REGULATORY TOXICOLOGY AND PHARMACOLOGY 21, 184–191 (1995)

Publication Bias in the Environmental Tobacco Smoke/ Coronary Heart Disease Epidemiologic Literature[1]

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Received June 11, 1994

Two approaches are used to assess publication bias in the environmental tobacco smoke/coronary heart disease (ETS/CHD) literature: (1) Statistical tests applied to all sex-specific relative risk (rr) estimates from 14 previously published studies indicate that publication bias is likely. A funnel graph of the studies' log relative risks plotted against their standard errors is asymmetrical, and weighted regression of the studies' log relative risks on their standard errors is significant ($P < 0.01$). (2) Previously unpublished ETS/CHD relative risks from the American Cancer Society's Cancer Prevention Studies (CPS-I and CPS-II) and the National Mortality Followback Survey (NMFS) do not show an increased CHD risk associated with ETS exposure. CPS-I: men, rr = 0.97 (0.90–1.05); CPS-I: women, rr = 1.03 (0.98–1.08); CPS-II: men, rr = 0.97 (0.87–1.08); CPS-II: women, rr = 1.00, (0.88–1.14); NMFS: men, rr = 0.97 (0.73–1.28); women, rr = 0.99 (0.84–1.16). Comparison of pooled relative risk estimates from 14 previously published studies (rr = 1.29; 1.18–1.41) and unpublished results from three studies (rr = 1.00; 0.97–1.04) also indicates that published data overestimate the association of spousal smoking and CHD ($\chi^2 = 25.1$; $P < 0.0001$).

INTRODUCTION

Many papers have appeared in recent years addressing the problem of publication bias (Rosenthal, 1979; Simes, 1986a,b; Chalmers *et al.*, 1987, 1990; Dickersin, 1990; Dickersin *et al.*, 1987, 1992; Light, 1987; Begg and Berlin, 1988; Peto, 1992). Nearly everyone agrees that publication bias tends to distort estimates of association obtained by pooling the results of published studies (i.e., quantitative meta-analysis), so that inferences about the presence and size of associations are rarely appropriately conservative. It is all but unanimously agreed that publication bias is a serious problem.

Publication bias is the systematic error in the published literature produced when the results of studies influence the decisions, by authors or by editors, to publish. It has long been suspected that chance, together with a preference for statistical significance when publishing small studies, plays a major role in publication bias (Rosenthal, 1979). However, the publication process is complex and is affected by the preferences of funding agencies, editors, and authors. Thus, publication of industry-financed research on environmental exposures may be conditioned on finding null results (Bross, 1981; Kotelchuk, 1974), publication of drug company-financed research may be conditioned on finding benefit from a new therapy (Davidson, 1986), and publication of agency-financed research may be conditioned on support for the agency's goals and objectives (Rennie and Flanagin, 1992). In addition, bias in favor of politically correct results may be a growing problem (Smith, 1980; Glass *et al.*, 1981; Feinstein, 1992).

Editors once openly stated a preference for statistically significant results (Melton, 1962). Today it is recognized that such standards are certain to produce publication bias, and editors are more inclined to publish null studies than they were a few years ago, especially if they contradict an earlier study and are of "equal or superior quality" (Angell, 1989). However, studies reporting null results will always have to meet a higher editorial standard of excellence than positive studies because, at a minimum, null results require high statistical power in order to be interpreted at all. It is probably safe to say that no scientific publication is free of bias. Therefore, we must learn to identify the effects of publication bias and adjust our inferences accordingly.

There are both empirical and methodological grounds for suspecting that publication bias may have inflated relative risk estimates derived from meta-analysis on results of published epidemiologic studies on ETS and various disease endpoints (Vandenbroucke, 1988; Peto, 1992; Dickersin and Berlin, 1992). In the case of coronary heart disease (CHD) most of the ETS/CHD studies are quite small, as seen in Table 1. Begg and Berlin (1988) noted that the presence of several small studies

[1] This work was supported in part by funding from Phillip Morris U.S.A. The views expressed represent the personal opinions of the authors and are not necessarily those of Phillip Morris U.S.A.

