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Short communication

Association of urban particle numbers and sources with lung function among children with asthma or allergies

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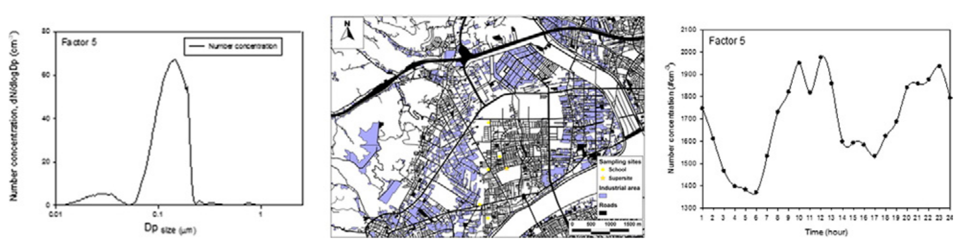
HIGHLIGHTS

- Particle numbers and sources were used for examining particle-induced health effects.
- Secondary particle is most responsible for deterioration in childhood lung function.
- Analyses that rely on only particle number may underestimate risks of particles.

GRAPHICAL ABSTRACT



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ABSTRACT

Previous studies have reported sources of particle number pollution in urban air, but have not evaluated relationships between respiratory health and these sources. We compared, among children with asthma or allergies, the associations of spirometric lung functions with increased exposure to source-specific versus size-segregated particle number concentrations (PNC). Hourly measurements of PNC were acquired from the aerosol Supersite in New Taipei, Taiwan. Spirometry (FVC, FEV_1, and FEF) was recorded monthly for 59 children with asthma or allergies at five schools during 2007–2008. After co-pollutant adjustment for ozone, we found a 0.21 and 0.17 L decrease in FVC [95% confidence interval (CI): −0.35, −0.06 L] and FEV_1 (95% CI: −0.32, −0.03 L), respectively, with an interquartile range increase (1879.7 #/cm^3) in secondary aerosol contribution observed on the previous day. In addition, we found no significant associations of FVC with accumulation mode (0.1 μm < aerodynamic diameter < 2.5 μm) PNC. Our findings suggest that PNC of secondary origin is most responsible for pollution-related respiratory effects among children living in urban Taipei. Studies that rely on exposure to size-segregated PNC may underestimate PM health impacts.

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1. Introduction

Ambient particulate matter (PM) air pollution is a well-documented environmental and public health problem (USEPA, 2009). Most studies on PM health risks have used the total mass within different size ranges as exposure indicators. However, there is an increasing interest in using particle number concentrations (PNC) as an alternative exposure indicator to assess air quality and associated health risks. In estimating risk of PM in terms of PNC, distributions of PNC data are commonly categorized into the nuclei/ultrafine [UFP; aerodynamic diameter (D_p) < 0.1 μm] and accumulation (AP; 0.1 μm < D_p < 2.5 μm) particle modes (USEPA, 2009). Alternatively, some studies categorized PNC into multiple size fractions with self-defined size bins, which differed considerably among studies (Leitte et al., 2011; Meng et al., 2013; Pekkanen et al., 1997; Penttinen et al., 2001a). Previous risk analyses using size-segregated PNC data have suggested that respiratory and cardiovascular effects might be associated with ambient UFP and/or AP levels, but no consistent and conclusive relationships have been determined (Andersen et al., 2008; Branis et al., 2010; Evans et al., 2014; Gong et al., 2014; Leitte et al., 2011; Meng et al., 2013; Pekkanen et al., 1997; Penttinen et al., 2001a; Penttinen et al., 2001b; von Klot et al., 2002). We hypothesize that the inconclusive finding is probably because these previous studies have not accounted for source differences in their examinations of PM health effects.

This hypothesis has been evaluated in studies using mass data resolved from source apportionment as inputs to epidemiologic models of the PM health effects (USEPA, 2009). One of the most common source apportionment methods is Positive Matrix Factorization (PMF), a factor analytic approach which seeks to partition the observed PM mass to its originating sources (Paatero and Tapper, 1994). These source-based studies have indicated clear association between human health effects and specific PM sources, especially primary combustions and secondary particles. Few studies, however, have used particle number data in their assessments of source-induced health effects (Yue et al., 2007).

