Environmental tobacco smoke exposure in the home and worksite and health effects in adults: results from the 1991 National Health Interview Survey

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Abstract

Objective—To determine the effect of environmental tobacco smoke (ETS) exposure in the home and worksite on the health of adults in the United States.

Design—Cross-sectional survey.

Setting—Nationally representative population.

Participants—43 732 adults who completed the Health Promotion and Disease Prevention supplement in the 1991 National Health Interview Survey.

Main outcome measures—Rate of restricted activity, bed confinement, and work absence in the two weeks preceding the survey and self-reported health status among adults with and without exposure to ETS.

Results—We found that only 20.2% of never-smokers and 23.1% of former smokers reported exposure to ETS at home or work, whereas 87.2% of current smokers reported exposure to ETS. Among never-smokers, after adjusting for covariates, people who were exposed to ETS were more likely to report one or more days of restricted activity (relative risk (RR) = 1.27, 95% confidence interval (CI) = 1.10 to 1.46), one or more days of bed confinement (RR = 1.43, 95% CI = 1.19 to 1.73), and one or more days of work absence (RR = 1.33, 95% CI = 1.05 to 1.73) in the two weeks before the survey than were people without such exposure. We detected smaller trends for one or more days of restricted activity among current and former smokers (RR = 1.16, 95% CI = 0.97 to 1.40; and RR = 1.11, 95% CI = 0.82 to 1.51), one or more days of bed confinement among current smokers (RR = 1.34, 95% CI = 0.95 to 1.88), and one or more days of work absence among former smokers (RR = 1.13, 95% CI = 0.84 to 1.50) in the two weeks before the survey than among people without such exposure, although the CIs were wide and chance cannot be excluded as an explanation for these findings. Never-smokers (RR = 1.47, 95% CI = 1.34 to 1.62), former smokers (RR = 1.22, 95% CI = 1.07 to 1.39) and current smokers (RR = 1.31, 95% CI = 1.10 to 1.56) exposed to ETS were all more likely to report a less than very good

health status than were people without such exposure.

Conclusions—This study demonstrates that never-smoking adults exposed to ETS report more acute health effects than unexposed, never-smoking adults, and suggests similar findings in current and former smoking adults.

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Keywords: adults, environmental tobacco smoke, respiratory tract diseases

Introduction

Environmental tobacco smoke (ETS) contains the same constituents that are known to cause respiratory disease in people who smoke.¹ Many studies have demonstrated an association between ETS exposure and respiratory disease in both children²⁻⁵ and adults.⁶⁻⁸ In this study, we used data from the 1991 National Health Interview Survey (NHIS) to investigate the relationship between ETS exposure and acute illnesses in adults. The NHIS was conducted among a probability sample of the civilian, non-institutionalised population of the United States.⁹ It consists of an extensive questionnaire that asks participants about their present health status, including whether they have had any recent illnesses. The first year in which all participants were asked whether they were exposed to ETS in their homes was 1991. We determined the number of adults exposed to ETS either in their homes or worksites and compared morbidity data (days of restricted activity, bed confinement and work absence, self-reported health status, and exacerbations of chronic respiratory and cardiac diseases) for these adults with the corresponding data for adults not exposed to ETS.

Methods

We searched the 1991 NHIS database for all subjects aged 18 years and older who completed the Health Promotion and Disease Prevention supplement. All respondents were asked about their smoking history. We classified subjects as current smokers (currently smoking cigarettes every day or some days or currently smoking a pipe or cigars), former smokers (smoked at least 100 cigarettes in their life but not currently smoking), or never-smokers (never smoked more than 100 cigarettes in their life). Former smokers were asked how many years ago they

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stopped smoking, but not when they started smoking or how many cigarettes they smoked daily, and current smokers were asked how many cigarettes they smoked daily. Each subject was asked: "Does anyone smoke cigarettes, cigars, or pipes anywhere inside this home?" and "On the average, about how many days per week is there smoking anywhere inside this home?" People who were currently working and either had a private enclosed office with a door, a shared office with a door, a cubicle with full or mid-height partitions, or an open office area were asked: "During the past two weeks, has anyone smoked in your immediate work area?". This question was not asked of people who had no regular work area, worked outdoors, travelled to different worksites, or worked in a motor vehicle. We excluded subjects who did not respond to questions about their personal smoking history or ETS exposure in their home. We compared adults with no reported ETS exposure either at home or at work with those with any reported ETS exposure.

Respondents were asked to estimate the number of days they had to decrease their usual activity, were confined to bed, or were absent from work because of illness during the two weeks preceding the survey. Total restricted activity days are the unduplicated sum of work absence, bed confinement, and decreased activity days. We limited our analysis of days people were absent from work to adults age 65 years and younger who were currently working. We multiplied the number of days by 26 to obtain annual estimates. We also classified each person as having one or more versus no days of restricted activity, bed confinement, or work absence in the two weeks before the survey for use in logistic regression models.

Chronic diseases could be reported in three ways: in response to a chronic disease questionnaire, as a reason for a hospitalisation or physician visit in the two weeks before the survey, or as a reason for activity limitation in the two weeks before the survey. As part of the NHIS protocol, the total sample was divided into six subgroups, each of which was asked about a different group of chronic illnesses: musculoskeletal conditions, impairments, digestive conditions, neurological and endocrine conditions, circulatory conditions, and respiratory conditions. We searched for chronic bronchitis, asthma, chronic sinusitis, and emphysema within the respiratory conditions subgroup; and ischaemic heart disease, cerebrovascular disease, hardening of the arteries, and aneurysm within the circulatory conditions subgroup. To determine the prevalence of these conditions, one would have to restrict the sample to the one-sixth subgroup that was asked about either respiratory or circulatory conditions (not used in this study). We considered subjects who had activity limitation or a physician's visit in the two weeks preceding the survey because of one of the chronic diseases noted above to have an "exacerbation" of this disease. To determine the rate of exacerbations of these diseases in the two

weeks preceding the survey, we limited the database to the people in approximately five-sixths of the sample who were not asked about chronic respiratory diseases or cardiovascular diseases.

The core NHIS contained the question: "Would you say your health in general is excellent, very good, good, fair, or poor?" We stratified the responses into two groups: very good and better, and good and worse.

