Original Contribution

Mortality among Lifelong Nonsmokers Exposed to Secondhand Smoke at Home: Cohort Data and Sensitivity Analyses

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Evidence is growing that secondhand smoke can cause death from several diseases. The association between household exposure to secondhand smoke and disease-specific mortality was examined in two New Zealand cohorts of lifelong nonsmokers ("never smokers") aged 45–77 years. Individual census records from 1981 and 1996 were anonymously and probabilistically linked with mortality records from the 3 years that followed each census. Age- and ethnicity-standardized mortality rates were compared for never smokers with and without home exposure to secondhand smoke (based on the reported smoking behavior of other household members). Relative risk estimates adjusted for age, ethnicity, marital status, and socioeconomic position showed a significantly greater mortality risk for never smokers living in households with smokers, with excess mortality attributed to tobacco-related diseases, particularly ischemic heart disease and cerebrovascular disease, but not lung cancer. Adjusted relative risk estimates for all cardiovascular diseases were 1.19 (95% confidence interval: 1.04, 1.38) for men and 1.01 (95% confidence interval: 0.88, 1.16) for women from the 1981–1984 cohort, and 1.25 (95% confidence interval: 1.06, 1.47) for men and 1.35 (95% confidence interval: 1.11, 1.64) for women from the 1996–1999 cohort. Passive smokers also had nonsignificantly increased mortality from respiratory disease. Sensitivity analyses indicate that these findings are not due to misclassification bias.

cohort studies; mortality; myocardial ischemia; neoplasms; New Zealand; respiratory tract diseases; tobacco smoke pollution

Abbreviations: ICD-9, *International Classification of Diseases*, Ninth Revision; SHS, secondhand smoke.

There is a substantial and growing body of evidence demonstrating the adverse health consequences of exposure to secondhand smoke (SHS) (1, 2). Adults exposed to SHS are at increased risk of developing lung cancer (1–6), ischemic heart disease (1, 2, 7–9), and stroke (10–12). Evidence is mounting that passive smoking also increases the risk of various respiratory diseases (1, 3, 10, 13).

A smaller collection of studies has examined mortality as the principal outcome of interest among those exposed to SHS. Cohort studies in the United States have produced evidence that passive smokers are at increased risk of death from ischemic heart disease (14–17), lung cancer (18, 19), and all causes (17, 20, 21). Relatively few studies have examined the association between SHS and mortality in population cohorts outside the United States (3, 10, 22–26).

We present findings from two large New Zealand cohorts of never smokers, describing the relation between exposure to SHS in the home and mortality over the subsequent 3 years. Data on all-cause mortality have previously been described in a short publication (27). Here, we report the

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FIGURE 1. Questions on smoking from 1981 (top) and 1996 (bottom) New Zealand censuses.

relation between household exposure to SHS and mortality from cardiovascular, respiratory, and malignant diseases. We also describe sensitivity analyses undertaken to correct for the effects of potential misclassification bias.

MATERIALS AND METHODS

Data sources

Data were derived from the New Zealand Census-Mortality Study, the methodology and structure of which are described in detail elsewhere (28). Briefly, two population cohorts were created by linking individual records from each of two New Zealand censuses (1981 and 1996) with individual mortality records from the 3 years following each census. Linkage was conducted anonymously and probabilistically by using sex, date of birth, ethnicity, country of birth, and area of residence as matching variables. Overall, about three quarters of all eligible mortality records were linked to their corresponding census record (71.0 percent for the 1981–1984 cohort and 78.2 percent for the 1996–1999 cohort) (29), with more than 97 percent of linked census-mortality pairs estimated to be correct (30). The percentage of mortality records linked to a census record varied by sex, age, ethnicity, neighborhood deprivation, rurality, and cause of death. (Deprivation was measured by an area-based index calculated from census data on socioeconomic characteristics (such as car access, housing tenure, and benefit receipt) at aggregations of about 100 people and was assigned to mortality data by using address (31).) Inverse probability weights were therefore applied to adjust for linkage bias. (For example, if records for 20 of 30 Māori male decedents aged 45–64 years and residing in moderately deprived,

small areas of New Zealand were linked to a census record, each of the 20 linked records received a weight of 1.5 (30/20). Similar inverse probability weights were calculated and applied to numerous strata (32).)

Study populations and exposure categories

The cohorts used in this study included persons aged 45–74 years who responded to the 1981 and 1996 censuses, identified themselves as lifetime nonsmokers, and lived in a private dwelling (i.e., not a prison, hospital, or other institution). We excluded persons for whom data on smoking status were not available for all other household members aged 15 years or older (figure 1). The resultant study populations comprised 87.0 percent of eligible never smokers from the 1981 census and 85.3 percent from the 1996 census.

