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Pulmonary inflammatory effects of source-oriented particulate matter from California's San Joaquin Valley

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Abstract

The EPA regulates ambient particulate matter (PM) because substantial associations have been established between PM and health impacts. Presently, regulatory compliance involves broad control of PM emission sources based on mass concentration rather than chemical composition, although PM toxicity is likely to vary depending upon PM physicochemical properties. The overall objective of this study was to help inform source-specific PM emission control regulations. For the first time, source-oriented PM was collected from the atmosphere in Fresno, CA, onto 38 source/size substrates. Mice were exposed via oropharyngeal aspiration to equivalent mass doses [50 μg] of two size fractions: ultrafine (Dp < 0.17μm) and submicron fine (0.17 < Dp < 1 μm) during summer and winter seasons. At 24 hours post-exposure, cellular and biochemical indicators of pulmonary inflammation were evaluated in the bronchoalveolar lavage fluid. Significant inflammatory responses were elicited by vehicle, regional background, and cooking PM sources that were dependent on season and particle size. This is the first study of source-oriented toxicity of atmospheric PM and supports source-specific emissions control strategies.

Keywords

air pollution; PM; Fresno; ultrafine; submicron fine

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1. Introduction

There is substantial evidence that exposure to ambient particulate matter (PM[1]) can result in adverse cardiopulmonary health effects (1), but it remains unclear which PM characteristics directly contribute to these effects. Current mass-based national ambient air quality standards are derived from epidemiological correlations between PM mass and adverse health outcomes (1, 2). However, studies suggest that PM toxicity is not just a function of mass, but is also due to size and composition (3–5).

Since current PM regulation is based on mass, it does not distinguish between potential differential toxicity of different sources. Yet, emissions controls could be more cost-effective if they reduce the toxicity of atmospheric PM in addition to or instead of just decreasing PM mass. Testing the toxicity of source-oriented PM can increase understanding of the associations between adverse health effects and PM exposure, and support such source-oriented emissions control strategies.

Identifying specific PM sources that cause adverse health effects is confounded by the wide variety of sources that contribute to the atmospheric mixture and atmospheric processing that may alter composition after emission. To address this challenge, we have combined a novel source-oriented sampling technique with a sensitive murine bioassay to investigate the relative toxicity of source-oriented ambient PM collected in Fresno, CA, an urban area in the San Joaquin Valley. Fresno consistently has some of the highest levels of ambient PM concentrations and asthma rates in the state (6); a fifth (20.2%) of all children and adolescents suffer from asthma in Fresno County, in which the city resides, and it is a consistent Environmental Protection Agency designated non-attainment area for PM2.5 (7). The source-oriented sampling technique utilizes temporal and compositional patterns in PM profiles to conditionally sample PM from the ambient air. Thus, each sample represents a particle composition that is associated with specific local or regional sources. Most importantly, the ambient source-oriented PM used in this study accounts for atmospheric processing and represents true population exposure, neither of which can be reproduced in the laboratory or with direct emissions testing. In this study, source-oriented PM samples were collected in two size fractions, ultrafine (UF; Dp < 0.17μm) and submicron fine (SMF; Dp < 1μm) and in two seasons (summer and winter). The goal of the particle collection was to collect fractions of ambient air that were representative of a specific source or source combination.

2. Materials and Methods

2.1 Source-Oriented Particle Collection, Filter Extraction and Source Attribution

The source-oriented sampling technique was used during summer 2008 and winter 2009 to directly sample the ambient air in Fresno, CA. Particle collection was performed according to methods described in detail elsewhere (8). Source-oriented PM samples were collected in two size fractions, ultrafine (UF; Dp < 0.17μm) and submicron fine (SMF; Dp < 1μm). This

[1]Abbreviations: bronchoalveolar lavage fluid (BALF), particle diameter (Dp), Hank's Balanced Salt Solution (HBSS), polycyclic aromatic hydrocarbons (PAHs), particulate matter (PM), sub-micron fine source-oriented particles (SMF), ultrafine source-oriented particles (UF).

was done using a single particle mass spectrometer (RSMS-II) that provided the chemical composition of individual particles in real-time and controlled 10 high-volume ChemVol samplers assigned to predetermined single particle composition classes to collect UF and SMF PM (8, 9). Collected PM was removed from the collection substrates using novel filter extraction techniques designed to maximize extraction efficiencies and minimize compositional biases (9). The dominant particle types and source(s) collected for source-oriented toxicity testing are presented in Table 1. Nocturnal inversion emissions were collected during the same hours for both summer and winter.

The major sources of PM in the Fresno air shed during the sampling periods were vehicular emissions, residential heating, highly processed regional background, and residential and commercial cooking. A detailed, retrospective source attribution analyses of the source-oriented sampling experiments are presented elsewhere (10).

2.2 Experimental Design: Bioassay

Male BALB/C mice (9–10 week old) were purchased from Charles River Laboratories, Inc. (Raleigh, NC) and were allowed to acclimate for two weeks prior to the beginning of the study. Animals were handled in accordance with standards established by the US Animal Welfare Acts as set forth in the National Institutes of Health Guidelines and in accordance with EU Directive 2010/63/EU. All procedures were approved by the University of California, Davis, Institutional Animal Care and Use Committee.