We classified people as having lower socioeconomic status if they were from a family in which the total family income was below the 1990 poverty level (as derived from the August 1991 Current Population Survey)¹⁰ and calculated on the basis of family size, number of children less than 18 years old, and family income, or if they had a 12th grade (high school) education or less. We excluded adults for whom data on poverty status or educational level was missing. In all of our regression analyses and our determination of national population estimates we used the NHIS weights. We age-adjusted the data used in the figures to the age distribution of the entire study sample because the age distribution of current, former and never smokers differed.

Because the NHIS is a complex sample design, we used SUDAAN (RTI, Research Triangle Park, North Carolina) to determine variances and to do significance testing in our regression models. The point estimates obtained with SUDAAN are identical to those obtained with SAS (SAS Institute, Cary, North Carolina), whereas the variances are larger with SUDAAN because SUDAAN, unlike SAS, accounts for the complex sample design of the NHIS survey. We used the SAS procedure GLM to calculate Bonferroni *t* tests for differences of mean exposures to ETS for region, season, and work status. We used the SUDAAN procedures RLOGIST (logistic regression), REGRESS (linear regression), CROSSTAB, and DESCRIB in these analyses. We also used the SAS procedure LOGISTIC to model the number of days of restricted activity, bed confinement, and work absence in a Poisson regression. The point estimates we obtained using this were similar to those obtained using the SUDAAN procedure REGRESS, although the variances were smaller, and we only present the linear regression results. In the logistic and linear regressions we included socioeconomic status, sex, people in household (living alone or not living alone), region of country, race, season during which the questionnaire was completed, age, work status (working and asked about ETS exposure at work, working and not asked about ETS exposure at work, or not working), number of cigarettes smoked daily among smokers, and years ago they stopped smoking among former smokers, as potential covariates and effect modifiers. In our preliminary regression models we analysed data on all participants for whom we had complete data, to determine the effect of current and former smoking on our outcome measures. In our final models, we ran separate analyses on never-smokers, former smokers, and current smokers.

Table 1 Study population of adults, age 18 and older, stratified by smoking status and reported environmental tobacco smoke (ETS) exposure at home and at work, from the 1991 National Health Interview Survey. The national estimates of these populations and the weighted percentages (WP) are also presented (columns may not add up to 100% because of rounding)

ETS exposure	Current smokers			Former smokers			Never-smokers			Total		
	Number	Estimated national population	WP	Number	Estimated national population	WP	Number	Estimated national population	WP	Number	Estimated national population	WP
Home: unknown	210	897 000	1.9	80	345 000	0.7	187	798 000	0.9	477	2 040 000	1.1
Home: no; work: no	1 139	5 259 000	10.9	7429	30 559 000	76.1	17 021	68 943 000	78.9	25 589	104 761 000	59.6
Home: no; work: yes	97	454 000	0.9	349	1 472 000	3.7	724	3 159 000	3.6	1 170	5 085 000	2.8
Home: <daily; work: no	842	3 119 000	6.4	491	1 875 000	4.7	921	3 600 000	4.1	2 254	8 594 000	4.8
Home: <daily; work: yes	64	257 000	0.5	30	109 000	0.2	45	188 000	0.2	139	554 000	0.3
Home: daily; work: no	8 633	35 576 000	73.4	1075	5 503 000	13.7	1 900	9 995 000	11.5	11 608	51 074 000	29.0
Home: daily; work: yes	716	2 935 000	6.0	58	321 000	0.7	104	584 000	0.7	878	3 840 000	2.3
Total: any ETS exposure	10 352	42 341 000	87.2	2003	9 280 000	23.1	3 694	17 526 000	20.2	16 049	69 147 000	39.2
Total	11 701	48 497 000		9512	40 184 000		20 902	87 267 000		42 115	175 948 000	

Table excludes 569 participants (representing 2 715 000 people) who did not have a smoking status listed and 1048 participants (representing 4 528 000 people) for whom we could not classify a socioeconomic status.

Results

We analysed data on 43 732 adults, representing an estimated 183 million American adults aged 18 years and older, who completed the Health Promotion and Disease Prevention supplement in 1991. After we excluded 569 participants who did not have a smoking status listed and 1048 participants for whom we could not classify a socioeconomic status, our database contained 42 115 people (table 1). We further excluded 477 people who had unknown ETS exposure at home and were left with 41 638 participants. On the basis of these data, we estimate that 69.1 million adults (39.8%, weighted percentage (WP)) were exposed to ETS in their homes or worksites (table 1). The percentage of people exposed varied dramatically by smoking status; only 20.2% (WP) of never-smokers and 23.1% (WP) of former smokers reported ETS exposure whereas 87.2% (WP) of current smokers did so (table 1). Of the participants reporting less-than-daily ETS exposure in the home, 20.6% (WP) reported being exposed four to six days weekly, 45.7% (WP) reported being exposed one to three days weekly, and 33.7% (WP) reported being exposed less than a day a week.

Of the people in our final dataset, 49.7% (WP) were never-smokers, 22.9% (WP) were former smokers, and 27.4% (WP) were current smokers. The median age of the never-smokers and current smokers in the study was 39 years, whereas the median age of the former smokers was 52 years. Of the former smokers, 28.6% (WP) had stopped smoking more than 15 years before the survey, 31.3% (WP) had stopped smoking 5–15 years before the survey, 22.2% (WP) had stopped smoking one to five years before the survey, and the remaining 17.9% (WP) had either stopped smoking in the year before the survey or did not answer this question.

Among adults under the age of 65, 25.9% (WP, age-adjusted) of never-smokers, 24.4% (WP, age-adjusted) of former smokers, and 28.3% (WP, age-adjusted) of current smokers

were currently not employed. After adjusting for age, sex, season, region, socioeconomic status and race, current smokers were more likely to be not employed (relative risk = 1.33, 95% confidence interval = 1.22 to 1.45) than were never-smokers.

Among never-smokers, the rate of ETS exposure in the home or worksite varied significantly by socioeconomic status, race, living alone, and age (table 2). These same variables, along with sex, and years ago stopped smoking or number of cigarettes smoked daily, were related to the rate of ETS exposure among current and former smokers (tables 3 and 4). Among current, former, and never-smokers, people in the west region of the United States reported less ETS exposure ($P<0.05$ for all, by Bonferroni t tests). Among former and never-smokers, people working and asked about ETS exposure reported more ETS exposure than among people working and not asked about ETS exposure or among people not working ($P<0.05$ for all, by Bonferroni t tests). We did not detect any significant difference in this measure among current smokers, nor did we detect any significant seasonal difference in ETS exposure in any smoking category ($P>0.05$ for all, by Bonferroni t tests).

