

Scientific Committee on Tobacco and Health (SCOTH)

Secondhand Smoke: Review of evidence since 1998

Update of evidence on health effects of secondhand smoke

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November 2004

Dear Sir Liam

It is my privilege to present to you this report of the Scientific Committee on Tobacco and Health (SCOTH), which reviews the evidence on secondhand smoke.

It has been a pleasure to chair this committee during a time of increased public and government awareness (nationally and internationally) of the dangers to non-smokers of secondhand smoke and the resultant debate over how people might best be protected from this risk.

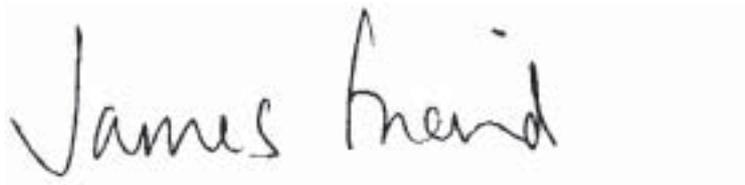
The Committee last reported on this issue to your predecessor Sir Kenneth Calman in 1998. That report covered three main areas, in each of which detailed reviews of the evidence were commissioned. These were: lung cancer, heart disease and children's health. Based on that thorough analysis of the evidence, SCOTH concluded that exposure to secondhand smoke was a cause of lung cancer and ischaemic heart disease among adults. Furthermore, smoking in the presence of infants and children, was a cause of serious respiratory illness and asthmatic attacks, sudden infant death syndrome, and glue ear. Since the 1998 report there has been further evidence published.

This new report summarises original scientific research and review papers that have appeared since 1998 to examine whether any revision to SCOTH's conclusions is required. We felt this was merited to provide further scientific underpinning to the debate around the health risks of secondhand smoke, which are still denied by a small number of campaigning groups. Eligible studies were identified by conducting literature searches on *Medline* using the terms 'passive smoking', 'environmental tobacco smoke', 'secondhand smoke' and 'second-hand smoke' and then examining the studies listed for their relevance and importance. The Committee also received oral evidence. We were grateful to those who made presentations to us and to the various witnesses, all of whom are listed in Annex A to the report. It is worth noting that we heard and received evidence from the Tobacco Manufacturers Association as part of this exercise.

The review of the evidence, which the Committee has provided here, does not claim to be exhaustive, as the volume of material on this topic is now very extensive. However, the Committee's conclusions are that the evidence published since 1998 reinforces the conclusions of the SCOTH report published at that time: confirming the causal effect of exposure to secondhand smoke on the risk of lung cancer, ischaemic heart disease and a strong link to adverse effects in children. There is no reason to revise SCOTH's conclusions relating to the number of causal effects. There is new evidence published since 1998 to an association between secondhand smoke and reduced lung function in adults.

I would like to thank the members of the Committee for their invaluable support, expertise and patience. I would also like to thank the members of the secretariat for their support and hard work in the preparation of the report.

Yours sincerely

A handwritten signature in black ink that reads "James Friend". The signature is written in a cursive style with a large initial 'J' and a distinct 'F'.

Emeritus Professor James Friend
Chair SCOTH

Copies to:
CMO Scotland
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Executive Summary

1. In 1998, the Scientific Committee on Tobacco and Health (SCOTH) issued a report which concluded that exposure to secondhand tobacco smoke causes lung cancer and heart disease in adult non-smokers and a variety of conditions including respiratory disease, cot death and middle ear disease in children. Now the Committee has considered evidence that has emerged in the past 5 years. This new evaluation has been assisted by a review of the scientific literature, published since 1998, prepared by Professor Martin Jarvis (Deputy Chair of SCOTH).

Lung cancer

2. The increased risk to non-smokers of lung cancer from secondhand smoke (SHS) was estimated at 24% in the overview of 37 studies and 4626 cases commissioned by SCOTH in 1998. An almost identical increased risk estimate emerged from the recent overview of 46 studies and 6257 cases carried out by the International Agency for Research on Cancer (IARC). Both these overviews looked at lung cancer cases in non-smoking women with partners who smoked. The results from all the new studies, when pooled together, lead SCOTH to conclude that SHS causes lung cancer, and to support the degree of risk found in the 1998 report. SCOTH concludes that there is an estimated overall 24% increased risk of lung cancer in non-smokers exposed to SHS. This estimate is only marginally reduced when the negative results of a recent Californian study published in the British Medical Journal are included.

