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Indoor–Outdoor Concentrations and Correlations of PM10-Associated Mutagenic Activity in Nonsmokers' and Asthmatics' Homes

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The indoor–outdoor relations of mutagenic compounds associated with airborne particles less than 10-μm aerodynamic diameter (PM10) were studied in the homes of 10 nonsmokers located in southern California. Asthmatics lived in nine of the 10 homes studied. Low-volume samplers (4 and 2 L/min) fitted with PM10 and cyclone (5-μm nominal particle size cut) particle preselectors were used to collect 24-h air samples indoors and outdoors. In addition, three individuals carried personal samplers for 24-h periods. Filter extracts were tested for mutagenicity using a sensitive *Salmonella* microsuspension assay. The concentrations of particle-associated mutagens were significantly higher outdoors compared to indoors. For the PM10- and cyclone-collected samples, indoor–outdoor ratios averaged 0.46 and 0.49, respectively. There were significant correlations between indoor and outdoor mutagenicity (revertants/m^3) for all samples (Spearman rank, $R_s = 0.74$; $n = 25$ pairs; $p < 0.001$). Further, the indoor and outdoor correlation of specific-mass mutagenic activity [revertants per unit particle mass (revertants/μg)] was highly significant [$p < 0.001$; $R_s = 0.90$ ($n = 25$ pairs)]. The mutagenic activity of personal samples was very similar to the fixed-site samples collected indoors. The results support the hypothesis that mutagen composition of the indoor and outdoor particles are similar and that outdoor particles which infiltrate into homes may be a significant source of respirable mutagenic particles in the homes of nonsmokers.

Introduction

The measurement of human exposure indoors and outdoors to airborne particles 10 μm or less (PM10) is important because particles this size enter and deposit on the trachea, the bronchi, and the alveoli (*1*) and are associated epidemiologically with increased respiratory infections, decreased lung function, and mortality (*2–4*). In California, PM10 has also been associated epidemiologically with cancer (*5*). A finding that is consistent with the potential carcinogenicity of PM10 is that these particles are known to contain numerous carcinogenic and mutagenic compounds (*6–8*). Although there are some prominent indoor sources of PM10 (cigarette smoking and cooking of food, for example), the contribution of outdoor PM10 to indoor air could be an important and significant source for day-to-day indoor exposure. People spend about 90% of their time indoors (*9*), and therefore, indoor air could represent a significant fraction of their total PM10 exposure. Further, the transport of toxic compounds associated with PM10 to the indoor environment from outdoors is not well understood.

In a previous paper from this study, Colome and colleagues (*10*) found that indoor concentrations of PM10 mass were moderately correlated with outdoor concentrations in the homes of asthmatics with no smokers in the family. Further, mass concentrations were generally lower indoors as compared to outdoors. A number of investigators have studied indoor PM10 mass concentrations (*11–15*). For example, Lioy et al. (*12*) reported that indoor–outdoor PM10 concentrations were correlated and that indoor concentrations were less than concurrently measured outdoor concentrations during a 14-day winter sampling period.

Airborne concentrations of specific compounds associated with particles have been measured indoors (*7, 16, 17*). For example, Waldman et al. (*16*) determined the concentrations of benzo[*a*]pyrene (BaP) in indoor and outdoor air and reported that indoor BaP concentrations correlated with ambient outdoor levels in most of the homes. Ando et al. (*17*) found that the concentrations of benzo[*k*]fluoranthene, benzo[*ghi*]perylene, and BaP in indoor air increased in proportion to the concentrations of these compounds measured in outdoor air. Although the measurement of these specific toxic compounds is important in the evaluation of health risk, the task is made difficult because PM10 consists of complex mixtures of compounds with different levels of biological activity.

One approach toward estimating exposure to the complex mixture of compounds in PM10 is to determine the mutagenic activity of collected PM10 samples. Mutagenic activity is an important characteristic of many, but not all, compounds that have been reported to present a risk of cancer in humans. Determining the extent of exposure to mutagenic compounds associated with indoor air particles can be helpful in assessing risks and also for identifying sources of mutagens in indoor air. Several approaches for measuring the mutagenic activity of airborne ambient particulate matter have been described, which typically use the *Salmonella*/microsome test of Ames et al. (*18–21*).

