



Title	Passive smoking: secondhand smoke does cause respiratory disease.
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- 1 International Agency for the Research on Cancer (IARC) Monograph on Tobacco Smoke and Involuntary Smoking. Volume 83, Lyon (in press).
- 2 Enstrom JE, Kabat, GC. Environmental tobacco smoke and tobacco related mortality in a prospective study of Californians, 1960-98. *BMJ* 2003;326:1057. (17 May.)
- 3 United States Department of Health and Human Services. *The health consequences of involuntary smoking a report of the surgeon general*. Washington, DC: US Government Printing Office, 1986.
- 4 United States Environmental Protection Agency. *Respiratory health effects of passive smoking: lung cancer and other disorders*. Washington DC: Office of Research and Development, 1992.
- 5 *Report of the Scientific Committee on Tobacco and Health*. London: Stationery Office, 1998.

Members of the IARC Working group: Patricia Buffler (USA), Richard Doll (UK), Elizabeth Fontham (USA), Yu-Tang Gao (China), Prakash Gupta (India), Allan Hackshaw (UK), Elena Matos (Argentina), Jonathan Samet (USA), Michael Thun (USA), Kurt Straif (France), Paolo Vineis (Italy), H-Erich Wichmann (Germany), Anna Wu (USA), David Zaridze (Russia).

Inverse correlation of smoking and education should have raised suspicion

EDITOR—It is well known that smoking is inversely correlated with education level; the highest percentage of smokers is found among those people who have not completed high school. This inverse correlation of smoking and education has been true for many years. It is referred to in the 15th edition (1977-9) of the *Encyclopedia Britannica*. Clearly, this casts suspicion on the data entry and the programming used by Enstrom and Kabat to perform their analysis,¹ because they find that the highest frequency of smoking is associated with the highest level of education.

From their table 2 (male never smokers) and table 3 (female never smokers) sorted by smoking status of spouse, they show that the heaviest smokers (≥40 cigarettes/day) are more likely to have completed high school than are non-smokers. Further, among smokers, they show that for those smoking a higher number of cigarettes the likelihood of completing high school is greater.

Because the “never smoked/formerly smoked” group does not show the expected higher proportion of high school graduates, this implies that there were a sizeable number of smokers included among the non-smokers; that would account for the spouses of “non-smokers” not exhibiting a lower rate of heart disease.

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Competing interests: None declared.

- 1 Enstrom JE, Kabat, GC. Environmental tobacco smoke and tobacco related mortality in a prospective study of Californians, 1960-98. *BMJ* 2003;326:1057. (17 May.)

Secondhand smoke does cause respiratory disease

EDITOR—The report by Enstrom and Kabat confirms that exposure to secondhand smoke causes injury to the respiratory system with the finding of a combined increased mortality risk for men and women for chronic obstructive pulmonary disease (relative risk 1.65, 95% confidence interval 1.0 to 2.73).¹ This is consistent with other investigations that show the sensitivity of the

respiratory system to secondhand smoke at all ages and in different settings. In Hong Kong several studies have shown that the exposure of infants to secondhand smoke in utero or postnatally in the home was linked to higher consultation rates and hospitalisation for respiratory and other illnesses.² Smoking in the home was clearly associated with bronchitic symptoms in a cohort of primary school children, independently of ambient air pollution.³ In an adult workforce, workplace exposures to passive smoking were associated with significant excess risks (66% to 212%) for all respiratory symptoms and increased healthcare costs.⁴ In a population survey the prevalence of secondhand smoke exposures at work was 47.5% among non-smoking full time workers compared with only 26% at home. People exposed at work were 37% more likely to consult a doctor for respiratory illness. The increased healthcare costs for primary care alone among three million workers was estimated at US\$29m (£18m; €26m) annually.⁵ Four independent case control studies on lung cancer and passive smoking in Hong Kong, reviewed by the United States Environmental Protection Agency, gave an overall relative risk of 1.48 (1.21 to 1.81).

In other words, we have epidemics of respiratory disease in Hong Kong caused by secondhand smoke. However, because of the way in which the Enstrom and Kabat paper was presented little or no attention will be paid in media reports to the findings on mortality risks from respiratory disease.

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Competing interests: AJH is a former chairman of the Hong Kong Council on Smoking and Health (COSH). THL is current vice chairman of COSH. All of the authors conduct research on the health effects of active and passive smoking and have received research funds, through their employer, the University of Hong Kong, to support their work.

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Doubts about effectiveness of age adjustment

EDITOR—According to Enstrom and Kabat's figures the greater had been a man's cigarette consumption in 1959 the less likely, it seems, was the death of his wife from coronary heart disease.¹ However, an age

bias existed in those women at the outset. In 1959 their mean age decreased with spousal smoking, such that the wives of men smoking 40 a day were a mean four years younger than wives of men smoking one to 19 a day, probably as a consequence of early death of smoking husbands of similarly aged wives (table 3 on bmj.com).

During the study period mortality from coronary heart disease fell by about 15% every four years.² The “passive” smokers were therefore predominantly from later cohorts for whom, age for age, mortality from coronary heart disease had fallen significantly in comparison to controls. The same argument applies to never smoking husbands of smoking women who had an average age four to five years lower than controls (table 2 on bmj.com). Adjusting for age alone will not remove this interaction of age and time of observation.

Moreover, the Cox proportional hazard model is critically dependent on assumed proportionality between two survival curves at all points following entry to the study.³ Mortality from coronary heart disease increases almost exponentially for most of adult life and the mortality curves of risk groups for coronary heart disease differ not only in scale but also in doubling time. As such their survival curves cannot be proportional, yet this was not tested.

The effectiveness of age adjustment in this study is questionable, the year of observation should have been taken into account, and the statistical method is potentially unsound. The biological implausibility of the trend in relative risk may well be an expression of systematic bias in the method.

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Competing interests: None declared.

- 1 Enstrom JE, Kabat, GC. Environmental tobacco smoke and tobacco related mortality in a prospective study of Californians, 1960-98. *BMJ* 2003;326:1057. (17 May.)
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Tobacco industry publishes disinformation

EDITOR—The American Cancer Society does not agree with the conclusions of Enstrom and Kabat in their analysis of environmental tobacco smoke in the cancer prevention study I (CPS-I).¹ Their study is fatally flawed because of misclassification of exposure. The cancer prevention study was started by the society in 1959 to measure the effects of active smoking, not to collect valid estimates of exposure to environmental tobacco smoke.² No information was obtained on sources of exposure to environmental tobacco smoke other than the smoking status of the spouse. Tobacco smoke was so pervasive in the United States in the 1950s and 1960s that virtually everyone was exposed, at home, at work, or in other