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THE EFFECTS OF ENVIRONMENTAL TOBACCO SMOKE ON ACUTE RESPIRATORY CONDITIONS

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ABSTRACT

Five years of the annual Health Interview Survey (HIS), conducted by the National Center for Health Statistics, are used to estimate the effects of air pollution, smoking, and environmental tobacco smoke on respiratory restrictions in activity for adults and bed disability for children. After adjusting for several socioeconomic factors, the multiple regression estimates indicate that an independent and statistically significant association exists between these three forms of air pollution and respiratory morbidity. The comparative risks of these exposures are computed and the plausibility of the relative risks is examined by comparing the equivalent doses with other studies that have used actual measurements of exposure taken in the homes of smokers. The results indicate that: (1) smokers will have a 55% to 75% excess in days with respiratory conditions severe enough to cause reductions in normal activity; (2) a one microgram (μg) increase in fine particulate matter air pollution is associated with a 3% excess in acute respiratory disease; and (3) a pack-a-day smoker will increase respiratory-restricted days for a nonsmoking spouse by 20% and increase the number of bed disability days for young children living in the household by 20%. The results also indicate that the estimates of the effects of environmental tobacco smoke on children are improved when the mother's work status is known and incorporated into the exposure estimate.

INTRODUCTION

Over the last three decades, researchers have been assessing the risks to the public health from cigarette smoking and air pollution. There is now considerable agreement that cigarettes and certain outdoor air pollutants adversely affect public health (U.S. DHHS 1984; National Research Council 1985). Cigarette smoke also significantly contributes to indoor air pollution levels, including potentially harmful gases and particulates. Nonsmokers, including both adults and children, exposed to environmental tobacco smoke may experience eye irritation, changes in pulmonary function, and increases in respiratory symptoms (U.S. DHHS 1986). There are few data sources containing health effects information that provide detailed individual-level estimates of smoking status, as well as exposure to both air pollution and environmental tobacco smoke. Epidemiologic research, designed to assess the risks from any one of these irritants, can be criticized because of the failure to control for other omitted exposures that may confound the estimates of risk. This paper addresses these shortcomings by empirically estimating the separate and concurrent risks from exposure to outdoor air pollution, environmental tobacco smoke, and mainstream smoke.

MATERIALS AND METHODS

To estimate the effects of smoking and air pollution on health, data from the Health Interview Survey (HIS), an annual survey conducted by the Census Bureau for the National Center for Health Statistics were used. It is a national, multistage probability survey of 50,000 households. Information on smoking habits was collected from a random one-third of the approximately 130,000 individuals in the survey. This analysis combined data from the 1976 through 1980 HIS and was limited to those households where at least two adults were sampled in the supplemental smoking questionnaire. With this information, the effects of smoking on one's nonsmoking spouse and the effects of parental smoking on children can be examined. The sample is drawn from metropolitan areas of all sizes and regions throughout the United States that are in the HIS and for which fine particulate matter air pollution measurements could be developed. From this remaining sample of roughly 7000 individuals, variables were constructed to estimate direct tobacco consumption and exposure to household tobacco smoke.

The multiple regression model and specification used in this analysis are similar to those used in Ostro (1987) and Hausman et al. (1984). Two groups are used in the analysis: adults, age 18 to 65, and children less than 6 years of age. Depending on the sample, different indicators of morbidity were used, based on the standard HIS two-week recall period. For adults, morbidity was indicated by the number of days of respiratory-related restricted activity in the two weeks prior to the day of the survey. Days of restricted activity, defined as any days where a respondent was forced to alter his or her normal activity, is a general indicator of health status. It includes days of work lost or bed disability as well as more minor restrictions. Respiratory conditions were determined from diagnoses reported in the HIS. For children, disability days spent in bed were used as the indicator of acute morbidity. While neither of these health outcomes are strict measures of respiratory impairment, any inclusion of nonrespiratory symptoms in the dependent variable will not bias the estimated regression coefficients. A dependent variable measured with an error that is unrelated to the independent variable will still produce slope coefficients that are unbiased and consistent (Pindyck and Rubinfeld 1976).

