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Day-to-Day Particulate Exposures and Health Changes in Los Angeles Area Residents with Severe Lung Disease

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ABSTRACT

We measured particulate matter ($PM_{2.5}$ and PM_{10}) exposures, home temperature, arterial blood oxygen saturation, blood pressure, and lung function in 30 volunteer Los Angeles area residents during four-day intervals. Continuous Holter electrocardiograms were recorded in a subgroup on the first two days. Subjects recorded symptoms and time-activity patterns in diaries during monitoring, and during a reference period one week earlier/later. All subjects had severe chronic obstructive pulmonary disease. PM_{10} (24-hr mean) at monitoring stations near subjects' homes averaged 33 $\mu g/m^3$, and ranged from 9 to 84 $\mu g/m^3$. In longitudinal analyses, day-to-day changes in $PM_{2.5}$ and PM_{10} outside subjects' homes significantly tracked concurrent station PM_{10} ($r^2 = 0.22$ and 0.44, respectively). Indoor and personal concentrations were less related to station readings ($r^2 \leq 0.1$), but tracked each other ($r^2 \geq 0.4$). In-home temperatures tracked outdoor temperatures more for lows ($r^2 = 0.27$) than for highs ($r^2 = 0.10$). These longitudinal relationships of subject-oriented and station PM measurements were generally similar to cross-sectional relationships observed previously in similar subjects. Among health measurements, only blood pressure showed reasonably consistent unfavorable longitudinal associations with particulates, more with station or outdoor PM than with indoor or personal PM.

IMPLICATIONS

In a small panel of chronically ill Los Angeles residents, subtle day-to-day health changes related more to outdoor background PM_{10} levels than to personal or in-home PM_{10} or $PM_{2.5}$ exposure levels. These findings suggest that particulate-associated ill health is caused not by inhaled particles per se, but by some component of outdoor particulate pollution (or some accompanying factor) that penetrates indoors more consistently than PM_{10} in general, or else acts during relatively brief periods spent outdoors.

INTRODUCTION

Increased ambient background concentrations of particulate matter (PM) often accompany increased rates of premature death or hospitalization in people with chronic cardiopulmonary disease. These statistical associations can be found in diverse atmospheric and socioeconomic circumstances, by cross-sectional or longitudinal analyses.[1–4] They are difficult to explain biologically because most laboratory studies of common PM components at realistic concentrations show either no toxic response, or only mild and subtle responses.[1,4] Thus, ambient PM might be a cause of ill health, or only an index for other unidentified causes. If the health/PM association is causal, the following hypotheses must be verifiable:

- Hypothesis 1: Personal PM exposures must track background ambient PM; that is, there must be significant cross-sectional (spatial, between-persons) associations and significant longitudinal (temporal, within-persons) associations between PM concentrations in the ambient background and in the personal environment.
- Hypothesis 2: Relatively common, mild health problems must increase with ambient PM levels, as do deaths and serious illnesses. (The alternative, that the toxic components of PM cause severe effects in a few without lesser effects in larger numbers of people who are less exposed and/or less susceptible, is biologically implausible.[5])

Some information relevant to hypothesis 1 is available. Brown and Paxton[6,7] reviewed five personal exposure studies involving both chronic obstructive pulmonary disease (COPD) patients and healthy volunteers under a variety of climatic conditions. They concluded that the relationship between personal and outdoor (ambient background) PM is variable and generally weak, and that indoor PM sources are more important contributors

to personal exposure. More recently, Janssen et al.[8] studied older adult nonsmokers in Amsterdam, and observed relatively strong longitudinal relationships between personal and outdoor PM_{10} concentrations (r^2 for a typical individual near 0.25 overall, near 0.5 on days without exposure to environmental tobacco smoke).

We previously studied personal exposures and short-term health changes in 45 Los Angeles area residents with severe COPD, during summer and autumn, aiming to test both hypotheses.[9] We found that PM_{10} or $PM_{2.5}$ concentrations outside subjects' homes showed strong cross-sectional relationships ($r^2 \geq 0.5$) with nearby monitoring stations' readings of ambient background PM_{10}. Personal concentrations showed weak cross-sectional correlations with station readings ($r^2 \leq 0.15$), and were more closely related to indoor concentrations ($r^2 \geq 0.35$), in reasonable agreement with the studies reviewed by Brown and Paxton. No subject-oriented longitudinal PM measurements were available, so day-to-day health changes could be compared only to station PM_{10} changes. Diastolic blood pressure showed a statistically significant ($P < 0.05$) longitudinal relationship, rising with station PM_{10}. One measure of lung function, peak flow, showed a significant negative longitudinal relationship with station PM_{10}; but other related measures—forced vital capacity (FVC) and forced expired volume in one second (FEV_1)—did not.