One or more restricted activity days in the two weeks before the survey was reported by 9.4% (WP, age-adjusted) of never-smokers, 10.4% (WP, age-adjusted) of former smokers, and 11.7% (WP, age-adjusted) of current smokers (figure 1). One or more days of bed confinement in the two weeks before the survey was reported by 4.9% (WP, age-adjusted) of never-smokers, 5.5% (WP, age-adjusted) of former smokers, and 6.3% (WP, age-adjusted) of current smokers (figure 1). Among workers under the age of 65, one or more days of work absence in the two weeks before the survey was reported by 5.3% (WP, age-adjusted) of never-smokers, 6.0% (WP, age-adjusted) of former smokers, and 6.4% (WP, age-adjusted) of current smokers (figure 2). In the logistic regression models, in which we determined the chance of people reporting one or more days of

restricted activity, bed confinement, or work absence in the two weeks before the survey, never-smokers exposed to ETS reported significantly more morbidity than unexposed never-smokers (table 5). These findings were consistent before and after adjusting for

Table 2 Study population of never-smoking adults, age 18 and older, stratified by socioeconomic status (SES), race, family size, region of country, season, age, and smoking status, with the number and weighted percentage (WP) of adults in each category (by row) with any reported exposure and no reported exposure to environmental tobacco smoke (ETS), from the 1991 National Health Interview Survey

		Any ETS exposure		No ETS exposure		P*
		n	WP	n	WP	
SES	Lower	2625	22.8	10 769	77.2	<0.001
	Higher	1069	16.2	6 252	83.8	
Race	White	3354	19.6	13 667	80.4	<0.001
	Non-white	840	22.9	2 854	77.1	
Lives alone	Yes	532	9.6	3 162	22.2	<0.001
	No	4989	90.4	12 032	65.8	
Sex	Female	2466	20.5	11 261	79.5	0.355
	Male	1228	19.9	5 760	80.1	
Region	Northeast	753	20.7	3 446	79.3	
	Midwest	1027	22.0	4 119	78.0	
	South	1303	22.1	5 555	77.9	
	West	611	15.3	3 901	84.7	
Season	Winter	891	21.6	3 868	78.4	
	Spring	967	20.4	4 385	79.6	
	Summer	933	19.5	4 311	80.5	
	Autumn	903	19.6	4 457	80.4	
Age (years)	18–24	859	30.4	2 345	69.6	<0.001
	25–34	925	18.9	4 280	81.1	
	35–44	762	20.6	3 219	79.4	
	45–54	383	18.1	1 841	81.9	
	55–64	337	20.0	1 555	80.0	
	65–74	268	14.5	1 847	85.5	
	≥75	160	8.8	1 934	91.2	
Work status	Working and asked about ETS	1384	28.1	3 898	71.9	
	Working, not asked about ETS	1145	18.0	6 185	82.0	
	Not working	1165	17.2	6 938	82.8	
Total		3694	20.3	17 021	79.7	

*Significance determined using χ^2 analyses or χ^2 test of trends.

Table 3 Study population of former smoking adults, age 18 and older, stratified by socioeconomic status (SES), race, family size, region of country, season, age, and years quit smoking, with the number and weighted percentage (WP) of adults in each category (by row) with any reported exposure and no reported exposure to environmental tobacco smoke (ETS), from the 1991 National Health Interview Survey

		Any ETS exposure		No ETS exposure		P*
		n	WP	n	WP	
SES	Lower	1342	24.8	4688	75.2	0.001
	Higher	661	20.9	2741	79.1	
Race	White	1726	22.8	6594	77.2	0.005
	Non-white	277	27.9	835	72.1	
Living alone	Yes	300	12.6	2077	87.4	<0.001
	No	1703	25.0	5352	75.0	
Sex	Female	1019	24.7	3516	75.3	0.014
	Male	984	22.3	3913	77.7	
Region	Northeast	497	25.5	1648	74.5	
	Midwest	564	26.0	1760	74.0	
	South	607	23.5	2239	76.5	
	West	335	17.9	1782	82.1	
Season	Winter	486	25.0	1685	75.0	
	Spring	519	22.5	1956	77.5	
	Summer	505	23.9	1874	76.1	
	Autumn	493	21.9	1914	78.1	
Age (years)	18–24	105	35.9	212	64.1	<0.001
	25–34	377	27.9	1023	72.1	
	35–44	439	24.9	1458	75.1	
	45–54	395	27.9	1145	72.1	
	55–64	324	21.2	1300	78.8	
	65–74	265	17.4	1448	82.6	
	≥75	98	12.0	843	88.0	
Years ago stopped smoking	<1	435	27.3	1249	72.7	<0.001
	1–5	536	28.0	1544	72.0	
	6–15	635	24.4	2301	75.6	
	>15	397	16.0	2335	84.0	
Work status	Working and asked about ETS	709	35.2	1380	64.8	
	Working, not asked about ETS	641	21.6	2665	78.4	
	Not working	653	18.2	3384	81.8	
Total		2003	23.3	7429	76.7	

*Significance determined using χ^2 analyses or χ^2 test of trends.

