A Study of Twelve Southern California Communities with Differing Levels and Types of Air Pollution

I. Prevalence of Respiratory Morbidity

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To study possible chronic respiratory effects of air pollutants, we initiated a 10-yr prospective cohort study of Southern California children, with a study design focused on four pollutants: ozone, particulate matter, acids, and nitrogen dioxide (NO_2). Twelve demographically similar communities were selected on the basis of historic monitoring information to represent extremes of exposure to one or more pollutants. In each community, about 150 public school students in grade 4, 75 in grade 7, and 75 in grade 10 were enrolled through their classrooms. Informed consent and written responses to surveys about students' lifetime residential histories, historic and current health status, residential characteristics, and physical activity were obtained with the help of the parents. In the first testing season, 3,676 students returned questionnaires. We confirmed associations previously reported between respiratory morbidity prevalence and the presence of personal, demographic, and residential risk factors. Rates of respiratory illness were higher for males, those living in houses with pets, pests, mildew, and water damage, those whose parents had asthma, and those living in houses with smokers. Wheeze prevalence was positively associated with levels of both acid (odds ratio [OR] = 1.45; 95% confidence interval [CI], 1.14–1.83) and NO_2 (OR = 1.54; 95% CI, 1.08–2.19) in boys. We conclude, based on this cross-sectional assessment of questionnaire responses, that current levels of ambient air pollution in Southern California may be associated with effects on schoolchildren's respiratory morbidity as assessed by questionnaire. Peters JM, Avol E, Navidi W, London SJ, Gauderman WJ, Lurmann F, Linn WS, Margolis H, Rappaport E, Gong H, Jr, Thomas DC. A study of twelve Southern California communities with differing levels and types of air pollution: I. Prevalence of respiratory morbidity.

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Despite significant improvements in ambient air quality over the past 30 yr, air pollution continues to pose a challenge to regulatory and health professionals. Millions of people breathe outdoor air containing pollutants at concentrations sufficient, judging from laboratory and field investigations, to provoke acute decreases in pulmonary function (1–3), respiratory symptoms (4, 5), and respiratory tract inflammation (6–8). Understanding chronic respiratory effects, and identifying specific pollutants responsible for them, has proved to be more elusive. Epidemiologic studies in California, including the UCLA Chronic Obstructive Respiratory Disease (CORD) studies (9, 10) and the Adventist Health Study (11), have reported declines in respiratory function and increases in chronic respiratory illness associated with increased exposure to ambient ozone (O_3) and/or particulate matter (currently measured as PM_{10}, particles less than 10 μm in aerodynamic diameter). However, issues of subject attrition and the adequacy of individual exposure assessment have made the results ambiguous. Excellent reviews of the voluminous literature on air pollution health effects have been published recently by Bates (12, 13), and by a committee of the American Thoracic Society for all common pollutants (14, 15).

In Southern California, episodic outdoor levels of O_3, PM_{10}, and nitrogen dioxide (NO_2) historically have been among the highest in the United States, and continue to exceed federal and state clean air guidelines, at least for O_3 and PM_{10}. Significant concentrations of strong acid vapor also occur, due primarily to photochemical conversion of nitrogen oxides to nitric acid. However, geographic variation is sub-

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stantial; different pollutants tend to be high in different localities, and all four pollutants are consistently low in one area near the coast northwest of Los Angeles. This heavy but nonuniform impact of air pollution makes Southern California well suited for epidemiologic studies.

To investigate the potential for chronic respiratory health effects from air pollution, we designed and initiated a multiyear investigation to document the respiratory health of children. Its goals are to determine whether significant chronic effects occur, and if so, to determine the relative importance of each of the aforementioned pollutants to the effects. Our study focuses on children because they spend more time outdoors and exercise more than adults, both of which increase their ambient air pollution exposures. In addition, the risk factors of smoking and occupational respiratory exposures are not as important for most young children. Public school students in grades 4, 7, and 10, residing in 12 middle-income communities with differing levels and patterns of pollution, were selected for study.

This report presents results from our initial questionnaire assessment of respiratory disease prevalence, residential history, housing characteristics, outdoor activity, and other potential risk factors such as passive smoking. We summarize the extensive air sampling effort aimed at characterizing and quantifying air pollution in the 12 communities and finally we relate the air pollution levels to respiratory disease prevalence in the 12 communities. The relationships between air pollution levels and pulmonary function are described in the companion paper (16).