0273-2300/95 $6.00

TABLE 1
Previously Published ETS–Heart Disease Epidemiologic Studies

Study	Sex	No. cases Un:Ex[a]	ETS by design[b]	Relative risk	95% CI Lower	95% CI Upper
1. Butler (1988)	F	60:4	Yes	1.40	0.51	3.84
2. Dobson *et al.* (1991)	F	117:43	Yes	2.46	1.47	4.13
	M	161:22		0.97	0.50	1.86
3. Garland *et al.* (1985)	F	2:17	No	2.70	0.90	13.60
4. He *et al.* (1989)	F	9:25	?	1.50	0.9	2.51
5. He *et al.* (1994)	F	11:15	Yes	1.24	0.56	2.72
6. Helsing *et al.* (1988)	F	437:551	No	1.24	1.1	1.4
	M	248:122		1.31	1.1	1.6
Sandler and Shore (1989)	F	(Same)	No	1.19	1.04	1.36
	M			1.31	1.05	1.64
7. Hirayama (1984)	F	118:376	No	1.15	0.94	1.42
8. Hole *et al.* (1989)	F & M	84	No	2.01	1.21	3.35
Gillis *et al.* (1984)	F	2:19		3.56	0.83	15.4
	M	18:14		1.29	0.64	2.64
9. Humble *et al.* (1990)	F	27:49	No	1.59	0.99	2.57
10. Jackson (1989)	F	20	Yes	4.00	1.35	13.1
	M	49		1.10	0.40	3.00
11. LaVecchia *et al.* (1993)	F	44	Yes	1.19	0.49	2.87
	M	69		1.43	0.59	2.94
12. Lee *et al.* (1986)	F	22:55	Yes	0.97	0.56	1.69
	M	26:15		1.34	0.64	2.80
13. Martin *et al.* (1986)	F	23	?	2.6	1.20	5.70
14. Svendsen *et al.* (1987)	M	8:5	No	2.23	0.72	6.92
Summary results[c]				1.29	1.18	1.41

[a] Number of heart disease cases—ETS unexposed:ETS exposed.
[b] Original design of study.
[c] Most recent results are used in cases of multiple reporting.

on the same issue increases the risk of publication bias. Lee (1992) observed that the ETS/CHD literature shows signs of publication bias, noting that in several large cohort studies there are relevant unpublished data. Bero *et al.* (1994) disputed this view, asserting that publication bias has not influenced the ETS epidemiologic literature. The study reported here was undertaken to evaluate the ETS/CHD literature for evidence of publication bias.

Statistical Tests of Publication Bias

Several statistical methods of detecting and quantifying publication bias have been recommended. Rosenthal (1979) proposed correcting pooled P values based upon an estimate of the number of unpublished studies. That estimate is computed by adding the standard normal deviates associated with the P values obtained and dividing by the square root of the number of studies being combined. This method is computationally simple, but it has been criticized on grounds that it forces the assumption of zero effect in the unpublished studies, it ignores possible variation in unpublished study size, and it produces neither an estimate of treatment effect nor a test of significance of the effect of publication bias (Begg and Berlin, 1988).

Hedges (1984), Hedges and Olkin (1985), and Berlin *et al.* (1989) proposed methods of evaluating publication bias based upon truncated sampling models. Iyengar and Greenhouse (1988) proposed methods based upon weighted distribution theory. These methods are computationally difficult, require an understanding of underlying models unfamiliar to many epidemiologists, and have not received much attention outside of statistical journals.

