

Acute Effects of Volcanic Ash from Mount Saint Helens on Lung Function in Children¹⁻⁴

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Introduction

With the massive eruption of Mt. St. Helens on May 18, 1980, about 3 cubic kilometers of mountain were blasted off the north side and about 1 cubic kilometer of pulverized magma was distributed over eastern Washington state and in a broad swath extending across the United States and around the world. Six subsequent, although much smaller, explosive eruptions during the summer of 1980 distributed volcanic ash over an even wider area of the Northwest. Ashfalls from the explosive eruptions ranged from a trace to almost 8 cm. After each ashfall, the ash was readily resuspended by traffic and the slightest gust of wind, so that the ambient levels for total suspended particulates (TSP) remained elevated throughout the summer in the affected parts of the Northwest. Because the city of Portland and its suburbs were in the ashfall area, a sizable population mass was involved.

There was immediate concern about the health effects of the ash, in particular about its ability to cause silicosis. There was no question that the ash was small enough to be respirable because early studies (1) had shown that over 90% of the ash particles by number were under 10 μ m in diameter. Analysis of the ash (1) showed that it contained about 65% silicon dioxide, but that only about 3 to 7% was in the free or crystalline form that is usually considered fibrogenic. The likelihood of silicosis developing in the general population from the ash exposure was, therefore, considered to be very remote simply because a large enough dose would not be delivered to the lungs over a long enough period of time (2).

With the concern about silicosis on the part of the general public somewhat allayed, there was still concern about the acute effects of the ash, both in persons with preexisting lung disease

SUMMARY To evaluate the acute effects of volcanic ash from Mt. St. Helens on the lung function of children, we studied 101 children 8 to 13 yr of age who were attending a 2-wk summer camp for children with diabetes mellitus in an area where about 1.2 cm of ash had fallen after the June 12, 1980, eruption. The outcome variables used were forced vital capacity, forced expiratory volume in one second, their ratio and mean transit time. Total and respirable dust levels were measured using personal sampling pumps. The children were tested on arrival and twice (early morning [A.M.] and late afternoon [P.M.]) every second or third day during the session. A within-day effect was measured by the P.M./A.M. ratio for the lung function variables; a between-day effect was measured by the change in the P.M. measurements over the 2 wk of camp. We found no strong evidence of either a within-day or a between-day effect on lung function, even in a subgroup of children who had preexisting lung disease or symptoms, despite daytime dust/ash levels that usually exceeded the Environmental Protection Agency's significant harm level for particulate matter.

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and, as a nonspecific irritant, in persons with hyperreactive airways. In a hospital surveillance system set up immediately after the May 18, 1980, eruption (3), there was a definite increase in the number of emergency room visits and admissions for asthma and bronchitis in communities with the heaviest ashfalls. There was particular concern about the effects of ash in children because they played outside and, in many instances, played in the ash, and in playing vigorously would tend to breathe by mouth, bypassing the filtering action of the nose. There was also some question as to whether children are particularly vulnerable to the effects of inhaled particulates, such as volcanic ash, because accounts of the acute health effects of ashfalls from other volcanoes (4) have specifically noted an increase in wheezy bronchitis in young children. In establishing standards for environmental pollutants, children are usually included in that portion of the population that may be at increased risk (5). This is, in part, because children will mouth breathe when playing vigorously and thereby increase the dose of a specific pollutant. Also, the frequent respiratory infections experienced during childhood may lead to an increased sensitivity or responsiveness of the respiratory tract

mucosa to particulates or to gaseous pollutants such as ozone, the oxides of nitrogen, and sulphur dioxide (6).

This study was, therefore, designed to evaluate the acute effects of inhaled volcanic ash on the lung function of children. The hypothesis being tested was that acute exposure to levels of volcanic ash in excess of existing environmental standards for TSP would result in some degree of impairment of lung function, probably airway narrowing caused by inflammation, increased mucus secretion, or bronchoconstriction. We chose children older than 8 yr of age because they would be able to perform spirometry.