METHODS

Experimental Design

The study was based on a quasi-factorial design that focused on O_3, PM_{10}, strong acid, and NO_2 as the pollutants of primary interest. Regions of Southern California were selected with the aim of maximizing the variability and minimizing the correlations in these four pollutants, based on historic routine air monitoring data and specialized monitoring studies, interpolating from more distant monitoring sites if no local data were available. In regions with pollution patterns of interest, cities or neighborhoods with stable, largely middle-income populations, ethnically representative of Southern California as a whole, were identified from 1990 census data. To address community-level sources of variability, we sought to maximize the number of participating communities, within existing financial constraints. Ultimately, 12 communities were selected. Four of these, in which public school officials declined to participate, were replaced by nearby communities with similar populations and air quality.

We sought to enroll as many participants as logistically feasible: about 300 per community, 3,600 in all. To permit cross-sectional assessment of age-related differences in chronic air pollution effects, we sought 150 fourth graders (aged 9–10 yr), 75 seventh graders (12–13 yr) and 75 tenth graders (15–16 yr) from each community.

Site Selection and Subject Recruitment

School district representatives in participating communities were consulted to identify suitable schools, based on demographic stability, likely parental cooperation, appropriate linkages between elementary, middle, and secondary schools, and absence of local pollution sources. Presentations were made to district administrators, school administrators, teachers, and students to explain the study and encourage participation. In any classroom targeted for participation, every student was invited to volunteer. In middle and secondary schools, science, health, or physical education classes were targeted, excluding any special classes for gifted or learning-disabled students.

Assessment of Past Exposures

The approach used to estimate past exposures to air pollutants was based on data collected in 1986–1990 by monitoring stations existing prior to our own study. This allowed estimates of O_3, PM_{10}, NO_2, and acid vapor, with spatial interpolation as needed, particularly for acid vapor. These data provided the estimates of relatively recent exposure upon which the communities were selected. We have also obtained estimates of lifetime exposure, based on residence histories and routine measurements nationwide. Results of comparisons of lifetime exposures between individuals will be reported separately.

Assessment of Current Exposures

An objective of the ambient air monitoring program established by our study team was to obtain continuous measurements of O_3, NO_2, and PM_{10}, with hourly averaging, in all communities. An additional objective was to obtain integrated (2-wk average) measurements of $PM_{2.5}$ (particulates less than 2.5 μm in diameter) (total mass, as well as nitrate, sulfate, and ammonium ion concentrations), and gaseous nitric, hydrochloric, formic, and acetic acids, for determination of seasonal and annual average concentrations. Ambient air quality monitoring was established in all 12 communities by adding instruments to seven pre-existing facilities operated by air pollution control agencies, and by constructing five new stations. All stations monitored hourly O_3 using ultraviolet photometers, hourly NO_2 (actually total NO_X minus NO) using chemiluminescent instruments, and hourly PM_{10} mass using tapered element oscillating microbalance (TEOM) instruments (17). The TEOM PM_{10} measurements were adjusted by 2 to 25% to account for losses from volatilization based on analysis of collocated TEOM and HiVol PM_{10} measurements in four of the 12 communities (18). A new aerosol/acid sampler was developed to collect 2-wk integrated samples of the aforementioned $PM_{2.5}$ and gaseous acid components at each station.

Health Effects Assessment

Two questionnaires were administered to the subjects in 1993. The questionnaire on medical history, residential history, and housing characteristics was distributed in early winter, and completed by parents or legal guardians. Questions on respiratory symptoms and illnesses were modeled after those used in the Harvard Six Cities and 24 Cities Studies (19, 20). Incentives were offered to classrooms to achieve high participation rates. This questionnaire was distributed in all communities simultaneously, in English or Spanish according to subject/parent preference. The time/activity pattern assessment questionnaire was administered in the spring. Subjects were given the forms by project staff following their lung function tests, and asked to complete and return them the following day. Seventh and tenth graders generally completed their questionnaires at the time of testing. Fourth graders were asked to take them home and enlist parents' help. Many fourth graders did not return forms, in which case their surveys were completed by telephone interviews with parents.

Definitions of Respiratory Conditions

Medically diagnosed asthma is a yes answer to the question "Has a doctor ever diagnosed this child as having asthma?" *Current/severe asthma* assumes medically diagnosed asthma, greater than one illness in the past 12 mo, or one illness in the past 12 mo and: (*1*) ever interrupted sleep; or (*2*) any medication in last 12 mo; or (*3*) overnight hospital stay in last 12 mo. *Wheeze* is a yes answer to the question "Has your child's chest ever sounded wheezy or whistling, including times when he or she had a cold?" (nonasthmatic subjects only). *Bronchitis* is a yes answer to the question "In the last 12 mo, did this child have bronchitis?" *Cough* is a positive response to either question: "During the past 12 mo, has this child had a cough first thing in the morning that lasted for as much as 3 mo in a row?" or "During the past 12 mo, has this child had a cough at other times of the day that lasted for as much as 3 mo in a row?"