Light and Pillemer (1984) recommended a simple and intuitively obvious method of evaluating publication bias that involves visual inspection for departure from what is termed a "funnel graph" appearance in a plot of the estimated size of effect against study sample size, for all studies providing data. More recently Vandenbroucke (1988) and Berlin *et al.* (1989) recommended a refinement of the funnel graph approach that uses the log relative risk for the estimated size of effect and the standard error of the log relative risk in place of the study sample size. The two methods of constructing a funnel graph are very similar. The latter approach has the advantage of using a more appropriate measure of precision of the effect estimate than study sample size.

If a collection of studies provides an unbiased estimate of the treatment effect, then random sampling error

should result in an approximately symmetrical distribution of results both above and below the summary relative risk from the pooled data, with scatter being greatest for the smallest studies and narrowing as the size of the studies increases. If substantial publication bias is present, then the plot will be markedly asymmetrical as study size decreases, and the top half of the funnel will contain most of the results.

Each recommended method for evaluating publication bias is based upon the same underlying assumption. "Our assumption is that among all studies, the effect sizes and the sample sizes should be independent . . ." (Berlin *et al.*, 1989, p. 383). This assumption constitutes a null hypothesis that was tested directly in the present study by weighted regression of the ETS/CHD studies' log relative risks against the standard errors of the log relative risks. This test is computationally simple, and the resulting regression coefficient provides a test of the statistical significance of publication bias.

Comparison with Unpublished Data

Several authors have also suggested that publication bias can be assessed by comparison of published results with results obtained from previously unpublished studies (Simes, 1986a,b; Begg and Berlin, 1988; Chalmers *et al.*, 1987; Dickersin, 1990). To make such a comparison, we analyzed data from two American Cancer Society (ACS) cohort studies, Cancer Prevention Study-I (CPS-I, sometimes referred to as the "million person study") and Cancer Prevention Study-II (CPS-II). Also, a relative risk from an analysis (Layard, 1995) of the National Mortality Followback Survey (NMFS) was used.

MATERIALS AND METHODS

Funnel Graph Test

In Fig. 1 sex-specific relative risks from all currently available ETS/CHD studies are used to produce a funnel graph by plotting log relative risks against their estimated standard errors (Vandenbroucke, 1988; Berlin *et al.*, 1989). Dashed lines are used to outline an imaginary funnel that illustrates the symmetry and dispersion of results expected from an unbiased sample of studies of different sizes.

Unpublished Data

In CPS-I more than one million men and women (456,000 men and 595,000 women) were enrolled by ACS volunteers in 25 states in 1959–1960. Information on study factors collected by questionnaire at the beginning of the study forms the basis for the present analysis. The cohort was followed for 13 years (1960–1972). Follow-up was complete for 98.4% of the cohort through June 1971 and was 92.7% complete for the 13th year of the study (1972). Causes of death were coded with the International Classification of Disease (ICD) Revision 7.

In CPS-II approximately 1.2 million subjects (509,000 men and 677,000 women) were enrolled by ACS volunteers in all 50 states, the District of Columbia, and Puerto Rico, in late 1982. Again, information on study factors collected by questionnaire at the beginning of the study forms the basis for the present analysis. The cohort was followed for 6 years (1983–1988). Vital status was ascertained for 98.2% of the cohort, and death certificates were obtained for 94% of decedents. Causes of death were coded with ICD Revision 9.

In both CPS studies, subjects were recruited by ACS volunteers from among friends, relatives, neighbors, and other acquaintances. Although volunteers came from all social classes, and tended to recruit subjects in the same socioeconomic class as themselves, the cohorts are not completely representative of the national population. For example, representative numbers of illiterate people, institutionalized people, itinerant workers, illegal aliens, military personnel, construction workers, and people who tend to move often were not included. Minority races and inner city residents were also underrepresented. Because of these differences, the socioeconomic level of the cohort was somewhat higher than that for the nation as a whole. In addition, sick people were likely to have been underrepresented in the study samples.

For the reasons stated above there is an apparent healthy person effect on the CPS death rates, as the age-specific death rates are lower for the CPS cohorts than those for the national population. Although death rates are not representative of the entire U.S. population, relative risks are based upon internal comparisons and should be reasonably reliable.

