# Number Concentration and Size of Particles in Urban Air: Effects on Spirometric Lung Function in Adult Asthmatic Subjects

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Daily variations in ambient particulate air pollution are associated with variations in respiratory lung function. It has been suggested that the effects of particulate matter may be due to particles in the ultrafine (0.01–0.1 μm) size range. Because previous studies on ultrafine particles only used self-monitored peak expiratory flow rate (PEFR), we assessed the associations between particle mass and number concentrations in several size ranges measured at a central site and measured (biweekly) spirometric lung function among a group of 54 adult asthmatics ($n$ = 495 measurements). We also compared results to daily morning, afternoon, and evening PEFR measurements done at home ($n$ = 7,672–8,110 measurements). The median (maximum) 24 hr number concentrations were 14,500/cm$^3$ (46,500/cm$^3$) ultrafine particles and 800/cm$^3$ (2,800/cm$^3$) accumulation mode (0.1–1 μm) particles. The median (maximum) mass concentration of $PM_{2.5}$ (particulate matter < 2.5 μm) and $PM_{10}$ (particulate matter < 10 μm in aerodynamic diameter) were 8.4 μg/m$^3$ (38.3 μg/m$^3$) and 13.5 μg/m$^3$ (73.7 μg/m$^3$), respectively. The number of accumulation mode particles was consistently inversely associated with PEFR in spirometry. Inverse, but nonsignificant, associations were observed with ultrafine particles, and no associations were observed with large particles ($PM_{10}$). Compared to the effect estimates for self-monitored PEFR, the effect estimates for spirometric PEFR tended to be larger. The standard errors were also larger, probably due to the lower number of spirometric measurements. The present results support the need to monitor the particle number and size distributions in urban air in addition to mass. *Key words*: air pollution, asthma, FVC, $FEV_1$, particles, particle size, peak expiratory flow rate, PEFR, spirometry. *Environ Health Perspect* 109:319–323 (2001). [Online 7 March 2001] *http://ehpnet1.niehs.nih.gov/docs/2001/109p319-323penttinen/abstract.html*

Short-term variation and levels of urban particulate air pollution are associated with declines in lung function and increased respiratory symptoms, hospital admissions, and mortality from cardiorespiratory causes (*1–6*). Recently, it has been suggested that ultrafine particles are responsible for the bulk of adverse health effects associated with particles in ambient air (*7*). This hypothesis has been tested in studies using self-monitored peak expiratory flow rates (PEFR) and respiratory symptoms as health end points (*8–10*). These studies have shown a 0.5–1.5% decrease in PEFRs among asthmatic children and adults in association with an interquartile range increase in ultrafine particulate number concentrations. However, measurement error is greater in self-monitored PEFRs than with spirometric PEFRs (*11*), and theoretically more accurate effect estimates could be obtained using more precise health end points. Only a few studies have used repeated spirometry to examine short-term respiratory health effects of particulate matter (*12,13*). These studies were focused on schoolchildren, and the authors reported small decreases in forced vital capacity (FVC) and forced expiratory volume in 1 sec ($FEV_1$) in association with elevations of particulate matter. Our first goal in the present analyses was to examine the associations between spirometric lung function indices (FVC, $FEV_1$, and PEFR) of adult asthmatics and ultrafine particulate number concentrations in ambient air.

In the subarctic climate, resuspended road dust has a major effect on particle mass measures, especially in the coarse range (particulate matter < 10 μm in aerodynamic diameter; $PM_{10}$) in late fall and early spring (*14,15*). However, coarse mineral or road dust particles appear to be less associated with self-monitored PEFR than combustion-related particles (*8,16*). Our secondary goal was to replicate these findings with spirometry.

## Material and Methods

The study was conducted in Helsinki, Finland, during the winter and spring season (1 November 1996–30 April 1997). Characteristic features of air pollution in Helsinki are low ozone levels, occasional episodes of meteorologic inversion situations with high levels of other pollutants, and seasonal episodes of resuspended road dust. The road dust phenomenon is seen particularly during spring when the streets are dry, the snow and ice on the ground have melted away, and the particulate matter deposited on the street is resuspended mechanically by traffic or wind. This particulate matter consists mainly of sand spread on the icy roads during the winter and matter ground from road surface by studded tires.