Study populations were divided into two categories according to imputed SHS exposure in the home. Households including at least one current smoker were regarded as "smoking," and never smokers living in these households were regarded as being exposed to SHS in the home. Never smokers living in households with no current smokers ("nonsmoking" households) were regarded as unexposed to SHS in the home.

Outcome measurement

Cause of death was derived from the death record according to the *International Classification of Diseases*, Ninth Revision (ICD-9). Disease-specific deaths were grouped as follows: cardiovascular disease (ICD-9 codes 393–459), including ischemic heart disease (ICD-9 codes 410–414) and cerebrovascular disease (ICD-9 codes 430–438); lung cancer (ICD-9 code 162); respiratory diseases (ICD-9 codes

470–478 and 490–519); and non-lung cancers (ICD-9 codes 140–161 and 163–209).

Analyses

All-cause and disease-specific mortality rates were calculated for each cohort of never smokers according to SHS exposure in the home. Mortality rates were controlled for age and ethnicity by direct standardization, using the 1996 census population as the standard. Age standardization was based on 5-year age bands and ethnicity standardization on three prioritized ethnic groups (i.e., Māori, Pacific, and neither Māori nor Pacific).

Poisson regression was used to adjust for age, ethnicity, marital status, and socioeconomic position (i.e., highest-level qualification, labor force status, household equivalized income, household car access, housing tenure, and small area deprivation index). The study populations used in regression analyses were slightly smaller than those used to calculate mortality rates (comprising 85 percent of the 1981–1984 cohort and 91 percent of the 1996–1999 cohort) because of missing data for some socioeconomic variables.

Statistical analyses were undertaken by using SAS version 8.0 software (SAS Institute, Inc., Cary, North Carolina).

Sensitivity analyses

Sensitivity analyses were conducted to explore the effects of possible misclassification of personal and household smoking status on relative risk estimates. Sensitivity analyses were undertaken by using crude data (person-time and death counts were required) for all-cause mortality among men from the 1996–1999 cohort. (Crude and adjusted relative risk estimates were very similar for all-cause mortality in this group.)

Misclassification of personal smoking status.   On the basis of previous research (33–36), we estimated that approximately 1.7 percent of current smokers and 6.8 percent of former smokers misreported themselves as lifetime non-smokers in the census. Since smokers tend to aggregate socially, we assumed that this misreporting was more common in smoking compared with nonsmoking households, at a ratio of 3:1 (4).

From the above assumptions, we calculated the proportions of misclassified current and former smokers in each exposure group of our study cohort, using smoking status data on all men aged 44–74 years in the 1996 census (refer to the Appendix). Using observed mortality rates by smoking status (Appendix), we then calculated the corrected numbers of persons and deaths by smoking status and SHS exposure, the numbers of deaths among misclassified current and former smokers, and (by subtraction) the corrected numbers of deaths among nonsmokers within each exposure group. Mortality by SHS exposure was recalculated by using these corrected figures, yielding the "true" mortality rate ratio adjusted for misclassification of personal smoking status.

Misclassification of SHS exposure. Misclassification of SHS exposure arises from three sources: misreported smoking status by household members (leading to misclassification of household smoking status), unmeasured SHS exposure outside the home (meaning that household SHS exposure will underestimate a person's total SHS exposure), and changes in SHS exposure over time. Considering these sources, we estimated this study's measure of SHS exposure to have a sensitivity of about 88 percent and a specificity of about 94 percent (refer to the Appendix for assumptions and estimations). Corrected mortality rates and rate ratios were calculated by using a method similar to that described by Greenland (37): the estimated sensitivity and specificity of the exposure measure were used to calculate corrected person-years, deaths, and mortality rates in each exposure group (Appendix).

RESULTS

The study cohorts comprised approximately 286,800 non-smokers from the 1981 census and 381,462 from the 1996 census, with 23.2 percent and 14.5 percent, respectively, living in households with at least one current smoker (table 1). Nonsmokers living in smoking households tended to be younger and were more likely to be married than those living in nonsmoking households. The two exposure groups had broadly similar income distributions, but persons from smoking households were slightly less likely to have a post-school qualification and slightly more likely to live in a deprived neighborhood. The prevalence of Māori and Pacific peoples was higher in smoking households. There were 0.84 million person-years of observation and 10,188 deaths in the 1981–1984 cohort, and 1.13 million person-years of observation and 9,153 deaths in the 1996–1999 cohort (weighted estimates) (table 2).