Indoor mutagenic activity has been measured in homes and restaurants with known mutagen exposures such as environmental tobacco smoke (*22–24*), and sensitive bioassays have been developed to detect indoor mutagenic activity (*25, 26*). There are, however, few reports of mutagenic activity in homes where there are no smokers and where there are no apparent known sources of particle-associated mutagens. Measurement of such homes would be helpful in establishing "baseline" levels of these toxic

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compounds indoors and could be used to investigate the influence of outdoor PM10-associated toxic compound concentrations on indoor concentrations.

The use of portable personal sampling pumps allows for measuring individual exposures to airborne mutagens and allows comparisons to be made with more traditional, fixed-site methods of assessing indoor air particle exposure. The use of these low-volume samplers, however, results in very low particle loading on filters and necessitates the use of a sensitive bioassay for mutagenicity determinations. We have previously reported on a microsuspension procedure (*27, 28*) of the *Salmonella*/microsome test. The assay was initially developed to measure urinary mutagenicity in smokers and nonsmokers and to detect airborne particle samples of limited sample mass (*28*). Airborne mutagenicity using this method has been reported for environmental tobacco smoke (ETS) exposure (*23, 29–32*), occupational environments, and for particles collected outdoors with less than 2.5-μm diameter (*28, 29*).

In the present study, we use mutagenicity as a surrogate of exposure for the complex mixture of toxic compounds associated with PM10. We investigated airborne mutagenicity of samples collected with low-volume air samplers and examined the relations of this activity indoors and outdoors. Some personal air samples were also measured. The data we report here provide an initial effort to measure baseline mutagenicity levels using low-volume air samplers and provide initial information on possible source contributions of PM10-associated mutagenic compounds detected indoors.

Materials and Methods

Indoor–Outdoor Air Sampling. Mutagenic activity of indoor and outdoor air was measured at 10 homes located in the southern California area during the fall season (asthmatics lived in nine of the homes sampled). Homes were located in Orange County cities centered around Anaheim. These cities are approximately 20–30 mi southeast of downtown Los Angeles. The asthmatic subjects were part of a California Air Resources Board-sponsored study (Pulmonary Function and Symptomatic Responses of Asthmatics to Ambient Acidic Atmospheres), in which a number of asthmatics were followed on a daily basis for changes in physiological function and health symptoms with respect to exposure to ambient acidic aerosols. Paired indoor–outdoor samples were collected simultaneously. The sampling period was from October to December 1987, and homes were measured sequentially. There were two cyclone samplers placed indoors and two cyclone samplers placed outdoors for all 10 homes. There were also two colocated PM10 samplers placed indoors and two outdoors at five of the homes. Due to concurrent measurements of ionic compounds, the other five homes had a single PM10 sampler placed indoors and a single sampler placed outdoors. The ionic compound concentrations and relations indoors and outdoors will be reported elsewhere. Personal samples were collected from three homes and were collected in parallel to fixed-site residential samples.

The air sampling devices used to collect PM10 (PM10 preselector) and particles nominally less than 5 μm (cyclone preselector) in indoor environments were used throughout (*33*). The sampling inlet for PM10 is designed to provide a sharp particle size cut at 10 μm, while for cyclone-associated particles, the 5-μm size cut collects 5-μm particles at 50% efficiency. The PM10 sampling device had a flow rate of 4 L/min (*33*), while the cyclone was connected to a personal sampling pump (SKC, Inc., Fullerton, CA) with a flow rate of 2 L/min. All flow rates were calibrated at the site before and after a 24-h sampling period. The filter used for the PM10 sample was a 47-mm-diameter Teflon membrane filter, and for the cyclone sample, a 37-mm-diameter Teflon filter (Gelman Filter Co., Ann Arbor, MI) was used. Both filters had nominal pore sizes of 2 μm.