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Methods

The study was carried out at a children's summer camp, located in an area of Oregon about 90 miles southwest of Mt. St. Helens, where about 1.2 cm of ash had fallen during the June 12, 1980, eruption. Vigorous clean-up of the immediate camp area greatly decreased the amount of ash on the ground. However, the ash still clung tenaciously to the trees that surrounded the camp and was thick in the field in which many of the activities took place. The slightest breeze would shake ash off the trees and resuspend the ash that was lying on the ground.

We studied 101 children attending two 2-wk summer camp sessions, the first for children 8 to 10 yr of age, the second for those 11 to 13. The camp was specifically for children with diabetes mellitus, but, apart from their diabetes, the campers were in good health. The children came from Oregon and the neighboring states. All but 6 came from areas that had no ashfall or areas in which the ash had been cleared. For 5 of these, time spent outside prior to camp had been less than 2 h a day; 4 of the 6 had worn masks while outside. The children were tested initially between 1:00 and 4:30 p.m., as they arrived at camp, after informed consent had been obtained from a parent or guardian. Subsequent testing was carried out a total of 4 days in Session 1 and 6 days in Session 2. On each follow-up day, testing was conducted between 7:30 and 9:00 a.m. and in the late afternoon or evening, usually between 4:30 and 6:00 p.m.

Testing consisted of a questionnaire and spirometry. The questionnaire used was the ATS-DLD Pediatric questionnaire (7) and was completed by a parent or guardian. For spirometry, we used the standardized methods recommended for epidemiologic studies by the ATS-DLD Epidemiology Standardization Project (7). All tests were carried out using one of two recording spirometers (Vitalograph Models 122000 and P) that conformed to the ATS recommended criteria for a reliable instrument when back extrapolation is used to determine zero time (8). Both spirometers were calibrated over a 6-L volume range at several different temperatures reflecting the ambient temperature range encountered in the study (10 to 30°C). Multiple linear regression equations using the Vitalograph reading, ambient temperature, their cross-product and squares were used to obtain true volume (ATPs) from the tracings and the measurement temperature. The inclusion of temperature in the equation was necessary because we found that the Vitalograph output, for any given ambient temperature input volume, decreased by approximately 5% for each 10°C increase in ambient temperature (data available on request). These ATPs volumes were then corrected to ATPs conditions.

As our outcome variables we used forced vital capacity (FVC), forced expiratory vol-

ume in one second (FEV_1), their ratio (FEV_1/FVC), and mean transit time, the last of which is thought to be a more sensitive indicator of small airway obstruction than the usual spirometric variables (9, 10). We report the FVC, FEV_1 , and mean transit time data as being representative of our results. Forced expiratory flow over the middle half of FVC ($FEF_{25-75\%}$) was used only for purposes of comparison with standard reference values (11). For the transit time analysis, we used the method proposed by Permutt and Menkes (9) to extrapolate each spirogram mathematically to infinite time in order to minimize the error that would be caused by early truncations of the spirogram (for example, in children with stiff chest walls or in children who fail to maintain sufficient effort during the whole of expiration). The Permutt and Menkes model assumes that the expired volume during a forced expiration comes from many lung compartments, each of which empties exponentially with a fixed time constant. The compartmental time constants are assumed to be distributed log-normally in a continuous fashion such that the distribution of these time constants (not of the transit times) can be completely represented by only 2 parameters: μ , the mean of the natural logarithms of the time constants, and σ , the standard deviation of the natural logarithms of the time constants. The mean transit time of the untruncated spirogram is, therefore, $\sigma_1(\infty) = e^{\mu + \sigma^2/2}$. The first term (e^{μ}) is the mean transit time of the median compartment. The second term ($e^{\sigma^2/2}$) is related to the degree of dispersion of the transit times.

