

# Respiratory Effects of Relocating to Areas of Differing Air Pollution Levels

EDWARD L. AVOL, W. JAMES GAUDERMAN, SYLVIA M. TAN, STEPHANIE J. LONDON, and JOHN M. PETERS

Department of Preventive Medicine, University of Southern California Keck School of Medicine, Los Angeles, California; and National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina

We studied 110 children (59 boys and 51 girls, who were 10 yr of age at enrollment and 15 yr of age at follow-up) who had moved from communities participating in a 10-yr prospective study of respiratory health (The Children's Health Study [CHS]) to determine whether changes in air quality caused by relocation were associated with changes in annual lung function growth rates. The subjects were given health questionnaires and underwent spirometry in their homes across six western states, according to a protocol identical to evaluations performed annually on the CHS cohort in school. Changes in annual average exposure to particulate matter with a mean diameter of 10  $\mu\text{m}$  ( $\text{PM}_{10}$ ) were associated with differences in annual lung function growth rates for  $\text{FEV}_1$ , maximal mid-expiratory flow, and peak expiratory flow rate. As a group, subjects who had moved to areas of lower  $\text{PM}_{10}$  showed increased growth in lung function and subjects who moved to communities with a higher  $\text{PM}_{10}$  showed decreased growth in lung function. A stronger trend was found for subjects who had migrated at least 3 yr before the follow-up visit than for those who had moved in the previous 1 to 2 yr. We conclude that changes in air pollution exposure during adolescent growth years have a measurable and potentially important effect on lung function growth and performance.

**Keywords:** air pollution; respiratory system; growth and development

The establishment of appropriate regulatory standards to protect the public health from ambient air pollution requires credible health information based on carefully performed studies. Long-term community studies tracking health outcomes in free-living populations (1, 2) offer a unique opportunity to make highly relevant health assessments in this regard. However, such studies may be complicated by practical limitations, such as loss to follow-up of portions of the study population through subject withdrawal or relocation (3). Subject withdrawal or relocation may inadvertently introduce bias through the loss of specific subgroups within the general study population, such as sensitive individuals who may perceive themselves as being at greater increased risk if they remain in the study area. Moreover, subjects who relocate from one region to another can experience appreciable changes in ambient pollutant exposure

by moving from an area of higher pollution to one characterized by lower ambient pollutant levels or vice versa.

In 1993, the Children's Health Study (CHS) was initiated to assess the potential chronic respiratory effects of ambient air pollution in children living in Southern California (4). Associations between air pollution and various health-related outcomes have been previously reported by several investigators (5–16). Our group recently reported that children aged 10 to 14 yr and living in areas of higher ambient air pollution (areas with higher levels of particulate matter with a mean diameter of 10  $\mu\text{m}$  [ $\text{PM}_{10}$ ],  $\text{NO}_2$ , and acids) have measurably slower annual rates of lung function growth, as measured by mean annual changes in  $\text{FEV}_1$ , forced expiratory flow at 75% of FVC ( $\text{FEF}_{75}$ ), or maximal midexpiratory flow (MMEF) (17).

Adolescence is a time of rapid lung function growth for both males and females, with age-related increases in lung function leveling off by the late teen years for females and by the early twenties for males (18). On the basis of our previous findings relating annual lung function growth rates to air pollution exposure during adolescence, we wanted to determine whether changes in pollution exposure during this period of rapid lung growth might affect rates of growth. If ambient air pollution was demonstrably affecting longitudinal lung function growth rates, we speculated that residential relocation (along with an increase or decrease in ambient air pollution exposure) might be reflected in measurable changes in lung function performance.

## METHODS

Subjects were drawn from a 10-yr longitudinal respiratory health study of children living in Southern California (4, 5), who enrolled in 1993 at the age of 10 yr or in 1994 at the age of 11 yr. Subject participation was approved by the University of Southern California Institutional Review Board for Human Studies, after receipt of written informed consent from subjects and their parents or guardians. To participate, subjects had to have been CHS participants, to have had one or more years of acceptable CHS lung function data, and to have moved away from the CHS communities at least 1 yr before follow-up. Ambient pollution data had to have been obtainable for the subject's current residential location at the time of the follow-up study. To control costs, subjects had to have moved no farther away than California, Arizona, Nevada, Oregon, Washington, or Utah. On the basis of these criteria, 164 subjects were eligible for study. Of these, 149 subjects responded to phone or mail contact, 15 declined to participate, and 110 subjects were tested.

Subjects were assigned pollution scores on the basis of annual average 24-h  $\text{NO}_2$ , daily average  $\text{PM}_{10}$  mass, and average daytime (10 A.M. to 6 P.M.)  $\text{O}_3$  levels in their current and former communities. Annual averages for former communities of residence were based on 1994 data, whereas averages for current communities of residence were represented by 1998 values. Differences between current and former community  $\text{O}_3$ ,  $\text{NO}_2$ , and  $\text{PM}_{10}$  levels were calculated and used in pollutant-specific analyses representing subject-specific changes in ambient exposure. Negative pollution scores reflected moves to areas of lower pollution (i.e., the current community of residence had lower ambient levels of  $\text{O}_3$ ,  $\text{NO}_2$ , or  $\text{PM}_{10}$  than did the former community). Similarly, positive pollution scores represented moves to communities with higher air pollution levels.

