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Editorial by Davey Smith

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Abstract

Objective To measure the relation between environmental tobacco smoke, as estimated by smoking in spouses, and long term mortality from tobacco related disease.
Design Prospective cohort study covering 39 years.
Setting Adult population of California, United States.
Participants 118 094 adults enrolled in late 1959 in the American Cancer Society cancer prevention study, who were followed until 1998. Particular focus is on the 35 561 never smokers who had a spouse in the study with known smoking habits.
Main outcome measures Relative risks and 95% confidence intervals for deaths from coronary heart disease, lung cancer, and chronic obstructive pulmonary disease related to smoking in spouses and active cigarette smoking.
Results For participants followed from 1960 until 1998 the age adjusted relative risk (95% confidence interval) for never smokers married to ever smokers compared with never smokers married to never smokers was 0.94 (0.85 to 1.05) for coronary heart disease, 0.75 (0.42 to 1.35) for lung cancer, and 1.27 (0.78 to 2.08) for chronic obstructive pulmonary disease among 9619 men, and 1.01 (0.94 to 1.08), 0.99 (0.72 to 1.37), and 1.13 (0.80 to 1.58), respectively, among 25 942 women. No significant associations were found for current or former exposure to environmental tobacco smoke before or after adjusting for seven confounders and before or after excluding participants with pre-existing disease. No significant associations were found during the shorter follow up periods of 1960-5, 1966-72, 1973-85, and 1973-98.
Conclusions The results do not support a causal relation between environmental tobacco smoke and tobacco related mortality, although they do not rule out a small effect. The association between exposure to environmental tobacco smoke and coronary heart disease and lung cancer may be considerably weaker than generally believed.

Introduction

Several major reviews have determined that exposure to environmental tobacco smoke increases the relative risk of coronary heart disease, based primarily on comparing never smokers married to smokers with never smokers married to never smokers. The American Heart Association, the California Environmental Protection Agency, and the US surgeon general have concluded that the increase in coronary heart disease risk due to environmental tobacco smoke is 30% (relative risk 1.30).[1–3] Meta-analyses of epidemiological studies have reported summary relative risks of about 1.30 for coronary heart disease[4–6] and about 1.25 for lung cancer.[7 8] The US Environmental Protection Agency has classified environmental tobacco smoke as a known human carcinogen.[7] Chronic obstructive pulmonary disease, primarily asthma, bronchitis, and emphysema, has been associated with exposure to environmental tobacco smoke, but the evidence for increased mortality is sparse.[2 3]

Although these reviews come to similar conclusions, the association between environmental tobacco smoke and tobacco related diseases is still controversial owing to several limitations in the epidemiological studies.[9–14] Exposure to environmental tobacco smoke is difficult to measure quantitatively and therefore has been approximated by self reported estimates, primarily smoking history in spouses. Confounding by active cigarette smoking is so strong that the association with environmental tobacco smoke can only be evaluated among never smokers. The relation between tobacco related diseases and environmental tobacco smoke may be influenced by misclassification of some smokers as never smokers, misclassification of exposure to environmental tobacco smoke, and several potential confounders. It is also unclear how the reported increased risk of coronary heart disease due to environmental tobacco smoke could be so close to the increased risk due to active smoking, since environmental tobacco smoke is much more dilute than actively inhaled smoke.

Most epidemiological studies have found that environmental tobacco smoke has a positive but not statistically significant relation to coronary heart disease and lung cancer. Meta-analyses have combined these inconclusive results to produce statistically significant summary relative risks.[4–8] However, there are problems inherent in using meta-analysis to establish a causal relation.[9–14] The epidemiological data are subject to the limitations described above. They have not been collected in a standardised way, and some relative risks have been inappropriately combined. Because it is more likely that positive associations get published, unpublished negative results could reduce the sum-

mary relative risks. Also, the meta-analyses on coronary heart disease omitted the published negative results from the large American Cancer Society cancer prevention study (CPS I).[10 11] We have extended the follow up for the California participants in this cohort, analysed the relation between environmental tobacco smoke and tobacco related diseases, and addressed concerns about this study.