Using an Apple II+ microcomputer and Apple Graphics Tablet (digitizer), each spirogram was divided into 100 equal volume units from zero volume to the point of truncation (usually FVC but often less than FVC because of discontinuities in the spirogram). The time to the midpoint of each volume division and the square of this time were then summed and divided by 100 to calculate the first moment (mean transit time = σ_1) and the second moment (σ_2), respectively, of the truncated spirogram. We calculated μ and σ from σ_1 , σ_2 , and the truncation time using a Fortran version of the digital computer algorithm used by Permutt and Menkes (9). The mean transit time of the untruncated spirogram, $\sigma_1(\infty)$, could then be calculated from the equation given above.

Of the 101 children attending the 2 camp sessions, 10 consistently had unacceptable spirometry results, usually because of poor cooperation or inconsistent effort, and had to be excluded from all analyses. Fifteen additional campers who only stayed for 1 wk and 12 who had incomplete data for FEV_1 and/or FVC were also excluded from the analysis. The main reasons for staying 1 wk were financial, other family commitments, or first time at camp. No children left early because of sickness. The 12 with incomplete

data had missing or technically unacceptable data on one or more test occasions. This left a total of 64 children with complete and technically acceptable data for both FVC and FEV_1 . Additional children had to be excluded from some of the transit time analyses because on at least one occasion μ and σ could not be estimated by the model we used, usually because of a discontinuous expiratory effort.

In order to define a "susceptible" subgroup within the study population who might be expected to show an exaggerated response to dust/ash, we used the questionnaire to identify those children who had frequent and/or exercise-induced wheezing, doctor-diagnosed asthma, chronic cough and/or phlegm, or a history of childhood chest infections before 3 yr of age. A total of 9 children in Session 1 and 10 in Session 2 had at least one of these characteristics and were, therefore, classified as susceptible.

Total and respirable dust levels were measured under the supervision of industrial hygienists from the National Institute for Occupational Safety and Health (NIOSH) using personal sampling pumps carried in backpacks with the end of the sampling tube clipped to the child's shirt collar. Each test day, 5 children wore pumps; 3 of the pumps contained a cyclone separator so that only dust in the respirable range (less than 10 μ m in diameter) was collected on the preweighed filter. The other 2 pumps collected dust of all sizes. Almost all of the campers carried a pump at least once. Because the camp activities were structured by groups or teams and took place in the general area of the camp, and because there was relatively little free time, this method of sampling was considered adequate. The pumps were worn for at least 8 h (from the a.m. test time to the p.m. test time), and a time-weighted average concentration (mg/m^3) of total and respirable inhaled dust was estimated from the noncontaminated filters. Nighttime dust levels were measured by placing sampling pumps in the dormitories.

We followed the usual method of exploring an occupational hazard by assessing a within-day effect and a between-day effect on lung function. The within-day effect was measured by the ratio of late afternoon to early morning test values (p.m./a.m.), using FVC, FEV_1 , and $\sigma_1(\infty)$. In the absence of any environmental factors that would affect lung function, we would expect this ratio to be approximately 1. This takes into account diurnal variation, which is usually described as having a sine wave periodicity with a peak in the early afternoon but approximately equal values at our a.m. and p.m. test times (12, 13). We would predict any dust effect to be bronchoconstrictive in nature, resulting in a fall in FEV_1 , probably a fall in FEV_1/FVC , and an increase in $\sigma_1(\infty)$. Because the children played in the dust during the day and were in a virtually ash-free environment at night, the p.m./a.m. ratio