(Received in original form February 5, 2001, accepted in final form September 27, 2001)

Supported by U.S. Environmental Protection Agency Cooperative Agreement CR824034, by contract CARB #94-331 from the California Air Resources Board, by National Institute of Environmental Health Sciences Center Grant #5 P30 ES07048, and by the Hastings Foundation.

Although the research described in this article was funded wholly or in part by the U.S. Environmental Protection Agency, through Cooperative Agreement CR824034 to the University of Southern California, it has not been subjected to review by that agency, and therefore does not necessarily reflect the views of the agency, and no official endorsement by the agency should be inferred from its support of this work.

Correspondence and requests for reprints should be addressed to Edward L. Avol, Department of Preventive Medicine, University of Southern California Keck School of Medicine, 1540 Alcazar Street, Suite 236, Los Angeles, CA 90033. E-mail: avol@hsc.usc.edu

Am J Respir Crit Care Med Vol 164, pp 2067–2072, 2001

DOI: 10.1164/rccm.2102005

Internet address: www.atsjournals.org

Study participation involved completing a written questionnaire, responding to a computerized health interview, and performing maximal-effort spirometry. Spirometry was accomplished with 12-L rolling seal spirometers (Model 232; Morgan Instruments, Andover, MA) interfaced to laptop computers. Subjects performed up to seven maximal exhalations from a seated position to provide three acceptable and consistent maneuvers. From these, the largest maneuver was used to determine FEV<sub>1</sub> in milliliters, FVC in milliliters, MMEF in milliliters per second, and peak expiratory flow rate (PEFR) in milliliters per second. Testing was preceded and followed by flow calibrations done with a 3-L volumetric syringe (Model FVC3000; Jones Instruments, Oakbrook, IL). This testing approach has been previously described and is being used in the CHS longitudinal study (5).

Testing was performed between January and June 1998, to parallel annual CHS testing. Testing of children who moved was typically accomplished on weekend mornings in their respective homes, whereas CHS testing was performed on weekday mornings in neighborhood schools. Testing involved 110 subjects who moved, consisting of 59 boys (age: 10.2 ± 0.5 [mean ± SD] yr at baseline and 15.1 ± 0.4 yr at follow-up) and 51 girls (age: 9.9 ± 0.4 yr at baseline and 14.9 ± 0.4 yr at follow-up).

Annual average changes in lung function were individually determined by subtracting subjects' baseline values from their follow-up values and dividing by the difference in age at testing. Linear regression was used to determine whether annual average changes in lung function correlated with average changes in pollution. Models included adjustments for sex, race, CHS entry year, annual average change in height, weight and body mass index (BMI), and the interaction of sex with annual average change in height. Hypothesis tests were performed at the p = 0.05 level, assuming a two-sided alternative hypothesis.

**RESULTS**

A summary of 1994 average air pollution levels for PM<sub>10</sub>, O<sub>3</sub>, and NO<sub>2</sub> in the 12 CHS communities appears in Table 1. Annual PM<sub>10</sub> levels varied from 15 µg/m<sup>3</sup> in Lompoc to more than 66 µg/m<sup>3</sup> in Mira Loma. Annual daytime (10 A.M. to 6 P.M.) ozone levels varied from 30 ppb in Santa Maria to 71 ppb in Lake Gregory. Daily 24-h average NO<sub>2</sub> levels ranged from 5 ppb in Lompoc to 43 ppb in Upland. Also listed in Table 1 are the numbers of subjects from each community that participated in the follow-up study, and their mean change in pollutant score.

As expected, children who originated in high-pollution communities tended to move to lower-pollution communities, giving them on average a negative pollution score. This underscores the observation that air quality in several Southern California communities has historically been poorer than in many other residential locations throughout the western United States. Conversely, there was a subset of communities (Atascadero, Santa Maria, and Lompoc) from which children who moved generally experienced a positive change in pollution score (i.e., they moved to an area of higher pollution).

As shown in Figure 1, increasing exposure to PM<sub>10</sub> was associated with decreased rates of annual growth in MMEF (p = 0.04), and PEFR (p = 0.007), and with marginally decreased rates of annual growth in FEV<sub>1</sub> (p = 0.06). For each increase of 10 µg/m<sup>3</sup> in the annual average 24-h PM<sub>10</sub>, annual lung function growth was estimated to decrease by 6.6 ml for FEV<sub>1</sub>, 16.6 ml/s for MMEF, and 34.9 ml/s for PEFR. These effect estimates, as well as those for NO<sub>2</sub> and O<sub>3</sub>, are shown in Table 2. Although increases in NO<sub>2</sub> and O<sub>3</sub> were also estimated to reduce lung function growth rates, none of these effects was statistically significant at the 5% level.