A Poisson distribution is used to model the event counts over time (i.e., the number of days ill) because of the large number of zeros (75% to 95%) in the dependent variables. Hausman et al. (1984) provide details on the efficiency and sensitivity of the Poisson assumption and a test, which indicated no misspecification, of the appropriateness of the model. The likelihood that N_{ijt} days of morbidity will occur in city i for individual j in the two-week time period t is described by (omitting subscripts):

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$$F(N) = e^{-Z} \cdot Z^N/N!$$

The expected value of N, which is Z, is allowed to vary among individuals according to the specification:

$$Z = exp\ (X \cdot b)$$

where X and b are vectors of explanatory variables and estimated parameters, respectively. The estimates of b are obtained by a maximum likelihood procedure.

Within each metropolitan area, different individuals were surveyed throughout the year. Their health survey response was merged with the two-week average of daily readings of outdoor particulate matter, a general indicator of ambient air pollution. Since current evidence (U.S. EPA 1982) implicates small particles rather than total suspended particles in causing health problems because of their potential to penetrate deep into the lung, estimates for fine particles (i.e., those less than 2.5 microns) were developed and used for this study. Based on regression equations developed by Trijonis (1982, 1983) fine particle concentrations were estimated using airport visibility data (see Ostro [1987] for details). Only airports within 9 km of the city center were used and all observations were corrected for humidity. This pollutant serves as an indicator for all pollutants resulting from fossil fuel combustion. The fine particulate concentration can be compared with the significant dose of particles (or tars) produced by cigarette smoking. Ostro (1985) has demonstrated that the relationship between fine particles and respiratory conditions is not affected by the inclusion of ozone or other pollutants.

Besides the air pollution variable, similar independent variables were included in each of the regression equations. These included age, sex, race, education (years of schooling), family income, quarter of the year in which the individual was surveyed, marital status, whether the individual was a current or former smoker, the existence of a chronic health condition, the maximum summer temperature and minimum winter temperature during the two-week recall period, and population density. When a subsample of only those currently working was analyzed, variables were added to control for paid sick leave (average number of days of sick leave disaggregated by industry group) and for occupation (white or blue collar).

Several different forms of the smoking variable were used to capture the effects of either direct or passive smoking. For direct effects, a binary variable was created to indicate whether any individual responded positively to the question, "Do you currently smoke?" There also was a measure of the number of cigarettes currently smoked every day. In each of the regressions for adults, a binary variable was included indicating whether the individual was a current or former cigarette smoker. For the measures of passive smoke exposure, different indicators were investigated. For adults, the alternative measures did not affect the general results, so the sum of the number of cigarettes smoked per day by all household members was used. For children, additional variables that detail the smoking and work status of the parents were considered in order to indicate the exposure to passive smoke.

Estimates of household smoking obviously are only an approximation of the real dose of tobacco smoke in the home and include a number of simplifications. First, the estimates assume that the number of cigarettes smoked during the day is independent of whether one is at work or not. Second, they do not differentiate between different types of cigarettes, the proximity and duration of the exposure to smoke, or ventilation rates in the home. Finally, the variables do not incorporate exposure to tobacco smoke at the workplace. This last shortcoming is mitigated by considering subsamples of the group, such as nonworkers. Thus, although the measures of passive smoke are crude, they do provide some index of the expected level of exposure to tobacco smoke.

TABLE 1

Correlation Coefficients for Sample of Current Workers

	Air Pollution	Current Smoker	Former Smoker
Age	−.02	−.06	.17
Education	−.04	−.14	.05
Income	−.03	−.11	.09
Married	−.01	−.07	.13
1st Quarter	−.07	.01	−.00
2nd Quarter	.01	.00	−.00
3rd Quarter	.16	−.03	.03
Density	−.17	.01	−.01
Winter Min Temp	−.03	.00	−.02
Summer Max Temp	.16	−.02	.02
Nonwhite	.07	.02	−.07
Chronic condition	.03	.01	.06
Air Pollution*	1.00	.01	−.00