In the present study, we aimed to test hypotheses 1 and 2 more rigorously than before. We employed a new panel of volunteers with severe COPD (including some previous volunteers), and performed more detailed longitudinal exposure and health measurements.

METHODS

Design

The study was conducted during autumn and winter, when the broadest possible range of ambient PM levels is expected in the Los Angeles area. (However, episodes of maximal atmospheric stagnation and PM concentrations never occurred on study days.) Thirty subjects were studied, each from a different home, usually three at a time. Each subject was monitored during four consecutive 24-hr periods, beginning between 8:00 and 11:00 a.m. on a Thursday, to document both weekday and weekend activity/exposure patterns. Concurrent, co-located PM_{10} and $PM_{2.5}$ samples were taken each 24 hr, inside and immediately outside the subject's home. Each subject wore a monitor to collect 24-hr personal PM samples concurrent with indoor and outdoor sampling; PM_{10} was monitored in half the subjects (randomly selected) and $PM_{2.5}$ in the others. Subjects were self-referred, and therefore not representative of any larger population; but they appeared typical of patients with advanced COPD, and exhibited a typical range of southern California home characteristics (see below).

Subjects

Volunteers (16 females, 14 males, age range 56–83) were recruited by advertisements, referrals from local chest physicians, and invitations to previous participants. All had clinically severe COPD (FEV_1 ranged from slightly above 50% to less than 20% of predicted values), presumably placing them at risk for PM effects, but were ambulatory enough to be exposed to ambient pollution. The majority of subjects used supplemental oxygen at least intermittently. Two were still smoking; all others were former smokers. They were screened by medical history, physical examination, lung function tests before and after bronchodilator, and resting and exercise electrocardiograms. During screening, subjects were trained in self-monitoring procedures to be used in the actual study. All subjects gave informed consent and were paid for participation. The protocol was reviewed and approved by the research committee at Rancho Los Amigos Medical Center. Three subjects lived in mobile homes, eight in apartments, and 19 in conventional detached single-family residences. Fourteen of the residences had central forced-air heating; 16 had space heaters. Six central-heated homes also had central air conditioning, while eight other homes had room air conditioners, three had evaporative coolers, and 13 had no cooling capability. None of the homes had any special air-cleaning equipment. Most used natural gas for cooking as well as heating.

Environmental Monitoring

Hourly PM_{10}, ozone (O_3), nitrogen dioxide (NO_2), carbon monoxide (CO), and temperature data were retrieved from the South Coast Air Quality Management District (SCAQMD) database for subject monitoring days and immediately preceding days. Data from the SCAQMD station nearest a subject's home were compared with his/her monitoring data. Not all stations had PM_{10} monitors, thus the nearest PM_{10} station might be more distant than the nearest station for other monitoring. Tapered element oscillating microbalance (TEOM) PM_{10} monitors and beta aerosol monitors (BAMs) for PM_{10} were considered equivalent in the data analysis.

Staff members visited each subject's home in turn on a Thursday morning to set up exposure and health monitors. Thus, the 24-hr monitoring interval began near 8:00 a.m. for the first subject and near 11:00 a.m. for the third subject studied in any given week. The staff revisited each home at 24-hr intervals to collect completed PM samples and start new ones, concluding on Monday morning. PM samplers were placed

in unobtrusive locations within the air "microenvironments" most frequented by subjects, with pumps in sound-insulated boxes, and inlets about 1 m above ground or floor level. Samples were collected on Gelman Teflo filters in Marple low-volume size-selective samplers. Personal samplers were placed in backpacks that subjects were told to wear, or otherwise keep as close to the breathing zone as possible, around the clock. Air sampling pumps were calibrated with bubble meters prior to field use. If flow readings at the beginning and end of a 24-hr sample differed, overall flow was estimated as the average. Gravimetric measurements of collected PM were made with a Cahn Electrobalance at controlled temperature and humidity. From aqueous extracts of exposed filters, sulfate (SO_4) and nitrate (NO_3) ions were quantitated in relation to a commercially-obtained standard (SPEX, Certi-Prep, Metuchen, NJ) using a Dionex DX-100 ion chromatograph (IC). Conventional analytical quality assurance procedures, including blank and replicate sampling, replicate analyses, and regular comparison of IC standard curves to detect drift, were followed. Because laboratory and field staff overlapped, analyses were not strictly blind. Quasi-blind conditions were maintained by coded labeling of samples, time lags between collection and analysis, and assigning different tasks to different people when practical. Temperature was recorded inside subjects' homes with battery-operated hygrothermographs. Personal O_3 and NO_2 exposures were estimated over entire 96-hr monitoring periods using passive badge samplers.