We then assessed the importance of community of origin in predicting changes in annual lung function growth. Based on PM<sub>10</sub> data collected during the early years of the study (and summarized in Table 1), we divided the 12 originating communities into the tertiles of "high," "medium," and "low." Changes in annual average lung function growth across these strata were compared with changes in PM<sub>10</sub> levels between former and current communities of residence. For subjects who moved from communities of high or low PM<sub>10</sub>, changes in PM<sub>10</sub> levels between the original and current communities of residence (in either a positive or negative direction) were reflected in statistically significant changes in rates of annual growth in MMEF (Figure 2). Most subjects originating in communities of medium PM<sub>10</sub> experienced modest changes in PM<sub>10</sub>, and for these subjects no significant association was detected between annual lung function growth and change in PM<sub>10</sub> levels. Similar associations were observed for PEFR and FEV<sub>1</sub>. We also explored the potential association between annual lung function growth rate

**TABLE 1. SUMMARY OF AMBIENT PM<sub>10</sub>, O<sub>3</sub>, NO<sub>2</sub> CONCENTRATIONS FOR SUBJECT BASELINE COMMUNITIES AND RELATIVE CHANGE IN POLLUTANTS AS A RESULT OF SUBJECT RELOCATION**

	Subject's Originating Children's Health Study Community											
	ML	UPL	RIV	LB	SD	LEL	LAN	ALP	LG	ATA	SM	LOM
No. of subjects	12	8	14	7	10	10	6	10	11	6	7	9
Baseline Levels*												
PM <sub>10</sub> , µg/m <sup>3</sup>	66.2	46.0	43.4	38.0	36.6	34.6	28.7	23.9	21.9	21.2	19.8	15.0
NO <sub>2</sub> , ppb	25.7	43.2	28.7	35.6	39.8	19.8	19.2	12.9	7.2	13.4	11.2	4.6
O <sub>3</sub> , ppb	56.7	54.0	63.0	30.7	60.8	58.9	47.3	58.1	70.8	42.2	30.4	41.0
Change in Pollutant Score†												
PM <sub>10</sub>												
Mean	-32.9	-9.7	-8.7	-0.5	-2.4	2.1	0.5	11.0	8.6	11.4	9.5	13.4
Range, min	-48.9	-17.0	-21.5	-2.2	-15.8	-2.9	-8.2	-4.0	-4.9	-1.5	1.0	5.7
Range, max	-21.1	5.5	20.5	5.0	21.8	14.7	10.3	28.1	32.4	27.0	28.4	37.5
NO <sub>2</sub>												
Mean	-1.3	-17.4	-1.6	-0.3	-14.1	7.7	1.8	9.2	16.5	4.4	7.6	13.9
Range, min	-10.6	-28.5	-13.6	-12.7	-28.2	0.6	-7.6	-1.3	8.0	-5.5	-4.1	5.4
Range, max	19.5	-0.2	19.3	4.6	3.7	23.9	11.5	20.5	32.8	13.8	32.2	25.1
O <sub>3</sub>												
Mean	-11.0	-7.6	-20.5	7.4	-14.5	-13.5	-3.7	-17.0	-27.0	1.8	11.7	-0.6
Range, min	-29.2	-14.4	-42.1	-0.4	-23.2	-22.1	-11.3	-30.3	-44.1	-10.8	2.5	-10.7
Range, max	2.3	-0.1	-2.1	27.8	-5.3	-5.1	5.8	-1.5	-10.0	8.9	22.7	6.8

Definition of abbreviations: ALP = Alpine; ATA = Atascadero; LAN = Lancaster; LB = Long Beach; LE = Lake Elsmore; LG = Lake Gregory; LOM = Lompoc; M = Mira Loma; RIV = Riverside; SD = San Dimas; SM = Santa Maria; UPL = Upland.

\* Mean 1994 ambient pollution levels for CHS community monitoring station: 24hrPM<sub>10</sub>, 24hrNO<sub>2</sub>, (10am to 6am) O<sub>3</sub>.

† Difference between mean 1998 ambient pollution level in community of relocation (follow-up testing) and 1994 (baseline) pollutant level.