Chemicals. Benzo[*a*]pyrene (BaP), 2-nitrofluorene (2-NF), and dimethyl sulfoxide (DMSO; spectrophotometric grade) were purchased from Aldrich Chemical Co. (Milwaukee, WI) and used without further purification. Dichloromethane (DCM; resi-analyzed grade) and methanol (HPLC grade) were purchased from Baker Chemical Co. (Phillipsberg, NJ).

Filter Handling and Extraction. Filters were precleaned using a 1:1 solution of dichloromethane and methanol, allowed to dry for 24–48 h, and preconditioned for an additional 24 h at 22–24 ° C, 30–40% relative humidity (RH) before weighing. Filters were, after each 24-h sampling period, wrapped in foil, placed in dry ice, and transported back to the laboratory. Filters were allowed to equilibrate to 22–24 °C for at least 1 h, reweighed, and placed in a −20 °C freezer until extraction. All filters were coded, and all handling was done under subdued or yellow lights to minimize potential photooxidation of adsorbed mutagens.

Filters were extracted by placing them in precleaned screw-capped bottles or flasks fitted with fluorocarbon-lined caps and adding 5 mL of dichloromethane (DCM). The container was shaken for 15 min and sonicated for 15 min, and the solvent was decanted into a precleaned 15-mL scintillation vial. The extraction procedure was repeated once more with DCM and finally with methanol, with all the extraction solvents pooled into a single prewashed vial fitted with a fluorocarbon-lined screw cap. The solvents were subsequently evaporated under a steady stream of nitrogen to about 1–2 mL of solvent, transferred to a minivial, and evaporated to dryness. The extract was then resuspended in DMSO.

Microsuspension Mutagenicity Assay. A microsuspension procedure previously reported (*27, 28*) was used. The assay, which is a straightforward modification of the *Salmonella*/microsome test, is approximately 10 times more sensitive than the standard Ames *Salmonella* procedure based on absolute amounts of material added per determination.

Tester strain TA 98 was kindly provided by Dr. B. N. Ames, Berkeley, CA. Bacteria were grown overnight in Oxoid Nutrient Broth No. 2 (Oxoid Ltd., Hants, England) to approximately 1–2 × 10^9 cells/mL and harvested by centrifugation (5000*g*, 4 °C, 10 min). Cells were resuspended in ice-cold phosphate-buffered saline (PBS, 0.15 M, pH 7.4) to a concentration of approximately 1 × 10^{10} cell/mL (determined spectrophotometrically at 550 nm).

The S9 and S9-mix were prepared according to the procedure of Ames et al. (*18*). The S9 from livers of Aroclor 1254-pretreated male Sprague–Dawley rats contained 52.4 mg of protein/mL as determined using the modified Biuret method of Ohnishi and Bar (*34*) was used throughout.

For the microsuspension assay, the following ingredients were added, in order, to 12 × 75 mm sterile glass culture tubes on ice: 0.1 mL of S9 mix, 0.005 mL of PM10 extract

Table 1. Indoor and Outdoor Mean (± SE) Mutagenic Activity per Cubic Meter of Air

particle preselector	indoor mean ± SE (rev/m³)[a]	(n)	outdoor mean ± SE (rev/m³)[a]	(n)	indoor/outdoor ratio mean ± SE
PM10	175.0 ± 21.1	18	337.7 ± 49.4	17	0.46 ± 0.05
Cyclone (PM5)	177.6 ± 23.4	27	341.6 ± 49.7	27	0.49 ± 0.05

[a] TA98 revertants + S9.

in DMSO, and 0.1 mL of concentrated bacteria in PBS (1 × 10 ^{10}/mL of PBS). The mixture was incubated in the dark at 37 °C with rapid shaking. After 90 min, the tubes were placed in an ice bath and taken out one at a time immediately before adding 2 mL of molten agar (*18*) containing 90 nmol of histidine and biotin. The combined solutions were vortex-mixed and poured onto minimal glucose plates. Plates were incubated at 37 °C in the dark for 48 h. Strain markers were routinely determined for each experiment. The spontaneous number of revertants were based on plates which had only DMSO added. All procedures were carried out in a room fitted with yellow fluorescent lights (G.E. F40Go) to minimize potential photooxidation.

