Original Article

Impact of Traffic Emissions on Local Air Quality and the Potential Toxicity of Traffic-related Particulates in Beijing, China*

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Abstract

Objective Air-borne particulates from different sources could have different physicochemical properties and inflammatory potentials. This study aims to characterize the chemical compositions and the toxicity of ambient particulate matter (PM) associated with traffic emissions.

Methods The concentrations of trace elements, organic carbon (OC), elemental carbon (EC) and polycyclic aromatic hydrocarbons (PAHs) in $PM_{2.5}$ and PM_{10} were measured in samples collected at sites in Beijing, China. Their toxic effects on the pulmonary system of rats were investigated. Biochemical parameters (LDH, T-AOC, TP) and inflammatory cytokine(IL-6, IL-1, TNF-α) levels were measured in the lungs of rats exposed to traffic-related PM. Oxidative damage was observed. PM samples were taken from a near road site and an off road site in summer time in 2006.

Results The concentrations of the USEPA priority pollutant PAHs in both PM_{10} and $PM_{2.5}$ were higher (299.658 and 348.412) at the near road site than those (237.728 and 268.472) at the off road site. The similar trend was observed for the concentrations of trace elements in PM. Compared to coarse particles (PM_{10}), fine particles ($PM_{2.5}$) have a greater adsorption capacity to enrich toxic elements than inhalable particles. Decrease in antioxidant capacity and an increase in the amount of lipid peroxidation products in rat lung tissues was observed.

Conclusion The findings of the present study suggest that the differing inflammatory responses of PM collected from the two road sites might have been mediated by the differing physicochemical characteristics.

Key words: Traffic-related PM; PAHs; Trace elements; Pulmonary toxicity

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INTRODUCTION

Recent epidemiological studies have provided ample evidence on a positive association between the inhalation of particulate matter (PM) and serious adverse health effects[1-4]. Many countries have adopted the World Health Organization (WHO) air quality guidelines which use the mass concentration of fine particles less than 2.5 μm in diameter ($PM_{2.5}$) or less than 10 μm in diameter (PM_{10}) as indicators of health risk. Current EU policies are targeted to reduce emissions of PM in general, but the current EU standards were established only for PM_{10} (annual average of 40 mg/m^3; daily average of 50 mg/m^3, with a permitted excess of 35 mg/m^3 per year). And so far,

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it is unclear whether the reduced ambient concentrations of PM_{10} would proportionally decrease its adverse health effects.

Many studies revealed that considerable heterogeneity in the characteristics of PM is attributed to differences in PM sources, composition and toxicity[5-6]. Changes in chemical composition of PM may change its impact on human health. To develop cost-effective policies for reducing PM and thus protecting public health, the PM fractions such as coarse particles, fine particles and ultrafine particles that have the most serious impact on health need to be identified based on their physicochemical and toxicological characteristics.

Traffic is known to be an important contributor to PM exposure. Therefore, it is important to understand the relationship between traffic emissions and characteristics of PM in the atmosphere[7-12]. In recent years, epidemiological studies have highlighted a possible link between traffic emissions and adverse health effects. The association of residential exposure to traffic with 8-year lung-function growth has been reported lately, which exhibits that local exposure to traffic on a freeway has adverse effects on children's lung development[13] . Hoek et al. 2002[14] reported the relationship between traffic-related air pollution and mortality in participants of the Netherlands Cohort study on Diet and Cancer (NLCS). Kan et al. (2007)[15] analyzed the relationship between traffic exposure and lung function, and the findings remained inconclusive in adults. Traffic emissions can affect both the amount and the properties of ambient PM, including its chemical, physical and toxicological characteristics[16-18]. The polycyclic aromatic hydrocarbon (PAH), and heavy metal concentrations and the radical generating capacity of PM are particularly,related to traffic intensity according to previous several studies[19-23].

Pulmonary inflammation, including the release of pro-inflammatory mediators and the recruitment of inflammatory cells,is known to be an important step in the body's response to particulate matter inhalation[24-25]. However, the quantitative study on the relationship between the physicochemical properties of PM and the impact on their inflammatory potential is relatively scarce.