Methods

CPS I is a prospective cohort study begun by the American Cancer Society in October 1959.[15–17] Long term follow up was undertaken at the University of California at Los Angeles on all 118 094 participants from California (see bmj.com for details).[18] In mid-1999 we sent out a two page questionnaire on smoking and lifestyle. The follow up period was from time of entry to the study (1 January to 31 March 1960) until death, withdrawal (date last known alive), or end of follow up (31 December 1998). The participants were aged 30-96 years at enrolment.

The underlying cause of each death was assigned according to the international classification of diseases (seventh, eighth, or ninth revision). For the analysis of environmental tobacco smoke we selected the 35 561 participants who had never smoked as of 1959 and who had a spouse in the study with known smoking habits.

Statistical analysis

The independent variable used for analysis was exposure to environmental tobacco smoke based on smoking status of the spouse in 1959, 1965, and 1972. Never smokers married to current or former smokers were compared with never smokers married to never smokers. The never smokers were defined as those who had never smoked any form of tobacco by the time of assessment. Never smokers married to a current smoker were subdivided into categories according to the smoking status of their spouse: 1-9, 10-19, 20, 21-39, ≥40 cigarettes consumed per day for men and women, along with pipe or cigar usage for women. Former smokers were considered as an additional category.

We calculated the age adjusted relative risk of death and 95% confidence interval as a function of smoking status of the spouse by using Cox proportional hazards regression.[18] A fully adjusted relative risk was calculated by using a model that included age and seven potential confounders at baseline: race (white, non-white), education level (<12, 12, >12 years), exercise (none or slight, moderate, heavy), body mass index (<20, 20-22.99, 23-25.99, 26-29.99, ≥30), urbanisation (five population sizes), fruit or fruit juice intake (0-2, 3-4, 5-7 days a week), and health status (good, fair, poor, sick).

Results

The personal and lifestyle characteristics and follow up status for 1959 never smokers were relatively independent of their spouse's smoking status (see bmj.com). Also, the baseline characteristics of the 1999 respondents in 1959 were similar to those for all participants in 1959, except for a younger age at enrolment. Although heavily censored by age, the 1999

Table 1 Level of smoking in spouse and deaths from selected causes among male never smokers in California cancer prevention study (CPS I) cohort, as of 1959 and 1972. Relative risk (95% confidence interval) comparing individuals with each level of exposure to those without exposure. Proportional hazards linear models adjusted for age and for age and seven confounders. For reference, 1960-98 death rate in deaths per 1000 person years adjusted to 1960 US population for attained ages 35-84 is given[18]

