measured tar...".