Groups of six mice were assigned to vehicle control, filter blank control, or source-oriented PM-exposure groups. Vehicle control mice were exposed to 50 μl of sterile Hanks Balanced Salt Solution (HBSS). Filter blank control mice were dosed with a 50 μl HBSS extract of borosilicate glass microfiber filters for UF controls or polyurethane foam substrates for SMF controls. The size-fractioned source-oriented PM samples listed in Table 1 were extracted, re-suspended in HBSS, and sonicated for a minimum of one hour to disperse the PM into solution and minimize PM aggregation. Samples were vortexed until dosing. Dynamic light scattering measurements (Microtrac Particle Sizer, Microtrac Inc., Malvern, PA) of the PM suspensions taken after sonication showed particle size distributions with mean particle diameters ranging from 170–600 nm. Mice exposed to source-oriented PM were dosed with 50 μg of UF or SMF PM in 50 μl of sterile HBSS.

Vehicle control, filter blank control, or source-oriented PM was delivered to the lungs of anesthetized mice via oropharyngeal aspiration (11) after mice were anesthetized via inhalation of isoflurane with oxygen (3:1 ratio). Following dosing, mice were closely monitored until they regained normal activity.

Approximately 24 hours after dosing, mice were anesthetized by an interperitoneal injection of pentobarbital (65 mg/kg). Bronchoalveolar lavage fluid (BALF) was collected by lavaging the entire lung with two 1 mL aliquots of HBSS. Recovered BALF was centrifuged at 2000 RPM for ten minutes at 4°C to isolate the BALF supernatant for freezing at −80°C. Cell pellets were re-suspended in 0.5 ml HBSS to determine total cell count and cell viability via trypan blue exclusion with a hemocytometer (Sigma, St. Louis, MO). A minimum of 100 μl of the cell suspension was used to prepare cytospins using a Shandon

Cytospin (Thermo Shandon, Inc., Pittsburg, PA). Air-dried cytospins were methanol fixed and stained with DiffQuick® (International Reagent Corp, Kobe, Japan) to determine cell profiles via light microscopy (500 cells per slide).

2.3 Statistics

JMP statistical software was used for data analysis (JMP, SAS Institute Inc., Cary NC). Descriptive statistics were calculated for all cellular and biochemical data. Data were log transformed to meet requirements for statistical analysis. Data in tables and figures are expressed as mean values ± standard error (SE) with a minimum of n = 6 per experimental group and n = 42 vehicle controls. Summer and winter data were evaluated independently. First, overall effect of particle size and source were analyzed using two-way analysis of variance (ANOVA). Second, an independent variable representing particle size and source was used to perform a one-way ANOVA with Tukey's post-test for pair-wise comparisons. Differences were considered statistically significant when $p < 0.05$. One-way ANOVA was used to compare sources across seasons, when appropriate. Four PM sources had comparable composition during summer and winter seasons; therefore, the toxicity of these sources could be directly compared.

3. Results

Significant pulmonary inflammatory responses were observed following exposure to the source-oriented PM samples that varied by source, size, and season, as depicted in Figures 1–7.

3.1 Summer

Seven of the nine collected summer sources of PM significantly increased the number of inflammatory cells recruited to the lungs compared to the control (approximately $33x10^3$ cells/mL) (Figure 1A).

The three most potent summer samples for total inflammatory cell recruitment (beginning with the most potent) were (1) UF vehicular emissions [gas and diesel: 8 times control], (2) UF regional background [5 times control], and (3) SMF metal-enriched PM [5 times control]. Diesel enhanced vehicular SMF, residential cooking (UF and SMF), daytime UF and nocturnal inversion UF and SMF emissions also significantly increased total inflammatory cell recruitment, but to a lesser degree.

Certain summer sources elicited significant neutrophil influx into the lungs over control levels (approximately $4 x10^3$ neutrophils/ml). The three most potent summer sources for neutrophil recruitment were the same three potent sources for total inflammatory cell recruitment: (1) UF vehicular emissions [60 times control] (Figure 2A), (2) UF regional background PM [36 times control] (Figure 3A), and (3) SMF metal-enriched PM [28 times control] (Figure 4A). Other summer PM samples produced neutrophil levels that were 20 times control or lower, such as SMF vehicle emissions (Figure 2A), regional background (Figures 3A), and nocturnal inversion (Figure 5A). Eosinophil influx, BALF protein and LDH levels were also significantly increased by UF samples from summer vehicular

emissions (Figure 2B, C, and D, respectively) and regional background PM (Figure 3B and 3C, respectively).

3.2 Winter

Total inflammatory cells recruited to the lungs were significantly increased by four of the ten winter sources of PM tested (Figure 1B). The two most potent winter sources were vehicular (gas and diesel) and regional background emissions, similar to the pattern observed for the corresponding sources in the summer. The most potent size fraction for winter vehicular (gas and diesel) and regional background emissions was the SMF (approximately 5–6 times greater than control), in contrast to UF in summer. The next most potent winter samples were the UF fraction of daytime and nocturnal inversion (approximately 4–5 times control).