Statistical Methods

We studied the relationship between community-specific ambient levels of pollutants and prevalence rates of the following questionnaire-based morbidity end points: ever asthma, current asthma, wheeze, cough, and bronchitis. Stepwise logistic regression was utilized to determine which personal variables were associated with each symptom, after adjustment for community and grade in school. The personal variables included as potential regressors were race, sex, height, body mass, reported asthma in parents, hay fever, membership in a health

TABLE 1

ESTIMATED AIR POLLUTION MEASUREMENTS FOR 12 COMMUNITIES BASED ON DATA COLLECTED DURING 1986–1990

	24-h Average O_3 (*ppb*)	Average Daily 1-h Max O_3 (*ppb*)	PM_{10} ($\mu g/m^3$)	NO_2 (*ppb*)	Acid Vapor (*ppb*)
Alpine	48.7	80.5	37.4	16.7	1.2
Atascadero	34.1	58.7	28.0	8.2	0.9
Lake Elsinore	38.4	82.7	59.5	21.3	2.1
Lake Gregory	54.2	98.9	38.3	23.6	2.3
Lancaster	38.4	70.8	47.0	13.2	3.2
Lompoc	25.9	34.8	30.0	1.6	0.9
Long Beach	18.4	45.2	49.5	44.8	2.4
Mira Loma	34.0	95.1	84.9	32.8	1.1
Riverside	34.0	95.1	84.9	32.8	1.1
San Dimas	41.2	109.2	67.0	39.1	2.9
Santa Maria	18.4	30.2	28.0	7.7	0.9
Upland	31.5	92.0	75.6	44.6	3.1

insurance plan, five or more plants in the house, water damage or mildew in the house, exposure to passive smoke at home, recent infestation of the house by pests or roaches, carpet in the subject's bedroom, current taking of vitamins, active smoking by the subject, pets in the house, and a gas stove or air conditioner in the house. Any personal covariates that were significant at the $p < 0.15$ level for a given symptom were included in all subsequent models of pollutant effects for that symptom.

To investigate the relationship between symptoms and air pollutants, we used a two-stage regression approach. In the first stage, we fit a single logistic regression model for the condition, including all of the significant personal covariates, and 12 separate intercept terms α_j for each community *j*. These intercept terms represent the logit of the community-specific prevalence rates, adjusted for the personal covariates. The adjusted prevalence rate can then be computed as $e^{\alpha j}/(1 + e^{\alpha j})$. In the second stage model, we regressed the community-specific parameter estimates (α_j, $j = 1, \ldots, 12$) on the community-specific ambient level of a given pollutant, using simple linear "ecologic" regression $\alpha_j = (\alpha_j = \alpha + \beta Z_j + E_j)$ where Z_j denotes the pollution variable(s) in community *j*. The expectation is that if there is a relationship between the condition and pollution, there will be a non-zero slope (β) in this model. The standard *t* test of zero-slope for a regression model is utilized to determine whether rates of symptoms are correlated with pollutants. The quantity, e^{β}, can be interpreted as the prevalence odds ratio of symptoms per unit change of the pollutant, adjusted for personal covariates. Both 1986–1990 and 1994 pollutant levels were tested in these models, and each model considered only one pollutant at a time. Analyses were performed on all subjects, and on subsets defined by sex.

RESULTS

Air Pollution Levels

Table 1 summarizes the distribution of the various air pollutants studied, based on the 1986–1990 estimates upon which the communities were selected. The 1994 measurements made during the study are presented in Table 2. Atascadero, Santa Maria, and Lompoc generally have low levels of all pollutants; Upland and San Dimas generally have high levels of all pollutants; and the other seven communities represent different profiles, with high levels of some but not all pollutants. Generally, for the 1986–1990 estimate, the correlations among the different pollutants tended to be relatively low across the 12 communities, the highest being between NO_2 and PM_{10} (0.75) and between O_3 and PM_{10} (0.69). The 1986–1990 estimates correlated well with our measurements in 1994. For 1994, the correlations of NO_2 and O_3 with PM_{10} measurements were similar to the 1986–1990 estimates ($r = 0.74$ and 0.68, respectively). Acid vapor was much more highly correlated with NO_2 in 1994 ($r = 0.83$) probably as a result of more accurate acid measurements. PM_{10} and $PM_{2.5}$ were highly correlated ($r = 0.90$). Acid vapor was correlated with peak O_3 ($r = 0.69$) but not 24-h O_3.

Participation Rates

Of 4,843 eligible participants, 3,676 returned medical questionnaires (76%). Return rates were highest for 4th graders (79%), and lowest for 10th graders (70%). Participation rates varied across communities from 65 to 86% but did not correlate with either pollution levels or disease prevalence.