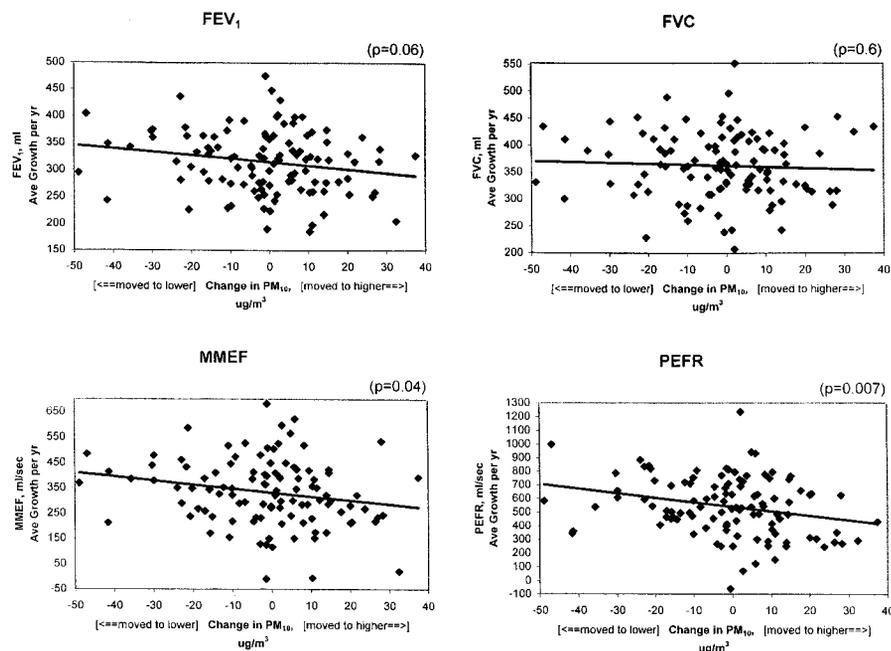


Figure 1. Effect of changes in  $PM_{10}$  on adjusted annual lung function growth for  $FEV_1$ , FVC, MMEF, and PEFR in all moved subjects studied. Annual lung function growth rates have been adjusted for sex, race, entry year into the CHS, annual average changes in height, weight, and BMI, and the interaction of sex with change in height.

and time elapsed since moving. To perform this analysis, we partitioned the study population into two groups, consisting of: (1) those who had moved away from their former communities within the past 1 or 2 yr, and (2) those who had moved away from their former communities at least 3 to 5 yr previously. A trend toward an increasing  $PM_{10}$  effect with increasing time away from the former community of residence was suggested for all lung function measures, but observed differences between the two groups were not statistically significant.

We compared several baseline medical and residential history characteristics of the 110 subjects who constituted the movers group in the study with those of their CHS peers who remained in the CHS communities and continued their study participation ("stayers"). As summarized in Table 3, there were no marked differences between the two groups in anthropomorphic, health history, or home exposure variables. There was a significant difference in the distribution of race between movers and stayers ( $p = 0.03$ ), with a higher percent-

age of Hispanic subjects in the stayers group and a higher percentage of non-Hispanic white subjects in the movers group. In general, however, these data suggest that movers were not a biased subset of the CHS participants.

We also compared the growth rates of movers with those of stayers. In this analysis, we focused on the relationship between growth in MMEF and change in  $PM_{10}$ . The changes in  $PM_{10}$  for stayers, computed as the difference between 1994 and 1998 levels in their respective communities, were small, ranging from  $-13.5 \mu\text{g}/\text{m}^3$  (Upland) to  $1.3 \mu\text{g}/\text{m}^3$  (Santa Maria). The annual-average growth in MMEF for stayers was computed from pulmonary function tests obtained in 1993 and 1998, which was identical to the period used for movers. The regression analysis applied to movers was augmented to include the combined sample of movers and stayers. Given the relatively large sample size of the stayers group, this analysis essentially provides a comparison of movers and stayers as a function of their respective changes in  $PM_{10}$  levels. Separate models were fit to subjects originating in communities of low, medium, or high  $PM_{10}$ , as was done in the analysis of movers alone, shown in Figure 2. In the low  $PM_{10}$  group, each increase of  $10 \mu\text{g}/\text{m}^3$  in annual average 24-h  $PM_{10}$  was estimated to reduce annual growth in MMEF by  $54.9 \text{ ml/s}$  ( $p = 0.002$ ). This implies that stayers in a low-pollution community have greater growth in MMEF than movers to a more polluted community. In the medium  $PM_{10}$  group, the  $PM_{10}$  effect estimate was only  $1.0 \text{ ml/s}$  ( $p > 0.99$ ), indicating that growth in movers was no different than that in stayers. In the high  $PM_{10}$  group, each decrease of  $10 \mu\text{g}/\text{m}^3$  in  $PM_{10}$  was estimated to increase annual growth in MMEF by  $19.1 \text{ ml/s}$  ( $p = 0.09$ ), providing marginal evidence that moving from a community with high pollution levels to one with low pollution levels leads to improved growth in MMEF.