TABLE 1
AGE, HEIGHT, AND BASELINE LUNG FUNCTION MEASUREMENTS*

| | Session 1 | | Session 2 | |
|--------------------------------|------------------|-------------------|------------------|-------------------|
| | Boys (n = 18) | Girls (n = 13) | Boys (n = 14) | Girls (n = 19) |
| Age, yr | 9.62 ± 0.18 | 9.99 ± 0.26 | 12.32 ± 0.28 | 12.19 ± 0.23 |
| Height, cm | 134.5 ± 1.3 | 137.5 ± 2.8 | 150.6 ± 2.5 | 150.3 ± 2.0 |
| FEV ₁ , % pred† | 103.6 ± 1.5 | 91.3 ± 3.0 | 96.7 ± 2.6 | 92.9 ± 1.8 |
| FVC, % pred† | 105.3 ± 2.0 | 97.3 ± 4.0 | 99.2 ± 2.8 | 97.0 ± 2.2 |
| FEV ₁ /FVC | 0.87 ± 0.01 | 0.88 ± 0.02 | 0.87 ± 0.01 | 0.90 ± 0.01 |
| PEF ₅₀₋₇₅ , % pred† | 95.5 ± 3.8 | 89.8 ± 5.1 | 94.4 ± 3.3 | 97.4 ± 4.0 |
| μ‡ | 0.90 ± 0.06 | 0.97 ± 0.09 | 0.88 ± 0.04 | 0.92 ± 0.05 |
| σ | 0.76 ± 0.07 | 0.83 ± 0.10 | 0.69 ± 0.07 | 0.51 ± 0.09 |
| α ₁ (=)§ | 0.59 ± 0.04 | 0.59 ± 0.04 | 0.55 ± 0.02 | 0.50 ± 0.02 |

Definition of abbreviations: FVC = forced vital capacity; FEV₁ = forced expiratory volume in one second; PEF₅₀₋₇₅ = forced expiratory flow over the middle half of the FVC; μ = mean of the natural logarithm of the time constants; σ = standard deviation of the natural logarithms of the time constants; α₁(=) = mean transit time of the untruncated spirogram.

* Results are mean values ± SEM.

† Predicted values calculated using formulas of Polgar and Weng (11, table 10).

‡ Sample sizes for the transit time variables (μ, σ, and α₁(=)) are 17, 13, 13, and 18.

would be less than 1 for FEV₁ and greater than 1 for α₁(=). The between-day effect was measured by the change in P.M. measurements over the 2 wk of camp.

For both the within-day and between-day measures, differences from day to day were assessed using a repeated measures analysis of variance (RM-ANOVA) adjusting for sex (14). To further adjust for individual differences in height and age, we analyzed the percent change for each child from baseline to first P.M. measurement and from first P.M. measurement to last P.M. measurement. These percent changes served as estimates of an overall ash/dust effect.

Results

The number of boys and girls with complete data at each session are listed in table 1, which gives mean ages, heights, and baseline spirometry data for each session/sex group. We included sex in our analysis of between- and within-day variability but found no significant differences between boys and girls; therefore, we have omitted sex as a factor in subsequent tables and figures.

The P.M./A.M. ratios, and their 95% confidence intervals, for FEV₁ and α₁(=) are shown in figure 1. We found, using a RM-ANOVA, that the P.M./A.M. ratios did not vary significantly from day to day for any of the lung function measurements, and that the patterns of variation that were observed were not related to day or TSP levels. To determine whether the ratios were in any way related to the ash/dust, we returned to the camp in 1981, by which time the ash had disappeared, and used the same study design and many of the same children tested in 1980; 53 of 56 children had acceptable

data. The 95% confidence intervals for the P.M./A.M. ratios for FEV₁ and α₁(=) in 1981 are shown by the dashed lines in figure 1, which serve as an ash-free comparison for the 1980 data. For both measures the 1980 values tended to be higher than those observed in 1981. Unpaired *t* tests indicated that 3 of the P.M./A.M. ratios for FEV₁ and 2 for α₁(=), were significantly greater (*p* < 0.06) than the respective ratios observed in 1981.