Mortality was higher among never smokers exposed to SHS at home (table 2, figures 2 and 3), with mortality differences by household SHS exposure most pronounced for women from the 1996–1999 cohort and least pronounced for women from the 1981–1984 cohort. Mortality from cardiovascular disease was higher for those with SHS exposure in every study group except 1981–1984 women. Mortality rates for ischemic heart disease and cerebrovascular disease were similarly elevated for SHS-exposed persons in all but the 1981–1984 group of women. Only a small number of lung cancer deaths occurred in any of the study groups, reflected in relatively low mortality rates. Deaths from respiratory diseases were also few, although in all instances mortality was higher in the SHS-exposed group. Mortality from non-lung cancer tended to be higher among those persons from smoking households.

Never smokers in smoking households had a consistently elevated relative risk of death from cardiovascular and respiratory diseases (table 3). Cardiovascular mortality risk was significantly higher for passive smokers in all but the 1981–1984 cohort of women, with rate ratios of 1.19 (95 percent confidence interval: 1.04, 1.38) for men and 1.01 (95 percent confidence interval: 0.88, 1.16) for women from the 1981–1984 cohort, and 1.25 (95 percent confidence interval: 1.06, 1.47) for men and 1.35 (95 percent confidence interval: 1.11, 1.64) for women from the 1996–1999 cohort. Statistical power was limited when cardiovascular mortality was broken down into more specific causes. Adjusted relative risk

TABLE 1.   Characteristics of 1981 and 1996 New Zealand census cohorts (never smokers aged 45–77 years) by sex and household smoking status

Household smoking status	1981 cohort				1996 cohort			
	Men		Women		Men		Women	
	Nonsmoking	Smoking	Nonsmoking	Smoking	Nonsmoking	Smoking	Nonsmoking	Smoking
Total no.*	72,504	19,578	147,822	46,893	131,163	21,450	194,958	33,891
Age (years) at census (%)								
45–64	74.2	85.6	64.7	81.8	77.6	86.0	68.9	83.2
65–74	25.8	14.4	35.3	18.2	22.4	14.0	31.1	16.8
Ethnicity (%)								
Māori	3.8	12.1	2.6	6.3	5.0	14.9	4.1	10.2
Pacific	1.2	2.5	0.8	2.5	2.1	5.9	2.0	7.9
Non-Māori non-Pacific	95.0	85.4	96.6	91.2	92.9	79.2	93.9	81.9
Marital status (%)								
Currently married	81.6	87.1	64.7	85.4	80.9	86.0	69.7	80.7
Previously married	9.3	7.2	27.6	12.4	9.8	7.8	23.3	15.3
Never married	8.8	4.8	7.6	1.9	7.5	4.2	5.0	2.2
Missing	0.3	0.8	0.2	0.3	1.9	2.0	2.0	1.8
Highest qualification (%)								
Postschool	26.8	21.6	15.0	11.3	44.6	36.0	29.4	22.5
School	10.6	10.1	10.0	9.7	20.1	19.4	25.8	25.0
None	61.1	66.5	72.9	77.1	34.7	43.5	43.8	51.3
Missing	1.5	1.7	2.0	1.9	0.7	1.1	1.0	1.2
Household income (quintiles) (%)†								
1 (highest)	25.3	26.9	18.3	22.5	28.0	26.4	20.4	21.4
2	19.6	21.4	15.6	20.3	20.6	22.5	18.1	21.7
3	17.0	16.2	14.7	17.0	16.9	19.7	16.1	20.6
4	15.4	11.5	17.6	14.1	15.2	10.4	23.2	12.6
5 (lowest)	11.8	6.8	22.9	10.3	13.5	9.7	16.6	12.6
Missing	10.9	17.0	10.8	15.8	5.8	11.2	5.7	11.1
Small-area deprivation (%)‡								
1–2 (least deprived)	23.9	19.7	21.3	18.1	29.6	21.8	26.7	20.4
3–4	22.3	20.1	21.3	20.2	23.5	20.2	22.9	19.5
5–6	20.7	19.4	21.3	21.0	19.5	19.2	20.5	19.9
7–8	18.8	20.1	20.0	20.7	15.9	19.0	17.5	20.0
9–10 (most deprived)	14.2	20.5	16.0	20.0	11.5	19.7	12.4	20.2
Missing	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1

* Raw numbers were randomly rounded to the nearest or second nearest multiple of three, as per Statistics New Zealand's protocol.

† Household income was equivalized by using the Luxembourg method (i.e., total household income divided by the square root of the number of household members (59)) and categorized into quintiles based on its distribution over the whole census population.

‡ New Zealand Deprivation Index, an index of material deprivation based on individual measures averaged across small geographically defined populations (31).

estimates for respiratory mortality ranged from 1.34 to 1.81, although 95 percent confidence limits were generally wide.