## DISCUSSION

The association reported here between  $PM_{10}$  and growth in lung function is consistent with results recently reported by our group for the larger cohort of subjects active in the CHS who remain in their original communities (17). That investigation found that children living in areas of higher ambient  $PM_{10}$ ,

TABLE 2. EFFECT OF CHANGES IN  $PM_{10}$ ,  $NO_2$ , or  $O_3$  ON AVERAGE ANNUAL LUNG FUNCTION GROWTH RATES

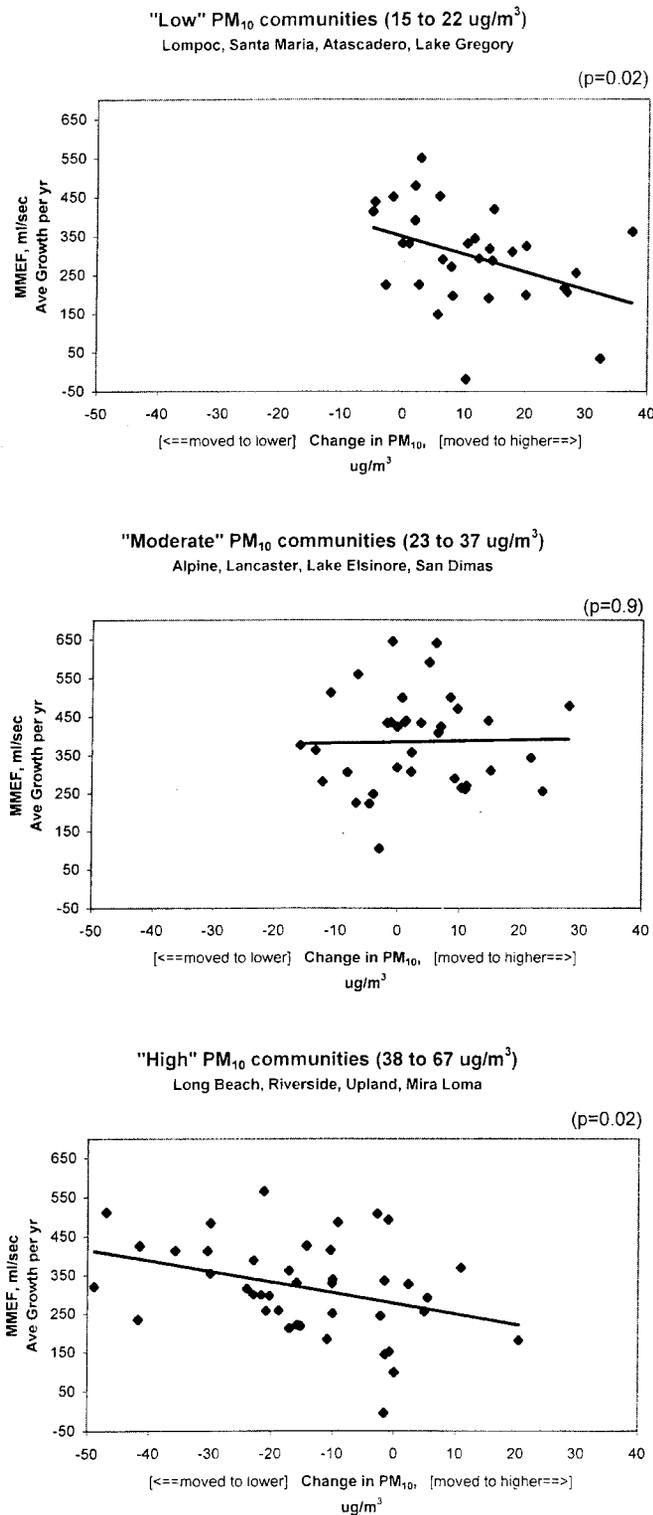
	$PM_{10}$ 24-h Average	$NO_2$ 24-h Average	$O_3$ 10 A.M. to 6 P.M. Average
FVC, ml			
Mean change <sup>1</sup>	-1.8	-2.7	-1.4
95% CI	-9.1, 5.5	-12.9, 7.5	-10.8, 8.0
$FEV_1$ , ml			
Mean change <sup>1</sup>	-6.6	-8.2	0.1
95% CI	-13.5, 0.3	-17.8, 1.4	-8.7, 8.9
MMEF, ml/s			
Mean change <sup>1</sup>	-16.6*	-10.7	-3.4
95% CI	-32.1, -1.1	-3.8, 11.4	23.6, 16.8
PEFR, ml/s			
Mean change <sup>1</sup>	-34.9†	-23.6	-8.9
95% CI	-59.8, -10.0	-59.5, 12.3	-41.6, 23.8

Definition of abbreviations: MMEF = maximal midexpiratory flow; PEFR = peak expiratory flow rate;  $PM_{10}$  = particulate matter with a mean diameter of  $10 \mu\text{m}$ .

Changes shown are per 10 units of pollutant, respectively.

\*  $p < 0.05$ .

†  $p < 0.01$ .



**Figure 2.** Effect of changes in PM<sub>10</sub> on annual lung function growth for MMEF, stratified by tertiles of PM<sub>10</sub> concentrations in subjects' baseline community of origin.

NO<sub>2</sub>, and acids had lower rates of annual lung function growth. Results from the current investigation indicate that during the teen years of development, the rate of lung function growth can be altered by a large change in exposure to air pollution.