Materials and methods employed in the conduct of the ACS studies are discussed in greater detail elsewhere (Hammond, 1966; Garfinkel, 1980; Garfinkel and Stellman, 1988).

The spousal smoking definition of ETS exposure and coding of CHD mortality employed in the present analysis are similar to the operational definitions of these variables used in other published ETS/CHD epidemiologic studies. These vary somewhat between CPS-I and CPS-II and are described in greater detail below. Only self-reported never-smokers who had spouses with known smoking habits were used in the analyses.

Both CPS-I and CPS-II collected data on self-reported smoking habits in terms of cigarettes smoked per day, which we grouped into the following categories: "Ex" (former smoker), 1–19, 20–39, and 40 or more. Relative risks for increasing spousal smoking levels were tested for trend in both the sex- and study-specific results and in the pooled results. Data on cigar and pipe smoking only were also collected for men. The category "Any" ETS exposure is the global spousal smoking exposure definition. It is the exposure measure reported

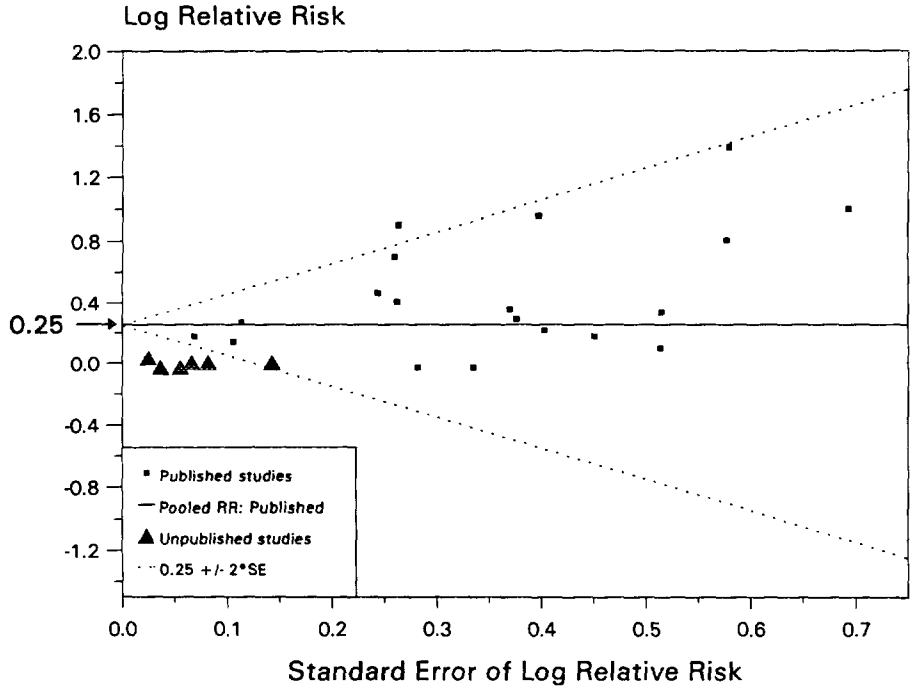

FIG. 1. Funnel graph. Sex-specific log relative risks for each study are plotted against their standard errors. Unpublished results are depicted as triangles. Previously published results are shown as squares. The solid horizontal line shows the summary log relative risk for the pooled published studies (rr = 1.29; log rr = 0.25). Dashed lines illustrate the expected dispersion of study results in the absence of bias.

in the largest number of published studies and is the definition used to summarize the sex- and study-specific relative risks and for pooling the unpublished results.

International Classification of Disease codes were used by ACS to code the cause of death listed on death certificates of deceased cohort members in both studies. These codes underwent two revisions between CPS-I and CPS-II. The definition of heart disease death for the CPS-I analysis includes the following ICD-7 codes: 420.0–420.2. The definition of heart disease death for the CPS-II analysis includes the following ICD-9 codes: 410–414. Both ICD code ranges primarily cover arteriosclerotic heart disease, myocardial infarction, and angina pectoris.