Results

Summary Statistics. All filters used in air sampling and subsequently extracted had detectable levels of mutagenic activity. The blank filters were generally at or near the spontaneous number of revertants. The airborne concentrations of genotoxic agents, based on mutagenic activity (revertants/m³), for cyclone or PM10 samplers are summarized in Tables 1 and 2.

The mutagenic activity of paired indoor and outdoor samples were evaluated on the basis of the number of revertants/m³ of air (revertants/m³). Mutagenic activity was significantly greater outdoors compared to indoors for both types of samplers ($p < 0.01$ for cyclones; $p < 0.01$ for PM10 samplers; Wilcoxon signed rank test). The average mutagenic activity ± standard error (± SE) measured for PM10 samples indoors was 175 ± 21 revertants/m³, while outdoors, the PM10-associated mutagenicity was 338 ± 49 revertants/m³. The filters from cyclone samplers had an average indoor mutagenic activity of 178 ± 23 revertants/m³, while the outdoor average activity was 342 ± 50 revertants/m³. The average mutagenic activity for both indoors and outdoors was very similar between the cyclone and PM10 samplers. The indoor to outdoor ratios (I/O ratio) for PM10 and cyclone samplers were calculated for paired means for each location. The mean I/O ratios ± SE were 0.46 ± 0.05 and 0.49 ± 0.05 for PM10 and cyclone samplers, respectively.

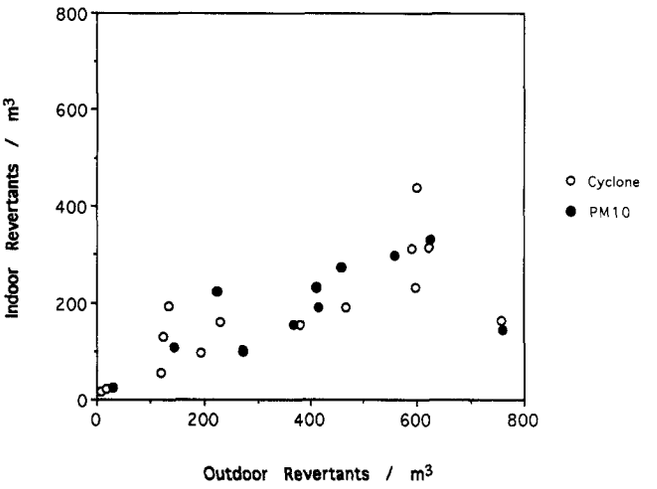

Figure 1. Paired indoor and outdoor specific mutagenic activity (revertants/m³) for all homes. Spearman rank correlation $R_s = 0.74$ ($n = 25$ pairs).

The mutagenic activity per microgram of particle mass (specific mass mutagenic activity) was evaluated for all samples and is presented in Table 2. The values were calculated by first determining the revertants per filter and dividing by the mass of that filter. The means were calculated from each per filter value. There also were no significant differences between the cyclone and PM10 samplers. However, the outdoor specific mass mutagenic activity was significantly higher than indoor values for paired samples ($p < 0.05$, Wilcoxon signed rank test).

Correlation of Mutagenic Activity Indoors and Outdoors. The results for the correlation of indoor and outdoor mutagenic activity based on airborne concentrations (revertants/m³) is presented in Figure 1. Each data point represents arithmetic mean mutagenic activities from mean cyclone or PM10 indoor and outdoor air samples from each sampling site. Duplicate indoor or outdoor samples were averaged or, if not available in duplicate, the single value for mutagenic activity was used in the matched pair. The correlation based on the Spearman rank between specific indoor and outdoor mutagenicity (revertants/m³) was $R_s = .74$ ($n = 25$ pairs; $p < 0.001$). The estimated slope for the correlation of combined PM10 and cyclone samplers was approximately 0.5, which is the ratio of indoor to outdoor concentrations reported for PM10 or cyclone preselectors separately summarized in Table 1. The concentrations indoors appeared to be linearly related to the concentrations outdoors.