Based on crude data for the 1996–1999 cohort of men, the unadjusted relative risk of death from any cause was 1.14 (table 4). Correction for estimated misclassification of personal smoking status reduced this relative risk to 1.10. Correction for estimated misclassification of SHS exposure increased the relative risk estimate to 1.24. When both types of misclassification were corrected for simultaneously, the resultant relative risk estimate was 1.18.

DISCUSSION

Data from two New Zealand cohorts of linked census and mortality records show that never smokers who live

TABLE 2.   Number of person-years of observation, number of deaths, and standardized* mortality rates for 1981–1984 and 1996–1999 New Zealand cohorts (never smokers aged 45–77 years) by sex, cohort, and cause of death

	Household smoking status	1981–1984 cohort			1996–1999 cohort		
		Unweighted	Weighted†	Mortality rate‡ (per 100,000 per year)	Unweighted	Weighted†	Mortality rate‡ (per 100,000 per year)
Men							
Person-years of observation	Nonsmoking	214,047	211,852		389,120	387,292	
	Smoking	57,824	57,344		63,555	63,244	
Deaths§							
Total	Nonsmoking	2,376	3,240	1,530.4	2,982	3,684	1,024.6
	Smoking	579	846	1,683.6	522	687	1,198.3
Cardiovascular¶	Nonsmoking	1,380	1,845	864.9	1,260	1,563	441.2
	Smoking	321	459	982.8	228	303	540.3
Ischemic heart disease	Nonsmoking	1,056	1,395	646.9	879	1,080	300.2
	Smoking	216	300	646.7	147	192	320.8
Cerebrovascular	Nonsmoking	174	243	112.3	165	210	60.9
	Smoking	57	84	178.5	39	51	116.3
Lung cancer	Nonsmoking	66	87	42.6	93	111	30.2
	Smoking	18	24	41.4	18	21	43.8
Respiratory	Nonsmoking	78	114	58.1	93	114	32.4
	Smoking	24	39	59.3	21	27	55.8
Non-lung cancer	Nonsmoking	513	681	312.2	1,032	1,245	339.8
	Smoking	132	180	347.8	144	180	333.4
Women							
Person-years of observation	Nonsmoking	438,383	435,423		580,059	578,216	
	Smoking	139,400	138,675		100,796	100,507	
Deaths§							
Total	Nonsmoking	3,555	4,902	1,009.8	3,333	4,026	671.6
	Smoking	855	1,200	1,050.4	591	756	854.8
Cardiovascular¶	Nonsmoking	1,716	2,358	475.4	993	1,212	205.0
	Smoking	357	498	469.8	177	231	269.4
Ischemic heart disease	Nonsmoking	1,083	1,458	286.8	564	681	113.6
	Smoking	216	300	279.8	90	111	143.3
Cerebrovascular	Nonsmoking	399	585	116.0	216	267	45.1
	Smoking	69	102	101.9	33	45	52.2
Lung cancer	Nonsmoking	48	69	17.5	102	129	20.9
	Smoking	15	21	17.4	21	27	24.3
Respiratory	Nonsmoking	87	126	30.3	111	138	24.6
	Smoking	30	45	36.1	27	36	43.2
Non-lung cancer	Nonsmoking	1,191	1,572	319.0	1,596	1,875	305.4
	Smoking	321	435	344.6	264	321	374.7

* Standardized by age (5-year age bands) and ethnicity (Māori, Pacific, and neither Māori nor Pacific).
† Weighted to adjust for linkage bias by age, sex, ethnicity, small-area deprivation, rurality, and cause of death (32).
‡ Mortality rates were calculated by using weighted data.
§ Raw numbers were randomly rounded to the nearest or second nearest multiple of three, as per Statistics New Zealand's protocol.
¶ Cardiovascular disease includes ischemic heart disease and cerebrovascular disease.