The same source-oriented winter PM samples that caused the greatest total inflammatory cell influx to the lung also caused the most neutrophil recruitment: (1) SMF regional background PM [50 times control] (Figure 3A), (2) SMF vehicular emissions [38 times control] (Figure 2A), (3) UF samples collected during the nocturnal inversion time period [37 times control] (Figure 5A) and (4) daytime [30 times control] (data not shown). Winter UF vehicle emissions also significantly increased the mean number of recovered neutrophils but to a lesser degree than the SMF vehicle emission fraction and did not significantly alter other endpoints (Figure 2). Regional background SMF elicited a significant increase in BALF protein levels but no other endpoints (Figure 3). Likewise, the UF fraction of winter nocturnal inversion PM significantly increased BALF protein levels compared to the control and the SMF fraction but not the other inflammatory endpoints (Figure 5).

3.3 Seasonal Comparisons of Vehicular, Regional Background, and Nocturnal Inversion Emissions

Vehicular and regional background UF emissions had comparable composition during summer and winter seasons; therefore, the toxicity of these sources could be directly compared, and summer was found to be more potent than winter for both sources. Summer UF vehicle emissions caused greater total cells ($p = 0.0099$), neutrophils ($p = 0.0459$), and LDH levels ($p = 0.048$) than winter UF vehicle emissions. Similarly, summer regional background UF was significantly more potent for total cells ($p = 0.008$) and eosinophils ($p = 0.0027$) than winter regional background UF.

In contrast, winter regional background SMF and winter nocturnal inversion UF caused significantly greater levels of total cells (winter nocturnal inversion UF, $p=0.0153$), neutrophils (winter regional background SMF, $p = 0.0426$), and BALF protein (winter regional background SMF, $p = 0.0385$; winter nocturnal inversion UF, $p = 0.0275$) than their summer counterparts.

3.4 Unique Seasonal Sources: Winter Biomass Combustion, Summer Cooking Emissions, and an Unknown Metals-Rich Source

Both winter biomass combustion and residential heating emissions significantly increased neutrophil influx regardless of PM size (Figure 6A). Residential heating emissions,

representing local and relatively unprocessed biomass combustion, in the UF size fraction also significantly increased eosinophil influx compared to the control and the corresponding SMF fraction (Figure 6B). No significant differences in BALF protein or LDH levels were observed.

In summer, cooking emissions from residential and commercial sources caused an increase in pulmonary inflammatory endpoints. Residential cooking emissions in both PM size fractions significantly increased total inflammatory cell (Figure 1A) and neutrophil influx (Figure 7A) compared to control levels. Residential cooking emissions also significantly increased neutrophil influx compared to the corresponding commercial cooking emissions. No significant differences in eosinophil influx, BAL protein, or LDH levels were observed.

An unknown metals-rich source in the summer significantly elevated levels of total cells and neutrophils (Figure 1A and 4A, respectively), but only the SMF fraction caused an increase in BAL protein levels (Figure 4C).

4. Discussion

The complex composition of ambient PM confounds efforts to identify which chemical, physical, and/or other characteristics drive adverse health effects. There is an urgent need to distinguish the most toxic sources of PM so that air quality can be improved and public health better protected. In this study, source-oriented PM was collected in Fresno, CA to evaluate pulmonary inflammatory and cytotoxic impact of exposure. The PM was characterized by source, size (UF and SMF), and season (summer and winter). Several summer and winter sources of PM were found to elicit significant pulmonary inflammatory responses *in vivo*. Regardless of season, vehicular emissions, and regional background PM were two of the most toxic sources evaluated in the study. These same two sources were similar in that the UF fraction of these two sources was more potent during summer, while the SMF fraction was more potent during winter. Summer sources of PM associated with significant pulmonary responses were vehicular emissions, regional background PM, residential cooking, and an unknown metals-rich source. Winter sources of PM associated with significant inflammatory responses were vehicular emissions, regional background PM, and nocturnal inversion PM. This data provides critical insight into how specific PM sources and size fractions might be associated with selected pulmonary injury and inflammatory responses. However, exposure to ambient PM is associated with a wide variety of adverse health outcomes, and it is important to note that relative toxicities are likely to vary based on the biological endpoints evaluated.

Vehicular emissions were a potent source during both summer and winter but there was a seasonal difference in which size fraction elicited the greatest pulmonary injury and inflammation. This difference may be due to partitioning of semi-volatile compounds, especially polycyclic aromatic hydrocarbons (PAHs), between the gas and particle phases. During cold, humid conditions that prevail in Fresno in the winter, these compounds partition into the particle phase; the lower temperature and higher humidity may grow particles from the UF to SMF size ranges. Vehicle emissions are rich in elemental carbon,

which can most likely be attributed to diesel emissions; however, they may also originate from light duty vehicle engines used in some landscaping equipment.

Regional background PM was also a highly potent inflammatory source during both summer and winter seasons. The inflammatory patterns observed in response to regional background PM was similar to the pattern observed with vehicular emissions; the UF fraction was the most potent in summer and the SMF fraction was the most potent in winter. Regional background PM arises from a mixture of primary emissions from vehicular and agricultural sources that have been highly processed in the atmosphere to form secondary emissions, such as ammonium nitrate and secondary organic aerosol that are transported throughout the region (10). A key aspect of this research was capturing atmospheric processing, which cannot be reproduced in the laboratory and is neglected in direct emissions testing. While not as technically source-specific as other sources in this study, regional background PM is certainly relevant to the regulatory conversation and probably one of the more complicated issues at hand.