Demography

Table 3 presents data on demographic characteristics, as reported by parents, by community. Whites were a plurality in all communities, and a majority in all except Long Beach, Riverside, and Santa Maria. Hispanics (who might be of any race, but usually placed themselves in the white or "other" categories) were a majority in Santa Maria. Sizable (> 10%) minorities of a specific race were present in Riverside (African Americans) and Long Beach (African and Asian Americans). Family income was higher in Upland, and lower in Santa Maria. A large majority of subjects (ranging from 72 to 95%

TABLE 2

AIR POLLUTION MEASUREMENTS FOR 12 COMMUNITIES BASED ON DATA COLLECTED IN 1994

					2-wk Average	
Community	24-h Average O_3 (*ppb*)	Average Daily 1-h Max O_3 (*ppb*)	24-h Average PM_{10} ($\mu g/m^3$)	24-h Average NO_2 (*ppb*)	Acid (HNO_3+HCl) (*ppb*)	$PM_{2.5}$ Mass ($\mu g/m^3$)
Alpine	42.3	71.3	21.3	13.2	2.6	9.2
Atascadero	24.7	50.1	20.7	14.1	1.3	7.6
Lake Elsinore	35.9	76.1	34.7	21.9	3.3	13.4
Lake Gregory	65.8	97.5	24.2	8.5	3.5	11.1
Lancaster	33.4	59.7	33.6	17.8	2.3	9.3
Lompoc	28.3	42.7	13.0	2.7	1.0	7.3
Long Beach	18.9	41.3	38.8	36.4	3.5	16.3
Mira Loma	27.3	76.3	70.7	31.3	3.1	31.5
Riverside	30.7	80.6	45.2	33.9	3.7	25.5
San Dimas	26.8	82.9	36.7	36.2	5.0	22.1
Santa Maria	20.0	35.5	29.2	4.3	1.3	6.7
Upland	25.2	73.3	49.0	42.6	4.7	24.0
Average	32.0	64.5	34.9	21.5	2.9	15.1

TABLE 3

NUMBER OF SUBJECTS AND DEMOGRAPHIC CHARACTERISTICS BY COMMUNITY

Community	Eligible Subjects	Subjects with Baseline Questionnaire Information (%)	White	Black	Asian	Other	Hispanic	Male	Income > $50,000
Alpine	396	298 (75)	84.0	0.4	0.8	14.8	12.8	49.8	37.5
Atascadero	371	260 (70)	84.1	0.4	0.4	15.1	11.4	40.5	36.3
Lake Elsinore	397	316 (80)	76.9	2.3	1.7	19.1	23.8	53.1	25.6
Lake Gregory	402	347 (86)	83.8	0.6	1.5	14.1	16.2	48.5	36.3
Lancaster	350	266 (76)	70.4	5.8	2.5	21.3	26.8	45.7	29.4
Lompoc	410	305 (74)	72.5	8.7	0.8	18.0	19.3	50.0	32.6
Long Beach	414	325 (79)	37.9	16.1	21.8	24.2	22.3	47.9	31.1
Mira Loma	438	308 (70)	66.8	1.1	1.4	30.7	34.0	46.3	29.4
Riverside	469	369 (79)	43.9	14.0	6.4	35.7	38.4	47.1	21.4
San Dimas	397	303 (76)	61.6	5.8	8.8	23.8	29.7	47.8	34.3
Santa Maria	371	300 (81)	46.3	1.6	2.8	49.3	60.1	48.1	12.9
Upland	428	279 (65)	69.4	2.6	8.7	19.3	16.7	49.6	65.6
Total	4,843	3,676 (76)	66.1%	5.1%	4.9%	23.9%	26.0%	47.9%	32.2%

across communities) lived in single-family houses. Slightly more than half of the children were female.

Housing and Personal Characteristics

Housing characteristics of potential health and/or exposure relevance were evaluated by community. House pets were common, but prevalence rates varied appreciably between communities, from 52.6% in Santa Maria to 91.6% in Atascadero. A large majority of homes had gas stoves, except in Alpine. A large majority had air conditioning, except in the two communities with mild coastal climates (Santa Maria and Lompoc) and the one with a cool mountain climate (Lake Gregory). A large majority of subjects' bedrooms were carpeted in all communities. Environmental tobacco smoke (ETS) exposure varied between 14.7 to 31.7% in the households, being least common in Atascadero and most common in Lake Elsinore.