Physiologic Measurements

Forced expiratory lung function tests were recorded with portable pneumotachograph spirometers (Creative Biomedics, San Clemente, CA), modified to store results without displaying them to the subject. Subjects were trained to perform three blows per test session, in three sessions per day, taking their respiratory medications after, not before, a session. Initially, a staff member calibrated the spirometer with a volumetric syringe, reviewed the procedure with the subject, and observed the first session to verify adequate performance. Collected data were reviewed using the manufacturer's visual display software, to edit out false readings due to pneumotachograph zeroing failures. The largest peak flow, FEV_1, and FVC recorded in each session were used in subsequent data analysis.

Blood pressure was measured with commercial home testing instruments (Sunbeam Model 7657), calibration-checked against a mercury-column sphygmomanometer. Subjects were instructed to take their blood pressure just before each lung function test. Results were printed out with time and date; thus, subjects got immediate feedback, in contrast to lung function measurements. Printed data were reviewed by a staff member, unaware of exposure conditions, who edited out physiologically implausible values.

Holter electrocardiogram (ECG) recording and semi-automated data analysis (Marquette Electronics, Jupiter, FL) were employed as in conventional diagnostic testing. The data analyst was not aware of subjects' exposure status. A field technician attached leads and started a 24-hr recording during the Thursday morning home visit, rechecked the ECG leads and started a second recording on Friday, and removed the leads and collected the equipment on Saturday. Hourly statistics for heart rates, ventricular ectopic beats, and supraventricular ectopic beats were obtained.

Subjects measured their arterial blood oxygen saturation (SaO_2) over 5-min intervals just before each of the three daily blood pressure and lung function tests, and continuously during sleeping hours, using Ohmeda pulse oximeters with fingertip sensors. Oximeters were factory-calibrated, and performed an automatic calibration check before each measurement session.

Diary Recording

Each subject filled out a diary each day, Thursday through Sunday, during health and exposure monitoring, and during a "reference period" one week before or after monitoring (order randomized). Monitoring and reference periods were compared to judge how monitoring influenced subjects' activity and symptoms. The diaries recorded hourly time-activity patterns, symptoms, and medication use, plus a daily summary of health and environmental-stress experience. They resembled previous diaries,[10,11] but required less time-activity recording and more symptom recording. At the final field visit, a staff member reviewed monitoring-period diaries and queried the subject as necessary to fill in missing information. Reference-period diaries were mailed; any missing information was obtained by phone. In statistical analyses, different location categories recorded in a given hour were assumed to account for equal portions of time. Time entries of "none," "some," "most," or "all" were estimated as 0, 20, 40, or 60 min, respectively. The subject estimated intensity of physical activity on a visual analog scale (rest = 0% to "hardest-ever" = 100%). That estimate, multiplied by the relevant time estimate, yielded a numerical index of hourly physical activity—the percentage of theoretical maximum activity level. From daily summary symptom reports, a numerical score for irritant symptoms likely to result from pollution exposure, and a separate score for lower respiratory symptoms, were calculated by a method used previously in controlled laboratory exposures.[12]

Statistical Analysis

To evaluate longitudinal relationships of background air quality, personal exposure, and health status, we used analyses of covariance with repeated measures on subjects and time-varying covariates (program 5V, BMDP Statistical Software, SPSS Inc., Chicago). This approach estimates time-related changes in a dependent variable (e.g., weekday–weekend differences) along with the linear longitudinal relationship of the dependent variable to a covariate (e.g., the change in a personal pollution measurement per unit change in a station measurement from day to day, or the change in blood pressure per unit change in a pollution measurement from day to day). Maximum likelihood estimation was used to replace missing data; and a general autoregressive data structure was assumed to allow for autocorrelation, the tendency of one day's health or environmental conditions to resemble the previous day's. To express longitudinal relationships independent of particular units of measurement, we estimated standardized slopes (r) and coefficients of determination (r^2), analogous to conventional correlation coefficients and coefficients of determination for cross-sectional data. We employed the simplest plausible analytical models, testing the effect of one covariate at a time on personal exposure or health variables measured over 24-hr intervals. For physiologic variables measured multiple times per day, preliminary analyses of variance were performed to estimate circadian variation among early (00:00–10:59), midday (11:00–17:59), and late (18:00–23:59) periods, chosen to correspond to typical testing schedules. Final analyses were performed on 24-hr averages, with each test result adjusted according to the group mean circadian change for its time interval. For selected pairs of environmental and health variables, we also performed ordinary least-squares regression analyses on each individual subject's data, and examined the distribution of individual correlation coefficients.[8]