Table 4 Study population of smoking adults, age 18 and older, stratified by socioeconomic status (SES), race, family size, region of country, season, age, and cigarettes smoked daily, with the number and weighted percentage (WP) of adults in each category (by row) with any reported exposure and no reported exposure to environmental tobacco smoke (ETS), from the 1991 National Health Interview Survey

		Any ETS exposure		No ETS exposure		P*
		n	WP	n	WP	
SES	Lower	8 084	90.9	725	9.1	<0.001
	Higher	2 268	83.1	414	16.9	
Race	White	8 339	88.6	952	11.4	0.023
	Non-white	2 013	90.5	187	9.5	
Living alone	Yes	2 658	92.9	198	7.1	<0.001
	No	7 964	88.3	941	11.7	
Sex	Female	5 502	92.1	436	7.9	<0.001
	Male	4 850	86.3	703	13.7	
Region	Northeast	2 084	89.9	208	10.1	
	Midwest	2 812	91.2	257	8.8	
	South	3 545	90.9	310	9.1	
	West	1 991	81.8	364	18.2	
Season	Winter	2 551	90.4	238	9.6	
	Spring	2 626	88.5	305	11.5	
	Summer	2 570	87.5	317	12.5	
	Autumn	2 605	89.3	279	10.7	
Age (years)	18-24	996	84.6	168	15.4	<0.001
	25-34	2 796	88.6	312	11.4	
	35-44	2 565	88.8	280	11.2	
	45-54	1 704	91.5	153	8.5	
	55-64	1 196	91.6	98	8.4	
	65-74	806	88.6	88	11.4	
	≥75	329	87.5	40	12.5	
Cigarettes smoked daily	≤10	3 746	79.9	829	20.1	<0.001
	11-20	4 371	93.8	243	6.2	
	20-30	1 212	96.6	41	3.4	
	>30	1 023	97.1	26	2.9	
Work status	Working and asked about ETS	2 193	88.9	244	11.1	
	Working, not asked about ETS	4 509	87.8	555	12.2	
	Not working	3 650	90.7	340	9.3	
Total		10 352	88.9	1 139	11.1	

*Significance determined using χ^2 analyses or χ^2 test of trends.

covariates (table 5). The corresponding fully adjusted models for current and former smokers all had confidence intervals that included

Table 5 The odds ratios of any restricted activity, bed confinement, work absence, exacerbations of chronic respiratory disease, and exacerbation of chronic cardiovascular disease, in the two weeks before the survey, among current, former, and never-smoking adults exposed to environmental tobacco smoke (ETS), compared with adults without such exposure. This table also displays the odds ratios for less than very good health status. Odds ratios are shown after adjusting for age only and after adjusting for age, socioeconomic status, race, living alone, sex, season, smoking status, region of country, cigarettes smoked daily (among current smokers), and years ago quit smoking (among former smokers), using logistic regression. From the 1991 National Health Interview Survey

	Adjusted for age		Adjusted for all covariates	
	Odds ratio	95% CI	Odds ratio	95% CI
Any restricted activity				
Current smokers	1.23	0.92-1.64	1.11	0.82-1.51
Former smokers	1.13	0.95-1.35	1.16	0.97-1.40
Never-smokers	1.23	1.07-1.41	1.27	1.10-1.46
Any bed confinement				
Current smokers	1.53	1.10-2.12	1.34	0.95-1.88
Former smokers	1.00	0.78-1.29	1.02	0.79-1.32
Never-smokers	1.41	1.17-1.70	1.43	1.19-1.73
Any work absence (among workers)				
Current smokers	1.08	0.74-1.58	0.99	0.66-1.47
Former smokers	1.16	0.86-1.57	1.13	0.84-1.50
Never-smokers	1.31	1.05-1.64	1.33	1.05-1.68
Any chronic respiratory disease exacerbation				
Current smokers	1.41	0.80-2.47	1.33	0.77-2.29
Former smokers	0.89	0.62-1.28	0.87	0.60-1.26
Never-smokers	1.42	1.05-1.91	1.44	1.07-1.95
Any chronic cardiovascular disease exacerbation				
Current smokers	1.44	0.53-3.95	1.25	0.41-3.79
Former smokers	1.26	0.90-1.77	1.23	0.86-1.77
Never-smokers	1.27	0.81-2.00	1.21	0.75-1.94
Less than very good health status				
Current smokers	1.66	1.41-1.97	1.31	1.10-1.57
Former smokers	1.27	1.13-1.43	1.22	1.07-1.39
Never-smokers	1.56	1.43-1.70	1.47	1.34-1.62

unity; thus, chance cannot be excluded as an explanation for these findings (table 5).

In the linear regression models, after adjusting for age and for all covariates, we found that current, former, and never-smokers who were exposed to ETS reported more days of restricted activity and bed confinement than unexposed people in the same smoking category, although the confidence intervals were wide for most of these findings, and chance cannot be excluded as an explanation (table 6). Former and never-smoking workers under age 65 who were exposed to ETS reported more days of work absence than workers not exposed, whereas smoking workers exposed to ETS reported fewer days of work absence than smoking workers not exposed (table 6), but again the confidence intervals were wide, and chance cannot be excluded as an explanation for these findings.

Exacerbations of chronic respiratory diseases in the two weeks preceding the survey were reported by 1.8% (WP, age-adjusted) of never-smokers, 2.6% (WP, age-adjusted) of former smokers, and 2.7% (WP, age-adjusted) of current smokers (figure 3). Exacerbations of chronic cardiovascular disease in the two weeks preceding the survey were reported by 1.3% (WP, age-adjusted) of never-smokers, 2.9% (WP, age-adjusted) of former smokers, and 2.0% (WP, age-adjusted) of current smokers (figure 3). In the age-adjusted and fully adjusted regression models, current and never-smokers exposed to ETS reported more

Figure 1 The age-adjusted, weighted percentage of never-smokers, former smokers, and current smokers, exposed and not exposed to environmental tobacco smoke, who reported any day of restricted activity or bed confinement during the two weeks preceding the survey. From the 1991 National Health Interview Survey.

Figure 2 The age-adjusted, weighted percentage of currently working never-smokers, former smokers, and current smokers, both exposed and not exposed to environmental tobacco smoke, who reported any day of work absence during the two weeks preceding the survey. From the 1991 National Health Interview Survey.

exacerbations of chronic respiratory diseases, whereas former smokers exposed to ETS reported fewer exacerbations of respiratory diseases; these findings reached significance only in never-smokers (table 5). Among current, former, and never-smokers, people exposed to ETS reported more exacerbations

of cardiovascular disease in the two weeks before the survey than people without such exposure (figure 3). After adjusting for covariates, we found that in all three smoking categories, people exposed to ETS reported more exacerbations of cardiovascular disease, although again the confidence intervals were wide and included unity (table 5).

A very good or better health status was reported by 66.3% (WP, age-adjusted) of never-smokers, 65.5% (WP, age-adjusted) of former smokers, and 56.6% (WP, age-adjusted) of current smokers. Current, former, and never-smokers who were exposed to ETS in the home or worksite were all more likely to report that their health status was less than very good, than were the unexposed people in the same smoking categories, both before and after adjusting for covariates (table 5).