Note:
* = measured as fine particulate matter

The existence of an omitted or confounding variable is always possible in an observational study. However, for an omitted variable to affect the results of this analysis, it must be related to both the explanatory variables of interest—air pollution, smoking status, and passive smoke—and the dependent variable. The inclusion of various socio-economic measures such as income, race, sex, education, and occupation will reduce the possibility of any omitted variable bias. In addition, as indicated in Table 1, the potential for confounding is low, since the correlations among the explanatory variables were low. The variables with the highest correlation with particulate matter air pollution were summer maximum temperature ($r = .16$), population density ($r = -.17$), and being surveyed in the third quarter (.16). Current smoking had the highest correlation with education (−.14) and income (−.11), while being a former smoker was correlated with age (.17), being married (.13), being male (.20), and income (.09). None of these correlations appears large enough to significantly confound the relationships between air pollution, smoking, and health. The problem was minimized further by considering successively smaller and more homogeneous subsamples of the adult population. Thus, results for the following subsets are reported:

1. All workers
2. All nonsmokers
3. All never-smoking nonworkers
4. Female, never-smoking nonworkers

First, the entire sample of those currently working, aged 18 to 65 ($n = 3634$) was used. This group has fairly similar activity patterns but, since it includes both smokers and nonsmokers, the effects of direct vs. passive smoke may be difficult to separate. Therefore, a sample consisting only of nonsmokers was analyzed next. Finally, to eliminate the in-

TABLE 2

Estimated Regression Coefficients for Respiratory-Related Restrictions in Activity, Workers, Age 18–65 (n = 3634) (standard errors in parentheses)

Variable	Mean	(Model 1)	(Model 2)	(Model 3)
FP	23.2	3.13 (0.26)	3.23 (0.27)	3.21 (0.27)
FOSMO	0.22	70.0 (11.0)	69.9 (11.6)	21.7 (8.6)
COSMO	0.41	74.1 (9.56)	55.5 (10.8)	
TOTCIG	17.17		0.83 (0.16)	
RRAD	2.5			

Note: FP = Two week average of fine particulate air pollution, lagged two two-week periods (micrograms per cubic meter).
FOSMO = Binary variable indicating former smoker (0, 1).
COSMO = Binary variable indicating current smoker (0, 1).
TOTCIG = Total number of cigarettes smoked by household members.
RRAD = Days of respiratory-related restrictions in activity per year.

All coefficients are significant, using two-tailed test, at $p < .01$. Coefficients are $\times 100$ and are interpreted as percent change in respiratory conditions due to a one unit change in the independent variable.

The other independent variables included in each regression (with their means) include:

Age (38.14), Race (17.6% nonwhite), Sex (40% female), Education (12.6 years), Family income ($22,046), Quarter of the year of survey (first 21.3%, second 23.4%, third quarter 27.6%), Marital status (65.6% married), Occupation (32.3% blue collar), Existence of chronic condition (8.5%), Minimum winter temperature (23.3), Maximum summer temperature (87.4), Paid sick leave (4.32 days), and Population density (7722 per square mile).

fluence of current exposure to occupational irritants, subsamples of never-smoking nonworkers were considered.

RESULTS

Table 2 presents the estimated coefficients for the smoking and air pollution variables for the regressions using the full sample of those currently working. The results of Model 1 (column 1) indicate a positive association between being either a former or current smoker and the number of days with a respiratory condition. In addition, the results indicate that exposure to fine particulate matter will affect respiratory health. In Model 2, a variable measuring the total number of cigarettes smoked daily by household members was added to the regression to incorporate the impact of environmental tobacco smoke. In Model 3, the effect of the omission of any of the smoking variables on the air pollution variable was measured. Specifically, the results indicate that the regression results are not affected by the omission of either direct or passive smoking; the coefficient for fine particulate air pollution remains unchanged from the specifications of Models 1 through 3.

To eliminate the problem of collinearity in the regressions between the measures for current smoking and for passive smoking (i.e., total number of cigarettes smoked by the household), a sample of currently nonsmoking adults was considered. The results of the multiple regression Model 4, shown in Table 3, again indicate that the air pollution and passive smoke variables are each significantly related to respiratory-related restrictions in activity.

Next, to better isolate the effects of passive smoke, former smokers were eliminated from the sample and a subsample consisting only of never-smokers, not currently working ($n = 979$), was considered. This sample is not exposed to occupational irritants and does not, by definition, include either current or former smokers. As displayed in Table 3, Model 5, the measures of air pollution and passive smoke each have a statistically significant relationship with respiratory illness.