The study group consisted of 78 adult asthmatic subjects from urban Helsinki. The group was recruited with newspaper announcements, direct mail, or through the local association of pulmonary disabled persons. Only nonsmoking adult asthmatics were admitted to the group. Asthma diagnosis was confirmed from the sickness insurance card supplied by the Social Insurance Institution of Finland. The entire study group resided within 2 km of the air quality monitoring site to ensure that the fixed-site measurement of pollutants reflected the pollutant exposure of the study subjects as well as possible.

The respiratory health of the subjects was monitored with daily self-monitored peak flow measurements and a supervised biweekly spirometric lung function test. In addition, the subjects recorded their daily symptoms and medication use in a diary.

The study subjects were instructed to measure PEFRs every day in a standing position immediately after getting up in the morning (600–1200 hr), after work (1400–1800 hr), and before going to sleep (1800–2400 hr) with a mini-Wright meter (Airmed; Clement Clarke International, Essex, UK). Each measurement included three blows, and all of them were recorded in the diary. The subjects were advised to do the measurements before taking any medication or before a meal. In addition, a supervised PEF maneuver was done at each biweekly clinic visit to verify correct performance of the measurement. The subjects were also characterized with a standard methacholine challenge test and a skin-prick test with the 13 most common local allergens.

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The biweekly spirometric lung function test was performed according to the American Thoracic Society protocol (*17*) with the subject in a sitting position and using a nose clamp; the test was performed at the study clinic set up in a local health care center and was supervised by a trained nurse. The participants were instructed to refrain from using bronchodilating medication, coffee, tea, cocoa, and cola drinks for 4 hr before the spirometry. Compliance to instructions was monitored with a written questionnaire. The maneuver was repeated at least two times with a MEDIKRO 909 portable spirometer using a heated pneumotachograph (MEDIKRO Ltd., Kuopio, Finland) and the best acceptable blow was evaluated and recorded. All spirometric parameters were corrected to body temperature, atmospheric pressure, and saturation with water vapor.

The results of 57 (73%) subjects out of 78 were used for the PEFR analysis. A total of 125 (60% of possible days) participation days was required for a subject to be included in the analysis; this was the reason for excluding 21 subjects, most of whom dropped out during the first week. Out of these 21 subjects, one subject was excluded because of unreliable reporting and one subject because asthma diagnosis could not be confirmed. Results of 54 (69%) were used for the analysis of spirometry data. Spirometry measurements were not performed for the excluded subjects because of nonconsent and exclusion criteria.

Air pollutants were monitored on a fixed monitoring site in central urban Helsinki, and meteorologic data and pollen counts were obtained from the existing metropolitan monitoring network. Particulate air pollution was monitored with five methods. Particle number concentration (PNC) in different size classes was measured continuously in 12 size ranges from 10 nm to 10 μm with an Electric Aerosol Spectrometer (EAS). We used the 8 smallest measured size ranges and aggregated them into two ranges: PNC in the ultrafine (0.01–0.1 μm; $PNC_{0.01–0.1}$) and accumulation range (0.1–1 μm; $PNC_{0.1–1}$). For quality control purposes, PNC was also monitored continuously with a condensation nuclear counter (CNC; TSI Inc, St. Paul, MN, USA). The correlation coefficient between particle number concentrations measured by CNC and EAS was 0.98. Twenty-four-hour, noon-to-noon particle mass concentrations were monitored with single-stage Harvard impactors (Air Diagnostics and Engineering, Naples, ME, USA) for particles < 10 μm ($PM_{10}$), < 2.5 μm ($PM_{2.5}$), and < 1 μm ($PM_1$) in aerodynamic diameter (*15*). The data for meteorologic parameters (wind speed, wind direction, relative humidity, and minimum temperature) were provided by the Helsinki Metropolitan Area Council.

We used daily, noon-to-noon mean values of pollutants for the statistical analyses. The data for continuously monitored pollutants were aggregated into 24-hr data. Lag 0 was defined as the 24 hr preceding the noon of the day when the lung function measurements were performed. Five-day average was defined as a mean of lag 0–lag 4.

We obtained data on influenza activity from the health authorities of Helsinki City. Influenza activity was reported to be increased during the end of January and the beginning of February. However, no serious epidemics were reported. Fever reporting was not increased during that period in our study group. To control for potential confounding, we obtained pollen count data collected with the Burkard volumetric pollen trap and provided to us by the Finnish Aerobiology Group (*18*). Because pollen counts were negligible during the whole study period, they were not considered confounders.