Discussion

Environmental tobacco smoke exposure is ubiquitous in the United States. Results from a recent nationally representative study¹¹ show that 89% of people in the United States age 6 or older had levels of cotinine in their blood greater than 5 ng/dl. The percentage of American children exposed to ETS in the home has decreased from 62% in 1970 to 37% in 1991.^{12,13} This decrement is probably related to the decreasing prevalence of smoking among American adults.¹⁴ We do not have nationally representative historical data for ETS exposure among adults, but a 1963 study of adults in Washington County, Maryland, showed that 30% of never-smoking men and 64% of never-smoking women were exposed to ETS at home,¹⁵ which is higher than the reported combined rates of ETS exposure (at home and at work) of 20.5% among never-smoking women and 19.9% among never-smoking men that we detected in our study. The reasons for these decreasing rates of exposure to ETS include the decreasing prevalence of smoking in the American population,¹⁴ the increasing numbers of worksites that have restricted or eliminated smoking,¹⁶ and the increasing number of communities in the United States that have enacted ordinances restricting or eliminating smoking in public places.^{17,18}

Table 6 The increased number of days annually of restricted activity, bed confinement, and work absence among and current, former, and never-smoking adults exposed to environmental tobacco smoke (ETS), compared with adults without such exposure, after adjusting for age only and after adjusting for age, socioeconomic status, race, living alone, sex, season, smoking status, region of country, cigarettes smoked daily (among current smokers), and years ago quit smoking (among former smokers), using linear regression, and the total mean annual days of restricted activity, bed confinement, and work absence among both current non-smoking and current smoking adults. From the 1991 National Health Interview Survey

	Adjusted for age		Adjusted for all covariates		Mean annual total (SE)
	Days annually	95% CI	Days annually	95% CI	
Days of restricted activity					
Current smokers	6.70	2.01-11.38	4.22	-0.72-9.16	20.4 (0.7)
Former smokers	2.12	-1.86-6.10	3.15	-1.00-7.30	20.9 (0.9)
Never-smokers	2.29	-0.66-5.24	2.87	-0.13-5.87	14.6 (0.6)
Days of bed confinement					
Current smokers	3.79	1.63-5.94	2.63	0.04-5.22	7.8 (0.5)
Former smokers	0.23	-2.20-2.66	0.57	-1.94-3.08	7.7 (0.5)
Never-smokers	1.58	-0.28-3.44	1.68	-0.22-3.58	5.5 (0.4)
Days of work absence (among workers)					
Current smokers	-0.62	-4.36-3.12	-0.83	-4.71-3.05	6.9 (0.4)
Former smokers	0.54	-1.95-3.03	0.99	-1.64-3.61	6.2 (0.6)
Never-smokers	0.48	-0.98-3.46	0.75	-0.75-2.25	4.8 (0.3)

CI = confidence intervals.

Figure 3 The age-adjusted, weighted percentage of never-smokers, former smokers, and current smokers, exposed and not exposed to environmental tobacco smoke, who reported any exacerbation of chronic cardiac disease or chronic respiratory disease during the two weeks preceding the survey. From the 1991 National Health Interview Survey.

We found that ETS exposure in the home or worksite varied dramatically by smoking status. Among current non-smokers (combining former and never-smokers) only 21.2% were exposed to ETS. This exposure rate is about half of that for children aged 1–10 from the same survey (37.0%).¹³ The most likely explanation for non-smoking adults having a lower rate of in-home ETS exposure than children is that adults can choose where to live and whom to live with, whereas children cannot. Fifteen per cent of the non-smoking adults in this survey lived alone, and many others chose to live with other non-smokers or to prohibit smoking in their homes. Conversely, among current smokers, 88.9% reported that they are exposed to ETS in their homes or worksites daily. Surprisingly, however, 12.0% of current smokers reported no ETS exposures in their home, indicating that neither they nor anyone else smokes in their homes.

Our primary finding in this study was increased morbidity, as measured by an increased rate of restricted activity, bed confinement, and work absence in the two weeks preceding the survey, among never-smokers exposed to ETS than among never-smokers not exposed to ETS. We also found that current, former, and never-smokers who were exposed to ETS classified themselves as being less healthy than those without this exposure. The relative risk we obtained for any restricted activity day in the two weeks preceding the survey (1.27, 95% CI = 1.10 to 1.46) is similar to the results we detected in children (RR = 1.26, 95% CI = 1.09 to 1.45)¹³ and the 20% increase in respiratory restricted days detected in a previous study of adults.¹⁹ The biological mechanism by which ETS causes respiratory illness in healthy adults is thought to be comparable with that of active light smoking,²⁰ and people with a history of atopy or respiratory illness have been shown to be more sensitive to acute effects of ETS.²¹ A longitudinal study of student nurses demonstrated that ETS exposure was associated with an

increased risk of phlegm production (RR = 1.41, 95% CI = 1.08 to 1.85).²²

Rates of restricted activity, bed confinement, and work absence in the two weeks preceding the survey among current and former smokers exposed to ETS were not significantly increased compared with those among current and former smokers not exposed to ETS. One explanation for these findings may be related to the smaller sample of current and former smokers compared with never-smokers, although it is possible that current and former smokers are less sensitive to ETS. Among smokers, evaluation of work absence days is probably influenced by smokers being more likely to be not working, compared with former and never-smokers, especially if working smokers represent a healthier subset of all smokers. In addition, all estimates of the effect of ETS exposures on the increased number of days of restricted activity, bed confinement, and work absence, except for days of bed confinement among current smokers, had wide confidence intervals. These wide confidence intervals are related to the sample size, the low prevalence of exposures and outcomes, and the influence of the design effect.

After adjusting for covariates, we found that current, former, and never-smokers who were exposed to ETS were more likely to have reported an exacerbation of chronic cardiovascular disease in the two weeks preceding the survey than those not exposed to ETS. In all of the adjusted models, confidence intervals included unity and chance cannot be ruled out as an explanation for these findings. ETS exposure among never-smokers has been linked to both atherosclerosis^{23 24} and ischaemic heart disease mortality.²⁵ ETS exposure has also been linked to acute cardiovascular effects, thought to be related to increased platelet aggregation and increased oxygen demand by the heart.²⁶ Clinical studies have demonstrated that ETS exposure exacerbates the symptoms of chronic cardiovascular disease.^{27–31} Increased carboxyhemoglobin levels resulting from passive smoking aggravate symptoms of angina by reducing myocardial oxygen delivery and decreasing contractility of the heart,^{28 30} and they also impair exercise performance among patients with chronic obstructive pulmonary disease by reducing the lung's ability to oxygenate the blood.³¹ Nicotine and other ETS components may further aggravate angina in patients with heart disease.²⁹ Most of the subjects in these clinical studies were ex-smokers or never-smokers; however, in the one study that included current smokers,²⁹ a decrease in time to exercise-induced angina was observed for all subjects, regardless of smoking status.