Our observations about longer-term effects of pollutants on respiratory health in subjects moving to different areas of

**TABLE 3. SELECTED BASELINE CHARACTERISTICS OF MOVERS (n = 91) AND STAYERS (n = 1002)**

Variable	Movers*		Stayers†		p Value
	Mean	SD	Mean	SD	
Age, yr	9.9	(0.4)	9.9	(0.4)	0.7
Height, cm	139.6	(7.3)	140.2	(6.8)	0.5
Weight, kg	35.2	(8.7)	36.4	(8.4)	0.2
BMI, m/kg <sup>2</sup>	17.9	(3.1)	18.4	(3.2)	0.2
Male	n	% Total	n	% Total	
Race‡	45	49.5	481	48.0	0.8
Asian	2	2.2	61	6.1	
Black	5	5.5	42	4.2	
Hispanic	15	16.5	284	28.3	
Other	1	1.1	9	0.9	
White	68	74.7	606	60.5	
Asthma	7	7.7	131	13.1	0.2
Bronchitis	13	14.8	122	12.7	0.6
Wheeze	25	28.4	320	33.8	0.3
Hay fever	15	17.9	157	17.2	0.9
Environmental tobacco smoke	21	23.1	154	15.8	0.08
Pests	72	81.8	746	81.4	1.0
Pets	74	81.3	769	76.8	0.4
Insurance	80	87.9	814	84.2	0.4
Gas stove	72	79.1	758	78.2	0.9

Definition of abbreviation: BMI = body mass index.

\* Eligible 10-yr-old subjects enrolled in 1993 who subsequently moved.

† Eligible 10-yr-old subjects enrolled in 1993 still active in Children's Health Study in 1998.

‡ As reported by parent.

ambient air pollution are consistent with a report by Kinney and Lippmann (19). Their study assessed the respiratory health of U.S. Military Academy cadets who trained in several different regions of the United States during summer and were therefore exposed to different levels and kinds of regional air pollution. Kinney and Lippmann observed seasonal declines in respiratory function, and related the observed changes to outdoor exposure to ozone and particles.

Our current study found changes in lung function of a similar magnitude to that in our analyses of 4 yr of follow-up of subjects still actively participating in the longitudinal health study (17). In the current study, only PM<sub>10</sub> was identified as playing a statistically significant role. The importance of acids could not be assessed because of the scarcity of available data on ambient acid levels in the communities to which our subjects had moved. The results of this study, together with those of the numerous previously reported investigations of PM<sub>10</sub> and its association with increased morbidity and mortality (20–23), underscore the national concern about particulate exposure and its relation to public health.

In the present study, we could not show that the duration of the period since the subject moved to a new community was statistically associated with changes in observed rates of annual lung function growth. The data did, however, show a trend consistent with this hypothesis. A more convincing test of this hypothesis would require larger sample sizes or longer follow-up periods. Plans are currently being made to accomplish this.

The analyses reported here suggest that previously observed changes in annual lung function growth rates (17) may be reversible during the period of rapid lung growth accompanying physical development during the teen years. Differences in annual respiratory growth rates during adolescence may be important predictors of respiratory health in later adult years. Hibbert and coworkers have argued that the development of several lung function indices in healthy children follows a consistent "track," increasing at a constant rate relative to those

of other healthy children (24). Data from the Six Cities Studies (25) also support the concept of tracking, but suggest that adolescents might deviate from predicted growth curves because of variations in the onset of the adolescent growth spurt. Nevertheless, the investigators who reported these data suggested that tracking was informative and of potential clinical use. Lebowitz and colleagues (26) reported that childhood respiratory illness and smoking (either active or passive) were important factors in childhood respiratory growth, but that with the exception of active smoking, these factors had negligible effects on tracking of subjects' values over time. Conversely, Borsboom and colleagues reported that the large intraindividual variation in the timing of growth spurts and the rates of growth precluded the application during adolescence of any averaged values to the study of ventilatory function (27). In their study of Dutch schoolboys, the authors found that the large variability in age of peak growth, and the time lag between growth in height and ventilatory function, were not adequately explained in commonly used reference values.

Among respiratory pollutants, tobacco smoke has been shown to adversely affect the growth of respiratory function in children. In a study of New Zealand children 9 to 15 yr of age who were exposed to passive smoking, Sherrill and associates reported a reduction in growth of the ratio of FEV<sub>1</sub> to VC, but no significant changes in absolute FEV<sub>1</sub> or VC related to either active or passive smoking (28). In a longitudinal health investigation of children in East Boston, Tager and coworkers reported a clear decline among children 9 to 14 yr old at the time of annual examination, in the predicted percent growth for those who were smokers versus those who were nonsmokers (29). In that study, predicted FEV<sub>1</sub> decreased by 2.8% per year in children who smoked as compared with those who did not smoke. Slightly larger decreases were predicted for FEF<sub>25-75</sub>. To compare the relative effects observed in the study by Tager and colleagues with the findings in our current work, we considered a white male CHS subject of average height, weight, and BMI who was a mover. For this hypothetical individual, an annual growth rate of 287 ml/yr in FEV<sub>1</sub> and 291 ml/s in MMEF would be expected. On the basis of the results presented in Table 2, we would predict a reduction of 2.3% per year in FEV<sub>1</sub> growth and a reduction of 5.7% per year in MMEF growth for every 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> for this child. This would suggest that ambient air pollution exposure has a similar magnitude of effect on lung function development to that previously observed for children who are active smokers.