TABLE 3

Estimated Regression Coefficients for Respiratory Related Restrictions in Activity, Age 18–65 (standard error in parentheses)

	All Nonsmokers (n = 3801)		Nonworking Never Smokers All (n = 979)		Nonworking Never Smokers Female (n = 782)	
Variable	(Model 4) Mean		(Model 5) Mean		(Model 6) Mean	
FP	23.54	1.35 (0.17)	24.14	1.07 (0.37)	24.33	1.38 (0.37)
FOSMO	0.34	71.6 (5.44)	—		—	
COSMO	—		—		—	
TOTCIG	8.26	0.85 (0.18)	8.3	0.94 (0.41)	8.5	1.14 (0.42)
RRAD	3.58		4.73		5.55	

Note: FP = Two week average of fine particulate air pollution, lagged two two-week periods (micrograms per cubic meter).
FOSMO = Binary variable indicating former smoker (0, 1).
COSMO = Binary variable indicating current smoker (0, 1).
TOTCIG = Total number of cigarettes smoke daily by household members.
RRAD = Days of respiratory-related restrictions in activity per year.

All coefficients are significant, using two-tailed test, at $p < .01$. Coefficients are $\times 100$ and are interpreted as percent change in respiratory conditions due to a one unit change in the independent variable.

The other independent variables included in each regression (with the means for model 4) include:

Age (39.13), Race (16.7% nonwhite), Sex (45% female), Education (12.7 years), Family income ($20,796), Quarter of year of survey (first 14.4%; second 24.6%; third 31.1%), Marital status (66.7%) Work status (64.5% working), Existence of chronic condition (12.8%), Minimum Winter Temperature (25.4), Maximum Summer Temperature (87.0), and Population Density (8245 per square mile).

In the final regression for adults, in Model 6, a sample of nonworking, never-smoking females ($n = 782$) was considered. Again, both air pollution and passive smoke are significantly related to restricted activity days. These last two subsamples produce the most convincing evidence about the ill effects of passive smoke in that the groups are not currently being exposed on the job and are not current or former smokers.

The results of the analysis of the effects of secondhand smoking on the number of disability days spent in bed by children age zero to six years are displayed in Table 4. To represent the effects of passive smoking, the same measure used above, the total number of cigarettes smoked by household members, was used. A number of refinements to this measure also were considered and are detailed below. The rest of the independent variables in the regression equation were similar to those used for the adults and include: age, sex, education of head of household, family income, national origin (Asian, Hispanic, Black), the existence of a chronic disease, family size, the quarter of the year of the survey interview, winter minimum and summer maximum temper-

TABLE 4

Estimated Regression Coefficient for Bed Days, Children Age 0–6 (n = 1575) (standard error in parentheses)

Variable	Mean	(Model 7)	(Model 8)	(Model 9)	(Model 10)
FP	23.86	0.88* (0.30)	0.86* (0.31)	0.87* (0.30)	0.86* (0.30)
TOTCIG	16.79	0.37* (0.14)			
DADCIG	9.76		−0.22 (0.14)		
MOMCIG	7.03		0.21# (0.11)		
MOMSMHO	0.23			18.7* (6.1)	
MOMCIGHO	4.24				0.76* (0.28)
BEDDAYS	6.28				

Note: FP = Two week average of fine particulate air pollution, lagged two two-week periods (micrograms per cubic meter).
TOTCIG = Total number of cigarettes smoked daily by household members.
DADCIG = Cigarettes smoked daily by male adult in household.
MOMCIG = Cigarettes smoked daily by female adult in household.
MOMSMHO = Binary variable indicating presence of a nonworking, smoking, female adult in the household.
MOMCIGHO = Number of cigarettes smoked daily by nonworking female adult in the household.
BEDDAYS = Bed disability days per year.

* = significant at $p < 0.01$, two-tailed test
# = significant at $p < 0.10$, two-tailed test

Coefficients are ×100 and are interpreted as percent change in respiratory conditions due to a one unit change in the independent variable.