	All 1959 participants, followed 1960-98			1959 participants aged ≥50, followed 1960-98		Participants defined in 1972, followed 1973-98	
Smoking in spouse and cause of death	No of deaths/No of participants	Age adjusted relative risk (95% CI)	Fully adjusted relative risk (95% CI)	No of deaths/No of participants	Age adjusted relative risk (95% CI)	No of deaths/No of participants	Age adjusted relative risk (95% CI)
Coronary heart disease (death rate 3.05/1000)							
Never (1)*	1860/7458	1.00	1.00	1534/5201	1.00	806/3404	1.00
Former (2)*	126/624	0.94 (0.78 to 1.12)	0.94 (0.77 to 1.14)	83/323	0.93 (0.74 to 1.16)	114/573	0.94 (0.77 to 1.14)
Current (cigarettes/day):							
1-9 (3)*	81/392	0.97 (0.78 to 1.21)	0.98 (0.78 to 1.24)	59/230	1.00 (0.77 to 1.30)	20/89	1.32 (0.84 to 2.06)
10-19 (4)*	99/513	0.86 (0.70 to 1.05)	0.82 (0.66 to 1.02)	73/282	0.91 (0.72 to 1.15)	33/153	1.02 (0.72 to 1.45)
20 (5)*	81/458	0.92 (0.74 to 1.15)	0.89 (0.70 to 1.13)	58/245	1.02 (0.78 to 1.32)	35/189	0.94 (0.67 to 1.32)
21-39 (6)*	27/129	1.16 (0.79 to 1.69)	1.13 (0.76 to 1.68)	19/62	1.30 (0.82 to 2.04)	14/58	1.20 (0.70 to 2.03)
≥40 (7)*	13/45	1.29 (0.75 to 2.22)	1.24 (0.70 to 2.19)	9/26	1.25 (0.65 to 2.41)	4/36	0.65 (0.24 to 1.73)
Total of current smokers	301/1537	0.94 (0.83 to 1.07)	0.92 (0.80 to 1.05)	218/845	1.00 (0.87 to 1.15)	106/525	1.04 (0.85 to 1.27)
Ever	427/2161	0.94 (0.85 to 1.05)	0.93 (0.83 to 1.04)	301/1168	0.98 (0.86 to 1.11)	220/1098	0.99 (0.85 to 1.15)
7 level index	2287/9619	0.99 (0.95 to 1.02)	0.98 (0.94 to 1.02)	1835/6369	1.00 (0.96 to 1.05)	1026/4502	1.00 (0.95 to 1.05)
Lung cancer (death rate 0.11/1000)							
Never	65	1.00	1.00	50	1.00	27	1.00
Former	5	0.92 (0.37 to 2.30)	0.82 (0.29 to 2.26)	3	0.89 (0.28 to 2.88)	3	0.63 (0.19 to 2.09)
Current	9	0.69 (0.34 to 1.39)	0.57 (0.26 to 1.26)	5	0.60 (0.24 to 1.52)	1	0.23 (0.03 to 1.68)
Ever	14	0.75 (0.42 to 1.35)	0.63 (0.33 to 1.22)	8	0.69 (0.32 to 1.46)	4	0.43 (0.15 to 1.24)
7 level index	79	0.94 (0.77 to 1.14)	0.88 (0.70 to 1.10)	58	0.91 (0.71 to 1.17)	31	0.68 (0.41 to 1.13)
Chronic obstructive pulmonary disease (death rate 0.12/1000)							
Never	69	1.00	1.00	59	1.00	30	1.00
Former	5	0.95 (0.38 to 2.37)	1.00 (0.40 to 2.50)	4	1.09 (0.40 to 3.02)	4	0.88 (0.31 to 2.50)
Current	17	1.40 (0.82 to 2.40)	1.28 (0.72 to 2.27)	13	1.51 (0.82 to 2.78)	7	1.80 (0.78 to 4.17)
Ever	22	1.27 (0.78 to 2.08)	1.20 (0.72 to 2.00)	17	1.39 (0.81 to 2.41)	11	1.29 (0.64 to 2.61)
7 level index	91	1.06 (0.91 to 1.25)	1.05 (0.88 to 1.24)	76	1.09 (0.91 to 1.30)	41	1.08 (0.86 to 1.38)

*Values in parentheses are index level of environmental tobacco smoke.

Table 2 Level of smoking in spouse and deaths from selected causes among female never smokers in California cancer prevention study (CPS I) cohort, as of 1959 and 1972. For reference, 1960-98 death rate in deaths per 1000 person years adjusted to 1960 US population for attained ages 35-84 is given[18]