Heart disease

3. New studies on SHS exposure and the risk of heart disease have strengthened the findings of the 1998 SCOTH overview which estimated that the excess risk in non smokers exposed to SHS compared to those not exposed was 23%. We now have greater understanding of the ways in which tobacco smoke inhalation damages the blood vessels. Whereas lung cancer risk increases in a linear fashion with the number of cigarettes smoked and the duration of smoking, damage to the heart and the arteries occurs disproportionately at the lower exposures experienced by those exposed to SHS. SCOTH therefore concludes that SHS causes heart disease and that the best estimate of increased relative risk of heart disease in non-smokers exposed to SHS remains at about 25%.

Health damage in infancy and childhood

4. A number of new studies have confirmed the range and extent of health damage in infancy and childhood. Children are at greatest risk in their homes and the evidence strongly links SHS with an increased risk of pneumonia and bronchitis, asthma attacks, middle ear disease, decreased lung function and sudden infant death syndrome. It has also been shown that babies born to mothers who come into contact with SHS have lower birth weights.

Exposure to SHS

5. Overall exposure to secondhand tobacco smoke in the population has declined somewhat as cigarette smoking prevalence has continued to come down. However, some groups, for example bar staff, are heavily exposed at their place of work and almost half of all children still live in households with at least one smoker.

Conclusion

6. SCOTH's conclusion is that knowledge of the hazardous nature of SHS has consolidated over the last five years, and this evidence strengthens earlier estimates of the size of the health risks. This is a controllable and preventable form of indoor air pollution. It is evident that no infant, child or adult should be exposed to SHS. This update confirms that SHS represents a substantial public health hazard.

Introduction

1. The Scientific Committee on Tobacco and Health (SCOTH) review of secondhand smoke (SHS), on which its 1998 report was based, covered three main areas, in each of which detailed reviews of the evidence were commissioned. These were: lung cancer (1), heart disease (2), and children's health (3). On the basis of this thorough analysis of the evidence, SCOTH concluded that exposure to SHS was a cause of lung cancer and ischaemic heart disease among adults. Furthermore, smoking in the presence of infants and children was a cause of serious respiratory illness and asthmatic attacks, sudden infant death syndrome, and glue ear.
2. SCOTH's conclusions were reached after careful consideration of the available scientific data. Since then, there has been further published evidence. This report summarises original scientific research and review papers that have appeared since 1998, in order to examine whether any revision to SCOTH's conclusions is required. The structure of the paper broadly follows SCOTH's earlier topic headings. Eligible studies were identified by conducting literature searches on *Medline* using the terms 'passive smoking', 'environmental tobacco smoke', 'secondhand smoke', 'second hand smoke' and 'second-hand smoke' and then examining the studies listed for their relevance and importance. The Committee has also received oral evidence (appendix 1). The review provided here does not claim to be exhaustive, as the volume of material published on this topic is very extensive. Throughout this review we will refer to SHS.

SHS AND LUNG CANCER

3. Several studies of the relationship between exposure to SHS and lung cancer have been published since 1998 (4-16). A definitive list is given in the International Agency for Research on Cancer (IARC) Monograph (17). Most of these studies have focused on women and have employed case-control methodology. The majority have investigated residential or spousal exposure, but some have also looked at occupational exposure. The authors of these publications have consistently reported an increased risk of lung cancer associated with exposure to other people's smoke, and in several studies dose-response effects were observed. In most studies considered individually the observed odds ratios failed to reach statistical significance. They were nevertheless comfortably within the confidence limits of the pooled odds from the 1997 meta-analysis presented to SCOTH (1) of 1.24 (95% confidence interval (CI) 1.13-1.36). That is an excess risk of 24% in non-smokers exposed to SHS compared to those not exposed.