Analysis of the P.M. measurements over time also failed to demonstrate any evidence of a between-day effect. The mean values of FEV₁, FVC, and α₁(=) for each test day for the 2 camp sessions are given in table 2. Although there were small but significant de-

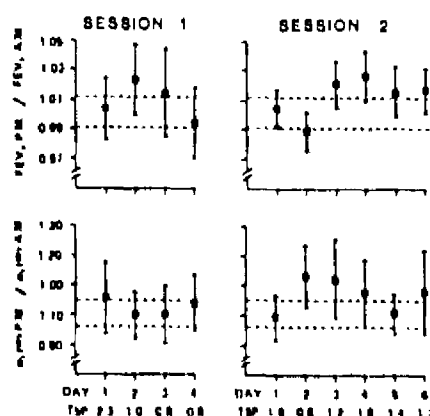


Fig 1 Daily 95% confidence intervals for P.M./A.M. ratios of FEV₁ and α₁(=) in 1980 based on the same children tested each day. Dashed lines indicate the corresponding 95% confidence intervals for a group of 53 children attending an ash-free camp in 1981. Total suspended particulate levels (μg/m³) are indicated for each observational day. For definition of abbreviations, see table 1.

creases (*p* < 0.01) in FVC and FEV₁, when expressed as a percent change, from baseline to the first P.M. measurements, there were no significant changes thereafter. In particular, there was no significant change from the first to the last P.M. measurement for any of the outcome variables. To test whether the drop from the baseline measurement was due to an ash effect or merely reflected the natural diurnal variation of the children, we used unpaired *t* tests to compare our study population with the campers we observed 1 yr later. In 1981, both FVC and FEV₁ showed a small, though not significant, increase from baseline to first P.M. measurement, with the percent changes being significantly different (*p* < 0.01) from the corresponding percent changes for the 1980 group. Similar findings were obtained using paired *t* tests on the 20 children who were present in both 1980 and 1981.

A comparison of susceptibles and nonsusceptibles for both within-day and among-day effects using a RM-ANOVA indicated significant differences for several of the response variables, although the differences were not consistent across sessions. The comparison for the FEV₁ and α₁(=) P.M. measurements is shown in figure 2. The mean FEV₁ P.M. values, corrected for height, for the susceptibles were significantly lower than those for nonsusceptibles (*p* = 0.013) in Session 1, although this trend was reversed in Session 2. An identical pattern was seen for the FVC P.M. values, corrected for height. Additionally the FEV₁/FVC P.M. values for susceptibles were consistently lower than for nonsusceptibles in both sessions, though in neither case were the differences significant. For α₁(=), the values for susceptibles were consistently greater than those for nonsusceptibles in both sessions. These differences were in the direction predicted, but were not significant. We note that the differences in the widths of the confidence intervals between susceptibles and nonsusceptibles was due partly to the smaller sample sizes in the susceptible groups, and partly to the increased variability of responses for susceptible persons. No consistent differences were observable for the P.M./A.M. ratios. Comparison of percent change from baseline to first P.M. measurement and from first to last P.M. measurement for susceptibles and nonsusceptibles gave results compar-

TABLE 2
P.M. MEASUREMENTS FOR FEV₁, FVC, AND $\alpha_1(\infty)^a$

| | Session 1 | | | Session 2 | | |
|------------|------------------|-------------|--------------------|------------------|-------------|--------------------|
| | FEV ₁ | FVC | $\alpha_1(\infty)$ | FEV ₁ | FVC | $\alpha_1(\infty)$ |
| Baseline | 1.93 ± 0.05 | 2.22 ± 0.07 | 0.59 ± 0.03 | 2.46 ± 0.08 | 2.80 ± 0.09 | 0.52 ± 0.02 |
| Test Day 1 | 1.87 ± 0.05 | 2.15 ± 0.07 | 0.60 ± 0.04 | 2.43 ± 0.07 | 2.73 ± 0.09 | 0.50 ± 0.02 |
| Test Day 2 | 1.85 ± 0.08 | 2.14 ± 0.07 | 0.62 ± 0.04 | 2.42 ± 0.08 | 2.71 ± 0.09 | 0.52 ± 0.02 |
| Test Day 3 | 1.84 ± 0.05 | 2.15 ± 0.07 | 0.58 ± 0.04 | 2.41 ± 0.08 | 2.72 ± 0.09 | 0.53 ± 0.03 |
| Test Day 4 | 1.83 ± 0.05 | 2.13 ± 0.07 | 0.60 ± 0.05 | 2.47 ± 0.08 | 2.76 ± 0.09 | 0.53 ± 0.03 |
| Test Day 5 | | | | 2.46 ± 0.08 | 2.57 ± 0.09 | 0.50 ± 0.02 |
| Test Day 6 | | | | 2.44 ± 0.07 | 2.73 ± 0.09 | 0.53 ± 0.03 |