Health Effects

Table 4 provides estimates of prevalence by age and sex, controlled for community. Asthma prevalence increased by grade in females but not in males. Males had higher rates of asthma than did females. Based on the assumption that migration patterns could be important, we divided the populations into lifetime residents and others. There were few differences in the prevalence rates based on this division. Table 5 presents the multivariate relationships between risk of respiratory morbidity and potentially relevant personal or residential characteristics. The risk of physician-diagnosed asthma (ever asthma) was higher for males, blacks, children of parents with asthma, those with insurance, and those living in houses with smokers or water damage. Risks were lower for Asians and those living in homes with plants. Patterns for current asthma were similar, but pests

TABLE 4

PREVALENCE OF RESPIRATORY SYMPTOMS BY SCHOOL GRADE AND SEX (ALL COMMUNITIES)

Grade	Medically Diagnosed Asthma	Current/ Severe Asthma	Wheeze	Bronchitis	Cough	Pneumonia
Boys						
4	17.3	9.4	25.4	14.4	7.7	1.8
7	16.7	12.4	21.8	13.6	6.1	2.0
10	16.0	8.2	20.6	10.9	7.6	1.2
p Value*	0.877	0.120	0.235	0.180	0.594	NC
Girls						
4	9.4	5.2	21.6	11.0	5.4	2.1
7	12.7	7.2	21.9	11.8	7.9	1.4
10	15.9	7.4	26.0	15.3	7.8	1.5
p Value*	0.002	0.153	0.330	0.087	0.118	0.440
Both sexes						
4	13.4	7.3	23.4	12.8	6.5	2.0
7	14.5	9.5	21.9	12.7	7.1	1.6
10	15.8	7.7	23.4	13.1	7.7	1.3
p Value*	0.183	0.131	0.679	0.941	0.566	0.409
p Value†	0.000	0.000	0.746	0.364	0.502	0.921

* p Value for the effect of grade controlling for community.
† p Value for the effect of sex controlling for community.

TABLE 5

ODDS RATIOS FOR HOUSING AND PERSONAL CHARACTERISTICS ASSOCIATED WITH RESPIRATORY MORBIDITY

	Ever Asthma	Current Asthma	Bronchitis	Cough	Wheeze
Black	1.60*	1.50*	0.79*	1.37*	0.82*
Asian	0.77*	0.36*	0.46*	1.07*	0.33†
Hispanic	1.14	1.10	0.87	1.26	1.10
Mixed/other race	1.08*	1.06*	0.60†	0.86*	0.92*
Height (per inch)	1.01	1.02	1.01	1.03†	1.00
BMI (lb/in^2)	1.13	0.95	1.26*	1.17	1.10
Asthma, mother	3.48*	4.10†	1.97†	1.19	1.59†
Asthma, father	3.49†	3.55†	1.63†	1.44	1.53†
Hay fever	2.08†	2.51†	2.23†	2.51†	2.34†
Insurance	1.77†	1.91†	1.75†	0.86	1.33
Plants	0.61†	0.46†	0.96	0.80	1.10
Water damage	1.33*	1.27	1.26	1.38*	1.15
Mildew	1.19	1.17	1.34†	1.45†	1.94†
Passive smoke	1.37†	1.30	1.12	1.23	1.23
Pests	1.06	1.46*	0.95	1.04	1.32
Roaches	0.90	0.80	1.70†	1.26	1.16
Carpet	0.78	0.58	0.65†	0.85	1.07
Vitamins	1.13	1.03	1.08	1.58†	1.17
Active smoker	0.93	0.70	1.07	1.82†	1.22
Pets	1.08	1.02	1.00	0.80	1.24
Gas stove	1.26	1.32	1.10	1.29	1.19
Air conditioning	1.05	1.24	0.72	1.07	1.16
More outdoors	0.95	1.04	1.17	0.85	1.01

* $p < 0.15$.
† $p < 0.05$.
Items with asterisks and daggers are included in the final adjustment model for this measurement. These items are adjusted for each other; remaining variables are adjusted only for the footnoted items, as well as for sex, age, grade in school, and community. Baseline race is white.

were associated with risks. Those spending more time outdoors did not have significantly higher risks for respiratory conditions.

Bronchitis risk was higher in males, in those with parents with asthma, and those with hay fever, household mildew, and health insurance. Risks were lower for blacks, Asians, and those living in bedrooms with carpet. There was a similar pattern for cough, with active smoking becoming a significant risk factor. Wheeze was associated with having asthmatic parents, hay fever, and household mildew.

The overall adjusted prevalence of specific chest conditions reported on questionnaires was analyzed by community. Prevalence rates varied considerably between communities, with no obvious relationship to community air quality. In particular, Atascadero, a community with good air quality, had the highest rates of asthma and bronchitis, more than double the lowest rates. The lowest rates for asthma occurred in Mira Loma, a community with poor air quality.

