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Mainstream and Environmental Tobacco Smoke

GIO BATTA GORI*,[1] AND NATHAN MANTEL†

*The Health Policy Center, 6704 Barr Road, Bethesda, Maryland 20816; and
†American University, Washington, D.C. 20016

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Environmental tobacco smoke (ETS) is derived from cigarette smoldering and active smoker exhalation. Its composition displays broad quantitative differences and redistributions between gas and respirable suspended particulate (RSP) phases when compared with the mainstream smoke (MSS) that smokers puff. This is because of different generation conditions and because ETS is diluted and ages vastly more than MSS. Such differences prevent a direct comparison of MSS and ETS and their biologic activities. However, even assuming similarities on an equal mass basis, ETS-RSP inhaled doses are estimated to be between 10,000- and 100,000-fold less than estimated average MSS-RSP doses for active smokers. Differences in effective gas phase doses are expected to be of similar magnitude. Thus the average person exposed to ETS would retain an annual dose analogous to the active MSS smoking of considerably less than one cigarette dispersed over a 1-year period. By contrast, consistent epidemiologic data indicate that active smoking of some 4–5 cigarettes *per day* may not be associated with a significantly increased risk of lung cancer. Similar indications also obtain for cardiovascular and respiratory diseases. Since average doses of ETS to nonsmoking subjects in epidemiologic studies are several thousand times less than this reported intake level, the marginal relative risks of lung cancer and other diseases attributed to ETS in some epidemiologic studies are likely to be statistical artifacts, derived from unaccounted confounders and unavoidable bias.

INTRODUCTION

During the last decade, considerable attention has been devoted to the question of whether environmental tobacco smoke (ETS) causes disease in nonsmokers (USSG, 1986; NRC, 1986; EPA, 1990a). Some epidemiologic studies of nonsmokers presumably exposed to ETS have suggested a marginal increase of risk for some diseases previously associated with active mainstream smoking (MSS). These reported risks, however, border on statistical and epidemiologic insignificance, and could easily derive from numerous and documented biases and confounders.

Official reviews have stopped short of implying a causal role of ETS in most of these associations, with a notable exception for lung cancer. This exception has been based not so much on admittedly questionable epidemiology, but on a public health stance of concern driven by perceived—but largely undocumented—compositional similar-

[1] To whom correspondence should be addressed.

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TABLE 6

REPORTED INDEPENDENT RISK FACTORS FOR LUNG CANCER

Factor	Reference	Maximum RR reported	95% CI
Family history of lung cancer	Samet *et al.* (1986)	5.3	(2.2–12.8)
	Ooi *et al.* (1986)	2.4	
	Horwitz *et al.* (1985)	2.8	(1.0–7.7)
	Wu *et al.* (1988)	3.9	(2.0–7.6)
Family history of tuberculosis	Wu *et al.* 1988)	10.0	(1.1–90.1)
	Sakurai *et al.* (1989)	6.4	
	Gao *et al.* (1987)	1.7	(1.1–2.4)
	Hinds *et al.* (1982)	8.2	(1.3–54.4)
β-carotene/vitamin A deficiency	Byers *et al.* (1987)	0.3	(P = 0.06 trend)
	Pastorino *et al.* (1987)	0.2	
	Wu *et al.* (1985)	0.4	(0.2–0.9)
	Ziegler *et al.* (1986)	2.2	
Alcohol intake	Pollack *et al.* (1984)	2.19	(1.3–5.0)
Dietary cholesterol/fat	Goodman *et al.* (1988)	2.2	(1.3–3.8)
Dietary fat intake	Wynder *et al.* (1987)	4–6	
Pork meat intake	Mettlin (1989)	2.4	(1.4–4.2)
Vegetable diet	Jain *et al.* (1990)	0.6	(0.4–0.88)
	Le Marchand *et al.* (1989)	0.3	(P = 0.009 trend)
Fruit intake	Koo (1988)	0.4	(0.2–0.9)
Milk intake	Mettlin (1989); Mettlin *et al.* (1990)	2.1	(1.4–3.2)
Hormone therapy in women	Adami *et al.* (1989)	1.3	
Cooking methods	Gao *et al.* (1987)	1.4–2.6	(1.1–5.0)
	Geng *et al.* (1988)	5.6	(3.4–9.1)
	Sobue *et al.* (1990)	1.9	(1.1–3.3)
	Mumford *et al.* (1987)	2–3	
Radon	Edlin *et al.* (1984)	4.3	(1.7–10.6)
	Lees *et al.* (1987)	2.4	(0.8–7.1)
Occupation	Kvale *et al.* (1986)	2.6	
Motor exhaust exposure	Hayes *et al.* (1989)	1.5	(1.2–1.9)
Socioeconomic class	Brown *et al.* (1975)	2.6–3.8	
Ventilatory function	Lange *et al.* (1990)	2–4	
Cardiac anomalies	Tenkanen *et al.* (1987)	2.4	
Physical inactivity	Albanes *et al.* (1989)	1.6	(1.2–3.5)
	Severson *et al.* (1989)	1.4	(1.0–2.1)
Psychosocial traits	Kulesza *et al.* (1989)	2–3	
Urban/rural risk ratio	Shy (1984)	1.2–2.8	

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REFERENCES

American Conference of Governmental and Industrial Hygienists (ACGIH) (1990). *Documentation of the Threshold Limit Values and Biological Exposures Indices.* 5th ed. plus supplements (1986–1990). ACGIH, Cincinnati.

ADAMI, H. O., PERSSON, I., HOOVER, R., SCHAIREK, C., AND BERGKVIST, L. (1989). Risk of cancer in women receiving hormone replacement therapy. *Int. J. Cancer* 44, 833–839.