RESULTS AND DISCUSSION

Environmental Monitoring

Table 1 gives summary statistics for PM_{10}, O_3, NO_2, and CO measured by monitoring stations nearest subjects' homes on study dates (Thursdays to Sundays). Air quality never violated U.S. Environmental Protection Agency (EPA) short-term standards, but occasionally violated California's PM_{10} standard (50 μg/m^3, 24 hr). Concentrations were appreciably lower here than in our previous panel study.[9] Table 2 gives summary statistics for PM_{10} and $PM_{2.5}$ total mass, NO_3, SO_4, and "other" components (total minus NO_3 minus SO_4), as determined from four successive 24-hr sample collections for each subject, beginning between 8:00 and 11:00 a.m., Thursday through Sunday. Concurrent PM_{10} data from nearest stations for the same 24-hr periods had mean 32 μg/m^3 and range 9–84 μg/m^3, similar to the conventional 24-hr data (measured midnight to midnight) in Table 1. PM_{10} concentrations outside subjects' homes averaged about 25% higher than concurrent station measurements; different methodology might account for part of that difference. PM_{10} concentrations indoors averaged 18% less than outdoors, while personal PM_{10} concentrations averaged 13% less than outdoors. $PM_{2.5}$ typically accounted for 60–70% of PM_{10}.

Table 1. Monitoring station data most relevant to subject monitoring (00:00 Thursday to 23:59 Sunday).

Pollutant	Avg. Time	Units	Mean	SD	Min	Median	Max
PM_{10}	24-h mean	μg/m^3	33	15	9	32	84
PM_{10}	1-h max	μg/m^3	78	41	23	62	198
O_3	24-h mean	ppb	13	7	0	11	34
O_3	1-h max	ppb	31	14	0	40	70
NO_2	24-h mean	ppb	33	11	14	33	57
NO_2	1-h max	ppb	54	18	20	50	100
CO	24-h mean	ppm	2.5	1.2	0.4	2.4	7.0
CO	1-h max	ppm	4.9	2.7	1	4	17

In contrast to a previous study of a general population in southern California,[13] and to the Dutch older adults' panel study,[8] we did not find a substantial excess of personal PM over indoor PM. This might reflect less activity/less particle generation by our subjects. Another possibility is that our subjects had more difficulty carrying the personal monitors, and so did not keep them in the personal environment at all times.

Indoor/outdoor (I/O) ratios averaged near 1 for PM_{10} or $PM_{2.5}$ mass, less than 1 for sulfate, still less for nitrate, and greater than 1 for other (chemically uncharacterized) PM. I/O varied widely between subjects and between days (Table 2). For some PM components, longitudinal analyses indicated that I/O decreased significantly ($P < 0.05$) with increases in an index of home heating (24-hr mean indoor temperature minus 24-hr mean outdoor temperature). This decreased I/O appeared to result from increased concentrations outside homes, with less change inside. Station PM_{10} concentrations also tended to increase with home heating, but the relationship did not attain statistical significance; thus, it is not clear whether the increased concentrations outside homes related to higher ambient background pollution on colder days, or to local emissions from heating. In any event, these results suggest that with heat on in typical subjects' homes (and windows presumably closed), penetration by outdoor particles was reduced appreciably, while generation/retention of indoor particles did not increase appreciably. The latter

Table 2. Subject-oriented particulate monitoring data.[a]