Layard (1995) reported results from an analysis of data from the NMFS. These data were collected by the National Center for Health Statistics in 1986. The NMFS was a representative 1% sample of U.S. adult deaths (>25 years). The 1986 Current Mortality Sample, a systematic sample of death certificates sent by state vital statistics offices to NCHS approximately 3 months after death, was used to select the sample for the NMFS. Materials and methods employed in the conduct of the NMFS are discussed in greater detail elsewhere (Seeman *et al.*, 1989).

In the CPS-I and CPS-II analyses the relative risk of death from CHD among never-smokers married to smokers compared to never-smokers married to never-smokers was calculated for men and women separately using Poisson regression methods (Breslow and Day, 1987). All CPS-I and CPS-II results were stratified by sex and adjusted for age and race. Relative risks were combined by computing a weighted average of the log relative risks, the weights being the inverse of the log relative risks (Woolf, 1955).

The difference between the pooled relative risks for published and unpublished results was tested for statistical significance by means of a χ^2 test.

RESULTS

Funnel Graph

Figure 1 presents the sex-specific log relative risk for each study plotted against its standard error. Unpublished results are depicted as triangles. Previously published results are shown as squares. The solid horizontal line shows the summary log relative risk for the pooled published studies (rr = 1.29; log rr = 0.25). Dashed lines illustrate the expected dispersion of study results in the absence of bias.

The plot shown in Fig. 1 is not symmetrical. Most of the results to the right of the unpublished results are in the top half of the funnel. As the standard error of the log relative risk becomes large, all of the published results are above the summary log relative risk.

Regression Analysis

Weighted regression of the log relative risks on their standard errors is statistically significant ($P < 0.01$).

TABLE 2

Age and Race of Never-Smoking Men and Women and the Smoking Status of Their Spouses in CPS-I and CPS-II

Never-smoking men		Never-smoking women	
	CPS-I		
Mean age at entry	56.1 years	Mean age at entry	52.8 years
Race	85,811 (97.0%) White	Race	259,429 (97.0%) White
	1,704 (1.9%) Black		4,852 (1.8%) Black
	943 (1.1%) Other		3,131 (1.2%) Other
Total	88,458 (100.0%)	Total	267,412 (100.0%)
Smoking status of wife	73,890 (83.5%) Never	Smoking status of husband	73,895 (27.6%) Never
	14,568 (16.5%) Ever		193,517 (72.4%) Ever
Total	88,458 (100.0%)	Total	267,412 (100.0%)
	CPS-II		
Mean age at entry	57.7 years	Mean age at entry	55.8 years
Race	98,579 (95.0%) White	Race	215,132 (95.2%) White
	2,745 (2.6%) Black		6,018 (2.6%) Black
	2,448 (2.4%) Other		4,917 (2.2%) Other
Total	103,772 (100.0%)	Total	226,067 (100.0%)
Smoking status of wife	77,339 (74.5%) Never	Smoking status of husband	77,455 (34.3%) Never
	26,433 (25.5%) Ever		148,612 (65.7%) Ever
Total	103,772 (100.0%)	Total	226,067 (100.0%)

Comparison with Unpublished Results

Table 2 gives the mean age and race of never-smoking men and women, and the smoking status of their spouses, in CPS-I and CPS-II. Table 3 gives the number of CHD deaths among never-smoking men and women in the two cohorts grouped according to the smoking status of the spouse.

In the CPS-I cohort there was a total of 88,458 male and 267,412 female never-smokers with spouses having known smoking habits. Among these subjects, there were 7758 CHD deaths in males and 7133 CHD deaths in females.

In the CPS-II cohort there was a total of 103,772 male and 226,067 female never-smokers with spouses having known smoking habits. Among these subjects there were 1966 CHD deaths in males and 1099 CHD deaths in females.