In this study, we measured the PAH and heavy metal concentrations of PM associated with traffic emissions in a selected road sites in some areas in Beijing, China. We also investigated the toxicity of PM by studying its effects on the lungs of rats in this area.

MATERIALS AND METHODS

Sample Collection

$PM_{2.5}$ and PM_{10} samples were collected from two road sites-one located near the Beiyuan Highway (referred to as the Highway site) with high traffic intensity and another situated away from traffic on an orchard (referred to as the Orchard site) (Figure 1). The vehicle flux on the Beiyuan Highway was 2639±1491.9/h during the study period, and the two sampling sites were about 200 m apart. The off road was sited northeast to the near road site. The wind directions and wind speeds of the two monitoring sites are listed in Table 1.

Table 1. The Wind Directions and Wind Speeds of the Two Monitoring Sites in August 1-30, 2006

Groups		N	NE	E	ES	S	WS	W	WN
Highway Site	Wind direction frequency	0.4	2.9	26.3	11.7	4.1	4.1	7.5	3.8
	Wind speed	2.1	1.1	1.6	1.9	1.8	1.7	1.5	1.1
Orchard Site	Wind direction frequency	0.4	2.7	24.6	10.9	3.9	3.9	7.0	3.5
	Wind speed	2.1	1.1	1.6	1.9	1.8	1.7	1.5	1.1

At each road site, two samplers were installed on 1.5 m height platforms, and 24 h samples were collected each day in August 01-30, 2006. The samples for chemical analysis were collected onto 47mm quartz filters (Adventec, UK) using a sampler made by Beijing Electrical Technology Co., Ltd with a flow rate of 77.7 L/min. For the toxicity studies, the PM samples were collected using a high-volume sampler (U.S. General), with a flow rate of 1500 L/min.

The filters were pre-heated at 600 °C for 2 h before sampling, to destroy any adsorbed organic compounds. They were conditioned in an electronic dessicator both before and after sample collection for 24 h and then weighed on a balance with precision of 0.1 mg (Shimadzu, Japan, AUX220). After collection, the loaded filters were stored in a freezer at about -24 °C until chemical analysis was conducted to limit the evaporation of volatile components. The filters were sectioned for individual analyses.

Figure 1. Maps of sampling sites near the 5th Beijing Ring Road (Beiyuan Highway and orchard). A: street map; B: satellite map; ■ Construction site.

PAH Analysis

Prior to the PAH analysis, the filters were cut in half, with one half extracted ultrasonically with dichloromethane (DCM), concentrated using a rotary evaporator, and purified using a silica gel cleanup technique[26]. PAH analysis was then conducted using a gas chromatograph (Thermo Finnigan DSQ Company, Trace GC2000) coupled with a mass spectrometer (Thermo Finnigan DSQ Company, Trace DSQ) run in selected ion monitoring (SIM) mode. The compounds of interest were separated on a HP-5MS capillary column (30 m × 0.25 mm × 0.25 μm, 19091s-433, Agilent). The flow rate of carrier gas was 1 mL/min, and the mass spectrometer was operated in the electron-impact mode (70 eV).

Elemental Analysis

Another half of the filters were digested with 3 mL concentrated HNO_3, 1 mL concentrated HCl, and 1 mL concentrated HF. The solutions were then evaporated and diluted to 10 mL with deionized water. The elemental concentrations were determined using inductively coupled plasma atomic emission spectroscopy (ICP-OES, Agilent 700, USA). For the blank, an unused membrane was processed and analyzed using the same methods as those for real samples. The elemental concentrations were subtracted from the sample concentrations.

Enrichment factors (EF) for each element were calculated by:

$$EF=(C_X/C_{Fe})_{PM}/(C_X/C_{Fe})_{crust}$$

Where: C_X is the concentration of the element in the PM; and C_{Fe} is the abundance of Fe in the crust. Fe was used as the reference material in this study.

The filters were analyzed with thermal/optical reflectance (TOR) method for organic carbon (OC) and elemental carbon (EC). The difference determined from replicate analyses was smaller than 10% for OC and EC.