	All 1959 participants, followed 1960-98			1959 participants aged ≥50, followed 1960-98		Participants defined in 1972, followed 1973-98	
Smoking in spouse and cause of death	No of deaths/No of participants	Age adjusted relative risk (95% CI)	Fully adjusted relative risk (95% CI)	No of deaths/No of participants	Age adjusted relative risk (95% CI)	No of deaths/No of participants	Age adjusted relative risk (95% CI)
Coronary heart disease (death rate 1.65/1000)							
Never (1)*	1053/7399	1.00	1.00	891/4230	1.00	428/3090	1.00
Former (2)*	1059/6858	1.02 (0.93 to 1.11)	1.03 (0.94 to 1.13)	909/4424	0.98 (0.89 to 1.08)	772/5079	1.03 (0.92 to 1.16)
Current:							
Pipe or cigar (3)*	389/2691	0.99 (0.88 to 1.11)	0.97 (0.86 to 1.10)	162/1735	0.97 (0.86 to 1.10)	24/173	0.99 (0.66 to 1.49)
1-9 cigarettes/day (4)*	183/1102	1.13 (0.97 to 1.33)	1.03 (0.86 to 1.23)	162/719	1.15 (0.97 to 1.36)	24/200	0.89 (0.59 to 1.34)
10-19 cigarettes/day (5)*	310/2117	1.03 (0.91 to 1.17)	0.99 (0.86 to 1.14)	272/1301	1.03 (0.90 to 1.18)	42/344	0.90 (0.66 to 1.24)
20 cigarettes/day (6)*	412/3288	1.04 (0.92 to 1.16)	1.02 (0.90 to 1.16)	309/1735	0.96 (0.84 to 1.10)	89/616	1.30 (1.04 to 1.64)
21-39 cigarettes/day (7)*	167/1646	0.95 (0.80 to 1.12)	0.88 (0.74 to 1.06)	127/792	0.95 (0.79 to 1.15)	25/239	1.14 (0.76 to 1.71)
≥40 cigarettes/day (8)*	72/841	0.83 (0.65 to 1.06)	0.80 (0.62 to 1.03)	49/399	0.74 (0.55 to 0.98)	20/211	0.89 (0.57 to 1.40)
Total of current smokers	1533/11685	1.01 (0.93 to 1.09)	0.97 (0.89 to 1.06)	1258/6681	0.98 (0.90 to 1.07)	224/1783	1.06 (0.90 to 1.25)
Ever	2592/18543	1.01 (0.94 to 1.08)	0.99 (0.92 to 1.08)	2167/11105	0.98 (0.91 to 1.06)	996/6862	1.04 (0.93 to 1.16)
8 level index	3645/25942	1.00 (0.98 to 1.01)	0.99 (0.97 to 1.00)	3058/15335	0.99 (0.97 to 1.01)	1424/9952	1.02 (0.98 to 1.05)
Lung cancer (death rate 0.08/1000)							
Never	51	1.00	1.00	31	1.00	25	1.00
Former	51	1.08 (0.73 to 1.60)	1.04 (0.69 to 1.57)	33	1.02 (0.62 to 1.66)	39	0.92 (0.56 to 1.53)
Current	75	0.93 (0.65 to 1.33)	0.88 (0.60 to 1.28)	44	0.86 (0.54 to 1.36)	14	1.00 (0.52 to 1.92)
Ever	126	0.99 (0.72 to 1.37)	0.94 (0.66 to 1.33)	77	0.93 (0.61 to 1.41)	53	0.95 (0.59 to 1.53)
8 level index	177	0.97 (0.91 to 1.04)	0.97 (0.90 to 1.05)	108	0.98 (0.89 to 1.07)	78	0.99 (0.87 to 1.13)
Chronic obstructive pulmonary disease (death rate 0.08/1000)							
Never	45	1.00	1.00	35	1.00	21	1.00
Former	50	1.17 (0.78 to 1.75)	1.24 (0.80 to 1.93)	37	1.01 (0.64 to 1.60)	36	1.00 (0.59 to 1.72)
Current	78	1.11 (0.77 to 1.60)	1.12 (0.74 to 1.69)	54	0.94 (0.61 to 1.44)	18	1.57 (0.84 to 2.96)
Ever	128	1.13 (0.80 to 1.58)	1.16 (0.80 to 1.70)	91	0.97 (0.66 to 1.44)	54	1.14 (0.69 to 1.89)
8 level index	173	0.99 (0.92 to 1.06)	0.98 (0.91 to 1.06)	126	0.97 (0.89 to 1.06)	75	1.06 (0.94 to 1.20)

*Values in parentheses are index level of environmental tobacco smoke.

respondents seemed reasonably representative of survivors. Race, education, exercise, height, weight, and fruit intake had also remained largely unchanged among the 1999 respondents since 1959. The proportion of participants who had withdrawn as of 1972, were lost as of 1999, or had an unknown cause of death was not related to the smoking status of spouses. However, widowhood (widowed as of 1999) increased substantially with increased smoking by the spouse.

Effects of exposure

Environmental tobacco smoke

Exposure to environmental tobacco smoke was not significantly associated with the death rate for coronary heart disease, lung cancer, or chronic obstructive pulmonary disease in men or women (tables 1 and 2). The relative risks were slightly reduced after adjustment for seven confounders. The relative risks were consistent with 1.0 for virtually every level of exposure to environmental tobacco smoke, current or former. Only the relative risks for chronic obstructive pulmonary disease suggested an association. An environmental tobacco smoke index based on seven or eight levels of smoking in a spouse gave a relative risk of about 1.0 for each level of change and no suggestion of a dose-response trend.