The set of PM10 and cyclone data points that are outliers in Figure 1 [high outdoor mutagenic activity (approximately 700 revertants/m³) and low indoor activity (approximately 140 revertants/m³)] are from a single home of an asthmatic. An electrostatic precipitator used by the asthmatic individual to decrease indoor airborne partic-

Table 2. Indoor and Outdoor Mean (± SE) Particle Mass Concentrations and Mean (± SE) Specific Mass Mutagenic Activity

particle preselector	indoor mean ± SE: particle mass concn (μg/m³)	indoor mean ± SE: specific mass mutagenicity (rev/μg)[a]	(n)	outdoor mean ± SE: particle mass concn (μg/m³)	outdoor mean ± SE: specific mass mutagenicity (rev/μg)[a]	(n)
PM10	46.8 ± 6.4	4.8 ± 0.6	18	61.5 ± 6.6	6.1 ± 0.9	17
Cyclone (PM5)	32.7 ± 3.5	5.9 ± 0.7	27	59.8 ± 8.3	7.1 ± 1.2	27

[a] TA98 revertants + S9.

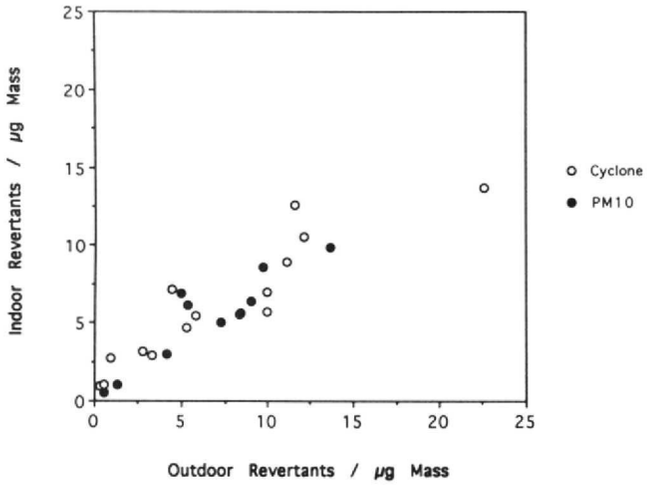

Figure 2. Paired indoor and outdoor mass specific mutagenic activity (revertants/μg) for all homes. Spearman rank correlation $R_s = 0.90$ ($n = 25$ pairs).

ulate matter was functioning during the collection of our samples. The same individual wore a personal sampler.

The specific mass mutagenic activity (revertants per microgram of particle; revertants/μg) and its indoor to outdoor relationships were examined to determine the similarities and differences between the mutagenic activity of particulate matter in the two environments. The correlation of specific mass mutagenic activity (revertants/μg) is presented for matched pairs of indoor and outdoor samples in Figure 2. Each data point represents matched arithmetic mean indoor and outdoor airborne specific mass mutagenicity from each sampling site for cyclone or PM10 samplers. The correlation between indoor and outdoor values is very strong with a Spearman rank coefficient of $R_s = 0.90$ ($n = 25$ pairs; $p < 0.001$). The standard Pearson correlation coefficient was $r^2 = 0.82$, and the slope of the best least-squares linear fit line for the cyclone and PM10 samples combined was 0.62 ± 0.12 (95% confidence interval).

Mutagenicity from Personal Sampling. The results of the personal sampling for PM10-associated mutagenicity are illustrated in Figure 3. Personal sampling apparatus dedicated for mutagenicity testing were carried by three of the nine asthmatic subjects. Each subject carried a single sampling pump with cyclone preselector. The relationship of filter duplicates for all cyclone samples for this study was $r^2 = 0.92$ with a slope of 1.05 ± 0.13 (95% CI). The subjects are represented by three bar graphs, where concurrent cyclone and PM10 sample measurements are presented as mean values of at least two samples. The corresponding indoor and outdoor measurements are also presented. The mutagenicity for the personal sample is from a single sample. The specific mutagenic activities (revertants/m^3) for the personal samples from all three homes (Figure 3) were very similar to the specific mutagenic activities of the fixed-site indoor cyclone samples. Two out of the three personal samples also matched very closely with their respective indoor fixed-site PM10 samples.