Significant pulmonary inflammatory and cell damage were observed in response to PM collected during the winter nocturnal inversion, with UF being the more potent size fraction for this source. Nighttime nocturnal inversion PM was collected during periods when the source mixture could not be definitively discerned or did not match one of the predetermined source combinations assigned to the source-oriented samples. Sampling occurred continuously during the hours of 17:00 and 9:00. The nighttime inversion layer is characterized by decreased mixing with the upper atmosphere, as emissions are trapped close to the earth’s surface. This meteorological process is common in the San Joaquin Valley and has the potential to increase pollutant concentrations and reduce the potential for exposure to secondary organic aerosol due to reduced atmospheric processing (12). Therefore, the nighttime inversion PM is relatively unprocessed and represents contributions from local sources.

PM size significantly influenced toxicity of several summer and winter sources. Previous reports from the San Joaquin Valley suggest that different sources emit PM with certain size profiles (13). The size distribution is likely impacted by seasonally dependent meteorological conditions that alter the production of secondary compounds that grow emitted particles.

Unique seasonal patterns of PM toxicity were noted, such as biomass combustion emissions (Figure 5) and summertime cooking. In this study, the residential heating category represents primary, unprocessed biomass combustion emissions from residential neighborhoods immediately surrounding the sampling site in Fresno. In contrast, the biomass combustion category represents highly processed emissions originating from regional scale sources. The highly processed biomass combustion emissions could originate from local sources if they were trapped aloft, mixed with regional source emissions, processed in the atmosphere and recirculated. Biomass combustion emissions related to residential heating were highly inflammatory, inducing a significant neutrophil and eosinophil influx to the lung. Biomass combustion emissions are derived from the use of woodstoves and fireplaces for residential heating during colder winter months, with

emissions beginning around 19:00 and decreasing towards the early morning hours (10). Wood smoke emissions contain significant amounts of organic carbon and secondary components. Eosinophil recruitment associated with exposure to the UF fraction of residential heating emissions, suggests that this source may pose a particular risk for those with existing allergic conditions (12). Zelikoff et al. (14) summarized a number of wood smoke exposure studies and concluded that prolonged wood smoke exposure, especially by children, can contribute to reduced pulmonary function and other respiratory symptoms. Wood smoke emissions are currently targeted by the San Joaquin Valley Air Pollution Control District with a “check before you burn” program that restricts wood-burning at times when meteorological patterns are likely to trap emissions close to the surface.

Residential cooking emissions collected in this study were mainly from backyard barbequing during the summer in the neighborhood of the sampling site. These emissions were rich in soot and potassium, low in organic species, and did not contain any secondary components, indicating that the particles came from a local primarily summertime source (10). The high potency associated with residential cooking emissions compared to commercial cooking emissions may be related to the heat source – residential cooks typically use solid fuels while commercial cooks typically use natural gas.

During summer, emissions from an unknown source combination enriched in metals (specifically zinc and lead) were highly inflammatory in both PM size fractions. The SMF fraction elicited significant cell damage. Metals have been implicated in a range of pulmonary health effects in numerous studies (3, 15, 16). This source was observed fairly infrequently and during the early night hours (21:00 – 1:00), and while the particular sources of this enriched metal PM are unclear, they appear to originate from three separate local, residential combustion sources (8, 10).

In conclusion, different source-oriented and size-segregated samples of sub-micron PM elicited differing levels of response in an array of toxicity measures, supporting the founding hypothesis for this study that different PM sources and their combinations have different levels of toxicity and are toxic in different ways.

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Highlights

- Toxicity of source-oriented PM collected in Fresno, CA, varied by size and season
- Vehicle, regional background, and cooking PM sources caused pulmonary inflammation
- Source-oriented PM control strategies may be more appropriate than mass-based