We also found that current and never-smokers, but not former smokers, who were exposed to ETS were more likely to have reported an exacerbation of chronic respiratory disease in the two weeks preceding the survey than those not exposed to ETS. This finding was significant only among never-smokers. An unexpected finding was that former smokers exposed to ETS did not report more

exacerbations of chronic respiratory disease. Because we did not have information of duration of disease or prior ETS exposure, we cannot determine whether or not some subjects altered their ETS exposure as a potential explanation for this finding. There is both epidemiological and clinical evidence that ETS exposure may increase the risk of respiratory disease and aggravate symptoms of pre-existing chronic respiratory disease among both non-smokers and smokers. Although many epidemiological studies have detected an increased risk for lung cancer among non-smokers exposed to ETS,^{1,32} several studies have also reported an increased risk for lung cancer among smokers who were exposed to ETS in the home,^{25,33-36} although two studies did not find such an association.^{37,38} None of the study results were statistically significant³⁹; however, meta-analyses of these studies reported significant pooled odds ratios of 1.3³⁹ and 1.25.¹ Because only two studies controlled for intensity of active smoking,^{25,37} confounding by amount smoked may explain these findings. Recent studies have also linked ETS exposure among non-smoking adults to obstructive respiratory disease and symptoms such as dyspnoea and bronchitis,^{20,22,40} acute worsening of respiratory diseases,⁴¹ and increased symptoms of asthma.⁶

It is biologically plausible that the ETS exposure could increase the health risks of smokers as well as non-smokers. The levels of many toxic and carcinogenic chemicals are substantially higher in concentrated sidestream smoke than in concentrated mainstream smoke, although the overall ambient levels are lower because sidestream smoke is typically diluted into a larger airspace.^{26,42-46} For example, the concentrations of the carcinogens N-nitrosodimethylamine, 2-naphthylamine, and 4-aminobiphenyl are 52, 39, and 31 times higher, respectively, in sidestream smoke than in mainstream smoke.⁴² On the other hand, the nicotine concentration in sidestream smoke is only 2.7 times higher than that in mainstream smoke.⁴² Thus, although cotinine levels in smokers may be almost entirely attributable to active smoking, passive smoking may contribute substantially to overall exposure to carcinogenic and other toxic components of tobacco smoke.^{46,47} Under conditions of heavy workplace ETS exposure, smokers may inhale a daily dose of benzo(a)pyrene equivalent to that obtained by actively smoking 1½–2 packs of cigarettes,^{48,49} and a daily dose of ammonia and pyridine equivalent to that obtained by smoking 2½ packs of cigarettes.⁴⁶ A one pack-per-day smoker who is exposed to ETS under these conditions could conceivably obtain a higher dose of these toxins from passive smoking than from active smoking. Another possible explanation for increased acute health effects among smokers exposed to ETS is that the people they live and work with, who are also being exposed to ETS, have more respiratory illnesses, thus increasing the likelihood that the smoker will be exposed to and develop these illnesses.

This study has several limitations. One is related to sample size. Even though the survey had almost 44 000 participants, only 21.2% of non-smokers reported exposure to ETS and only 11.1% of current smokers reported no exposure to ETS. These low rates, in conjunction with several infrequent outcomes that we were evaluating, resulted in wide confidence intervals. For example, to detect a 20% increase in the incidence of chronic cardiovascular disease exacerbations among non-smokers (from 2.0% to 2.4%) with a power of 0.80 and a 95% confidence interval, we would have needed more than 66 000 non-smokers in the study. To detect a similar increase among smokers, we would have needed more than 112 000 current smokers.

Another limitation in this analysis is the measure of workplace exposure, which was limited to currently employed adults who worked in a specific type of work environment. This restriction led to the series of questions on workplace exposure being asked of less than 25% of the survey sample and only about 50% of current workers. Workers who were not asked about worksite ETS exposure included those who worked in one building with no regular work area, those who travelled to different worksites, and those who worked in a motor vehicle. Furthermore, among subjects asked about workplace exposure to ETS, about 15% reported exposures. This level of exposure can be contrasted with reported exposures of 52–70% in a recent European study.⁴⁰ Results from previous research have shown that people who have no regular work area are twice as likely to report ETS exposure as are people working in a private office.¹⁶

Another problem with this survey's evaluation of workplace exposures to ETS is that these exposures are probably under-reported. Workers were asked only whether their immediate work area was smoke-free or not. Thus, smokers who are not allowed to smoke at their worksite might be exposed to ETS in other indoor or outdoor designated smoking areas.³⁹

We did not have biochemical measures of ETS exposure or primary smoking, such as measurements of cotinine in serum or urine.^{11,50} Previous studies have shown that misclassification of subjects' self-reported smoking status can occur.^{12,51} Lack of biochemical measures also meant that we were unable to validate ETS exposures. Although another nationally representative study has demonstrated that people reporting exposures generally have higher serum cotinine levels than people not reporting such exposures,¹¹ self-reported ETS exposures do not necessarily correlate with serum or urine levels of cotinine.^{49,50} People can be exposed to ETS in places other than their homes or workplaces, such as stores or restaurants; the 1991 NHIS did not ask about these potential exposure sites and this is probably why our overall rate of exposure to tobacco smoke of 42.8% (100% of current smokers and 21.2% of current non-smokers) is much lower than the exposure rate of 89% as indicated by measurable

cotinine levels in a recent nationally representative study.¹¹

We also do not have data on the amount of ETS that subjects were exposed to or whether they had exposures before the two weeks asked about in the survey. In this database, daily exposures in the home to one cigarette would be counted the same as daily exposure to 40 cigarettes. NHIS surveys subsequent to the 1991 survey asked about intensity of exposure. Similarly, workplace exposures, which were not asked of all workers, were also not quantified. We did not attempt to examine dose-response relationships in this analysis because we did not have good data on intensity or duration of exposure. It is possible that people reporting less than daily exposure to ETS had higher levels of exposure than those reporting daily exposures. Another study of ETS exposure in adults, which had data on hours of exposure daily, years of exposure, and number of smokers exposed, was able to demonstrate a dose-response relationship for respiratory symptoms such as wheeze, dyspnoea, and bronchitis symptoms.⁴⁰