TABLE 3

Deaths from Coronary Heart Disease among Never-Smokers Grouped by Cigarettes per Day Smoked by the Spouse in CPS-I and CPS-II

	Cigarettes per day smoked by the spouse						
	None	Ex[a]	1–19	20–39	40+	Pipe/ cigar[b]	Total
CPS-I							
Men	6954	206	400	186	12	0	7758
Women	2217	1685	949	980	110	1192	7133
CPS-II							
Men	1566	223	90	77	10	0	1966
Women	376	470	56	60	19	118	1099

[a] Quit smoking before the beginning of the study.
[b] Smoked pipes and cigars only.

Table 4 presents the relative risks and 95% confidence intervals calculated from the CPS study data. Relative risks were adjusted for age and race. Further adjustment using a weight index, exercise, highest level of education, dietary factors, alcohol consumption, history of hypertension, and history of diabetes had no appreciable effect on any of the reported associations. It is evident from Table 4 that most of the relative risks are very near 1.00, regardless of sex or spousal smoking behavior. There are four relative risk estimates with confidence intervals that exclude 1.00, all of which are in men. In the CPS-II cohort, never-smoking men with exsmoking wives experienced significantly lower CHD death rates than never-smoking men with never-smoking wives (rr = 0.81, CI 0.70–0.93). Among men with wives who smoked 1–19 cigarettes per day, the relative risk was rr = 1.36 (1.10–1.68), but lower, rr = 1.26 (1.00–1.58), among men with wives who smoked 20–40 cigarettes per day, and still lower, rr = 1.13 (0.61–2.11), among men with wives who smoked 40+ cigarettes per day. No significant trend was observed in any of the separate or combined results.

After the relative risks for the two cohorts were combined, only the reduction in risk among men married to exsmokers was significant (rr = 0.88, CI 0.79–0.97). No relative risk was significant for any women married to a smoker or for any level of exposure after both sexes, and both studies, were combined.

Table 5 gives the number of never-smoking male and female cases and controls in the NMFS analysis according to the smoking status of their spouses, as well as the

TABLE 4

Relative Risks for Death from Coronary Heart Disease According to Cigarettes per Day Smoked by the Spouse in CPS-I and CPS-II[a]

Men			Women		
Exposure	rr	95% CI	Exposure	rr	95% CI
		CPS-I			
Ex[b]	0.95	0.83, 1.09	Ex[b]	0.99	0.93, 1.05
1–19	0.99	0.89, 1.09	1–19	1.04	0.97, 1.12
20–39	0.98	0.85, 1.13	20–39	1.06	0.98, 1.15
40+	0.72	0.41, 1.28	40+	0.95	0.78, 1.15
Any	0.97	0.90, 1.05	P/cigar[c]	1.06	0.99, 1.14
			Any	1.03	0.98, 1.08
		CPS-II			
Ex[b]	0.81	0.70, 0.93	Ex[b]	0.99	0.86, 1.13
1–19	1.36	1.10, 1.68	1–19	1.14	0.86, 1.51
20–39	1.26	1.00, 1.58	20–39	0.98	0.75, 1.29
40+	1.13	0.61, 2.11	40+	1.27	0.80, 2.01
Any	0.97	0.87, 1.08	P/cigar[c]	0.98	0.79, 1.20
			Any	1.00	0.88, 1.14
		Combined			
Ex[b]	0.79	0.80, 0.97	Ex[b]	0.99	0.93, 1.05
1–19	1.05	0.96, 1.15	1–19	1.05	0.97, 1.13
20–39	1.06	0.93, 1.19	20–39	1.06	0.98, 1.14
40+	0.89	0.58, 1.35	40+	0.99	0.83, 1.18
Any	0.97	0.91, 1.03	P/cigar[c]	1.05	0.98, 1.12
			Any	1.02	0.98, 1.07
		Both sexes, both cohorts, combined			
	Ex[b]	0.96		0.91, 1.01	
	1–19	1.05		0.99, 1.11	
	20–39	1.06		0.99, 1.12	
	40+	0.97		0.83, 1.15	
	Any	1.00		0.97, 1.04	