Discussion

A sensitive *Salmonella* microsuspension assay was used to measure the level of mutagenic activity collected on low-volume filter samples in the houses where there were

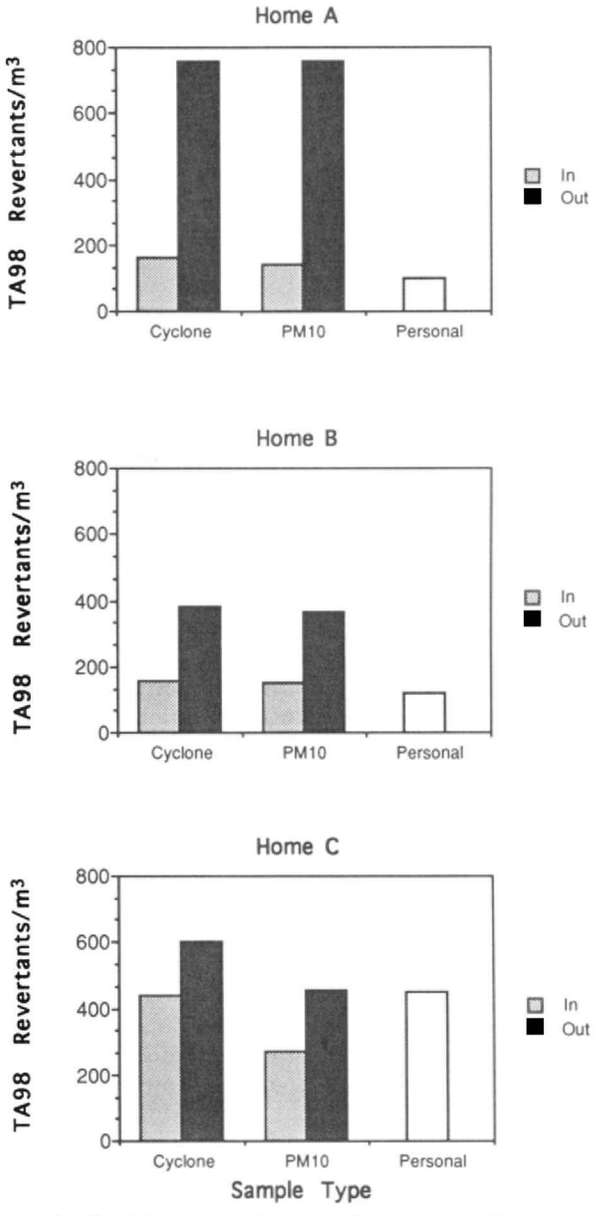

Figure 3. Particle-associated mutagenicity representing personal exposure. Cyclone samplers are worn by asthmatic volunteers and are compared to fixed-site cyclone and PM10 samplers. Each subject carried a single sampling pump. Residents in home A used an electrostatic precipitator located in the same room where the indoor sampler was located during the sampling period.

no smokers or other known indoor sources of particle-associated mutagens. Mutagenic activity was statistically higher outdoors as compared to indoors. There were no indoor or outdoor differences in the mutagenicity (revertants/m^3) associated with the particles collected by cyclone or PM10 samplers. This finding is consistent with the suggestion that mutagens are associated with airborne particulate matter in the finer size ranges, primarily on particles less than 1 μm in aerodynamic diameter (*35, 36*). Previously, Colome et al. (*10*) found that indoor and outdoor concentrations of PM10 and cyclone mass were correlated. Furthermore, in the homes of asthmatics, mass concentrations were consistently lower as compared to outdoor concentrations. Similarly, we found in these same homes that indoor mutagenic activity was considerably lower than the outdoor levels.