Active cigarette smoking

As expected there was a strong, positive dose-response relation between active cigarette smoking and deaths from coronary heart disease, lung cancer, and chronic obstructive pulmonary disease during 1960-98 (see bmj.com). These relative risks were consistent with those for the full CPS I cohort followed until 1972.[15 17] As it is generally considered that exposure to environmental tobacco smoke is roughly equivalent to smoking one cigarette per day,[4] we extrapolated the relative risk due to exposure to environmental tobacco smoke from the relative risks for smoking 1-9 cigarettes per day. These extrapolated relative risks were about 1.03 for coronary heart disease and about 1.20 for lung cancer and chronic obstructive pulmonary disease. Based on these findings, exposure to environmental tobacco smoke could not plausibly cause a 30% increase in risk of coronary heart disease in this cohort, although a 20% increase in risk of lung cancer and chronic obstructive pulmonary disease could not be ruled out.

Discussion

Our findings are based on the California cohort from the large American Cancer Society cancer prevention study (CPS I), followed during 1960-98. Although participants in CPS I are not a representative sample of the US population, the never smokers in this cohort had a total death rate that was close to that of US white never smokers.[19] Furthermore, the relative risks were based on comparisons within the cohort and should be valid.

Strengths of study

CPS I has several important strengths: long established value as a prospective epidemiological study, large size, extensive baseline data on smoking and potential confounders, extensive follow up data, and excellent long term follow up. None of the other cohort studies on environmental tobacco smoke has more strengths, and none has presented as many detailed results (see bmj.com). Considering these strengths as a whole, the CPS I cohort is one of the most valuable samples for studying the relation between environmental tobacco smoke and mortality.

Concern has been expressed that smoking status in spouses in 1959 does not accurately reflect total exposure to environmental tobacco smoke because there was so much exposure to non-residential environmental tobacco smoke at that time.[6] The 1999 questionnaire showed that smoking status of spouses was directly related to a history of total exposure to environmental tobacco smoke. It also showed that the extent of misclassification of exposure was not sufficient to obscure a true association between environmental tobacco smoke and coronary heart disease among women (see bmj.com).

Comparison with other studies

Our results for coronary heart disease and lung cancer are consistent with those of most of the other individual studies on environmental tobacco smoke,[4–8] including the results for coronary heart disease and lung cancer in the full CPS I.[10 16] Moreover, when our results are included in a meta-analysis of all results for coronary heart disease, the summary relative risks for current and ever exposure to environmental tobacco smoke are reduced to about 1.05, indicating a weak relation.

Widowhood was strongly correlated with smoking status of spouses, owing to the reduced survival of smokers. Since widowers have higher death rates than married people, controlling for widowhood would be expected to reduce the relative risks in this and other studies of smoking in spouses.[20 21] The precise effect of widowhood due to smoking in spouses still needs to be determined, but it may partially explain the positive relative risks found in other cohorts.

Conclusion

The results of the California CPS I cohort do not support a causal relation between exposure to environmental tobacco smoke and tobacco related mortality, although they do not rule out a small effect. Given the limitations of the underlying data in this and the other studies of environmental tobacco smoke and the small size of the risk, it seems premature to conclude that environmental tobacco smoke causes death from coronary heart disease and lung cancer.

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Contributors: See bmj.com

Funding: The American Cancer Society initiated CPS I in 1959, conducted follow up until 1972, and has maintained the original database. Extended follow up until 1997 was conducted at the University of California at Los Angeles with initial support from the Tobacco-Related Disease Research Program, a University of California research organisation funded by the Proposition 99 cigarette surtax (www.ucop.edu/srphome/trdrp). After continuing support from the Tobacco-Related Disease Research Program was denied, follow up through 1999 and data analysis were conducted at University of California at Los Angeles with support from the Center for Indoor Air Research, a 1988-99 research organisation that received funding primarily from US tobacco companies.[22]

Competing interests: In recent years JEE has received funds originating from the tobacco industry for his tobacco related epidemiological research because it has been impossible for him to obtain equivalent funds from other sources. GCK never received funds originating from the tobacco industry until last year, when he conducted an epidemiological review for a law firm which has several tobacco companies as clients. He has served as a consultant to the University of California at Los Angeles for this paper. JEE and GCK have no other competing interests. They are both lifelong non-smokers whose primary interest is an accurate determination of the health effects of tobacco.