To pursue potential air quality/respiratory health relationships further, we performed ecologic regression analyses on the community intercept terms (log odds ratios) from the individual level models in Table 5. Table 6 includes the results based on the 1986–1990 exposure data. Prevalence of wheeze was associated with exposure to NO_2 (odds ratio [OR] = 1.48; 95% confidence interval [CI], 1.08–2.02) and acid (OR = 1.55; 95% CI, 1.09–2.21) in males only. In Table 7 we present a similar analysis based on the 1994 exposure measurements. Again we see a positive association of NO_2 (OR = 1.54; 95% CI, 1.08–2.19) and acid (OR = 1.45; 95% CI, 1.14–1.83) with wheeze in boys only. Peak ozone exposure was associated with decreased prevalence of doctor-diagnosed asthma in females only.

DISCUSSION

Effects of Household and Personal Characteristics

We found that certain residential, demographic, and family characteristics were associated with higher respiratory disease prevalence in Southern California schoolchildren, independent of outdoor air quality. Risk factors included being male, African American or American Indian ethnicity, asthmatic parents, personal history of hay fever, house pets, household pests, dampness in the home, and smokers in the home. Associations of respiratory disease with high family income, health insurance, or taking vitamins suggest that the relevant conditions are under-diagnosed in subjects of lower socioeconomic status, that such subjects under-report their respiratory problems on questionnaires, and/or that parents acquire insurance or administer vitamins in response to children's respiratory illness.

Most of the risk factors identified in our children have been found in previous questionnaire assessments of children living in different climates (usually colder) and exposed to different air pollution (usually with more sulfur compounds, but otherwise less severe than in our most polluted communities). In the 24 Cities Study, a significantly increased risk of wheeze was found to be related to parental smoking (21), but (in contrast to our results) no significant association of parental smoking with asthma was observed. The earlier Six Cities Study also showed lower respiratory symptoms and illnesses related to parental smoking (22, 23). It showed no significant effect of gas stove usage (22), in agreement with our findings. Our study is not ideal for addressing the gas stove issue, due to our small proportion of subjects without gas stoves. The Six Cities Study also showed a significant effect of home dampness on respiratory symptoms (24, 25). Similar effects of dampness (or more specifically, of mold- and mildew-derived allergens) have been observed in a variety of climates, from subtropical (26) to very cold (27). Animal-derived allergens, whether from pets or insect pests, likewise have been implicated in children's respiratory morbidity by multiple studies (28, 29). Concerning race and sex differences, Gold and colleagues (30), studying over 9,000 children in four cities, found more asthma and lower respiratory symptoms in black than in

TABLE 6

ODDS RATIOS OF RESPIRATORY ILLNESS ON 1986–1990 AMBIENT AIR POLLUTANTS*

		All Subjects		Males		Females	
Symptom	Pollutant†	OR	(CI)	OR	(CI)	OR	(CI)
Ever asthma	Peak O_3	0.93	(0.72, 1.21)	1.15	(0.91, 1.44)	0.72	(0.50, 1.04)
	PM_{10}	0.93	(0.76, 1.13)	1.00	(0.82, 1.21)	0.85	(0.62, 1.17)
	NO_2	0.95	(0.72, 1.26)	0.94	(0.72, 1.22)	0.99	(0.62, 1.57)
	Acid	1.00	(0.72, 1.38)	1.03	(0.76, 1.40)	0.99	(0.58, 1.69)
Current asthma	Peak O_3	0.95	(0.70, 1.29)	1.02	(0.75, 1.39)	0.84	(0.50, 1.40)
	PM_{10}	1.09	(0.86, 1.37)	1.09	(0.87, 1.37)	1.03	(0.69, 1.55)
	NO_2	1.14	(0.83, 1.56)	1.16	(0.85, 1.58)	1.03	(0.59, 1.80)
	Acid	1.02	(0.70, 1.49)	1.01	(0.69, 1.47)	1.04	(0.54, 1.98)
Bronchitis	Peak O_3	1.14	(0.84, 1.55)	1.15	(0.85, 1.53)	1.23	(0.82, 1.83)
	PM_{10}	0.94	(0.74, 1.19)	0.95	(0.75, 1.21)	0.93	(0.67, 1.29)
	NO_2	0.85	(0.62, 1.17)	0.84	(0.62, 1.13)	0.87	(0.56, 1.36)
	Acid	1.16	(0.79, 1.68)	1.06	(0.73, 1.54)	1.31	(0.81, 2.12)
Cough	Peak O_3	0.98	(0.82, 1.17)	0.93	(0.66, 1.33)	1.03	(0.71, 1.48)
	PM_{10}	1.06	(0.93, 1.21)	0.98	(0.74, 1.29)	1.16	(0.89, 1.51)
	NO_2	1.14	(0.96, 1.34)	1.25	(0.88, 1.76)	1.05	(0.71, 1.55)
	Acid	1.13	(0.92, 1.38)	1.05	(0.68, 1.63)	1.14	(0.73, 1.76)
Wheeze	Peak O_3	1.08	(0.87, 1.35)	1.30	(0.89, 1.89)	0.95	(0.67, 1.35)
	PM_{10}	1.05	(0.89, 1.25)	1.26	(0.96, 1.66)	0.91	(0.70, 1.19)
	NO_2	1.09	(0.86, 1.37)	1.47	(1.04, 2.09)‡	0.85	(0.59, 1.21)
	Acid	1.26	(1.01, 1.57)‡	1.55	(1.03, 2.32)‡	1.08	(0.71, 1.66)