with a smoker have higher rates of cardiovascular, respiratory, and overall mortality compared with those living in smoke-free homes. Cardiovascular diseases (including ischemic heart disease and cerebrovascular disease) account for the majority of excess mortality among those exposed to SHS in the home. The large size of these cohorts enhances the precision with which we were able to estimate the mortality effect of passive smoking. Sensitivity analyses
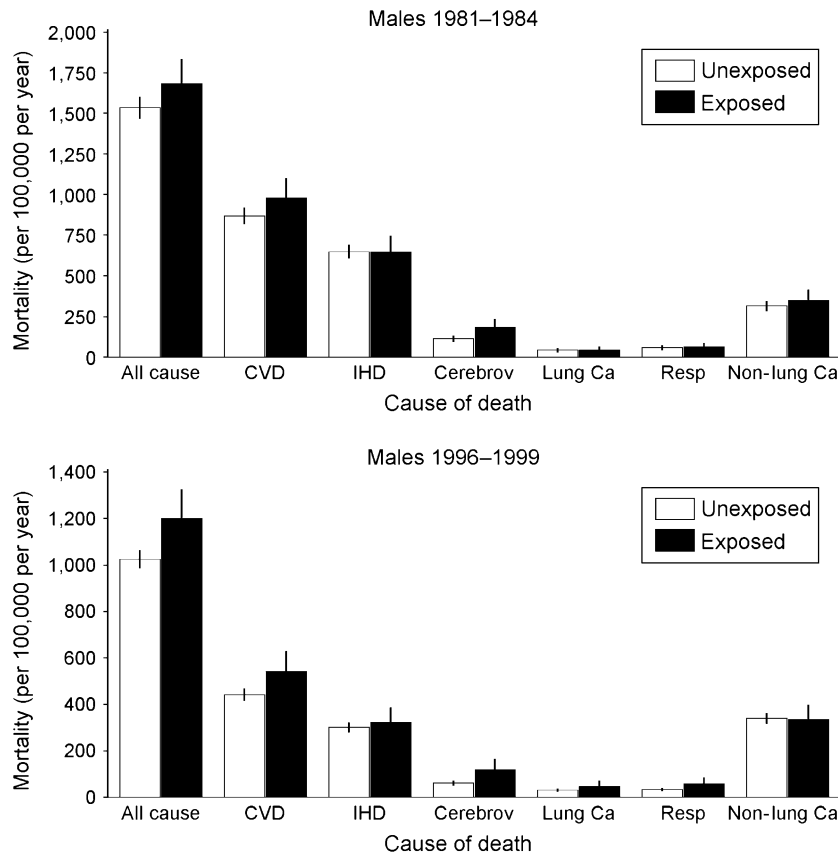
FIGURE 2. All-cause and disease-specific mortality in 1981–1984 and 1996–1999 male New Zealand cohorts (nonsmokers aged 45–77 years) by household exposure to secondhand smoke. Mortality rates (deaths per 100,000 per year) are standardized by age (5-year age bands) and ethnicity (Māori, Pacific, and neither Māori nor Pacific). Vertical lines at the top of each column, 95% confidence intervals. CVD, cardiovascular disease (*International Classification of Diseases*, Ninth Revision (ICD-9) codes 393–459); IHD, ischemic heart disease (ICD-9 codes 410–414); Cerebrov, cerebrovascular disease (ICD-9 codes 430–438); Lung Ca, lung cancer (ICD-9 code 162); Resp, respiratory disease (ICD-9 codes 470–478, 490–519); Non-lung Ca, non-lung cancer (ICD-9 codes 140–161, 163–209).

indicate the increased mortality risk observed for passive smokers is unlikely to be due to misclassification bias.

Our results are consistent with previous studies of SHS exposure and cardiovascular disease, strengthening estimates of cardiovascular mortality risk for passive smokers (10, 14–17, 24). These data also add to the growing evidence for an association between SHS exposure and adult respiratory disease (3, 10, 13).

The comparatively short duration of follow-up somewhat limited our study's power and means that it is probably best suited to detecting increased mortality risk for diseases with relatively acute mechanisms. Since our cohorts were created by linking census and mortality records, duration of follow-up was constrained by the need to maximize the accuracy and success of record linkage (which tended to decline over time). Unfortunately, we were not permitted to link records across censuses. A 3-year follow-up is probably adequate for deaths from cardiovascular diseases, where relevant biologic processes (increased platelet aggregation, impaired endothelial function, and impaired oxygen delivery) are triggered by relatively short-term exposure to environmental

tobacco smoke (38–42). However, our study is likely to have underestimated the association between SHS and diseases with a long latency (such as lung cancer), where the relevant exposure probably occurred many years before this study was undertaken. Had more detailed information been available on smoking intensity in each household, our analyses might have been enhanced by testing for a dose-response relation. The association between SHS exposure and increased mortality risk becomes clearer with greater degrees of aggregation (e.g., by grouping all cardiovascular deaths) and is most consistently apparent for all-cause mortality (reported previously by Hill et al. (27)).