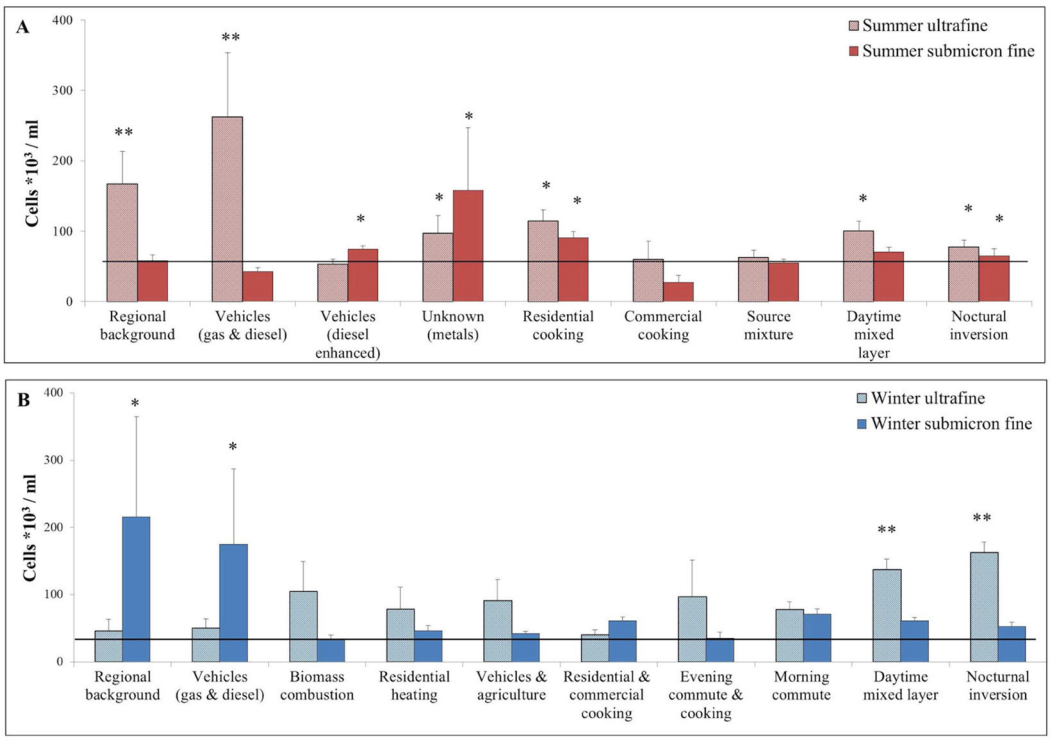

Figure 1. Total pulmonary cells recovered in BALF 24 hours post-exposure to 50 μg summer (A) or winter (B) UF (light shade) or SMF (dark shade) source-oriented PM

Asterisks (* $p < 0.05$; ** $p < 0.0001$) indicate significant differences from control (solid line). Data expressed as mean ± SEM.

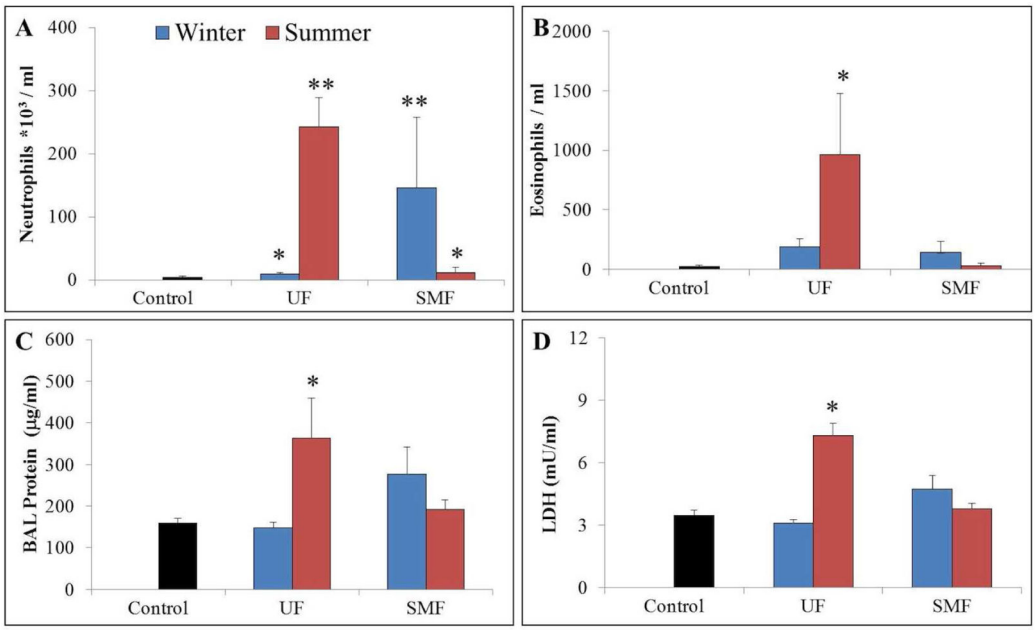

Figure 2. Pulmonary response to summer and winter vehicle emissions

(A) Neutrophils, (B) Eosinophils, (C) BALF protein, and (D) LDH, expressed as mean ± SEM, recovered in BALF 24 hours post-exposure to 50 μg winter (blue) or summer (red) UF or SMF vehicle emissions. Asterisks (* $p < 0.05$; ** $p < 0.0001$) indicate significant differences from control (black bar).