4. Other meta-analyses have been published since 1998, for example Zhong *et al* (18) and Taylor *et al* (19). Both reported similar pooled relative risks and these were consistent with Hackshaw *et al* 1997 (1).
5. Investigators who recently examined the data for California from the US Cancer Prevention Study 1 (CPS1) reported no increase of lung cancer among either men or women with smoking spouses (20). The relative risk of lung cancer in never smoking women married to ever smokers was reported as 0.99 (95% CI 0.72-1.37) based on 177 cases. The authors concluded that there was no increased risk associated with SHS. Although this paper was published in a high profile medical journal (the British Medical Journal) and received media attention, subsequent comment has pointed to serious methodological problems that raise major questions over the validity of the conclusions (21, 22). The size of the sample studied (177 cases compared to 6257 considered by IARC) also needs to be considered in giving appropriate weight to the findings. The observed result is consistent with previous pooled estimates of risk, and adding this finding to the IARC meta-analysis reduces the pooled estimate only marginally.
6. Several papers have been published which question the interpretation of the association of exposure to SHS with lung cancer as causal (23-30). These authors have emphasised problems of potential misclassification of exposure. However, empirical investigations of this issue have suggested that it is unlikely to constitute a major problem (31, 32).
7. In June 2002, IARC issued the summary of its conclusions on the carcinogenicity of SHS, pending publication of its full monograph (17). A scientific working group of 29 experts from 12 countries had reviewed all significant published evidence related to tobacco smoking, both active and involuntary. The conclusions reached were that there is:
 - sufficient evidence that involuntary smoking (exposure to SHS) causes lung cancer in humans.
 - limited evidence in experimental animals for the carcinogenicity of mixtures of sidestream and mainstream tobacco smoke.
 - sufficient evidence in experimental animals for the carcinogenicity of sidestream smoke condensates.

8. IARC's conclusion, that exposure to SHS is causally associated with lung cancer, was based on consideration of both toxicological and epidemiological data. The results of an updated meta-analysis were also reported. Among never smoking women married to ever smokers the relative risk of lung cancer was 1.24 (95% CI 1.14-1.34) based on 46 studies and 6257 cases. This is almost identical to the 1997 analysis included in the SCOTH report that yielded a relative risk of 1.24 (95% CI 1.13-1.36), but based on 37 studies and 4626 cases (1). It is reassuring that although the number of studies has increased since 1997 the results have not materially altered. The IARC monograph also reported that among never smoking men married to ever smokers the relative risk was 1.37 (95% CI 1.02-1.83), based on 11 studies and 442 cases. Further, that there is an increased risk associated with exposure at the workplace; relative risk of 1.19 (95% CI 1.09-1.30) pooled over 19 studies of never smoking women based on 3588 lung cancer cases.
9. In December 2002 the US National Toxicology Program (NTP) issued its 10th report on carcinogens (33). The report lists SHS as a known human carcinogen, based on sufficient evidence of carcinogenicity from studies on humans that indicate a causal relationship between exposure to SHS and human lung cancer. SHS was first listed as a carcinogen in the 9th report of the National Toxicology Program published in December 2000.

CONCLUSION

The evidence published since 1998 continues to point to a causal effect of exposure to SHS on risk of lung cancer. The pooled increased relative risk remains in good agreement with that estimated by Hackshaw, Law and Wald (24%; 13-36%, 95% CI) (1).