Variable	No. of Samples	Mean	Sd	Min	Median	Max
Outdoor PM_{10}:						
Total mass	118	39.8	18.3	7	38	97
NO_3	118	6.5	6.5	0.2	4.6	25.0
SO_4	118	2.4	1.5	0.3	2.0	7.8
Other	118	30.9	13.5	4.7	31.5	79.0
Outdoor $PM_{2.5}$:						
Total mass	118	24.8	14.5	4	22	63
NO_3	118	4.8	5.4	0.1	2.8	21.7
SO_4	118	1.8	1.4	0.2	1.4	6.6
Other	118	18.1	9.0	3.4	17.7	40.7
Indoor PM_{10}:						
Total mass	120	32.6	15.6	9	30	105
NO_3	120	1.6	1.8	0.1	1.0	9.7
SO_4	120	1.7	1.1	0.2	1.4	6.5
Other	120	29.3	14.5	7.6	27.0	96.7
Indoor $PM_{2.5}$:						
Total mass	116	23.5	15.3	4	19	92
NO_3	116	1.2	1.5	0.1	0.7	7.9
SO_4	116	1.5	1.1	0.1	1.1	5.9
Other	116	20.8	14.4	3.6	14.4	89.2
Personal PM_{10}:						
Total mass	59	34.8	14.8	5	33	85
NO_3	59	1.5	1.5	0.2	1.1	9.1
SO_4	59	1.6	1.0	0.3	1.3	4.8
Other	59	31.7	14.6	4.1	30.5	83.0
Personal $PM_{2.5}$:						
Total mass	60	23.8	15.1	4	18	65
NO_3	60	1.7	2.3	0.1	0.7	10.7
SO_4	60	1.5	1.2	0.2	1.2	5.6
Other	60	20.6	13.3	3.7	16.9	61.6
Indoor/Outdoor (I/O) Ratio, PM_{10}:						
Total mass[b]	118	0.94	0.53	0.31	0.75	2.84
NO_3[b]	118	0.39	0.38	0.04	0.30	2.50
SO_4	118	0.73	0.23	0.28	0.73	1.60
Other[b]	118	1.09	0.65	0.37	0.73	3.66
Indoor/Outdoor (I/O) Ratio, $PM_{2.5}$:						
Total mass	114	1.13	0.77	0.22	0.86	4.25
NO_3[b]	116	0.48	0.49	0.03	0.48	3.00
SO_4	116	0.86	0.27	0.17	0.85	2.00
Other	114	1.31	0.90	0.18	0.98	4.66

[a]All measurements in $\mu g/m^3$, from 24-h integrated samples.
[b]I/O ratio decreased ($P < 0.05$) with increased home heating (see text).

observation is not surprising in homes of COPD patients, who presumably try to minimize indoor pollution sources, but it may not be generalizable to other homes.

Table 3 summarizes longitudinal analyses relating each subject-oriented PM measurement with concurrent station PM_{10}. As judged from r^2, day-to-day changes in station PM_{10} accounted for 44% of day-to-day variance in PM_{10} outside subjects' homes, and 22% of variance in $PM_{2.5}$ outside homes. In regressions of outside-home PM_{10} versus station PM_{10} for individual subjects, the median r value was 0.84 and the median slope was 0.91. In indoor or personal environments, station PM_{10} accounted for considerably lower percentages of day-to-day variance. In individual regressions of indoor PM_{10} versus station PM_{10}, the median r value was 0.54 and the median slope was 0.22. The relationship to station PM_{10} levels was somewhat closer for personal $PM_{2.5}$ than for personal PM_{10}. At least in part, this difference reflects measurement of personal PM_{10} and personal $PM_{2.5}$ in different subjects.

Table 3. Longitudinal regression relationships of subject-oriented PM_{10} or $PM_{2.5}$. Measurements with concurrent PM_{10} readings at nearest monitoring stations.

Variable	PM_{10} Slope	PM_{10} r^2	$PM_{2.5}$ Slope	$PM_{2.5}$ r^2
PM total mass outside home	0.870	0.44	0.373	0.22
PM total mass inside home	0.364	0.10	0.294	0.07
PM total mass personal	0.250	0.05	0.299	0.07
PM NO_3 outside home	0.105	0.07	0.052	0.03
PM NO_3 inside home	0.020	0.04	0.015	0.03
PM NO_3 personal	0.047	0.05	0.028	0.06
PM SO_4 outside home	0.033	0.13	0.019	0.05
PM SO_4 inside home	0.019	0.07	0.020	0.09
PM SO_4 personal	0.005	0.01	0.019	0.07

Day-to-day changes in total mass concentration inside homes significantly ($P < 0.001$) tracked changes outside homes for PM_{10} ($r^2 = 0.27$) and for $PM_{2.5}$ ($r^2 = 0.19$). The I/O longitudinal relationship was stronger for nitrate ($r^2 = 0.45$ for both size ranges) than for total mass, despite generally reduced concentrations of nitrate indoors. The relationship was still stronger for sulfate ($r^2 > 0.8$), suggesting that sulfate was mostly of outdoor origin, and penetrated indoors more consistently than other PM components. Co-located PM_{10} and $PM_{2.5}$ measurements closely tracked each other, for total mass, nitrate, or sulfate, with r^2 ranging from 0.61 to 0.96. Personal and inside-home measurements showed highly significant longitudinal relationships for all measured PM components, with r^2 values ranging from 0.36 to 0.92, reflecting the fact that most subjects spent 90% or more of their time in their homes, according to diaries. The close relationship of personal to indoor PM may be partly an artifact of the monitoring process, which appeared to inhibit subjects from leaving their homes (see next section).