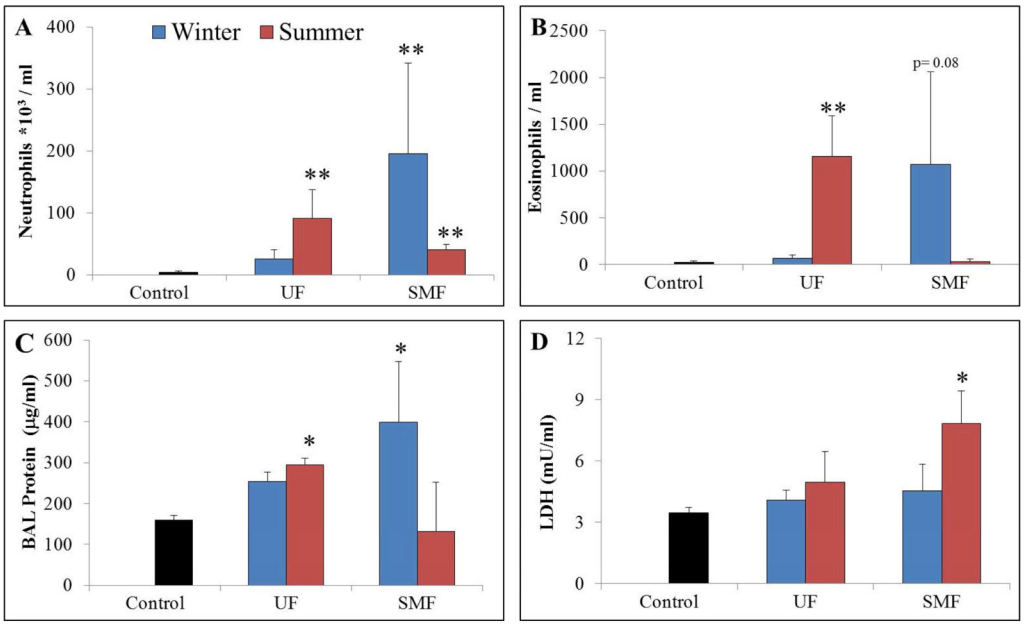

Figure 3. Pulmonary response to summer and winter regional background PM

(A) Neutrophils, (B) Eosinophils, (C) BALF protein, and (D) LDH, expressed as mean ± SEM, recovered in BALF 24 hours post-exposure to 50 µg winter (blue) or summer (red) UF or SMF highly processed regional background PM. Asterisks (* $p < 0.05$; ** $p < 0.0001$) indicate significant differences from control (black bar).

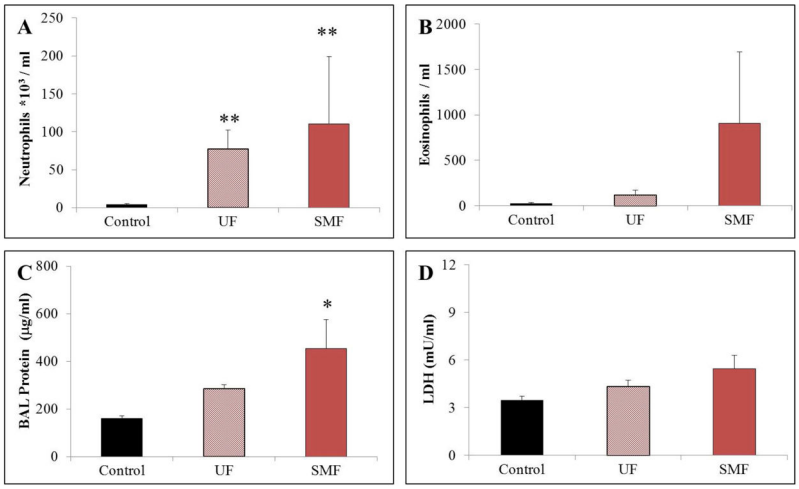

Figure 4. Pulmonary response to unknown metals-rich source

(A) Neutrophils (B) Eosinophils (C) BALF protein, and (D) LDH, expressed as mean ± SEM, recovered in BALF 24 hours post-exposure to 50 µg summer UF (light red) or SMF (dark red) metal-rich PM. Asterisks (* $p < 0.05$; ** $p < 0.0001$) indicate significant differences from control (black bar); # indicates significantly different from the SMF fraction as indicated by brackets.