All lung function parameters were transformed into deviation (%) variables by first subtracting the median value of the individual from the absolute value of the measurement, dividing the total by the median value of the individual, and finally multiplying this by 100. All regression coefficients and standard errors were calculated per one interquartile range of the original pollutant measurement.

Preliminary analyses were performed using linear regression with only individual pollutants or meteorologic variables and their lags up to 3 days as dependent variables. Linearity was confirmed from scatter plots of lung function versus variables of interest. The preliminary analyses and visualization of data were done with S-Plus 4.0 (Mathsoft Inc., Cambridge, MA, USA).

The selection of a covariate for the models was based on the variable having a clear effect on the regression coefficient of the pollutant. The models for spirometric lung functions included a linear variable for temperature and relative humidity to adjust for meteorologic factors, a day-of-study variable and a squared day-of-study variable to adjust for long-term time trend, and a variable to adjust for the time of spirometry. We used the MIXED procedure (SAS Institute Inc., Cary, NC, USA) to model the linear regression in the final analyses. The same base model was used for all the pollutants.

The models for self-monitored PEFR included a variable for temperature and relative humidity to adjust for meteorologic factors, a day-of-study variable and a squared day-of-study variable to adjust for long-term time trend, and a variable for weekends. Residual plots for the individual pollutants were examined for autocorrelation, heteroscedasticity, and potential outliers. Autocorrelation was accounted for in the final analyses, which were done with the MIXED procedure.

The ethics committees of the Skin and Allergy Hospital at Helsinki and the National Public Health Institute approved the study. Written informed consent was obtained from all of the participants. The procedures used in the study were in accordance with the Helsinki Declaration.

**Table 1.** Descriptive statistics of study variables during 1 November 1996–30 April 1997 (181 days).

| | *n*[a] | Minimum | 25% | Median | 75% | Maximum |
|---|---|---|---|---|---|---|
| FVC (L) | 495 | 0.9 | 2.6 | 3.2 | 3.8 | 6.8 |
| Deviation from personal median (%) | | –48.4 | –2.6 | 0.0 | 2.6 | 24.6 |
| $FEV_1$ (L) | 495 | 0.7 | 1.7 | 2.5 | 3.0 | 6.0 |
| Deviation from personal median (%) | | –28.1 | –2.2 | 0.0 | 2.6 | 27.6 |
| PEFR (spirometry, L/min) | 495 | 154.8 | 332.4 | 405.0 | 459.0 | 703.2 |
| Deviation from personal median (%) | | –28.3 | –2.9 | 0.0 | 3.1 | 38.7 |
| Self-monitored morning PEFR (L/min) | 8,225 | 120 | 350 | 430 | 480 | 725 |
| Daily deviation from personal median (%) | | –53.9 | –3.2 | 0.0 | 2.5 | 63.0 |
| Self-monitored afternoon PEFR (L/min) | 7,672 | 135 | 370 | 435 | 500 | 730 |
| Daily deviation from personal median (%) | | –50.0 | –2.8 | 0.0 | 2.6 | 36.7 |
| Self-monitored evening PEFR (L/min) | 8,100 | 120 | 365 | 435 | 495 | 730 |
| Daily deviation from personal median (%) | | –56.4 | –2.6 | 0.0 | 2.4 | 39.1 |
| Bronchodilator use (doses/person/day) | 6,262 | 0 | 2 | 4 | 6 | 23 |
| Corticosteroid use (doses/person/day) | 7,093 | 0 | 2 | 4 | 4 | 16 |

[a]Spirometry results (FVC, FEV1, PEFR) are for 54 subjects, and self-monitored PEFR results are for 57 subjects.

**Table 2.** Descriptive statistics of the pollutants and meteorology during 1 November 1996–30 April 1997.

| | No. of days | Minimum | 25% | Median | 75% | Maximum |
|---|---|---|---|---|---|---|
| $PM_{10}$ (μg/m$^3$)[a] | 169 | 3.8 | 10.2 | 13.5 | 19.5 | 73.7 |
| $PM_{2.5}$ (μg/m$^3$)[a] | 169 | 2.4 | 5.5 | 8.4 | 12.1 | 38.3 |
| $PM_1$ (μg/m$^3$)[a] | 170 | 1.0 | 3.4 | 5.6 | 7.5 | 22.9 |
| $PNC_{0.01–0.1}$ (1,000/cm$^3$)[b] | 151 | 3.7 | 10.4 | 14.5 | 17.7 | 46.5 |
| $PNC_{0.1–1}$ (1,000/cm$^3$)[b] | 151 | 0.1 | 0.6 | 0.8 | 1.2 | 2.8 |
| Minimum temperature (°C) | 178 | –20.3 | –3.4 | 0.3 | 2.8 | 8.8 |
| Relative humidity (%) | 168 | 40.1 | 76.6 | 87.5 | 93.5 | 99.2 |

[a]24-hr particulate mass concentrations. [b]Daily mean particle number concentrations.