The other independent variables included in each regression (with their means) include:

Age (3.06), Race (74% white; 16% black; 8% Spanish; 2% Asian), sex (48% male), Education of head of household (12.65 years), Family income ($17,770), Quarter of year of survey (1st: 18%; 2nd: 28%; 3rd: 26%), Existence of chronic condition (2%) Minimum winter temperature (23.8), Maximum summer temperature (92.5) and Population density (8,316).

atures, and air pollution (measured in terms of fine particulate matter).

The results indicate a generally positive and significant relationship between the passive smoke measure and the number of days spent in bed for children. In Model 7, for example, coefficients for air pollution and the total number of cigarettes smoked daily by household members are both positive and significantly related to bed disability days. This estimated effect is significant since the HIS data indicate that 62.3% of the children have at least one parent who smokes, while 7.3% have two parents who smoke.

In regression Model 8, variables were included indicating the number of cigarettes smoked separately by the male and female adult at the home. As may be expected, only the adult females' smoking status had a positive association with child illness. The coefficient for the male adult was not different from zero. With this indication that the female adult's smoking status was a better indicator of a child's exposure to secondhand smoke, in Model 9 a binary variable was included that took on a value of one if the female adult in the household was a smoker and also currently not working. This greater detail in the exposure measure increases the efficiency of the estimate. Specifically, the regression indicates that children with a smoking, nonworking female adult in the home will have 18.7% more bed days than children with "other" (either smoking and working, or nonsmoking) female adults. Finally, Model 10 considers the number of cigarettes smoked by the nonworking, smoking female adults. Overall, these estimated effects are important since the HIS data indicate that 20% of the children in this age bracket live with a nonworking female adult who smokes.

The impact of acute illness of the parents was also tested. A binary variable was created that indicated whether either the adult male or female living in the household had a respiratory-related restriction in activity during the two-week recall period. While parental respiratory illness was significantly associated with the child's bed days, the estimated coefficient of the passive smoking variable was not affected by the inclusion of this variable.

DISCUSSION

The results of the above analysis indicate that all three sources of "air pollution"—outdoor ambient air pollution, direct consumption of cigarettes, and environmental tobacco smoke—are statistically associated with respiratory-related restrictions in the activity of adults. There is also an indication that air pollution and passive smoke are associated with an increase in illness in children, measured as the number of days spent in bed for health reasons.

From the empirical evidence, an exposure-response relationship was estimated for all three sources of air pollution. Also, concerns about the potential confounding between the effects of smoking and outdoor air pollution on health were addressed. Therefore, the comparative risks from the three different air pollutants and the equivalent doses can be determined.

In interpreting these results, one must be particularly aware of selection bias problems. For example, some people will select themselves out of the labor market or out of tobacco consumption because of health conditions. It is possible, therefore, that for some subgroups, the current smokers may appear healthier than former smokers, since the latter may have either quit because of respiratory problems or, in extreme cases, may have died. There is also a possibility of reporting bias regarding smoking status; specifically, that smokers may not admit to currently smoking. However, this would bias the air pollution estimate only if smokers in either high *or* low air pollution cities consistently tended to misrepresent their status. In addition, if a current or former smoker is misrepresented as a "never" smoker, this would tend to underestimate the effects of smoking.

The Poisson regression results for adults indicate that the estimated relationship between outdoor air pollution, measured as fine particulate matter, and days with respiratory-related restrictions in activity, is not affected by the exclusion or inclusion of any of the smoking variables (see Tables 2 and 3). The persistence of the effect, regardless of the specification of the smoking status, may be due to the relatively low correlation between smoking and air pollution. A lag of two two-week periods prior to the survey recall period (i.e., the third and fourth weeks before the two-week recall period) provided the best fit between air pollution and health.