Previous studies of the etiology of respiratory disease have suggested that individuals with limited respiratory capacity are at increased risk for earlier onset of a range of respiratory maladies. The reduction in annual respiratory growth rates with increased pollution exposure observed in the present study may reduce the level of lung function attained and lead ultimately to an increased risk of respiratory events in adulthood. However, it is also possible that early deficits will be reversed with subsequent accelerations in growth rate or a longer growth period. Earlier work by Borsboom and coworkers revealed that adolescents with a history of respiratory symptoms exhibited annual lung growth on a growth curve parallel with but lower than that of asymptomatic peers (30). Burrows and colleagues have suggested that "as the twig is bent, the tree inclines" (31). Our data suggest that annual lung function growth rates can change with exposure to pollutants, but whether these changes in rates somehow compensate for slowed growth is unknown. Drawing upon data for the respiratory effects of smoking (32), one might conclude that in the absence of the exposure insult, a return to nominal (but not accelerated) rates of growth (or decline) would ensue. If this is true, then periods of slowed lung growth,

even during periods of peak respiratory growth (such as those experienced by males and females in their teen years), may have lifelong implications for health.

The results reported here support the view that changes in ambient pollution levels (in this case, PM<sub>10</sub>) may have measurable effects on longer-term lung function (and health) outcomes. The relative importance of the changes observed in the present study, and the potential health implications of these observed changes for the later lives of children, support the need for studies to confirm and extend these observations.

**Acknowledgment:** The authors thankfully acknowledge the students and families of the Children's Health Study cohort for allowing us into their homes. We thank Deborah Kim, Steve Howland, Christina Gallop, and Lupe Valencia for doing the field testing; Ed Rappaport and Jun Manila for software and hardware technical support; Bill Linn for overseeing the quality assurance of health testing; and Hita Vora and Duncan Thomas for analytical support and guidance in the study. We also acknowledge William McDonnell, who served as project officer.

## References

1. Ware JH, Ferris BG, Dockery DW, Spengler JD, Stram DO, Speizer FE. Effects of ambient sulfur oxides and suspended particles on respiratory health of preadolescent children. *Am Rev Respir Dis* 1986;133:834-842.
2. Abbey DE, Mills PK, Petersen FF, Beeson WL. Long-term ambient concentrations of total suspended particulates and oxidants as related to incidence of chronic disease in California Seventh-Day Adventists. *Environ Health Perspect* 1991;94:43-50.
3. Detels RD, Tashkin DP, Sayre JW, Rokaw SN, Coulson AH, Massey FJ, Wegman DH. The UCLA population studies of chronic obstructive pulmonary disease. *Chest* 1987;92:594-693.
4. Peters JM, Avol E, Navidi W, London SJ, Gauderman WJ, Lurmann F, Linn WS, Margolis H, Rappaport E, Gong H Jr, *et al.* A study of twelve Southern California communities with differing levels and types of air pollution: I. Prevalence of respiratory morbidity. *Am J Respir Crit Care Med* 1999;159:760-767.
5. Peters JM, Avol E, Gauderman WJ, Linn WS, Navidi W, London SJ, Margolis H, Rappaport E, Vora H, Gong H Jr, *et al.* A study of twelve Southern California communities with differing levels and types of air pollution: II. Effects on pulmonary function. *Am J Respir Crit Care Med* 1999;159:768-775.
6. Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD, Ferris BG Jr. Effects of inhalable particles on respiratory health of children. *Am Rev Respir Dis* 1989;139:587-594.
7. Pope CA III, Dockery DW, Spengler JD, Raizenne ME. Respiratory health and PM<sub>10</sub> pollution. A daily time series analysis. *Am Rev Respir Dis* 1991;144:668-674.
8. Vedral S, Petkau J, White R, Blair J. Acute effects of inhalable particles in asthmatic and nonasthmatic children. *Am J Respir Crit Care Med* 1998;157(Pt 1):1034-1043.
9. Romieu I, Meneses F, Ruiz S, Huerta J, Sierra JJ, White M, Etzell R. Effects of intermittent ozone exposure on peak expiratory flow and respiratory symptoms among asthmatic children in Mexico City. *Arch Environ Health* 1997;52:368-376.
10. McConnell R, Berhane K, Gilliland FG, London SJ, Vora H, Avol EL, Gauderman WJ, Margolis H, Lurmann F, Thomas D, *et al.* Air pollution and bronchitic symptoms in Southern California children with asthma. *Environ Health Perspect* 1999;107:757-760.
11. Gilliland FD, Berhane K, McConnell R, Gauderman WJ, Vora H, Rappaport EB, Avol E, Peters JM. Maternal smoking during pregnancy, environmental tobacco smoke exposure and childhood lung function. *Thorax* 2000;55:271-276.
12. Gilliland FD, Berhane K, Rappaport E, Thomas DC, Avol E, Gauderman J, London SJ, Margolis HG, McConnell R, Islam KT, *et al.* The effects of ambient air pollution on school absenteeism due to respiratory illnesses. *Epidemiology* 2001;12:43-54.
13. Berhane K, McConnell R, Gilliland F, Islam T, Gauderman WJ, Avol E, London SJ, Rappaport E, Margolis HG, Peters JM. Sex-specific effects of asthma on pulmonary function of children. *Am J Respir Crit Care Med* 2000;162:2097-2104.
14. Braun-Fahrlander C, Vuille JC, Sennhauser FH, Neu U, Kunzle T, Grize L, Gassner M, Minder C, Schindler C, Varonier HS, *et al.* Respiratory health and long-term exposure to air pollutants in Swiss school-children. SCARPOL Team. Swiss study on childhood allergy and re-