^aFor definitions of abbreviations, see table 1.

^bBased on 31 children in Session 1 and 33 in Session 2 with consistently acceptable FVC and FEV₁ data, and 26 children in Session 1 and 28 in Session 2 with consistently acceptable transit time data. Results are mean values ± SEM.

able to those observed for the study population as a whole. In particular, none of the changes from first to last P.M. measurement was significantly different from zero.

Our study was based on the assumption that the camp would be dusty. We were certainly very aware that the air was dusty, especially when the children were playing yard games or hiking in the woods. The research team found that the dust was very irritating to the throat. Daytime TSP levels averaged 1.24 mg/m³ for Session 1 and 1.46 mg/m³ for Session 2. Nighttime TSP levels, obtained from area sampling pumps in the dormitories, were at or below the limit of detection (0.01 mg/m³). The average respirable dust levels at the camp during the day were 0.179 mg/m³ for Session 1 and 0.163 mg/m³ for Session 2. Nighttime values, however, were close to zero.

Discussion

We found no strong evidence of a within-day or overall change in lung function in a group of children 8 to 13 yr of age who lived in a moderately dusty environment for a 2-wk period. This finding is reassuring because the particulate levels in their environment were typical of or higher than the levels in many of the ashfall areas of the Northwest in the weeks following the eruption of Mt. St. Helens, and were above the significant harm level designated by the Environmental Protection Agency (EPA) for particulate matter (1 mg/m³).

We used data collected in the same way at the same camp in 1981 as an ash-free comparison for the 1980 data. If ash had no within-day effect on the P.M./A.M. ratios in 1980, the 1980 and 1981 P.M./A.M. ratios for the outcome variables should be identical. In the presence of a bronchoconstrictor ash

effect, we would expect the ratios for FEV₁ to be lower and for $\alpha_1(\infty)$ to be higher in 1980 than in 1981. Although the results are inconclusive, further evidence against an ash effect derives from the observation that no appropriate dose-response relationship appears to exist between the P.M./A.M. ratios and the TSP levels.

We found a small but significant decrease in FVC and FEV₁ between the afternoon the children arrived at camp and the first P.M. measurement, although it is unlikely that this was an ash effect because there was a mean increase in FEV₁/FVC. Although it could have been a diurnal effect because the children were tested somewhat earlier in the afternoon on the

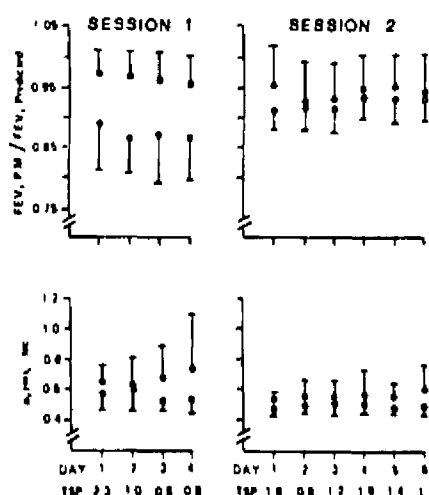


Fig. 2. Comparison of lung function measurements for susceptible (●) and nonsusceptible (○) subgroups. Top curves present 95% confidence intervals for the ratios of the FEV₁ P.M. measurements to the values predicted (11) on the basis of the children's height. Bottom curves present 95% confidence intervals for the unadjusted P.M. measurements of $\alpha_1(\infty)$. Total suspended particulate levels (mg/m³) are indicated for each observational day.

first day of camp than on follow-up days, this conclusion is not supported by 1981 data, which showed a small but significant increase in FVC and FEV₁ between the first day of camp and the first follow-up day. We are, therefore, unable to offer a satisfactory explanation for the observed decreases. Analogous changes were not observed in the transit time variables.