A degree of exposure misclassification is inevitable (43). Since we were unable to estimate SHS exposure outside the home, our "nonexposed" study groups included a proportion of persons with significant SHS exposure. The probable effect of this misclassification is to dilute mortality differences between exposure groups, thus reducing the observed association between SHS and mortality. The opposite effect arises from misclassification of personal smoking status (44): smokers misclassified as nonsmokers are more likely
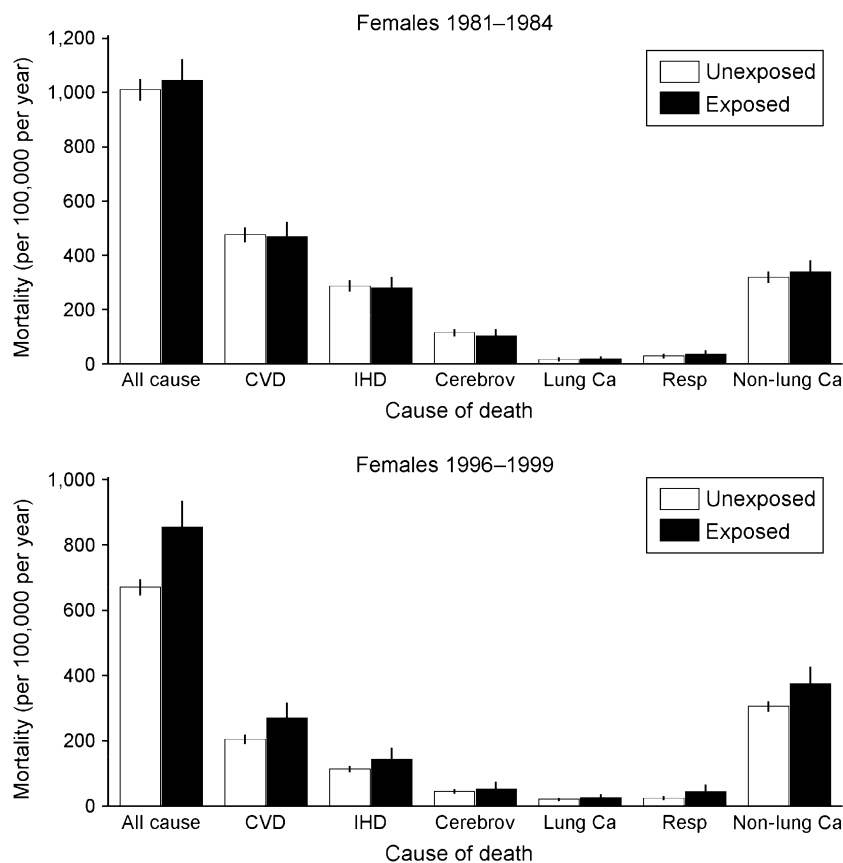
FIGURE 3.   All-cause and disease-specific mortality in 1981–1984 and 1996–1999 female New Zealand cohorts (nonsmokers aged 45–77 years) by household exposure to secondhand smoke. Mortality rates (deaths per 100,000 per year) are standardized by age (5-year age bands) and ethnicity (Māori, Pacific, and neither Māori nor Pacific). Vertical lines at the top of each column, 95% confidence intervals. CVD, cardiovascular disease (*International Classification of Diseases*, Ninth Revision (ICD-9) codes 393–459); IHD, ischemic heart disease (ICD-9 codes 410–414); Cerebrov, cerebrovascular disease (ICD-9 codes 430–438); Lung Ca, lung cancer (ICD-9 code 162); Resp, respiratory disease (ICD-9 codes 470–478, 490–519); Non-lung Ca, non-lung cancer (ICD-9 codes 140–161, 163–209).

to be found in households with other smokers (4), producing a positive bias on the SHS-mortality association. We attempted to address both kinds of misclassification through sensitivity analyses, which suggest that the two tend to cancel one another out (table 4). Although our estimates were based on a number of assumptions, we believe that we were conservative in estimating bias due to unmeasured SHS exposure outside the home. If this is the case (i.e., if significant SHS exposure outside the home affects a greater proportion of never smokers than we assumed in our calculations), the true association between household SHS exposure and mortality risk may be greater than reported here.

The absence of an observed association between SHS exposure and lung cancer is probably a reflection of the above limitations. Lung cancer has a long latency period, and the relevant exposure in study participants will have occurred some decades prior to our census-derived measure of passive smoking. Misclassification of exposure will probably be greater for analyses of lung cancer mortality than for mortality from diseases with a shorter latency. This effect is

exacerbated by our definition of "nonsmoking" households, which will produce greater misclassification of past compared with current SHS exposure ("nonsmoking" households included those containing former smokers, which would logically have been "smoking" households in the past).