Overnight low temperatures inside homes showed a highly significant longitudinal relationship with outdoor low temperatures at stations (slope = 0.29, $r^2 = 0.27$). Indoor lows ranged from 59 to 75 °F; outdoor lows from 38 to 69 °F. For daily high temperatures, the I/O relationship was weaker (slope = 0.11, $r^2 = 0.10$) but still highly significant. Indoor highs ranged from 64 to 85 °F (88 and 100 °F in two homes with apparent heating anomalies, which were excluded from longitudinal analyses); outdoor highs ranged from 50 to 95 °F.

Table 4. Group mean time-activity statistics by time of day.

Variable	< 6:00 a.m.	6:00 a.m. –noon	noon– 6:00 p.m.	> 6:00 p.m.
% of clock hours spent all or partly away from home	0.5	7.5	23.5	7.0
min/hr outdoors	0.07	3.2	7.5	1.6
min/hr in vehicles	0.04	1.6	4.5	1.4
min/hr physically active	0.8	12.0	15.9	9.6
% of maximum physical activity	0.2	6.1	7.8	4.0

Table 5. Group mean time-activity statistics, monitoring week versus reference (diary-only) week.

Variable	Monit. Wk.	Ref. Wk.	P Value[a]
% of clock hours spent all or partly away from home	7.4	11.8	0.001
min/hr outdoors	2.6	3.6	0.005
min/hr in vehicles	1.5	2.3	0.006
min/hr physically active	9.2	10.0	0.07
% of maximum physical activity	4.3	4.7	0.13

[a]Interaction term from analysis of variance with week (first or second) as repeated-measures factor and order (monitoring-control or control-monitoring) as grouping factor. Main effects of week and order were nonsignificant for all variables.

Cross-sectional analyses of personal NO_2 badge readings versus concurrent four-day-average station readings showed a nonsignificant relationship ($r^2 = 0.01$, $P > 0.6$). For O_3, the corresponding relationship was significant ($r^2 = 0.26$, $P < 0.005$), but lost significance after exclusion of the two subjects with highest exposure levels. The previous panel study[9] had shown stronger relationships between personal and station O_3 or NO_2, probably because ambient concentrations were higher.

Diary Time-Activity Records

Most subjects' diaries indicated that they spent most of their time indoors at home and were sedentary. Table 4 summarizes reports of time spent in other circumstances, between 00:00 Thursday and 23:59 Sunday, for the reference period plus the monitoring period. Physical activity time was appreciable, but activity was of low intensity, as judged either from diary reports or from recorded heart rates. Comparison between monitoring and reference periods (Table 5) showed that monitoring inhibited subjects from leaving home, reducing their percentage of hours away from home, minutes per hour outdoors, and minutes per hour in vehicles by roughly one-third. Monitoring did not significantly limit physical activity, according to diaries.

Health Monitoring

Daily mean diastolic blood pressure increased significantly with same-day or previous-day 24-hr mean PM_{10} at the nearest monitoring station, although the relationship explained little of the variance in blood pressure. Figure 1 represents the longitudinal regression relationship to same-day PM_{10}, estimated from all subjects' data, as a solid line with PM_{10} median and extremes indicated by blocks. Each individual's regression relationship, from ordinary least-squares analysis, is shown by a broken line encompassing his/her range of PM_{10} concentrations. The median r value for individual regressions was 0.33. The group slope, 0.095 mm Hg increase in diastolic pressure for each $\mu g/m^3$ increase in PM_{10} ($r^2 = 0.04$, $P = 0.03$), agreed reasonably well with the previous study's[9] estimated slope of 0.077 mm Hg/($\mu g/m^3$). The relationship to previous-day station PM_{10} was still more positive (slope 0.122 mm Hg/($\mu g/m^3$), $r^2 = 0.05$, $P = 0.01$). Systolic pressure showed a positive relationship to previous-day station PM_{10} (slope 0.172 mm Hg/($\mu g/m^3$), $r^2 = 0.06$, $P = 0.006$), but not to same-day PM_{10} (slope 0.08, $r^2 = 0.01$, $P = 0.22$). A typical subject's blood-pressure change (in mm Hg) over the observed range of station PM_{10} (Table 1) would be 6 systolic and 7 diastolic based on same-day PM_{10}, or 13 systolic and 9 diastolic based on previous-day PM_{10}.