- spiratory symptoms with respect to air pollution, climate, and pollen. *Am J Respir Crit Care Med* 1997;155:1042-1049.
15. Jedrychowski W, Flak E. Effects of air quality on chronic respiratory symptoms adjusted for allergy among preadolescent children. *Eur Respir J* 1998;11:1312-1318.
  16. Chen PC, Lai YM, Wang JD, Yang CY, Hwang JS, Kuo HW, Huang SL, Chan CC. Adverse effect of air pollution on respiratory health of primary school children in Taiwan. *Environ Health Perspect* 1998;106:331-335.
  17. Gauderman WJ, McConnell R, Gilliland F, London S, Thomas D, Avol E, Vora H, Berhane K, Rappaport EB, Lurmann F, *et al.* Association between air pollution and lung function growth in Southern California Children. *Am J Respir Crit Care Med* 2000;162:1383-1390.
  18. Schwartz JD, Katz SA, Fegley RW, Tockman MS. Analysis of spirometric data from a national sample of healthy 6- to 24-year olds (NHANES II). *Am Rev Respir Dis* 1998;138:1405-1414.
  19. Kinney PL, Lippmann M. Respiratory effects of seasonal exposures to ozone and particles. *Arch Environ Health* 2000;55:210-216.
  20. Pope CA. Particulate pollution and health: a review of the Utah Valley experience. *J Expos Anal Environ Epidemiol* 1996;6:23-34.
  21. Schwartz J, Dockery DW, Neas LM. Is daily mortality associated specifically with fine particles? *J Air Waste Manag Assoc* 1996;46:927-939.
  22. Vedal S. Ambient particles and health: lines that divide. *J Air Waste Manag Assoc* 1997; 47:551-581.
  23. Ostro BD, Hurley S, Lipsett MJ. Air pollution and daily mortality in the Coachella Valley, California: a study of PM<sub>10</sub> dominated by coarse particles. *Environ Res* 1999;81:231-238.
  24. Hibbert ME, Hudson IL, Lanigan A, Landau LI, Phelan PD. Tracking of lung function in healthy children and adolescents. *Pediatr Pulmonol* 1990;8:172-177.
  25. Wang X, Dockery D, Wypij D, Fay ME, Ferris BG. Pulmonary function between 6 and 18 years of age. *Pediatr Pulmonol* 1993;15:75-88.
  26. Lebowitz MD, Holberg CJ, Knudson RJ, Burrows B. Longitudinal study of pulmonary function development in childhood, adolescence, and early adulthood. *Am Rev Respir Dis* 1987;136:69-75.
  27. Borsboom GJJ, Van Pelt W, Quanjer PH. Interindividual variation in pubertal growth patterns of ventilatory function, standing height, and weight. *Am J Respir Crit Care Med* 1996;153:1182-1186.
  28. Sherrill DL, Martinez FD, Lebowitz MD, Holdaway MD, Flannery EM, Herbison GP, Stanton WR, Silva PA, Sears MR. Longitudinal effects of passive smoking on pulmonary function in New Zealand children. *Am Rev Respir Dis* 1992;145:1136-1141.
  29. Tager IB, Munoz A, Rosner B, Weiss ST, Carey V, Speizer F. Effect of cigarette smoking on the pulmonary function of children and adolescents. *Am Rev Respir Dis* 1985;131:752-759.
  30. Borsboom GJJM, Van Pelt W, Quanjer PH. Pubertal growth curves of ventilatory function: relationship with childhood respiratory symptoms. *Am Rev Respir Dis* 1993;147:372-378.
  31. Burrows B, Taussig LM. As the twig is bent, the tree inclines (perhaps). *Am Rev Respir Dis* 1980;122:813-815.
  32. Fletcher CM, Peto R. The natural history of chronic airflow obstruction. *Br Med J* 1977;1:1645-1648.