An interesting and perhaps surprising finding is the apparently increased mortality from non-lung cancers in those exposed to SHS (figures 2 and 3), with a significantly increased risk of non-lung-cancer mortality among women from the later cohort (table 3). Although lung cancer is the malignancy most commonly coupled with smoking, tobacco smoke is also a major risk factor for other kinds of cancer (45, 46), and there is evidence that passive smoking increases the risk of all-cancer mortality (10). With reference to the US population, Glantz and Parmley (38) estimated that non-lung cancers account for a greater proportion of SHS-related deaths than does lung cancer. Nevertheless, it is surprising that we found an association for non-lung cancers but not lung cancer (both presumably having long

TABLE 3. Standardized* and adjusted† mortality relative risk estimates for nonsmokers living in smoking households, 1981–1984 and 1996–1999 New Zealand cohorts (never smokers aged 45–77 years) by sex, cohort, and cause of death

	1981–1984 cohort				1996–1999 cohort			
	Standardized RR‡	95% CI‡	Adjusted RR	95% CI	Standardized RR	95% CI	Adjusted RR	95% CI
Men								
All cause	1.10	0.99, 1.22	1.17	1.05, 1.30	1.17	1.05, 1.31	1.16	1.04, 1.30
Disease specific								
Cardiovascular§	1.14	0.99, 1.30	1.19	1.04, 1.38	1.22	1.03, 1.46	1.25	1.06, 1.47
Ischemic heart disease	1.00	0.85, 1.18	1.04	0.88, 1.23	1.07	0.86, 1.32	1.18	0.96, 1.44
Cerebrovascular¶	1.59	1.14, 2.21			1.91	1.23, 2.96	1.82	1.20, 2.77
Lung cancer¶	0.97	0.53, 1.77	1.08	0.56, 2.09	1.45	0.75, 2.81		
Respiratory	1.02	0.62, 1.67	1.38	0.79, 2.42	1.72	0.99, 2.98	1.81	1.00, 3.28
Non-lung cancer	1.11	0.90, 1.38	1.19	0.95, 1.49	0.98	0.80, 1.20	0.98	0.80, 1.20
Women								
All cause	1.04	0.96, 1.13	1.06	0.97, 1.16	1.27	1.15, 1.41	1.28	1.16, 1.42
Disease specific								
Cardiovascular§	0.99	0.87, 1.12	1.01	0.88, 1.16	1.31	1.09, 1.58	1.35	1.11, 1.64
Ischemic heart disease	0.98	0.83, 1.15	0.98	0.83, 1.17	1.26	0.98, 1.63	1.27	0.98, 1.66
Cerebrovascular	0.88	0.66, 1.17	0.90	0.67, 1.21	1.16	0.75, 1.79	1.17	0.76, 1.82
Lung cancer¶	1.00	0.49, 2.01			1.16	0.70, 1.92	1.38	0.78, 2.41
Respiratory	1.19	0.74, 1.92	1.34	0.81, 2.21	1.75	0.99, 3.10	1.59	0.95, 2.68
Non-lung cancer	1.08	0.94, 1.24	1.04	0.90, 1.21	1.23	1.06, 1.42	1.21	1.05, 1.40

* Standardized (directly) by age (5-year age bands) and ethnicity (Māori, Pacific, and neither Māori nor Pacific).

† Adjusted for age, ethnicity, marital status, and socioeconomic position (i.e., education, labor force status, household equivalized income, household car access, housing tenure, and small-area deprivation index).

‡ RR, relative risk; CI, confidence interval.

§ Cardiovascular disease includes ischemic heart disease and cerebrovascular disease.

¶ Because of small numbers of deaths in some covariate substrata, we were unable to accurately calculate adjusted relative risk estimates for cerebrovascular mortality in the 1981–1984 cohort of men or lung cancer mortality in the 1996–1999 cohort of men and the 1981–1984 cohort of women.

lag times). It may be that some non-lung cancers have a shorter latency than lung cancer does and are thus less subject to misclassification of past SHS exposure. It is also possible that chance played some role in these results.

In keeping with other studies (47–52), we found lower average educational levels and higher levels of neighborhood deprivation among never smokers living with smokers. Relative risk estimates were adjusted for a wide range of

TABLE 4. Relative risk estimates for all-cause mortality in men aged 45–77 years (1996–1999 New Zealand cohort): unadjusted and adjusted for misclassification of personal smoking status and misclassification of secondhand smoke exposure

		Misclassification of secondhand smoke exposure	
		Unadjusted	Adjusted
Misclassification of personal smoking status	Unadjusted	1.14	1.24
	Adjusted	1.10	1.18

socioeconomic variables, which are well recorded in the New Zealand census. Other potential sources of confounding (such as diet) could not be adjusted for directly; however, given that these factors tend to be patterned by socioeconomic position, their influence on relative risk estimates should have been largely addressed in the above analyses (47, 53, 54). It is possible that some confounding remains from unmeasured risk factors, but, in our view, this factor is unlikely to explain the results reported here. Unlike all-cause mortality, few factors other than tobacco smoke would be expected to raise mortality risk across the range of conditions examined in this study (including cardiovascular disease, cancer, and nonmalignant respiratory disease).