Unlike station PM_{10}, subject-oriented PM exposure measurements did not relate to blood pressure closely, except for outside-home PM_{10} (which closely tracked station PM_{10}, as described earlier). No significant relationships with blood pressure were found for outside-home $PM_{2.5}$, inside-home $PM_{2.5}$, inside-home PM_{10}, or any nitrate or sulfate

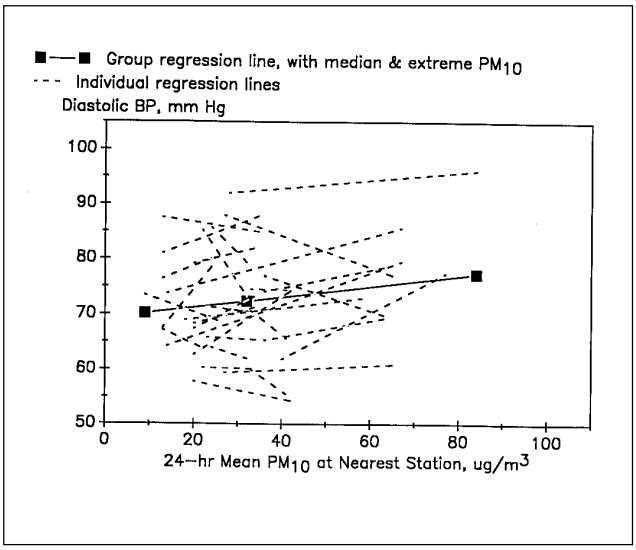

Figure 1. Longitudinal relationship of diastolic blood pressure to station PM_{10} on the same day. Solid line = estimated group mean slope, allowing for autocorrelation and day-of-week effects ($r^2 = 0.04$, $P = 0.03$). Blocks = median and extreme PM_{10} concentrations. Broken lines = individual slopes estimated by ordinary least-squares regression.

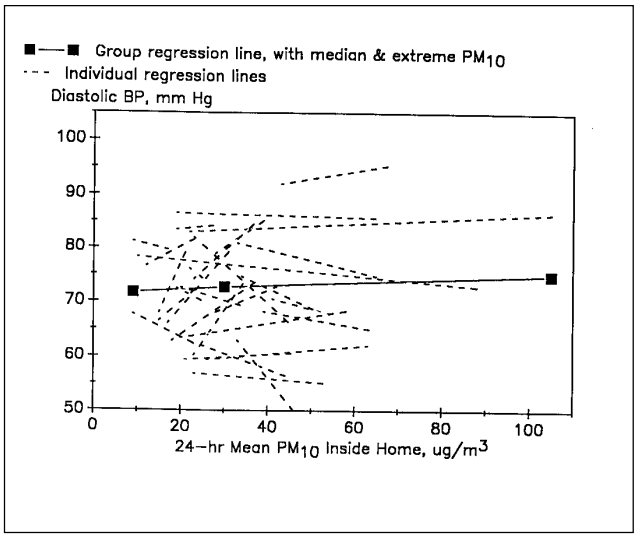

Figure 2. Longitudinal relationship of diastolic blood pressure to in-home PM_{10} on the same day plotted in the manner of Figure 1. Group mean slope is not significantly different from zero ($r^2 = 0.005$, P = 0.4).

measurement. The nonsignificant relationship of diastolic pressure to inside-home PM_{10} (slope = 0.034, r = 0.07, P = 0.43) is shown in Figure 2, as a contrast to the significant relationship with station PM_{10} shown in Figure 1. Blood pressures showed no significant relationships to station O_3, NO_2, or CO. Diastolic pressure significantly tracked daily maximum indoor temperature (slope 0.42 mm Hg/°F, $r^2 = 0.04$, P = 0.03).