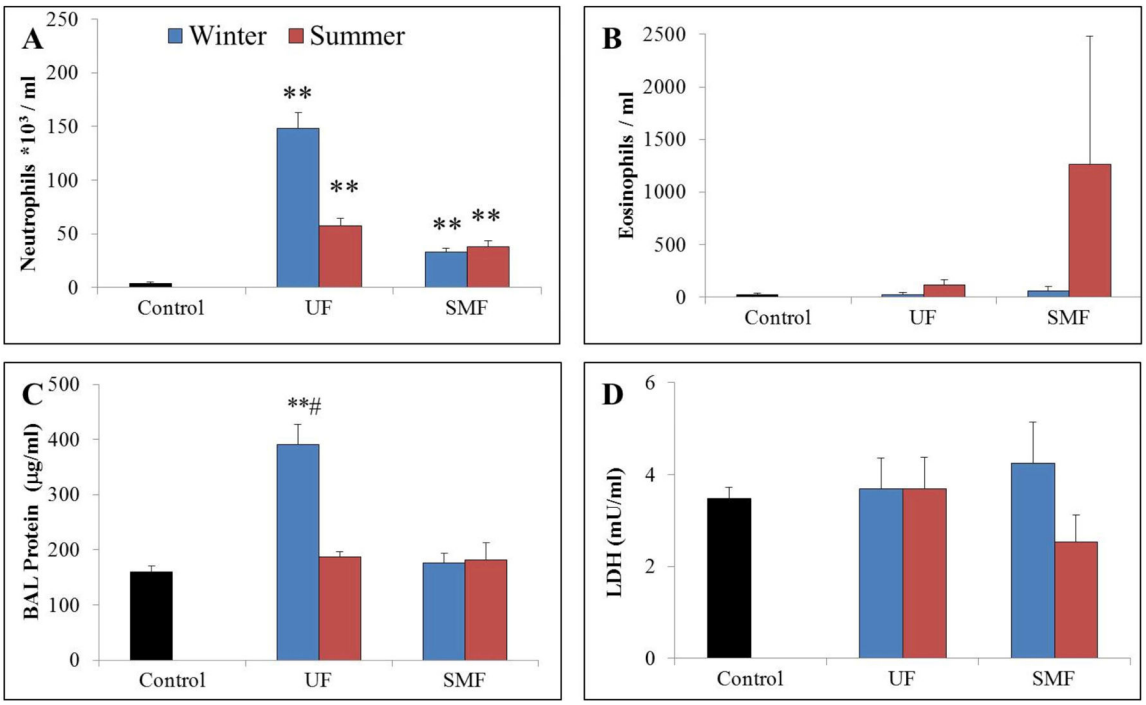

Figure 5. Pulmonary response to summer and winter nocturnal inversion PM

(A) Neutrophils, (B) Eosinophils, (C) BALF protein, and (D) LDH, expressed as mean ± SEM, recovered in BALF 24 hours post-exposure to 50 µg winter (blue) or summer (red) UF or SMF nocturnal inversion PM. Asterisks (* $p < 0.05$; ** $p < 0.0001$) indicate significant differences from control (black bar); # indicates significantly different from the SMF fraction.

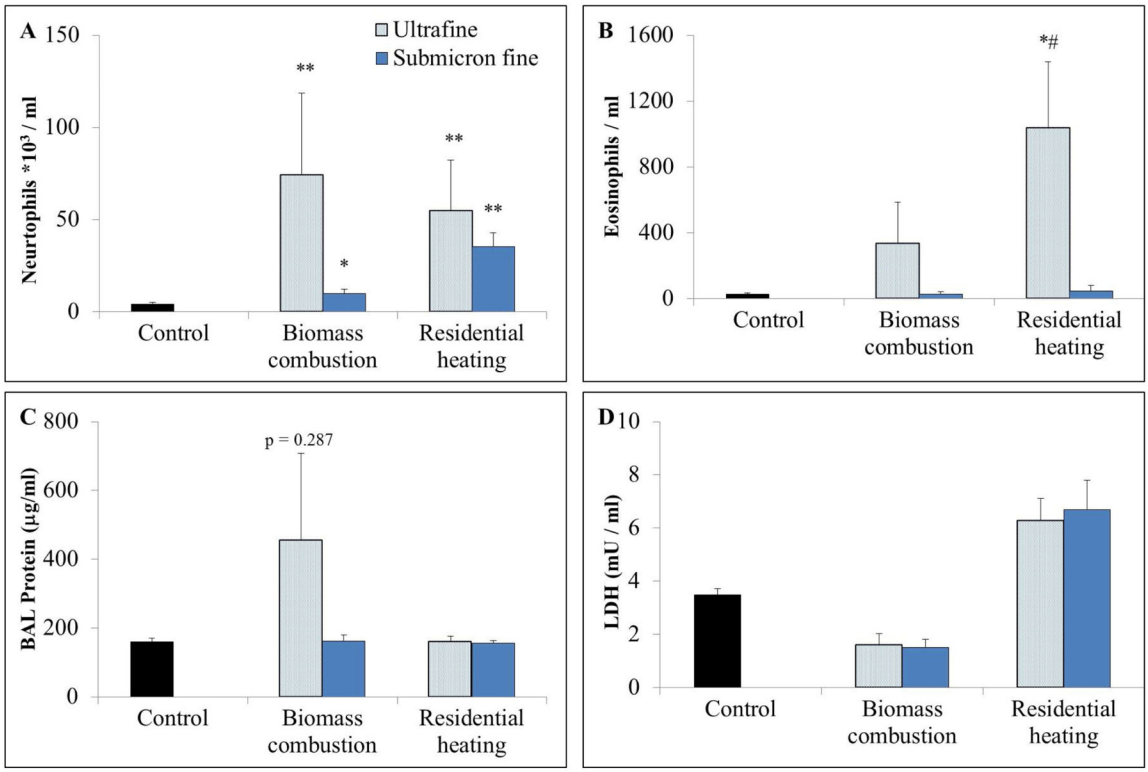

Figure 6. Pulmonary response to winter biomass combustion emissions related to residential heating

(A) Neutrophils, (B) Eosinophils, (C) BALF protein, and (D) LDH, expressed as mean ± SEM, recovered in BALF 24 hours post-exposure to 50 mu;g or summer UF (light blue) or SMF (dark blue) biomass combustion and residential heating PM. Asterisks (* $p < 0.05$; ** $p < 0.0001$) indicate significant differences from control (black bar); # indicates significantly different from the SMF fraction from the same source.