Another limitation of our study is that we could not validate the presence of diseases, the accuracy of the diagnoses, or the accuracy with which respondents reported their health status or morbidity. It is possible that people who smoke or are exposed to ETS simply report more problems than non-smokers or people who are not exposed to ETS.⁵² Conversely, a positive response bias, in which people who have a disease, such as asthma, are more likely to notice and report ETS exposure, is also possible. Our measure of chronic disease exacerbations, which included physician visits, may have resulted in the misclassification of some people who merely had routine, scheduled physician visits unrelated to any worsening of their chronic disease.

A final limitation of our analysis is that we could not include all potential confounders in our models. Recent studies have shown that people exposed to ETS may have poorer diets than people without such exposure.^{53 54} Other confounders we could not control for in this analysis included workplace exposures to other agents causing health effects, outdoor air pollution, familial history of respiratory or cardiac disease, and past exposure to ETS.

Despite these limitations, this study demonstrates that never-smoking adults exposed to ETS report more morbidity than adults not exposed to ETS. ETS exposure can be reduced in worksites and other public areas through corporate and public policy as well as through educational efforts, and ETS has been eliminated in many of these areas; however, educating people about the dangers of ETS is the principal way to reduce ETS exposures in homes.

- 1 US Environmental Protection Agency. *Respiratory health effects of passive smoking: lung cancer and other disorders*. Washington, DC: Environmental Protection Agency, 1992. (Publication EPA/600/6-90/006F.)
- 2 Dockery DW, Ware JH, Spiro A, Speizer FE, Ferris BG. Passive smoking, gas cooking, and respiratory health of children living in six cities. *Am Rev Respir Dis* 1984; 129:366-74.
- 3 Ogston SA, Florey CduV, Walker CM. Association of infant alimentary and respiratory illness with parental smoking and other environmental factors. *J Epidemiol Community Health* 1987;41:21-5.
- 4 Wright AL, Holberg C, Martinez FD, Taussig LM. Relationship of parental smoking to wheezing and nonwheezing lower respiratory tract illnesses in the first year of life. *Am J Epidemiol* 1989;129:1232-46.
- 5 Ehrlich R, Kattan M, Godbold J, et al. Childhood asthma and passive smoking. Urinary cotinine as a biomarker of exposure. *Am Rev Respir Dis* 1992;145:594-9.
- 6 Jindal SK, Gupta D, Singh A. Indices of morbidity and control of asthma in adult patients exposed to environmental tobacco smoke. *Chest* 1994;106:746-9.
- 7 Xu X, Li B. Exposure response relationship between passive smoking and adult pulmonary function. *Am J Respir Crit Care Med* 1995;151:41-6.
- 8 Robbins AS, Abbey DE, Lebowitz MD. Passive smoking and chronic respiratory disease in non-smoking adults. *Int J Epidemiol* 1993;22:809-17.
- 9 Massey JT, Moore TF, Parsons VL, Tadros W. Design and estimation for the National Health Interview Survey, 1985-1994. *Vital Health Stat* 1989;2:1-4.
- 10 US Bureau of the Census. *The current population survey*. Washington, DC: US Government Printing Office, 1991.
- 11 Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR. Exposure of the US population to environmental tobacco smoke. The Third National Health and Nutrition Examination Survey, 1988 to 1991. *JAMA* 1996;275:1233-40.
- 12 Bonham GS, Wilson RW. Children's health in families with cigarette smokers. *Am J Public Health* 1981;71:290-3.
- 13 Mannino DM, Siegel M, Husten C, Rose D, Etzel R. Environmental tobacco smoke exposure and health effects in children: results from the 1991 National Health Interview Survey. *Tobacco Control* 1996;5:13-18.
- 14 US Centers for Disease Control and Prevention (CDC). Trends and recent patterns in selected tobacco-use behaviors—United States, 1900-1993. *MMWR CDC Surveill Summ* 1994;43:1-42.
- 15 Sandler DP, Helsing KJ, Comstock GW, Shore DL. Factors associated with past household exposure to tobacco smoke. *Am J Epidemiol* 1989;129:380-7.
- 16 Borland R, Pierce JP, Burns DM, Gilpin E, Johnson M, Bal D. Protection from environmental tobacco smoke in California: the case for a smoke-free workplace. *JAMA* 1992;268:749-52.
- 17 US Centers for Disease Control and Prevention. *State tobacco control highlights—1996*. Atlanta, Georgia: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 1996. (CDC Publication No 99-4895.)
- 18 National Cancer Institute. *Major local tobacco control ordinances in the United States*. Washington, DC: National Institutes of Health, 1993. (NIH Publication No 93-3532.)
- 19 Ostro BD. Estimating the risks of smoking, air pollution, and passive smoke on acute respiratory conditions. *Risk Analysis* 1989;9:189-96.
- 20 Dayal HH, Khuder S, Sharrar R, Tribf N. Passive smoking in obstructive respiratory diseases in an industrialized urban population. *Environ Res* 1994;65:161-71.
- 21 Cummings KM, Zaki A, Markello S. Variation in sensitivity to environmental tobacco smoke among adult non-smokers. *Int J Epidemiol* 1991;20:121-5.
- 22 Schwartz J, Zeger S. Passive smoking, air pollution and acute respiratory symptoms in a diary study of student nurses. *Am Rev Respir Dis* 1990;141:62-7.
- 23 Diez-Roux AV, Nieto FJ, Comstock GW, Howard G, Szklo M. The relationship of active and passive smoking to carotid atherosclerosis 12-14 years later. *Prev Med* 1995;24:48-55.
- 24 Howard G, Burke GL, Szklo M, et al. Active and passive smoking are associated with increased carotid wall thickness. The Atherosclerosis Risk in Communities Study. *Arch Int Med* 1994;154:1277-82.
- 25 Hole DJ, Gillis CR, Chopra C, Hawthorne VM. Passive smoking and cardiorespiratory health in a general population in the west of Scotland. *BMJ* 1989;299:423-7.
- 26 Glantz SA, Parmley WW. Passive smoking and heart disease: epidemiology, physiology, and biochemistry. *Circulation* 1991;83:1-12.
- 27 Aronow WS. Effect of passive smoking on the cardiovascular and respiratory systems. In: US Department of Health, Education, and Welfare. *Proceedings of the third world conference on smoking and health, 2-5 June 1975*. Washington, DC: Public Health Service, 1975.
- 28 Aronow WS. Effect of cigarette smoking and of carbon monoxide on coronary heart disease. *Chest* 1976;70:514-8.
- 29 Aronow WS. Effect of passive smoking on angina pectoris. *N Engl J Med* 1978;299:21-4.
- 30 Aronow WS. Aggravation of angina pectoris by two percent carboxyhemoglobin. *Am Heart J* 1981;101:154-157.
- 31 Aronow WS, Ferlinz J, Glauser F. Effect of carbon monoxide on exercise performance in chronic obstructive pulmonary disease. *Am J Med* 1977;63:904-8.
- 32 Fontham ET, Correa P, Reynolds P, et al. Environmental tobacco smoke and lung cancer in nonsmoking women. A multicenter study. *JAMA* 1994;271:1752-9.
- 33 Akiba S, Kato H, Blot WJ. Passive smoking and lung cancer among Japanese women. *Cancer Res* 1986;46:4804-7.