SHS AND ISCHAEMIC HEART DISEASE

10. A paper prepared for SCOTH by Law *et al* in 1997 (2) estimated a relative risk of ischaemic heart disease (IHD) attributable to SHS of 1.23 – that is, an excess risk of 23% in non-smokers exposed to SHS compared to those not exposed.
11. Another meta-analysis published in 1999 in the *New England Journal of Medicine* (34) found a similar estimate of the relative risk of coronary heart disease attributable to exposure to SHS. Overall the relative risk was estimated at 1.25 (95% CI 1.17-1.32), based on 18 studies. This is close to the result reported in the SCOTH report.
12. The article in 1999 also reported a relative risk of 1.22 (95% CI 1.10-1.35) in men, 1.24 (95% CI 1.15-1.34) in women, 1.17 (95% CI 1.11-1.24) for home exposure and 1.11 (95% CI 1.00-1.23) for exposure in the workplace. A significant dose-response relationship was identified.
13. A case-control study in Greece reported an odds ratio of 1.47 (95% CI 1.26-1.80) for myocardial infarction or unstable angina in non-smokers exposed to SHS. There was an excess risk with exposure at work as well as exposure at home (35). In Sweden, a case-control study of myocardial infarction risk found an odds ratio of 1.58 (95% CI 0.97-2.56) for an average daily exposure of 20 cigarettes or more from the spouse, with evidence of a dose-response relationship (36).
14. A feature of the epidemiological data on SHS and heart disease is the consistent evidence for non-linear relationships between dose and risk. The increased risk associated with exposure to SHS is about 25%, a substantial fraction of the risk from active smoking, although uptake of smoke by non-smokers is typically only about 1% of that by active smokers. Thus it appears that a substantial risk arises from quite modest exposure to SHS. A recent review by Law and Wald has discussed this apparent anomaly and offered an explanation (37).
15. The evidence of increased risk at low levels of exposure has also been highlighted by Whincup and colleagues who have recently examined cardiovascular risks of SHS in non-smokers in the British Regional Heart Study (38). This cohort was recruited in 1978-1980 and has now been followed up over a period of 20 years. Exposure to SHS in non-smokers was quantified by the concentration of plasma cotinine at baseline. Relative hazards for coronary heart disease (CHD) in the second, third and fourth quartiles of cotinine level compared with the first were 1.46 (95% CI 1.01-2.13), 1.52 (95% CI 1.04-2.22) and 1.65 (95% CI 1.12-2.43) after adjustment for established CHD risk factors. Observed risks were particularly increased during the

first (3.82, 95% CI 1.36-10.73) and second (1.85, 95% CI 1.04-3.29) five year follow up periods compared with later periods. A strength of this study is that it used an objective marker of the extent of SHS that integrates all sources of exposure.

16. Investigators of the California data gathered as part of the American Cancer Society's CPS1 (see the section on lung cancer above) reported no relationship between exposure to SHS and heart disease (20). The American Cancer Society has made clear its view that this data set was inappropriate for examining cardiovascular risks of exposure to SHS (21). A number of concerns have been raised, in the same way that they were for lung cancer. In particular, that many spouses who smoked at baseline would have given up during the course of the study period yet still be recorded as a smoker.
17. There is continued scientific discussion as to the mechanism by which SHS results in heart disease. Evidence against platelet aggregation as a mechanism has been presented (39, 40). Arterial endothelial dysfunction has been reported to be associated with exposure to SHS (41-43). Accelerated atherogenesis was found in mice exposed to sidestream smoke (44), confirming the results of eight other experimental studies showing a pronounced effect of low dose tobacco smoke exposure on atherogenesis in various animal species (37). Progression of atherosclerosis was observed in non-smokers exposed to SHS followed over a period of 3 years (45). Exposure to SHS was reported to be associated with an acute deterioration in the elastic properties of the aorta (46).

CONCLUSION

The evidence published since 1998 continues to point to a causal effect of exposure to SHS on the risk of ischaemic heart disease. The weight of evidence now is stronger than at the time of the SCOTH report. The increased risk associated with exposure to SHS is estimated to be in the order of 25%.

SHS AND STROKE

18. Associations between stroke and exposure to SHS have been little studied. Two studies have looked at the relationship. No association with stroke was seen in the study by Whincup and colleagues (38). One study, however, a case-control study from New Zealand published in 1999 (47) identified an increased risk of stroke in never-smokers and long-term ex-smokers exposed to SHS. The overall odds ratio (OR) was 1.82 (95% CI 1.34-2.49), with increases seen in both men (OR 2.10) and women (OR 1.66). There is insufficient evidence to date to confirm an association between stroke and exposure to SHS.