[a] Adjusted for age and race.
[b] Quit smoking before the beginning of the study.
[c] Smoked pipes and cigars only.

associated odds ratios and 95% confidence intervals (Layard, 1995). There was a total of 475 CHD deaths among never-smoking men in the case group and 998 nonsmoking-related deaths among never-smoking men in the control group. There was a total of 914 CHD deaths among never-smoking women in the case group and 1930 nonsmoking-related deaths among never-smoking women in the control group. In both men and women, the risk of CHD death was nearly the same regardless of the smoking status of the spouse (men, rr = 0.97 (0.73–1.28); women, rr = 0.99 (0.84–1.16)).

The pooled relative risk for published results, rr = 1.29 (1.18–1.41), is significantly higher than the pooled relative risk for the unpublished results, rr = 1.00 (0.97–1.04); $\chi^2 = 25.1$ ($P < 0.0001$).

DISCUSSION AND CONCLUSIONS

Statistical tests performed on results from 14 published ETS/CHD epidemiologic studies and on previously unpublished data from three studies indicate that publication bias is present in the ETS/CHD literature. A funnel graph of study results does not exhibit the symmetry expected from an unbiased collection of studies. Study size, expressed as the standard error of the log relative risk, and estimated effect size, expressed as the log relative risk, were found to be highly significantly correlated in a weighted regression analysis ($P < 0.01$) of the previously reported ETS/CHD study results. Both observations support the inference that publication of ETS/CHD results is more likely if the results are positive than if they are negative or null.

Comparison of published results with previously unpublished data from three large studies provides additional support for this conclusion. Meta-analysis of results from 14 currently published ETS/CHD epidemiologic studies produced a pooled relative risk estimate of rr = 1.29 (1.18, 1.41). Meta-analysis of previously unpublished results from the two large ACS cohort studies, and the NMFS, produced a pooled relative risk estimate of rr = 1.00 (0.97, 1.04). The difference between these two pooled relative risk estimates is highly significant ($\chi^2 = 25.1$; $P < 0.0001$). This discrepancy between the relative risk estimates derived from published and unpublished data provides further support for the inference that publication of ETS/CHD results is more likely if the results are positive than if they are negative or null.

Given the strong indications of bias in the published literature, and the complete absence of association between ETS and CHD observed in previously unpublished results described here, it is possible that publication bias alone could account for the 29% excess risk reported in the published literature. This conclusion is in sharp contrast to the conclusion of Bero *et al.* (1994) that "There is no publication bias against statistically nonsignificant results on ETS in the peer-reviewed literature."

TABLE 5

National Mortality Followback Survey Coronary Heart Disease/Environmental Tobacco Smoke Case–Control Study[a]

Spousal smoking	Cases	Controls	rr[b]	95% CI
		Men[c]		
No	378	783	1.0	
Yes	97	215	0.97	0.73–1.28
		Women[c]		
No	459	969	1.0	
Yes	455	961	0.99	0.84–1.16

[a] Layard (1995).
[b] Adjusted for age.
[c] Never-smokers.

Reasons for publication bias in the ETS/CHD literature are unclear. Many factors could cause publication bias in this field. It is generally assumed that both authors and editors favor statistically significant study results, particularly if the study is small, and that this preference accounts for most publication bias. However, most of the ETS/CHD studies are small and report nonsignificant results, so achieving statistical significance alone cannot account for the observed publication bias.

Another possible explanation for the observation of publication bias in the ETS/CHD literature is that there is relatively ample institutional financing of tobacco-related health effects research and virtually no institutional support for discussing contrary findings when reporting results. Given the large, and rapidly growing, number of studies with data that could be tested for an ETS/CHD association, and the willingness of authors and editors to publish small positive studies in this field, the effects of publication bias in the ETS/CHD literature are likely to become even greater in the future.

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