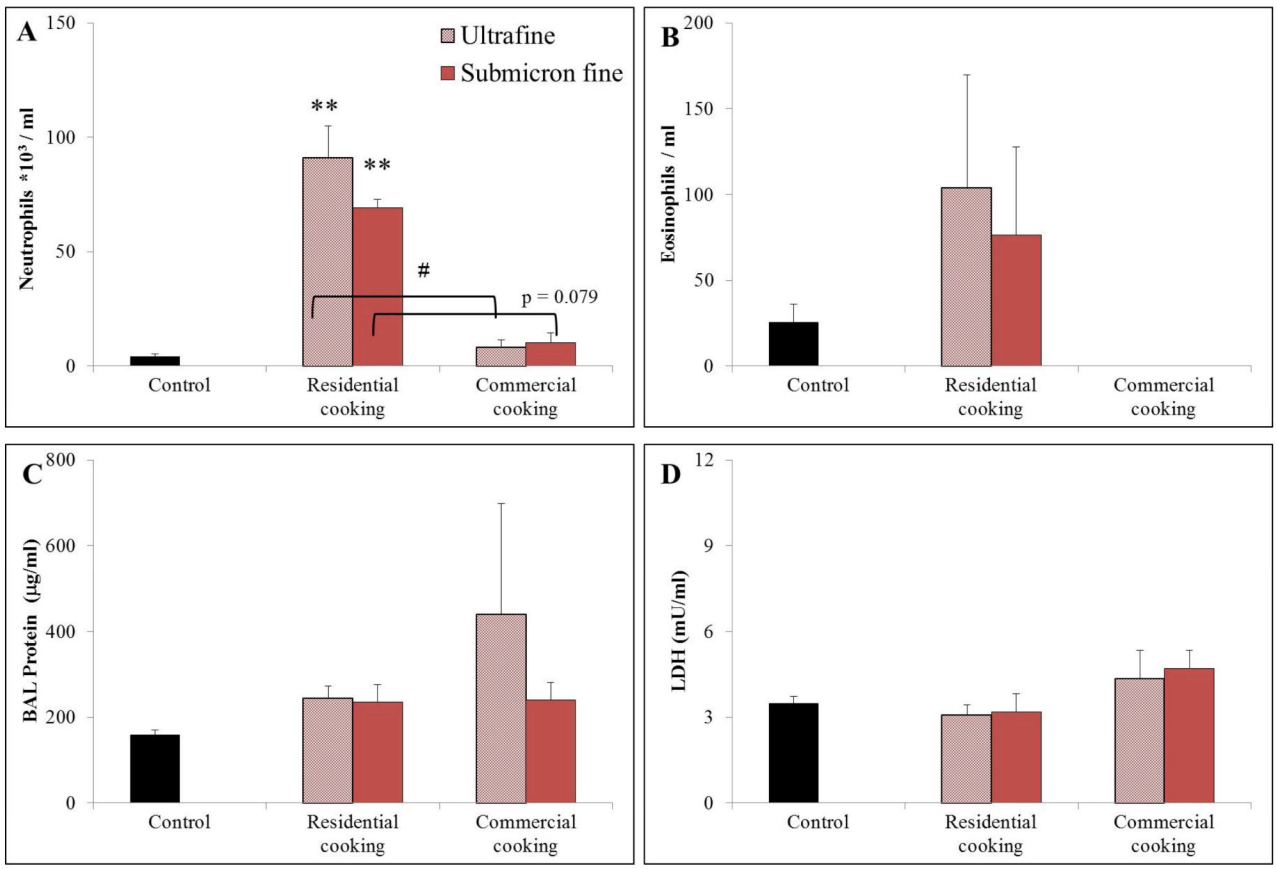

Figure 7. Pulmonary response to summer residential and commercial cooking emissions

(A) Neutrophils, (B) Eosinophils, (C) BALF protein, and (D) LDH, expressed as mean ± SEM, recovered in BALF 24 hours post-exposure to 50 µg summer UF (light red) or SMF (dark red) residential and commercial cooking PM. Asterisks (* $p < 0.05$; ** $p < 0.0001$) indicate significant differences from control (black bar); # indicates significantly different from the SMF fraction as indicated by brackets.

Table 1

Summary of the dominant source(s) and particle type(s) collected for source-oriented toxicity testing.

ChemVol Number*	Summer		Winter	
	Dominant Particle Type(s)	Dominant Source(s)	Dominant Particle Type(s)	Dominant Source(s)
1	K	Residential cooking	K/EC/OC	Residential heating
2	CAN	Regional background	CAN	Regional background
3	EC	Vehicles (diesel enhanced)	EC; EC/OC	Vehicles (gas & diesel)
4	CAN; K; EC/OC	Source mixture	K/CAN	Biomass combustion
5	EC; EC/OC	Vehicles (gas & diesel)	CAN; K/CAN	Vehicles and agriculture
6	Metals	Unknown (metals)	K/EC/OC	Residential and commercial cooking
7	K; Na/K	Commercial cooking	Operated ~ 17:00–20:00	Evening commute and cooking
8	Not used	Not used‡	Operated ~ 06:00–09:00	Morning commute
9	Operated ~ 11:00–15:00	Daytime mixed layer	Operated ~ 09:00–17:00	Daytime mixed layer
10	Uncertainty ChemVol	Nocturnal inversion	Uncertainty ChemVol	Nocturnal inversion

*ChemVol number is included in this table to provide easy references to related publications.

‡there were not enough source combinations identified during the summer to use all 10 ChemVols in the sampling train (8); K, potassium; CAN, carbonaceous ammonium nitrate (8); EC, elemental carbon; OC, organic carbon; Na, sodium;