What is already known on this topic

Exposure to environmental tobacco smoke is generally believed to increase the risk of coronary heart disease and lung cancer among never smokers by about 25%

This increased risk, based primarily on meta-analysis, is still controversial due to methodological problems

What this study adds

In a large study of Californians followed for 40 years, environmental tobacco smoke was not associated with coronary heart disease or lung cancer mortality at any level of exposure

These findings suggest that the effects of environmental tobacco smoke, particularly for coronary heart disease, are considerably smaller than generally believed

Active cigarette smoking was confirmed as a strong, dose related risk factor for coronary heart disease, lung cancer, and chronic obstructive pulmonary disease

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Prevalence of hardcore smoking in England, and associated attitudes and beliefs: cross sectional study

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ELPS

This is an abridged version; the full version is on bmj.com

Abstract

Objective To quantify the prevalence and characteristics of hardcore smokers in England.
Design Cross sectional survey.
Setting Interview in respondents' household.
Participants 7766 adult cigarette smokers.
Main outcome measures Hardcore smoking defined by four criteria (less than a day without cigarettes in the past five years; no attempt to quit in the past year; no desire to quit; no intention to quit), all of which had to be satisfied.
Results Some 16% of all smokers were categorised as hardcore. Hardcore smoking was associated with nicotine dependence, socioeconomic deprivation, and age, rising from 5% in young adults aged 16-24 to 30% in those aged ≥65 years. Hardcore smokers displayed distinctive attitudes towards and beliefs about smoking. In particular they were likely to deny that smoking affected their health or would do so in the future. Prevalence of hardcore smoking was almost four times higher than in California.
Conclusion Hardcore smoking presents a serious challenge to public health efforts to reduce the prevalence of smoking, but the proportion of hardcore smokers does not necessarily increase as overall prevalence in a population declines. More hardcore smokers could be persuaded to quit, but this will require interventions that are targeted to the particular needs and perceptions of both socially disadvantaged and older smokers.

Introduction

The idea that there might exist a group of cigarette smokers who are especially resistant to giving up has attracted considerable interest.[1 2] No generally accepted definition of a hardcore smoker exists, but by consensus they are those who are very unlikely to give up, either because they are determined not to or because they lack any confidence in their ability to do so successfully.

There have been few attempts to quantify the extent of hardcore smoking. Recent estimates from California have indicated that about 5% of smokers aged 26 and above could be considered hard core.[3] The Californian study adopted an operational definition based on three principal characteristics: no attempts to quit in the past 12 months; an expectation of never quitting in the future; and cigarette consumption of at least 15 cigarettes per day. Typical hardcore smokers were older, white, male, of low income, poorly educated, and living alone.

We examined the prevalence and demographic correlates of hardcore smoking in Britain. We did not include cigarette consumption as one of our criteria but placed additional weight on the absence of quitting in the past and on the lack of desire to give up smoking as well as lack of intention. The main justification for including cigarette consumption as a criterion is as an indicator of dependence on tobacco. We prefer a concept that is based entirely on measures reflecting motivation. However, for purposes of comparison, we also estimated the prevalence of hardcore smoking using the Californian definition.

Methods

Data were gathered in four surveys of adults in England by the Health Education Authority in 1994, 1995, 1996, and 1997. These surveys monitored smoking prevalence and consumption, provided information about smokers' attitudes and beliefs, and examined smokers' recent attempts at quitting and their desires, intentions, and confidence of succeeding in future attempts. Households were selected by a random probability sampling technique, using the postcode as the sampling frame.

Interviewers collected basic demographic details and smoking habits for each adult in the household. In households where it was established that a current cigarette smoker (or someone who had given up within the past six months) lived, a more detailed interview was attempted with that person. Response rates for the initial household interview averaged just over 80%

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