PM Suspension Preparation

The 30 filtration membranes collected using the High-Volume Sampler were recovered from filters by sequential sonications (four cycles of 20 min each at 10000×g at 4 °C) in sterile water according to Francesca et al.[27]. The supernatant was removed, and PM was maintained in a low temperature refrigerator for further study.

Animal Treatment

Thirty healthy male Wistar rats (8 weeks of age, weighing 180-220 g) were obtained from the Academy of Military Medical Sciences (Beijing, China). All procedures concerning the usage of animals were reviewed and approved by the Institutional Animal Care and Use Committee of the Academia. The rats were randomly divided into five groups: (1) the control group; (2) the Highway site $PM_{2.5}$ group; (3) the Highway site area PM_{10} group; (4) the Orchard site area $PM_{2.5}$ group; and (5) the Orchard site area PM_{10} group. The rats were anaesthetized with ether, and exposed to the PM suspension through intratracheal instillation at a dose of 7.5 mg/kg BW. The control group was treated with 7.5 mg/kg BW of normal saline. Each group received an intratracheal instillation once per day for 14 days.

Analysis of Biomarkers

Collection and Analysis of BALF 24 h after the final intratracheal instillation, rats were anaesthetized with ether, bled from the femoral artery and sacrificed by cervical decapitation. The lung and trachea were exposed by dissection, and the left lung was temporarily clamped. The right lung was lavaged with 6 mL of warm normal saline, and the recovered bronchoalveolar lavage fluids (BALF) were centrifuged at 400×g for 10 min. The concentrations of lactate dehydrogenase (LDH), total antioxidant capacity (T-AOC) and total protein (TP) in the BALF were analyzed using biochemical analysis kits (Shangbo, Beijing), and the results were measured by UV/VIS spectrometry (Shimadzu, UV-2550).

Collection and Analysis of Lung Homogenates The left lungs of the rats were excised, immediately cooled in ice, and homogenized in Teflon-glass homogenizer. The homogenates were then centrifuged at 700×g for 15 min. The levels of interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) in the lung homogenates were analyzed using Enzyme Linked Immunosorbent Assay (ELISA) kits (Shangbo, Beijing), and the results were measured using an ELISA Reader.

Histopathological Evaluation The middles of the left lungs were embedded in paraffin, thin-sectioned coronally, and the sections were then stained with hematoxylin-eosin and were examined by light microscopy.

Statistical Analyses

The data are reported as mean±standard deviation (SD). All statistical analyses were performed using SPSS software, version 13.0 (SPSS Inc., Chicago, IL, USA). A one-way analysis of variance (ANOVA) and Bartlett's test were calculated for each sampling value. If the F-statistic from the ANOVA was significant, Dunnett's test was used to compare the means from the control group to each of the groups exposed to the particulates. Differences in the means were considered significant if $P<0.05$.

RESULTS

PAH Concentrations in PM

130 PAHs were detected in the four types of samples from the two sites. According to the size distribution, PAHs were divided into 10 stages (Figure 2). Other PAHs was the most predominant which comprised many kinds of USEPA priority PAH, followed by Chrysene and Pyrene.

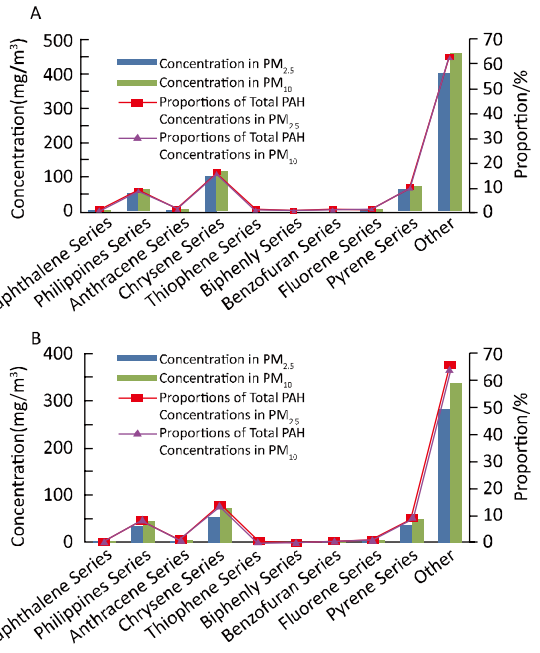

Figure 2. PAHs concentrations and proportions in PM_{10} and $PM_{2.5}$. (A) Highway site PM; (B) Orchard site PM.