We were surprised to find no consistent difference in response between the group we designated as susceptible and the nonsusceptibles. Persons who met our criteria for susceptibility might be expected to show an increased bronchial responsiveness and, therefore, an exaggerated response to an inhaled irritant. We were not able to demonstrate this.

In examining the validity of our data and conclusions, it is important to scrutinize the study design and to look for possible biases that might confound our results. First, our subjects: We chose children older than 8 yr of age because our chance of getting a consistent maximal expiration in this age group was excellent. We chose the camp because it was in an ashfall area and because the wooded surroundings made it likely that the camp would be dusty despite vigorous attempts to remove the surface ash. In comparing the children's baseline measurements to accepted reference values for lung function (11), as shown in table 1, it can be seen that our children tended to have lower values than predicted. However, a group of 29 nondiabetic children tested separately using the same equipment and procedures gave results not significantly different from our study population.

Results from 2 recent animal studies show that diabetes mellitus can affect lung function as a result of the extensive microvascular changes that occur in many organs (15, 16). Reinila and coworkers (15) reported finding triglyceride deposits in the pulmonary artery in streptozotocin-diabetic rats, and Vracko and associates (16) reported the finding that epithelial and capillary basal laminae of alveoli were significantly thicker in the lungs of 14 diabetics who came to autopsy than in a control group of 10 who had no history or evidence of diabetes mellitus. However, because we were looking specifically for airway narrowing caused by inflammation, increased mucus secretion, or bronchoconstriction, it seems

unlikely that the existence of such changes in the vessels and alveolar epithelium would affect our results. Also, it would be surprising if extensive microvascular disease is present in this age group because many of the children had developed overt diabetes mellitus within the previous 4 yr.

Second, our methods: We used standardized methods (7) for lung function tests and historical information, and a spirometer that met recently proposed criteria for accuracy (8). As far as possible, children were tested on the same instrument throughout the study. We corrected for the temperature sensitivity of our spirometers and made corrections to BTPS conditions. Although specific conductance may be a more sensitive test of changes in large airway caliber than spirometric variables, we used spirometry for practical reasons and because it is the approach most commonly used in the evaluation of an occupational hazard. We used transit time analysis because it has been proposed as a sensitive way of detecting minor degrees of air-flow obstruction (9, 10). Experience with this approach in children, however, has not been extensive (17-19). As can be calculated from tables 1 and 2, the standard error of the mean (when expressed as a fraction of the mean) was consistently 2 to 3 times larger for $\sigma_1(\infty)$ than for FEV₁ or FVC, and usually larger than FEF₂₅₋₇₅, indicating a high degree of intersubject variability. Intrasubject variability for $\sigma_1(\infty)$ was also quite large in comparison with the standard spirometric variables. This variability could have been decreased by further training of both subjects and technicians so that maximal effort was sustained for 6 s. This is particularly important in children who empty their lungs very rapidly and have difficulty sustaining an expiratory effort for a full 6 s. The intrasubject variability could also be decreased by digitizing the volume signal on-line or from an analog tape recording, whereas we digitized the volume-time curve by hand from paper tracings using a digitizer. A third source of variability may lie in the fact that a small percentage of the curves could not be fit by the Permutt model because σ could not be calculated, and a value of zero was used instead. Permutt and Menkes (9) found this in 10 to 15% of their young subjects and noted that the cause was an increasing slope of the flow-volume

curve at the terminal portion of expiration. We conclude that, even with the extrapolation to infinite time, transit-time analysis was neither as useful nor as reliable as FEV₁ or FVC; however, we do feel it is worth considering when the volume signal can be digitized accurately and the subjects provide a smooth, complete volume-time curve.