* Single pollutant models adjusted for personal and environmental factors. Models for all subjects are adjusted for the variables with asterisks and daggers in Table 5.

† Odds ratios are scaled to the interquartile range for each pollutant as follows: 40 ppb of O_3, 25 $\mu g/m^3$ of PM_{10}, 15 $\mu g/m^3$ of $PM_{2.5}$, 25 ppb of NO_2, and 1.7 ppb of acid ($HCl+HNO_3$, measured on a mole basis). Four models are fit for each symptom or condition, one for each pollutant.

‡ $p < 0.05$.

TABLE 7

ODDS RATIOS OF RESPIRATORY ILLNESS ON 1994 AMBIENT AIR POLLUTANTS*

		All Subjects		Males		Females	
Symptom	Pollutant†	OR	(CI)	OR	(CI)	OR	(CI)
Ever asthma	Peak O_3	0.86	(0.61, 1.21)	1.18	(0.85, 1.62)	0.58	(0.37, 0.91)‡
	PM_{10}	0.87	(0.67, 1.14)	0.92	(0.71, 1.19)	0.80	(0.52, 1.25)
	NO_2	0.98	(0.71, 1.34)	0.98	(0.72, 1.32)	0.99	(0.58, 1.67)
	$PM_{2.5}$	0.79	(0.48, 1.29)	0.94	(0.58, 1.54)	0.63	(0.29, 1.40)
	Acid	0.95	(0.75, 1.19)	1.04	(0.84, 1.29)	0.87	(0.60, 1.25)
Current asthma	Peak O_3	0.84	(0.56, 1.26)	0.89	(0.59, 1.34)	0.77	(0.38, 1.54)
	PM_{10}	1.11	(0.81, 1.54)	1.12	(0.81, 1.55)	1.00	(0.57, 1.77)
	NO_2	1.21	(0.85, 1.71)	1.25	(0.90, 1.75)	1.07	(0.57, 2.02)
	$PM_{2.5}$	1.15	(0.63, 2.09)	1.27	(0.71, 2.27)	0.86	(0.31, 2.42)
	Acid	1.02	(0.78, 1.34)	1.03	(0.79, 1.35)	0.96	(0.61, 1.53)
Bronchitis	Peak O_3	1.22	(0.81, 1.84)	1.19	(0.80, 1.78)	1.37	(0.80, 2.33)
	PM_{10}	0.90	(0.65, 1.26)	0.91	(0.66, 1.26)	0.91	(0.58, 1.43)
	NO_2	0.88	(0.61, 1.28)	0.87	(0.61, 1.24)	0.91	(0.55, 1.52)
	$PM_{2.5}$	0.80	(0.44, 1.45)	0.83	(0.46, 1.50)	0.78	(0.35, 1.76)
	Acid	1.01	(0.77, 1.34)	0.98	(0.75, 1.29)	1.08	(0.75, 1.56)
Cough	Peak O_3	0.93	(0.73, 1.18)	0.89	(0.55, 1.44)	0.98	(0.60, 1.62)
	PM_{10}	1.14	(0.96, 1.35)	0.95	(0.65, 1.39)	1.33	(0.95, 1.87)
	NO_2	1.14	(0.94, 1.39)	1.20	(0.79, 1.80)	1.11	(0.72, 1.71)
	$PM_{2.5}$	1.17	(0.83, 1.63)	1.03	(0.51, 2.07)	1.37	(0.69, 2.72)
	Acid	1.05	(0.90, 1.23)	1.14	(0.85, 1.54)	1.01	(0.73, 1.39)
Wheeze	Peak O_3	1.10	(0.81, 1.49)	1.26	(0.73, 2.17)	1.02	(0.63, 1.65)
	PM_{10}	1.01	(0.79, 1.29)	1.25	(0.82, 1.90)	0.83	(0.58, 1.19)
	NO_2	1.12	(0.86, 1.45)	1.54	(1.04, 2.29)‡	0.86	(0.57, 1.29)
	$PM_{2.5}$	1.03	(0.66, 1.61)	1.62	(0.77, 3.40)	0.71	(0.37, 1.36)
	Acid	1.13	(0.94, 1.35)	1.44	(1.12, 1.87)§	0.93	(0.69, 1.26)

* Single pollutant models adjusted for personal and environmental factors. Models for all subjects are adjusted for the variables with asterisks and daggers in Table 5.