This study examined the health effects of source-specific PNC. We decomposed urban PNC data into source factors using PMF and explored association between the source-specific PNC exposures and spirometric indices. In addition, we examined health effects of PNC for UFP, AP, and total particle (TP; the sum of UFP and AP) size fractions, in order to determine whether there are any differences in estimated risks between source-specific and size-segregated PNC impacts. This analysis is the first to apply source-specific PNC in the examinations of PM respiratory health effects in humans. In this Short Communication, we demonstrated this source apportionment-epidemiology approach through analyzing data collected in a longitudinal study of lung function in schoolchildren (Chen et al., 2011; Chen et al., 2014).

2. Materials and methods

2.1. Data collection

The study was conducted in Xinzhuang District, New Taipei, Taiwan, during September 2007–June 2008. The study area is a mixed-use residential and commercial district, with several heavily trafficked roads and two major highways passing through or in the periphery.

Respiratory health measurements were adopted from a previous study (Chen et al., 2011; Chen et al., 2014). Briefly, in September 2007, a pediatric health study of air pollution recruited 4221 children from three elementary schools and two middle schools in Xinzhuang District. In the present study, we focused on the 59 children who had current asthma and/or allergic rhinitis and participated voluntarily in a longitudinal follow-up lasting for nine months. Their respiratory health conditions were confirmed by an otolaryngologist at the beginning of the follow-up. Trained medical technicians visited children's schools once a month from October 2007 through June 2008 to perform spirometric testing of the children, following the protocols of American Thoracic Society (Miller et al., 2005). We recorded six measures of lung function: forced vital capacity (FVC), forced expiratory volume in 1 s (FEV_1), forced expiratory flow at 25%, 50%, and 75% of FVC ($FEF_{25\%}$, $FEF_{50\%}$, and $FEF_{75\%}$), and average expiratory flow over the middle half of FVC ($FEF_{25–75\%}$). Information on demographic characteristics, household exposures to environmental factors, and symptomatic attacks were obtained through questionnaires. The study was approved by the institutional review board of the National Taiwan University Medical Center, and informed consent was obtained from each child's parents.

Air pollutants and meteorological parameters were monitored at the Aerosol Supersite, operated by the Taiwan Environmental Protection Administration (TEPA), in Xinzhuang District. All the five schools in this study were located within a 2.5 km radius of the Supersite. Size distributions of PNC were measured by a scanning mobility particle sizer spectrometer (TSI Inc., St. Paul, MN), covering the 0.01–0.2 μm size range, and an optical aerosol spectrometer (PMS Co., Inc., Boulder, CO), covering the range between 0.2–2.5 μm. Ozone concentrations were measured at a TEPA air quality monitoring station nearby.

We computed hourly averages of air pollution data for source apportionment analysis. Hourly source-apportioned data were then pooled to form daily average concentrations for use in the risk analysis. Data between October and December 2007 were excluded from the risk analysis due to a significant amount of missing and abnormal values existing in the PNC size distributions. Spirometric tests were not performed during the Winter Break in January 2008. Thus, data from February 2008 through June 2008 were included in the source apportionment and risk analyses.

2.2. Statistical analysis

2.2.1. Source apportionment modeling

The PMF model was applied for source apportionment based on measurements at the Supersite. The input data for the PMF model included the size distributions of PNC and the corresponding uncertainties. The theoretical basis and implementation details are provided in the supplementary information (SI), Part I. Source apportionment procedures were performed in the EPA PMF software package version 3.0 (Norris et al., 2008).

2.2.2. Health effects modeling

The mixed-effects model was used for analyzing the association between lung function and exposure to source-specific PNC. Random effects were included to account for the repeated lung function measurements of each child. We first built single-pollutant models by individually adding each of the source factors or size-segregated PNC (TP, AP, and UFP) to the models. We then constructed two-pollutant models by adding ozone as a co-pollutant to the single-pollutant models, as our previous analysis identified that ozone was a significant gaseous pollutant affecting pediatric lung functions (Chen et al., 2011). Following the model setup of our previous analysis, a first-order autoregressive correlation structure was chosen as the assumed correlation between repeated health measurements of the same child. Similarly, the one-day lag exposure model was identified as producing the most stable results among the zero- to three-day lag assumptions in our previous analysis (Chen et al., 2011) and thus was adopted in the present study. Both the single- and two-pollutant models included covariate adjustments for gender, school, body mass index (the weight in kilograms divided by the square of the height in meters), upper-respiratory infection, asthma/allergic rhinitis symptomatic attack, medication use, parental education, parental atopy (a history of asthma, allergic rhinitis, or atopic eczema in parents), day of the week, and household exposure to environmental tobacco smoke. Analyses were performed using the software SAS (version 9.3; SAS Institute Inc., Cary, NC, USA). The regression coefficients and their 95% confidence intervals (CI) were presented as estimated change in lung function levels