## Results

In our group of 54 adult asthmatics, the median values for spirometric lung function indices were 3.2 L for FVC, 2.5 L for $FEV_1$, and 405 L/min for PEFR (Table 1). The deviation of spirometric lung function indices ranged between –48.4% and 38.7%, and the deviation of self-monitored PEFR values from the personal median ranged between –56.4% and 63.0%.

During the 181 study days, the median concentrations for $PM_{10}$, $PM_{2.5}$, and $PM_1$ were 13.5, 8.4, and 5.6 μg/m$^3$, respectively (Table 2). The median concentrations of $PNC_{0.01–0.1}$ and $PNC_{0.1–1}$ were 14,500 and 800 particles/cm$^3$, respectively. The minimum daily temperature ranged from –20.3°C to 8.8°C and the relative humidity ranged from 40.1% to 99.2 %.

The particle mass concentrations ($PM_{10}$, $PM_{2.5}$, $PM_1$) were highly intercorrelated (Table 3). A high correlation was also observed between $PM_{2.5}$, $PM_1$, and $PNC_{0.1–1.0.}$ No high correlations were observed between meteorologic parameters and particle mass or particle number concentrations.

The spirometric lung function indices (FVC, $FEV_1$, and PEFR) tended to be inversely, but mostly nonsignificantly, associated with ultrafine particle number concentrations measured on the same day, the previous day, and with a mean concentration of the past 5 days (Table 4). The strongest associations were observed in the size range of 0.1–1 μm. These associations were predominantly nonsignificant. The spirometric PEFR also tended to be inversely associated with $PM_{2.5}$ and $PM_1$ concentrations.

The regression coefficients from the models for self-monitored PEFR were smaller than the regression coefficients from the models for spirometric PEFR and $FEV_1$ (Table 5).

When looking at the eight measured particle size classes separately, spirometric PEFR was most strongly associated with the particle number concentrations in size classes between 0.10 and 1.0 μm (Figure 1, Table 6).

## Discussion

Using biweekly spirometry over 6 months on a group of 54 adult asthmatics we found that FVC, $FEV_1$, and spirometric PEFR were inversely, but mostly nonsignificantly, associated with particle number concentrations on the preceding days. The standard errors were large, and only the associations with particles in the accumulation mode were statistically significant.

The median values for particle number concentrations in the ultrafine and accumulation ranges were 14,500 and 800/cm$^3$, respectively. The concentration of ultrafine particles is comparable to ultrafine number concentrations measured in Erfurt, Germany (median 11,230/cm$^3$), Birmingham, United Kingdom (mean 36 600 /cm$^3$), and Pasadena, California (mean 13,000/cm$^3$), (*10,19,20*). In contrast, levels of $PM_{10}$, $PM_{2.5}$, and $PM_1$, and the number concentrations of accumulation mode particles were lower than levels usually measured in urban settings. This phenomenon is probably explained by different source profiles at different sites together with the interactions between ultrafine and larger particles in the urban atmosphere.

Ultrafine particle number concentrations tended to be inversely but nonsignificantly associated with FVC, $FEV_1$, and PEFR. The large standard errors leading to low statistical significance were mainly due to the small observed effect on lung function and the relatively low number of observations. The most clear inverse association of the spirometric PEFR was observed with accumulation mode particles. The PEFR decreased by –0.84% for an interquartile range increase in $PNC_{0.1–1}$ measured on the previous day. The corresponding effect estimates for $PM_1$ and $PM_{2.5}$ were somewhat smaller: –0.15% and –0.12%, respectively.