SHS AND RESPIRATORY DISEASE IN CHILDREN

19. The 1998 SCOTH report concluded that smoking in the presence of children is a cause of serious respiratory illness and asthmatic attacks. It also concluded that sudden infant death syndrome, the main cause of post-neonatal death in the first year of life, is associated with exposure to SHS and that the association is judged to be one of cause and effect. In 1999, the World Health Organization (WHO) convened an international consultation on SHS and child health (48). Its conclusions were similar to those of the 1998 SCOTH report. The WHO found that SHS is a real and substantial threat to child health, causing a variety of adverse health effects including increased susceptibility to lower respiratory tract infections such as pneumonia and bronchitis, worsening of asthma, middle ear disease, decreased lung function, and sudden infant death syndrome.
20. A considerable number of studies have been published since 1998 confirming adverse effects of exposure to SHS on a variety of endpoints in children. These include impairment of lung function (49-51), respiratory symptoms in adolescents (52), wheezing (53), school absence due to respiratory illness (54), middle ear disease (55) and recurrent ear infections (56). A meta-analysis of studies examining the association of SHS exposure with serious lower respiratory tract infections found odds ratios of 1.71 (95% CI 1.33-2.20) for infants aged 0-2, and 1.57 (95% CI 1.28-1.91) for age 0 to 6 (57). In a meta-analysis, babies born to mothers who were non-smokers but exposed to SHS had a birthweight 28 grams less (95% CI (-41)-(-16)) than babies born to non-exposed mothers (58).

CONCLUSION

The evidence published since 1998 continues to point to a strong link between exposure to SHS and adverse health effects in children. There is no reason to revise SCOTH's conclusions relating to a number of causal effects.

EXPOSURE MEASUREMENT

21. Exposure to SHS has frequently been quantified by use of cotinine, which provides a sensitive and specific marker of nicotine uptake and has proved of value for documenting the extent of exposure in a variety of population groups. One question mark over the use of cotinine has been whether it gives an indication of risk-relevant exposure, that is whether cotinine levels are related to those of compounds more likely to be implicated in health effects. Recent data suggest that this is indeed the case.
22. Cotinine concentrations have been reported to correlate well ($r = 0.71$) with those of metabolites of the tobacco-specific nitrosamine NNK, a potent tobacco carcinogen (59). Non-smoking women exposed to tobacco smoke from their partners have been shown to have higher concentrations of NNK metabolites than unexposed women (60).
23. Studies of cotinine concentrations in representative population samples in Britain indicate that smoking by partners is an important determinant of exposure, with clear dose-response trends with partner's consumption evident (61). It has been pointed out that the extent of exposure from partner smoking is in reasonable agreement with estimates of the extent of lung cancer risk (62). Some population groups, in particular bar workers, continue to be much more heavily exposed (63, 64).
24. There is good evidence that children's exposure to SHS (which largely occurs in the home) has for some years been on a declining trend, as the prevalence of adult smoking has declined (65). However, close to half of all children still live in households with at least one smoker, and the magnitude of exposure from mothers and fathers in children whose parents still smoke does not appear to have declined. In other words, fewer children are exposed, but those that are receive the same exposure.

SHS AND RESPIRATORY SYMPTOMS AND LUNG FUNCTION IN ADULTS

25. An issue that was not considered in the previous SCOTH report was that of a link between exposure to SHS and an increase in respiratory symptoms in adults. A study of 7882 adults in 16 countries found that exposure to SHS was associated with a variety of respiratory symptoms (66). Significant dose-related trends were found between exposure to SHS and bronchial responsiveness and forced expiratory volume (FEV1). A Scottish study found that exposure to SHS was associated with lowered FEV1 and forced vital capacity (FVC) (67). There was a significant exposure-response relation for SHS at work, total exposure, and duration of daily exposure. A study of some 11,000 adults participating in the US National Health and Nutrition Survey (NHANES III) found no evidence that SHS exposure was related to a decrease in lung function in men. However, some impairment of lung function with SHS exposure was found in women, especially those with asthma (68). In an experimental study, significant declines in FVC and FEV1 were observed in both men and women after an acute exposure to sidestream tobacco smoke (69). Small reductions in FEV1 were associated with higher cotinine concentrations in a longitudinal study of non-smoking adults in Britain (70). A recent population-based case control study from Finland has provided evidence that adult-onset asthma is significantly increased by recent exposure to SHS at work (OR 2.16, 95% CI 1.26-3.72) and at home (OR 4.77, 95% CI 1.27-17.7) (71).