The sample consisting of current workers (Models 1 through 3) may comprise the best sample for determining the risk of fine particulate air pollution since their normal activity patterns are relatively similar and restrictions in their activity are easier to note. The results indicate that a one μg per m^3 change in fine particles results in a 3.2% excess in acute respiratory disease, or an annual increase of 8000 days of respiratory-related restrictions per 100,000 (3.2% change in

TABLE 5

Effects of Smoking on Acute Respiratory Symptoms

Author, reference	Effect	Percent Increase In Prevalence for Smokers
Bland et al.[a] (1978)	Morning cough, boys	29
	Morning cough, girls	233
	Cough at other times, boys	70
	Cough at other times, girls	84
	Breathlessness, boys	82
	Breathlessness, girls	71
Bouhuys et al.[b] (1981)	Cough, men	195
	Cough, women	98
Burghard et al. (1979)	Morning cough	88
	Day or night cough	72
Dean et al.[c] (1978)	Phlegm, men	136
	Phlegm, women	244
Gulsvik (1979)	Morning cough	227
	Daytime cough	300
	Phlegm when coughing	180
Park (1981)	Morning cough	113
	Daytime cough	160
	Nightime cough	47
	Morning phlegm	30
	Daytime phlegm	−43
	Nightime phlegm	80
	Breathlessness on exertion	28
Neukirch et al. (1982)	Cough and/or phlegm, boys	34
	Cough and/or phlegm, girls	66
Kristein (1983)	Work loss days	33–45

Notes:
a = comparing nonsmoker to occasional smoker
b = uses average for all cities
c = assumes 20 cigarettes consumed by smoker

respiratory illness/μg per m^3 x 2.5 mean annual days of respiratory illness x 10^5). For children (Table 4), a 1 μg increase in fine particles results in an increase of 0.86% days in bed, or an annual increase of 5400 days per 100,000 (0.86% x 6.28 mean annual bed days x 10^5). The sample of individuals currently working also was used to estimate the direct effect of smoking. Table 2 indicates that relative to never-smokers, this group has a 55% to 75% excess in days with respiratory conditions severe enough to cause reductions in normal activity. By definition, this estimate compares two groups that are similar except for smoking status, and controls for several socioeconomic factors including age, race, sex, income, education, occupation, and number of days with sick leave. This range is similar to that reported in other studies of acute respiratory symptoms. While smokers have two to four times more chronic respiratory disease (U.S. DHHS 1984), existing research indicates that they have 20% to 80% more acute respiratory symptoms (see Table 5 for a summary of these studies). Thus, the estimates predicted from this analysis are within the range provided by the earlier studies.

This analysis also indicates that former smokers may continue to have up to 70% more respiratory-restricted activity days (or 2.5 days per person) than the population of never-smokers. This large effect may be a result of the smoking cessation by those with existing chronic disease.

The best estimate of the impact of secondhand smoke is obtained from the sample of nonsmokers or of never-smoking nonworkers. For the latter group there is no confounding from direct smoking or from acute occupational exposure. As indicated by Model 5 (Table 3), for example, there may be almost a 1% increase in acute respiratory illness per cigarette smoked per day by household members. This amounts to 0.062 days per year per cigarette (i.e., estimated coefficient indicating 0.94% increase in respiratory illness days per cigarette x mean annual days of respiratory illness for this group of 6.6). The results also indicate that the exposure estimate was not improved by weighting the amount smoked by an individual by a national average of the expected time spent at home for a given sex and work status.

The estimated acute effect of environmental tobacco smoke is larger relative to the impact of smoking (i.e., from Table 2, 0.83% per passive cigarette vs. 75% per pack, or 3.75% per direct cigarette) than existing estimates of exposure would suggest. For example, the National Research Council (1981) estimated that the average particulate matter "dose" from passive smoke is about 1% of the direct dose from smoking. The larger-than-proportional impact on health from environmental tobacco smoke indicated by this analysis may be due to several factors including: the smoking spouse being visited at the home by other smokers; the nonsmoking spouse frequently being close to the plume generated by the smoker (the NRC estimate was based on average ambient levels in a public place or in the smoker's home); differences by smoking status in particle retention and clearance efficiency; and differences in constituents between mainstream and sidestream smoke. Further research is indicated regarding the effective dose from sidestream vs. mainstream smoke.