Our results are consistent with two previous studies on the health effects of particle number concentrations. Peters et al. (*10*) reported inverse associations between ultrafine and accumulation mode particle number concentrations and PEFRs in asthmatic subject. Peters et al. reported that the effect estimates for 5-day averages of ultrafine and accumulation mode particle number concentrations ranged from –1.57 to –4.04 L/min for an interquartile range increase in the pollutant. In comparison, the corresponding effect estimates from our spirometric PEFR models are –2.96 L/min and –9.19 L/min for ultrafine and accumulation mode particle number concentrations, respectively. In addition, we previously observed inverse

**Table 3.** Spearman correlation coefficients for pollutants and meteorologic variables.

| | $PM_{10}$[a] | $PM_{2.5}$ | $PM_1$ | $PNC_{0.01–0.1}$ | $PNC_{0.1–1}$ | Temperature | Relative humidity |
|---|---|---|---|---|---|---|---|
| $PM_{10}$ (μg/m$^3$)[a] | 1.00 | 0.75 | 0.63 | 0.24 | 0.57 | 0.21 | –0.15 |
| $PM_{2.5}$(μg/m$^3$)[a] | | 1.00 | 0.92 | 0.26 | 0.85 | 0.10 | 0.31 |
| $PM_1$(μg/m$^3$)[a] | | | 1.00 | 0.32 | 0.86 | –0.07 | 0.30 |
| $PNC_{0.01–0.1}$ (1/cm$^3$) | | | | 1.00 | 0.39 | –0.35 | –0.10 |
| $PNC_{0.1–1}$ (1/cm$^3$) | | | | | 1.00 | –0.05 | 0.30 |
| Temperature | | | | | | 1.00 | 0.19 |
| Relative humidity | | | | | | | 1.00 |

[a]24-hr mean particulate mass concentrations. All coefficients > 0.15 or below –0.15 are statistically significant at $p < 0.05$.

**Table 4.** Associations of biweekly spirometric lung function indices and particle number concentrations measured on previous days.

| | FVC[a] | | $FEV_1$ | | PEFR | |
|---|---|---|---|---|---|---|
| | β[b] | SE | β | SE | β | SE |
| $PNC_{0.01–0.1}$ | | | | | | |
| Lag 0 | 0.00 | 0.45 | –0.40 | 0.44 | –0.52 | 0.50 |
| Lag 1 | –0.25 | 0.27 | –0.37 | 0.27 | –0.27 | 0.30 |
| Lag 2 | 0.31 | 0.36 | 0.59 | 0.35 | 0.34 | 0.41 |
| 5-Day average | –0.68 | 0.75 | –0.91 | 0.72 | –0.72 | 0.84 |
| $PNC_{0.1–1}$ | | | | | | |
| Lag 0 | –0.06 | 0.42 | 0.14 | 0.42 | –0.29 | 0.47 |
| Lag 1 | –0.60 | 0.32 | –0.44 | 0.32 | –0.84 | 0.36* |
| Lag 2 | 0.14 | 0.44 | 0.45 | 0.43 | –0.17 | 0.50 |
| 5-Day average | –1.20 | 0.93 | –0.86 | 0.90 | –2.27 | 1.04* |
| $PM_1$ | | | | | | |
| Lag 0 | –0.04 | 0.10 | –0.04 | 0.10 | –0.23 | 0.12* |
| Lag 1 | –0.07 | 0.08 | 0.00 | 0.08 | –0.15 | 0.09 |
| Lag 2 | 0.12 | 0.08 | 0.18 | 0.08 | 0.04 | 0.09 |
| 5-Day average | 0.15 | 0.16 | 0.21 | 0.16 | –0.22 | 0.18 |
| $PM_{2.5}$ | | | | | | |
| Lag 0 | 0.00 | 0.07 | 0.04 | 0.07 | –0.06 | 0.08 |
| Lag 1 | –0.06 | 0.05 | –0.02 | 0.05 | –0.12 | 0.06* |
| Lag 2 | 0.07 | 0.04 | 0.10 | 0.05* | 0.02 | 0.05 |
| 5-Day average | 0.03 | 0.10 | 0.00 | 0.10 | –0.17 | 0.11 |
| $PM_{10}$ | | | | | | |
| Lag 0 | 0.05 | 0.04 | 0.07 | 0.04 | 0.04 | 0.04 |
| Lag 1 | –0.01 | 0.04 | 0.01 | 0.04 | –0.03 | 0.04 |
| Lag 2 | 0.05 | 0.03 | 0.06 | 0.03 | 0.04 | 0.03 |
| 5-Day average | 0.05 | 0.06 | 0.06 | 0.06 | 0.02 | 0.06 |