The USEPA priority PAH concentrations in the PM from the Highway sites and the Orchard sites are shown in Table 2. The average total USEPA priority PAH concentrations ranged from 237.72 to 348.41 ng/m^3. For the whole data set, the concentrations of all the USEPA priority PAHs except naphthalene were greater than 1 ng/m^3.

Benzo[g,h,i]perylene (BghiP), a highly carcinogenic compound, had the highest levels for all groups, with the average levels over an experimental group ranging from 46.160 to 76.106 ng/m^3. Benzo[k]fluoranthene (BkF) and Benzo[b] fluoranthene (BbF) were also detected at relatively high concentrations.

For all samples, especially those from the Highway site, the high molecular weight homologs (4-6 rings) had higher concentrations compared to the low molecular weight homologs, indicating that combustion of petroleum fuels might be the potential sources of PAHs and PM measured at this area (Table 2). When comparing samples from the two sites, the Highway site usually had a higher concentration for each PAH compound. The average concentrations of total USEPA priority PAHs for PM_{10} and $PM_{2.5}$ at the Highway site were 348.412 and 299.658 ng/m^3, respectively, which were about 1.3 times higher than those at the Orchard site.

PM Elemental Concentrations

Different proportions of 22 element species were detected in the four sample types ($PM_{2.5}$ and PM_{10} from the two sites). As shown in Table 3, for each sample group, the EFs of As, Cd, Cu, Zn, S, and Pb were higher than 10. Sulfur was found to be the most enriched

Table 2. USEPA Priority PAH Concentrations in PM_{10} and $PM_{2.5}$ (n=5, ng/m^3)

PAHs	Highway Site		Orchard Site	
	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}
Fluorene	2.357±0.264	2.543±1.430	1.485±0.641	1.954±0.958
Phenanthrene	18.334±7.395	19.946±10.857	13.187±10.039	16.705±11.382
Naphthalene	0.542±0.381	0.423±0.115	0.253±0.138	0.475±0.381
Pyrene	39.970±13.482	38.432±20.974	30.452±20.004	35.294±14.264
Benzo[a]pyrene	11.432±7.342	14.057±11.351	8.751±3.582	9.268±4.889
Indeno[1,2,3-cd]pyrene	19.219±10.649	23.224±17.372	13.218±10.742	14.323±10.391 [a]
Anthracene	1.362±0.472	1.466±0.589	0.894±0.365	1.255±0.674
Benz[a]anthracene	8.519±3.841	11.078±9.379	7.255±2.099	8.614±2.481
Dibenz[a,h]anthracene	8.262±4.682	11.346±7.325	6.424±2.964	7.074±2.275
Fluoranthene	36.965±21.084	36.722±18.490	28.673±12.298	33.360±15.427
Benzo[b]fluoranthene	20.835±9.839	25.697±14.733	19.346±10.946	20.404±10.058
Benzo[k]fluoranthene	26.118±17.361	33.728±21.845	22.841±10.573	24.340±16.539
Benzo[ghi]perylene	61.376±22.375	76.106±40.197	46.160±21.465 [a]	48.524±15.327 [a]
Chrysene	44.279±21.479	53.652±26.395	38.805±14.271	46.884±33.945
Total	299.658±114.976	348.412±138.954	237.728±138.436 [a]	268.472±139.825

Note. [a]P<0.05 between different sampling sites.