Regarding children, the results indicate that a child with a nonworking, smoking female adult in the home will have almost one more day per year (a 19% increase) spent in bed for health reasons than would other children. These numbers are consistent with Ware et al. (1984) who suggested a 20% to 35% increase in the risk of all respiratory illnesses and symptoms due to maternal smoking; Bland et al. (1978) (16% increase in day or night cough); Schenker et al. (1983) (18% increase in chest illness before age two and 34% increase in the prevalence of more than three days of chest illness); Cameron et al. (1969) (33% increase in rates of respiratory illness with restricted activity); and Bonham and Wilson (1981) (6% increase in restricted activity days for children under six). The latter paper was based on the 1970 HIS and used analysis of variance and comparisons of the means to indicate that children in families that smoked more tended to have more bed-disability days and restricted activity days. However, Bonham and Wilson (1981) did not provide quantitative estimates of risk and only controlled for a few of the potentially confounding influences on the children.

The results for children indicate that the exposure estimate is dramatically improved when maternal smoking habits are used in the model. These findings are consistent with those reported by Tager et al. (1983), Weiss et al. (1980), and Ware et al. (1984), and are probably a result of the greater amount of time children spend with their mothers. Further improvements in the estimates occur when the mother's work status is known and incorporated into the exposure estimate.

Besides comparing these results with those obtained from previous research, one can check the plausibility of the results by computing the equivalent dose (in μg per m^3 of respirable particulates) of secondhand smoke relative to ambient par-

ticulate matter. Although there are many different measures that can be used to classify the nonsmokers' exposure to tobacco smoke, total respirable particulates may be the best measure of the potential risk for acute respiratory conditions.

Thus, to test the plausibility of the passive smoke results, fine particles (FP) were first converted to respirable particles (RSP) (i.e., particles roughly below 10 microns) using the ratio FP/RSP = 0.95 reported by Sexton (1984). Using the results derived earlier indicating a 1 μg increase in fine particles relates to an increase of 0.08 days with respiratory restrictions, 1 μg of respirable particulates relates to 0.08 restricted days. Since it also has been determined (Model 6) that one passive cigarette relates to 0.062 restricted days, this implies that one passive cigarette has the same impact as 0.79 (i.e., 0.062/0.084) μg of respirable particulates. Stated differently, a pack-a-day smoker would generate the equivalent health impact of 14.8 μg per m^3 of respirable particulates. Dockery and Spengler (1981a, b) have empirically determined from actual exposure measurements that in the average house, a pack-a-day smoker contributes 18 μg per m^3 of respirable particles. Other equivalent doses could be calculated using other subsamples. Regardless of the specific sample used, however, the estimates appear plausible when compared with the observed pack-a-day exposure of 18 μg.

CONCLUSIONS

In sum, evidence from the Health Interview Survey indicates a consistent statistical association between respiratory conditions and exposures from air pollution, smoking, and passive smoke. The results indicate that: (1) smokers will have a 55% to 75% excess in days with respiratory conditions severe enough to cause reductions in normal activity, (2) a 1 microgram increase in fine particulate matter air pollution is associated with a 3% excess in acute respiratory disease, and (3) a pack-a-day smoker will increase respiratory restricted days for a nonsmoking spouse by 20% and increase the number of bed disability days for young children living in the household by 20%. The results also indicate that the estimates of the effects of environmental tobacco smoke on children are improved when the mother's work status is known and incorporated into the exposure estimate.

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DISCUSSION

Carl N. Lawson, LRW Engineers Inc., Tampa, FL: Did you take into consideration teenagers or other smokers in a home? If not, why? That could play a very important part in your continued study.

B.D. Ostro, California Public Health Foundation, Berkeley, CA: No, teenagers were not included since their activity patterns, specifically the amount of time and time of day they spend at home, is so variable. Obviously, they would contribute to the overall dose of ETS.

Behzad Samimi, Graduate School of Public Health, San Diego State University, San Diego, CA: You stated in your presentation about assessment of visibility for submicroscopic particles. I wonder what technique you used for this assessment? Did you measure the visibility in terms of COH (coefficient of haze)? Did you use a tape sampler for this purpose? What technique was used to assess and characterize the fine particles? Since less than 10 μm particulate were measured, what type of size selector did you employ?

Ostro: Airports are required to report visibility every hour, based on visual markings, not direct ambient measurements. Equations involving atmospheric chemistry have been developed that relate the inverse of visibility to fine particles (less than 2.5 microns) after correcting for humidity and other meteorologic factors.