Regression coefficients (β) and standard errors (SE) are adjusted for time trend, temperature, relative humidity, and diurnal variation.
[a]Lung function indices (FVC, FEV1, and PEFR) are defined as deviation (%) from personal median. [b]Regression coefficients and SEs were calculated per interquartile range of each particle measurement. *$p < 0.05$.

associations between ultrafine and accumulation mode particle number concentrations and PEFRs on asthmatic children (*9*). The effect estimates from this study are not directly comparable to the present study because the PEFRs of children are smaller than the PEFRs of adults.

We previously reported that the particle effect on self-monitored PEFR tended to increase with decreasing particle size (*8*); that is, the largest inverse effect was observed for ultrafine particles. This was not observed in the present study for spirometric PEFR. In comparing these two studies, we found that the confidence intervals of the effect estimates in the ultrafine range overlap, but the estimates in the accumulation mode differ. This discrepancy could be due to chance or because self-monitoring of PEFR was done daily, but spirometry was performed only on selected weekdays. Also, because the blowing techniques differ between the two lung function measurements, they may reflect slightly different aspects of lung function. Furthermore, there was a poor within-person correlation between self-monitored and spirometric PEFR (average within-person correlation between afternoon PEFR and spirometric PEFR; mean $r = 0.21$).

In addition to the size distribution of particles, respiratory health effects may be explained by the typical chemical composition of each size range. Ultrafine particles are formed during combustion processes, and in urban settings they are mostly derived from exhaust of automobile engines. The main source of particles in the accumulation mode is the coagulation of ultrafine particles. They are also formed from condensation of water or different vapors onto existing ultrafine particles, causing them to grow into this size range. This takes time, and most of the accumulation mode particles are from long-range transport.

In recent literature, transition metals such as iron, vanadium, and nickel (*21*); diesel exhaust with its components (*22*); endotoxin (*23*); and particle acidity (*24*) have been described as the characteristics of fine and ultrafine particles most likely to cause cellular damage. In the study by Dusseldorp et al. (*1*), increased concentrations of iron tended to be associated with a decline in PEFR among adult asthmatics. In contrast, the study among children by Roemer et al. (*25*) provided only weak support for the hypothesis that daily fluctuations in soluble elemental concentrations in ambient particulate matter are responsible for acute health effects. Neas et al. (*26*) reported that acutely lower peak flows in children were associated with fine sulfate particles, but only weakly associated with the acidity of the fine particles. However, neither toxicologic nor epidemiologic evidence on the specific effects of the composition of particulate matter is conclusive to date.

Numerous studies verify the associations of various respiratory health end points with $PM_{10}$ on the previous days (*5,6*). We did not observe negative associations between either self-monitored PEFR or spirometric lung function indices and $PM_{10}$. This is probably due to the effect of coarse, road-dust related particles, which influence the particle mass measurements in the subarctic spring and fall conditions of Helsinki (*15*).

Most of the associations reported in this paper are nonsignificant or are borderline significant. It is therefore evident that cautious interpretation should be applied to these effect estimates. Chance may explain these findings. Three aspects of these results support a true effect of PNC on the lung function of adult asthmatics: *a*) the consistency of the results using three daily self-monitored PEFR maneuvers and several spirometric lung function indices; *b*) the consistency of the results over lag 0, lag 1, and 5-day mean values of PNC; and *c*) the consistency of the presented results with our previous studies on Finnish schoolchildren (*9,27,28*).