34 Buffler PA, Pickle LW, Mason TJ, Contant C. The causes of lung cancer in Texas. In: Mizell M, Correa P, eds. *Lung cancer: causes and prevention*. New York: Verlag Chemie International, 1987:83-99.

35 Correa P, Pickle LW, Fontham E, Lin Y, Haenszel W. Passive smoking and lung cancer. *Lancet* 1983;2:595-7.

36 Geng G, Liang ZH, Zhang AY, Wu GL. On the relationship between smoking and female lung cancer. In: Aoki M, Hisamichi S, Tominaga S, eds. *Smoking and health, 1987. Proceedings of the sixth world conference on smoking and health, 9-12 November 1987*. Amsterdam: Elsevier, 1988:483-6.

37 Humble CG, Samet JM, Pathak DR. Marriage to a smoker and lung cancer risk. *Am J Public Health* 1987;77:598-602.

38 Koo LC, Ho JH, Saw D. Is passive smoking an added risk factor for lung cancer in Chinese women? *J Exp Clin Cancer Res* 1984;3:277-83.

39 Siegel M, Husten C, Merritt RK, Giovino GA, Eriksen MP. Effects of separately ventilated smoking lounges on the health of smokers: is this an appropriate public health policy? *Tobacco Control* 1995;4:22-9.

40 Luenberger P, Schwartz J, Ackermann-Liebrich U, et al. Passive smoking exposure in adults and respiratory symptoms (SAPALDIA Study). Swiss study on air pollution and lung diseases in adults, SAPALDIA Team. *Am J Respir Crit Care Med* 1994;150:1221-8.

41 Danuser B, Weber A, Hartmann AL, Krueger H. Effects of a bronchoprovocation challenge test with cigarette sidestream smoke on sensitive and healthy adults. *Chest* 1993; 103:353-8.

42 US Department of Health, Education, and Welfare. *Smoking and health. A report of the Surgeon General, 1979*. Rockville, Maryland: Public Health Service, Office on Smoking and Health, 1979. (DHEW Publication No (PHS) 79-50066.)

43 US Department of Health and Human Services. *The health consequences of involuntary smoking. A report of the Surgeon General, 1986*. Rockville, Maryland: Public Health Service, Centers for Disease Control, 1986. (DHHS Publication No (CDC) 87-8398.)

44 Adams JK, O'Mara-Adams J, Hoffmann D. Toxic and carcinogenic agents in undiluted mainstream smoke and sidestream smoke of different types of cigarettes. *Carcinogenesis* 1987;8:729-731.

45 Luceri F, Pieraccini G, Moneti G, Dolara P. Primary aromatic amines from side-stream cigarette smoke are common contaminants of indoor air. *Toxicol Ind Health* 1993;9:405-13.

46 Guerin MR, Jenkins RA, Tomkins BA. *The chemistry of environmental tobacco smoke: composition and measurement*. Chelsea, Michigan: Lewis Publishers, 1992.

47 Hammond SK, Coghlin J, Gann PH, et al. Relationship between environmental tobacco smoke exposure and carcinogen-hemoglobin adduct levels in nonsmokers. *J Natl Cancer Inst* 1993;85:474-8.

48 Cuddeback JE, Donovan JR, Burg WR. Occupational aspects of passive smoking. *Am Ind Hyg Assoc J* 1976;37:263-7.

49 Bridbord K, Finklea JR, Wagmer JK, et al. Human exposure to polynuclear aromatic hydrocarbons. In: Freudenthal RI, Jones PW, eds. *Carcinogenesis. Volume 1: polynuclear aromatic hydrocarbons: chemistry, metabolism, and carcinogenesis*. New York: Raven Press, 1976:319-24.

50 O'Conner TZ, Holford TR, Leaderer BP, Hammond SK, Bracken MB. Measurement of exposure to environmental tobacco smoke in pregnant women. *Am J Epidemiol* 1995; 142:1315-21.

51 Tunstall-Pedoe H, Brown CA, Woodward M, Tavendale R. Passive smoking by self report and serum cotinine and the prevalence of respiratory and coronary heart disease in the Scottish heart health study. *J Epidemiol Community Health* 1995;49:139-43.

52 Friedman GD, Siegel AB, Dales LG. Cigarette smoking and chest pain. *Ann Intern Med* 1975;83:1-11.

53 LeMarchand L, Wilkens LR, Hankin JH, Halsey NJ. Dietary patterns of female nonsmokers with and without exposure to environmental tobacco smoke. *Cancer Causes Control* 1991;2:11-6.

54 Matanoski G, Kanchanaraks S, Lantry D, Chang Y. Characteristics of nonsmoking women in NHANES I and NHANES I epidemiologic follow-up study with exposure to spouses who smoke. *Am J Epidemiol* 1995;142:149-57.

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