There have been a few reports measuring indoor and outdoor mutagenicity simultaneously and over at least a

12–24 h period. For example, preliminary results of diurnal measurements of indoor and outdoor mutagenicity have been reported for an office environment where there were no smokers present (*37*). Indoor levels followed the diurnal pattern of the outdoor mutagenic activity, and the mutagenic activity indoors was on the average about 70% of that of the outdoor activity.

In the current study, there was a statistically significant correlation between airborne indoor and outdoor concentrations of mutagenic activity (revertants/m^3) for particles less than 10-μm aerodynamic diameter. The Spearman rank correlation coefficient was 0.74 (n = 25 pairs; $p <$ 0.001). The specific mutagenic activity (revertants/m^3) based on the ratio of indoor to outdoor activity was, on the average, 50% of that of the outdoor air. This may be due to losses of particles during infiltration into the home and, possibly, also due to losses of particles on indoor surfaces (adsorption to indoor "sinks"). Further, since there were different individual homes measured on different days and locations, a relationship of mutagenicity based on airborne concentrations indoors and outdoors supports the concept that there were similar percentages of outdoor particles infiltrating indoors at all locations.

The specific mass mutagenic activity (revertants/μg; or mutagenic activity per mass of particle) was highly correlated (Figure 2) indoors with outdoors (Spearman rank correlation coefficient = 0.90, n = 25 pairs, $p < 0.001$). One hypothesis to explain this result is that the mutagenic compounds associated with indoor and outdoor particles were similar. From previous related studies, Colome et al. (*10*) reported significant correlations between particle mass concentrations indoors with outdoors, and similar findings were seen for nitrate and sulfate concentrations indoors and outdoors. These findings taken together suggest that outdoor sources contribute significantly to indoor particle concentrations and that the chemical composition of the indoor and outdoor particles sampled in this study were similar. This also suggests that mutagenicity is associated with infiltrating outdoor particles in a size range which is inefficiently removed from the air.

The indoor particles had lower mutagenic activity per microgram than the outdoor particles, averaging about 83% for the cyclone samples and 79% for the PM10 samples. This lower activity per mass found indoors can be the result of many factors including, for example: (1) loss of the mutagenic particles relative to nonmutagenic particles indoors; (2) deactivation of mutagenic compounds indoors; and (3) increase of nonmutagenic particles relative to the mutagenic particles indoors. Several of these and additional factors are probably responsible for this decrease in activity per unit of particle mass.

The mutagenic activity measured from personal sampling demonstrated that personal sampling for mutagens is possible under low-exposure conditions using conventional personal sampling methods. Also, the personal samples closely reflected the mutagenic activity from the fixed-site sampler indoor mutagenicity values. Since the individuals measured are asthmatics, their behavior could possibly favor the indoor environments as compared to outdoors, which could explain this finding.

One home (A) illustrated in Figure 3 had an unusually high ratio of outdoor to indoor mutagenic activity compared with all other homes sampled. The cyclone and PM10 samples had almost identical mutagenic activities (also see in Figure 1 as the outlier). This home was equipped with an electrostatic precipitator system located near where the sampling took place. Although we cannot rule out the possibility that there was a lower air-exchange rate for this home, it is also possible that the active removal of particulate matter indoors is effective for removing indoor air mutagenic particles.

In summary, the results of this study demonstrate that baseline levels of mutagenic compounds associated with PM10 and using low-volume air samplers can be measured over a range of ambient levels. This can be important in evaluating incremental exposure to mutagenic/carcinogenic compounds indoors. The results of this study also suggest that mutagenic compounds associated with particles found outdoors may be an important source of mutagenic activity indoors and that mutagenic activity is associated with the fine particle fraction. Future studies of the variability of mutagenic concentrations with time, season, location, and different homes would be helpful in evaluating the relative significance of these variables on mutagenic activity. The approaches used here show promise to be a useful tool for future personal sampling measurements of human exposure to airborne mutagens.

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