No lung function measure showed a meaningful relationship to PM_{10} at the nearest station on the same or the preceding day. Some analyses of lung function versus subject-oriented PM measurements showed statistically significant negative relationships, usually eliminated by excluding two or three "outlying" subjects. The most robust result was the relationship of peak flow to the previous day's indoor $PM_{2.5}$ (slope = -0.0093 (L/sec)/(μg/m^3), $r^2 = 0.09$, P = 0.001). From the lowest to the highest observed indoor $PM_{2.5}$ (Table 2), this relationship would predict a loss of 0.8 L/sec—medically significant in people with impaired lung function. However, the regression lost significance if subjects with unusually high indoor $PM_{2.5}$ on one or more days were excluded. Also, significance depended on inclusion of Monday lung function data, based on only one morning measurement. Lung function showed no significant relationships with O_3, NO_2, or CO. Warmer early-morning low temperatures showed a small positive effect on that day's peak flow (regression slope 0.05 (L/sec)/°F, $r^2 = 0.05$, P = 0.01).

Daytime SaO_2 showed no significant longitudinal relationships with concurrent PM, O_3, NO_2, or temperature. Overnight SaO_2 did not vary significantly with PM, O_3, NO_2, or temperature, except that it tracked indoor $PM_{2.5}$ in the "wrong" direction, improving with increasing concentration (slope 0.016%/(μg/m^3), r = 0.03, P = 0.04). This relationship remained significant after exclusion of "outlying" subjects. Periods of clinically subnormal arterial oxygenation (SaO_2 < 90%), usually transient, were observed in 25 of 30 subjects.

Satisfactory ECG data were obtained only in a minority of subjects. To improve analytical power, data were pooled with similar data from a pilot study. In all, 18 subjects had analyzable recordings on two successive days. Their incidence of supraventricular ectopic beats (log-transformed) increased significantly with station PM_{10} ($r^2 = 0.15$, P = 0.01); the regression predicted a doubling of incidence for each 14-μg/m^3 increment in PM_{10}. The relationship was weaker with outside-home PM_{10} or $PM_{2.5}$ ($r^2 = 0.07$, P = 0.11) and essentially zero with indoor PM. The public health implications of this relationship (if it is real) are uncertain, in that supraventricular ectopic beats are considered relatively unimportant in clinical practice. Ventricular ectopic beats, more strongly associated with cardiovascular morbidity and mortality, showed no significant relationships to PM measures.

Day-to-day changes in irritant symptoms or lower respiratory symptoms during monitoring periods showed no significant relationship to any measure of air pollution. However, lower respiratory symptoms, analyzed over the monitoring period plus the reference period, showed a marginal relationship to station PM_{10} ($r^2 = 0.014$, P = 0.05). The regression slope predicted a minimally perceptible symptom increase from a PM_{10} increment of 40 μg/m^3.

CONCLUSIONS

Los Angeles area monitoring stations appeared to give meaningful estimates of PM exposures outdoors at the homes of our "high-risk" subjects, with respect to temporal as well as spatial variation. Predictions of indoor exposures from station data were statistically significant, but explained no more than 10% of the variance over time. Indoor PM appeared to be the predominant influence on total personal exposures, not only in this study, but also in previous personal exposure studies elsewhere.[6,7] Thus, our findings provide only limited support for hypothesis 1, that personal exposures track ambient background PM.

Concerning hypothesis 2, we found some evidence suggesting that slight day-to-day health changes in our COPD patients tracked PM changes. The evidence was strongest for blood pressure, more doubtful for lung function, supraventricular ectopic heartbeats, and respiratory symptoms. Surprisingly, these possible health effects usually tracked ambient background PM, and did not track

PM measures more representative of personal exposure. However, peak flow losses related (equivocally) to previous-day indoor $PM_{2.5}$. Our previous panel study[9] suggested a relationship of peak flow with station PM_{10} (the only longitudinal measurement available) on the same day. Either finding might be interpreted as a real PM effect, or an effect of an unknown confounder on measurements made in less than ideal circumstances, or a chance significant finding among many statistical tests. For clarification, larger-scale, longer-duration studies of COPD patients may be indicated, since peak flow is relatively easy to measure.

This study was limited by a small, self-selected subject group, short follow-up period, nonideal monitoring circumstances, and location in an area with atypically mild climate (advantageous, however, in terms of relatively high pollution levels and relatively little confounding by weather stresses). Within these limits, the results argue that particle inhalation per se does not cause the morbidity associated with ambient background PM, in that the measurements more representative of inhaled particles were less associated with day-to-day health changes. If some component of ambient PM did cause the health changes in our subjects, it presumably either acted during their relatively brief periods outdoors or penetrated indoors more readily than other outdoor PM components. More investigation is needed to identify specific health risk factors (whether components of ambient PM, accompanying gaseous pollutants, or other environmental factors) and exposure conditions under which they exert their effects.

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