Third, our definition of the study population: We only used data for the 64 children who had complete, technically acceptable, data on FEV₁ and FVC for all measurements. To determine whether our conclusions would have changed if we had included more children in the analysis, we analyzed the data using the 78 children who had complete data on FEV₁ and FVC for the first week. This represented 85.7% of the 91 children who had any usable data. We found no appreciable differences in results between the 2 analyses, and concluded that no biases had been introduced by our selection procedure.

We believe that our dust data are reliable because for the first 2-wk session the data were collected by experienced NIOSH industrial hygienists, and all filters from both camps were weighed by a NIOSH-approved laboratory. Only 8 of the 90 individual dust samples were not included in the analysis: 4 total and 2 respirable because of obvious contamination and 1 of each kind because they had also been used as area samples the day before. The sampling during the second camp session was supervised by a volunteer who had been instructed by the NIOSH team. We used total dust as an index of dustiness, rather than respirable dust, because we were not concerned as much with the dust that was small enough to penetrate into the alveoli as in the larger particles that would be deposited in the nasopharynx and airways and be liable to act at those sites as nonspecific irritants (20, 21).

Dust levels, measured as total suspended particulates, ranged between 0.8 mg/m³ and 2.3 mg/m³, whereas respirable dust averaged 0.17 mg/m³. The EPA primary standards for particulate matter (5) are 0.075 mg/m³ annual geometric mean and 0.260 mg/m³ maximal 24-h concentration, not to be exceeded more than once per year. In addition, states are required to set their own standards for an air pollution alert, warning, and emergency level. For Oregon, these levels are set as 24-h averages and are as follows: air pollu-

tion alert, 0.375 mg/m³; warning, 0.625 mg/m³; emergency, 0.875 mg/m³. All states are required to set the significant harm level at 1.0 mg/m³ for a 24-h average. The EPA has not yet set standards for respirable dust but estimates that the annual TSP standard of 0.075 mg/m³ is roughly equivalent to a respirable dust level of 0.050 mg/m³ (5) and the 24-h TSP standard of 0.260 mg/m³ is roughly equivalent to 0.140 mg/m³ of respirable dust. However, these standards assume the existence of other urban pollutants, such as sulphur dioxide and the oxides of nitrogen. Because the camp was in a rural setting, and forested, it is likely that the levels of these pollutants were very low. Andersen and coworkers (22) recently found a decrease in the FEV₁ of 16 young healthy subjects exposed for 5 h to 2 mg/m³ of inert dust in an environmental chamber; 98% of their dust particles by number and 70% by mass were below 12.5 μ m, so they were probably using dust of smaller particulate size than the resuspended volcanic ash. However, their subjects were at rest and were only exposed for 5 h. Ours were exercising and were exposed for about 14 h each day.

Dust levels in an occupational setting are often much higher than the levels we observed. The Occupational Safety and Health Administration (OSHA) standards for nuisance dusts are 10 mg/m³ for total dust and 5 mg/m³ for respirable dust (23). These standards are for an occupational setting where exposure is limited to 40 h a week over a working lifetime. Clearly, we were dealing with much lower levels. However, it is important to point out that the OSHA nuisance dust standards are designed to prevent the development of pneumoconiosis, rather than as protection against the irritant and bronchoconstrictor effect of dust. It is commonly thought that persons with hyperreactive airways find a dusty environment so irritating that they usually choose not to work in a dusty job (24). Also worth noting is that nuisance dust standards are not applicable to volcanic dust because one of the criteria for a nuisance dust is that it not contain free silica (2).

In summary, we were unable to detect a measurable change in lung function that we could attribute to ash/dust, even in a susceptible subgroup of children whom we considered to be at increased risk, despite 2 wk of exposure

to daytime dust/ash levels that usually exceeded the EPA significant harm level for particulate matter.

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