**Table 5.** Associations of daily self-monitored PEFRs and particle number concentrations measured on previous days.

| | Morning PEFR[a] | | Afternoon PEFR | | Evening PEFR | |
|---|---|---|---|---|---|---|
| | β[b] | SE | β | SE | β | SE |
| $PNC_{0.01–0.1}$ | | | | | | |
| Lag 0 | –0.017 | 0.094 | –0.231 | 0.085** | –0.151 | 0.080 |
| Lag 1 | –0.240 | 0.090** | 0.019 | 0.081 | –0.002 | 0.078 |
| Lag 2 | 0.068 | 0.099 | 0.057 | 0.087 | –0.119 | 0.084 |
| 5-Day average | –0.307 | 0.283 | –0.770 | 0.254** | –0.596 | 0.252* |
| $PNC_{0.1–1}$ | | | | | | |
| Lag 0 | –0.061 | 0.104 | –0.164 | 0.094 | –0.125 | 0.089 |
| Lag 1 | –0.086 | 0.104 | 0.070 | 0.094 | 0.045 | 0.091 |
| Lag 2 | 0.033 | 0.110 | –0.095 | 0.097 | –0.204 | 0.093* |
| 5-Day average | 0.053 | 0.321 | –0.521 | 0.289 | –0.528 | 0.287 |
| $PM_{2.5}$ | | | | | | |
| Lag 0 | 0.113 | 0.112 | 0.049 | 0.100 | –0.072 | 0.096 |
| Lag 1 | –0.076 | 0.112 | 0.134 | 0.100 | 0.129 | 0.097 |
| Lag 2 | –0.001 | 0.110 | –0.059 | 0.100 | –0.100 | 0.096 |
| 5-Day average | 0.146 | 0.142 | 0.063 | 0.138 | 0.019 | 0.132 |

Regression coefficients (β) and standard errors (SE) are adjusted for long-term time trend, temperature, relative humidity, weekends, and autocorrelation.
[a]PEFR is defined as deviation (%) from personal median. [b]Regression coefficients and SEs were calculated per interquartile range of each particle measurement. *$p < 0.05$; **$p < 0.01$.

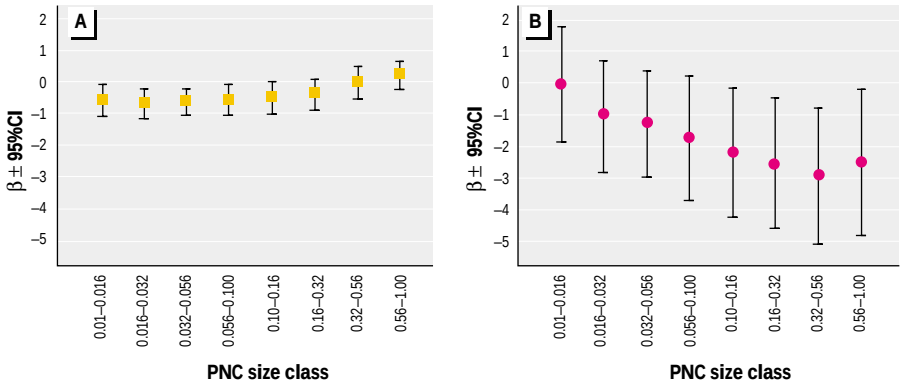


**Figure 1.** Adjusted regression coefficients (β) and 95% confidence intervals (CI) for the association of lung function with 5-day mean particle number concentrations in eight size classes. Lung function is defined as the percent of deviation from the individual median using (*A*) daily self-monitored mini-Wright PEFR measurements or (*B*) biweekly supervised spirometric PEFRs. Regression coefficients were calculated per interquartile range of each sizeclass.

**Table 6.** Association of PEFR[a] (spirometry) with the size classes of the particle number concentration (5-day mean).

| Size class (μm) | β[b] | SE |
|---|---|---|
| 0.010–0.018 | 0.03 | 0.93 |
| 0.018–0.032 | –0.96 | 0.90 |
| 0.032–0.056 | –1.23 | 0.86 |
| 0.056–0.100 | –1.68 | 1.01 |
| 0.10–0.18 | –2.13 | 1.05* |
| 0.18–0.32 | –2.49 | 1.06* |
| 0.32–0.56 | –2.89 | 1.12* |
| 0.56–1.00 | –2.46 | 1.19* |

Regression coefficients (β) and standard errors (SEs) are adjusted for long-term time trend, temperature, relative humidity, and diurnal variation.
[a]PEFR is defined as deviation (%) from personal median. [b]Regression coefficients and SEs were calculated per interquartile range of particle number concentration. **p* < 0.05.

In conclusion, the number concentrations of ultrafine particles in ambient air in Helsinki are comparable to concentrations measured at other urban sites, whereas the concentrations of accumulation mode and larger particles are generally lower. We observed inverse, mainly nonsignificant associations between spirometric lung function indices (FVC, $FEV_1$, and PEFR) and ultrafine and accumulation mode particle number concentrations in ambient air, but no association with coarse particles. These results support the need to monitor the size distribution and number concentrations of particles, in addition to mass, in ambient air.

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