CONCLUSION

The evidence published since 1998 points to an association between SHS and respiratory symptoms and reduced lung function in adults.

Overview

CONCLUSION

The evidence published since 1998 reinforces the conclusions of the SCOTH report published at that time:

- The causal effect of exposure to SHS on risk of **lung cancer** has been confirmed by further original studies and by the authoritative review conducted by IARC. The pooled increased relative risk remains in good agreement with that estimated by Hackshaw, Law and Wald at 24%.
- The causal effect of exposure to SHS on risk of **ischaemic** heart disease has been confirmed and the weight of evidence is stronger now than at the time of the SCOTH report. The increased risk associated with exposure to SHS is in the order of 25%.
- There is a strong link between exposure to SHS and **adverse health effects in children**. There is no reason to revise SCOTH's conclusions relating to a number of causal effects.

The Committee would like to thank all who helped in the preparation and review of this report, in particular Professors Britton, Cook, Hackshaw, Law and Wald and Dr Dawn Milner.

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Appendix 1

Tobacco Manufacturers Association (TMA) Presentations on SHS

List of Presenters:

Peter Lee

Chris Ogden (TMA)

Chris Proctor (BAT)

Paul Sadler (ITL)

Jeff Jeffrey (Gallahar)

Witnesses:

Professor James Friend

Professor Martin Jarvis

Professor Godfrey Fowler

Professor Christine Godfrey

Professor Marion Hall

Professor Newell Johnson

Professor David Purser

Dr Stuart Logan

Ms Cathy Weatherup

Nick Adkin

Tony Doole

Keith Darrall

Appendix 2

Membership of SCOTH

Professor James Friend (Chairman)

Professor Emeritus, University of Aberdeen: Formerly Honorary Professor, Clinical Medicine and Therapeutics, University of Aberdeen and Consultant in Thoracic Medicine, Grampian Health Board

Professor Martin Jarvis (Deputy Chairman): Cancer Research UK Health Behaviour Unit, Department of Epidemiology and Public Health, University College London

Professor Ilana Crome: Professor of Addiction Psychiatry at Keele University Medical School

Professor Godfrey Fowler: Professor Emeritus of General Practice, Oxford University

Professor Hamid Ghodse: International Centre for Drug Policy, St. George's Hospital Medical School, University of London

Professor Christine Godfrey: Department of Health Sciences, University of York

Professor Roger Greenhalgh: Imperial College School of Medicine, Charing Cross Hospital, London

Professor Marion Hall: Aberdeen Maternity Hospital, Aberdeen

Professor Tim Higenbottam: AstraZeneca R&D Charnwood, Discovery Bioscience, Loughborough and Sheffield University.

Professor Newell Johnson: Director, WHO Collaborating Centre for Oral Cancer and Precancer; Department of Oral and Maxillofacial Medicine and Pathology, Guy's, Kings and St Thomas' Schools of Medicine, Dentistry and Biomedical Sciences of King's College London

Professor Stuart Logan: Peninsular Medical School, St Luke's Campus, Exeter

Professor Sir Richard Peto: Clinical Trial Service Unit, Radcliffe Infirmary, Oxford University

Professor Rosalind Smyth: Division of Child Health, School of Reproductive and Development Medicine, University of Liverpool Institute of Child Health, Alder Hey Children's Hospital, Liverpool

Professor Richard Carter (until July 2003): Royal Marsden Hospital and University of Surrey

Other Health Departments

Ginny Blakey: National Assembly for Wales, Cardiff

Dr Lesley Wilkie: Scottish Executive Health Department, Edinburgh

Cathy Weatherup: National Assembly for Wales, Cardiff

Department of Health

Nick Adkin Administrative Secretary

Dr Rob Shayer: Scientific Secretary



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