† Odds ratios (ORs) are scaled to the interquartile range for each pollutant as follows: 40 ppb of O_3, 25 $\mu g/m^3$ of PM_{10}, 15 $\mu g/m^3$ of $PM_{2.5}$, 25 ppb of NO_2, and 1.7 ppb of acid ($HCl+HNO_3$, measured on a mole basis). Five models are fit for each symptom or condition, one for each pollutant.

‡ $p < 0.05$.

§ $P < 0.01$.

white children, independent of socioeconomic status, and more in boys than in girls. Maternal smoking and family history of respiratory illness were also significant risk factors. In contrast to our results, they found that air conditioner use predicted respiratory illness.

The overall consistency between our results and previous findings suggests that most or all of the known personal and household risk factors operate over a considerable range of population and environmental characteristics. On the basis of this evidence, a broad-based campaign to abate airborne hazards inside homes, including smoke, animal-derived allergens, and allergens from dampness-dependent organisms, would be an effective intervention on behalf of children's respiratory health.

Effects of Outdoor Air Pollution

We found some associations between outdoor air pollution exposure and prevalence of respiratory diseases, when we defined exposure over five fairly recent years, or by one year's very recent air quality data collected specifically to relate to our health data. The statistically significant association of wheeze prevalence with acid and NO_2 in boys might be important, but since a large number of comparisons were made, this association could be entirely compatible with chance.

Neas and coworkers (31) have reported the association between indoor NO_2 concentrations and symptoms, including wheeze. This contrasts with the findings of Dijkstra and colleagues (32). While Braun-Fahrlander and colleagues (33) found no relationship between indoor and outdoor NO_2 levels and incidence of symptoms, outdoor NO_2 was associated with longer duration of symptoms. This is compatible with our findings in which prevalence of wheeze was associated with outdoor NO_2. Results from the 24 Cities Study (34) showed that bronchitis symptoms were associated with particulate exposure (specifically the strong acid component). Prevalence rates were not broken down by sex. No significant excess morbidity was reported on questionnaires in polluted Canadian communities (35). In our study, NO_2 was highly correlated with acid vapor ($r = 0.83$). One recent publication shows air pollution effects being more common for girls than boys (36).

The overall results concerning outdoor air pollution show no consistent or large excesses of morbidity in subjects who lived in the most polluted communities and/or had the highest estimated exposures. This might indicate: (*1*) little effect of even the most severe outdoor pollution; (*2*) an increase of uncontrolled risk factors in cleaner communities, offsetting any reduced risk from pollution; (*3*) our inability to detect important effects, because of exposure misclassification, inadequate sensitivity of health measures, or bias in diagnostic practices between communities; or (*4*) effects of self-selection of place of residence inherent in cross-sectional comparisons. Other possible risk factors notable in the cleanest communities included relatively cool summers in Lompoc and Santa Maria, and relatively cold winters in Atascadero, as well as farmlands and grasslands, increasing the potential for airborne allergen or pesticide exposure near all three communities. However, each of those potential risks was present in one or more polluted communities also. Thus, a "tradeoff" of effects between other risk factors and air pollution seems unlikely, although it cannot be ruled out. We sought to minimize exposure misclassification through an intensive outdoor air monitoring effort. However, individuals' exposures indoors (where typical Cali-

fornia children spend 85% or more of their time [37]) may not have reflected outdoor conditions, and individuals' outdoor exposures should have varied with their time–activity patterns, which we could document only crudely. Thus, we cannot rule out the possibility that either indoor exposures or outdoor activity varied systematically with outdoor air quality. If so, we might have misclassified many individuals' exposures, and thereby failed to detect more real effects of outdoor pollution.

It is possible that perceptions of air pollution or health sensitivities influence some subjects' choices of place of residence in ways that could produce spurious associations when a population is examined cross-sectionally. For example, the high rate of asthma in Atascadero (one of our less polluted communities) theoretically could be influenced by families with respiratory conditions seeking cleaner communities, although this does not seem to be a likely explanation in this study. The longitudinal follow-up of this population should provide more accurate assessment of the true relationships between air pollution and respiratory disease.

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