The association between household SHS exposure and mortality was notably stronger in the later cohort (1996–1999). This finding may be explained by greater misclassification in the earlier cohort (1981–1984) due to greater (unmeasured) SHS exposure outside the home. Potential health risks from passive smoking were virtually unknown in the early 1980s, and smoking was socially acceptable in most circumstances (55); by the mid-1990s, public attitudes and behavior had started to shift against exposure to other people's tobacco smoke (56). In 1990, the New Zealand

Government passed legislation banning smoking in shared workplaces and many public areas (57). These social and legal changes mean that SHS exposure outside the home was probably lower for our 1996–1999 cohort, resulting in a less biased estimate of the association between home SHS exposure and mortality. An alternative explanation for the differing strength of association is a greater degree of uncontrolled confounding in the 1996–1999 cohort, which might have occurred if household SHS exposure was more strongly correlated with other unhealthy lifestyle factors in later years. In our view, this is unlikely to be the main explanation, given the modest change in relative risk estimates with adjustment for socioeconomic position (which is closely correlated with lifestyle (47, 53, 54)).

In summary, findings from two cohorts of never smokers show increased cardiovascular, respiratory, and overall mortality among nonsmoking adults exposed to SHS in the home compared with those living in smoke-free homes. Excess mortality risk for passive smokers was not accounted for by confounding or misclassification bias. These findings add to the weight of evidence for a causal association between SHS and mortality from tobacco-related disease.

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APPENDIX

In the 1996 census, male respondents in the age group 44–74 years comprised 20.2 percent current smokers, 39.1 percent former smokers, and 40.7 percent never smokers. Crude all-cause mortality rates (per 100,000 per year) were 1,645 among smokers, 1,397 among former smokers, and 788 among never smokers (59).

Calculating proportions of misclassified current and former smokers in study exposure groups

The proportion of misclassified current and former smokers in the unexposed cohort is given by the formula $M_U = M_T N_T/(N_U + RN_E)$, where M_U is the proportion of misclassified current and former smokers in the unexposed cohort, M_T is the proportion of misclassified current and former smokers in the total cohort (i.e., unexposed plus exposed cohorts), N_T is the number of persons (or person-years of observation) in the total cohort, N_U is the number of persons in the unexposed cohort, R is the ratio of misclassification in exposed compared with unexposed cohorts (in this case assumed to be 3:1), and N_E is the number of persons in the exposed cohort.

Estimated sensitivity and specificity of SHS exposure categorization

Misclassification of SHS exposure arises from three sources: misreported smoking status among household members (leading to misclassification of household smoking status), unmeasured SHS exposure outside the home (meaning that household SHS exposure will underestimate a person's total SHS exposure), and changes in SHS exposure over time. The first source of error will reduce sensitivity by about 2 percent and specificity by about 1 percent (an estimated 1.7 percent of current smokers misreport themselves as nonsmokers (33); we assume that the propor-

tion of nonsmokers misreporting as current smokers is half as much). The second source of error will further reduce sensitivity by approximately 5 percent (a conservative "best guess" for the proportion of nonsmokers in nonsmoking households who have significant SHS exposure outside the home). The specificity of our SHS exposure measure will be unaffected. The third source of error we estimate to account for a further 5 percent reduction in both sensitivity and specificity, on the assumption that background SHS exposure differed from exposure status at the time of measurement for about 5 percent of nonsmoking census respondents. (This last source of error will be greater in the case of diseases with a long latency period (such as lung cancer), where the relevant SHS exposure occurred some time prior to exposure measurement.) These estimates yield an overall sensitivity of 88 percent and specificity of 94 percent in our study measure of SHS exposure.

Calculating study numbers corrected for misclassification of SHS exposure

Having assigned values to the sensitivity (Se) and specificity (Sp) of the exposure measure, the numbers of deaths in the true exposed and true unexposed categories are given by the following two formulae:

1. $U_t = (E_r - SeE_t)/(1 - Sp)$
2. $E_t = (U_r(1 - Sp) - SpE_r)/((1 - Se)(1 - Sp) - SeSp)$,

where E_t is the number of deaths among true exposed persons, U_t is the number of deaths among true unexposed persons, E_r is the number of deaths among reported exposed persons, and U_r is the number of deaths among reported unexposed persons. The same two formulae can be used to calculate numbers of persons (or person-years) in the true exposed and true unexposed categories, using E and U to represent numbers of persons (or person-years) in the exposed and unexposed categories.