Table 3. Major Element Concentrations and EFs in PM_{10} and $PM_{2.5}$ (n=5, μg/m^3)

	Highway Site				Orchard Site			
	$PM_{2.5}$	EF	PM_{10}	EF	$PM_{2.5}$	EF	PM_{10}	EF
As	0.022±0.015	647.8±365.9	0.030±0.017	340.5±131.3	0.015±0.006	556.5±152.1	0.018±0.006	353.3±145.8
Cd	0.003±0.001	673.4±238.7	0.003±0.000	470.9±243.5	0.003±0.001	583.5±263.7	0.003±0.000	317.6±189.3 [a]
Cr	0.043±0.016	18.1±6.0	0.059±0.026	11.3±5.3	0.023±0.011	20.9±11.4	0.032±0.016	13.0±5.8
Cu	0.077±0.039	144.7±59.7	0.127±0.047	93.6±42.4	0.113±0.047	56.4±24.9 [a]	0.162±0.069	40.5±29.1 [a]
Fe	1.301±0.731	1.0±0.6	2.936±0.949	1.0±0.5	0.721±0.228	1.0±0.3	1.598±0.574 [a]	1.0±0.3
Ni	0.002±0.000	1.0±0.3	0.009±0.004	1.0±0.4	0.001±0.000	1.3±0.4	0.002±0.000	1.5±1.3
Pb	0.128±0.057	559.5±325.0	0.199±0.042	341.1±115.9	0.090±0.0310	424.1±119.0 [a]	0.124±0.084	297.9±116.8
Zn	0.433±0.193	339.1±143.2	0.677±0.271	209.7±131.9	0.311±0.153	241.5±108.3	0.434±0.261 [a]	175.0±53.3 [a]
S	4.893±2.885	1207.8±42.7	7.382±2.164	639.2±238.5	4.197±2.270	732.0±258.0 [a]	4.884±1.008 [a]	522.5±215.8 [a]
Al	0.852±0.013	0.4±0.2	1.697±0.852	0.4±0.2	0.578±0.277	0.5±0.3	1.056±0.219	0.4±0.1
B	0.027±0.017	130.8±109.3	0.032±0.029	69.0±31.4	0.017±0.009	134.4±84.7	0.022±0.083	75.8±31.9
Ba	0.027±0.020	2.9±1.3	0.072±0.039	3.1±1.0	0.019±0.011	3.5±1.9	0.038±0.015	3.0±1.1
Ca	3.776±1.008	4.0±2.6	9.709±4.517	4.5±1.7	1.852±0.935	3.5±2.0	4.305±2.181	3.6±1.4
K	1.440±0.743	2.9±1.7	2.153±1.193	1.9±0.6	0.724±0.531	2.6±1.0	1.077±0.813	1.8±1.0
Mg	0.780±0.355	1.5±0.6	2.107±1.004	1.8±0.9	0.508±0.142	1.7±0.3	1.201±0.531	1.8±0.8
Mn	0.062±0.021	2.9±1.3	0.116±0.062	2.3±1.1	0.046±0.015	3.7±1.3	0.060±0.008	2.2±0.9
Na	0.565±0.284	1.1±0.5	0.830±0.530	0.7±0.1	0.418±0.286	1.4±0.3	0.551±00.179	0.8±0.2
P	0.087±0.019	3.7±1.7	0.143±0.084	2.7±1.0	0.051±0.028	3.8±2.0	0.072±0.031	2.4±1.0
Sr	0.022±0.014	2.6±1.2	0.035±0.027	1.9±0.3	0.011±0.006	2.2±1.0	0.020±0.007	1.8±0.6
Ti	0.039±0.016	0.3±0.1	0.082±0.029	0.3±0.0	0.024±0.011	0.3±0.1	0.044±0.019	0.3±0.1
V	0.001±0.000	0.3±0.2	0.006±0.002	0.8±0.4	0.002±0.000	1.3±0.5	0.003±0.000	0.9±0.3
Zr	0.016±0.009	4.1±2.8	0.033±0.012	4.1±1.9	0.004±0.001	1.6±0.3	0.005±0.002	1.1±0.5
Total	14.595±8.392		28.437±10..847		9.729±5.483		15.711±6.926	
T/M(%)	11.5±7.4		12.8±5.3		8.6±2.7		10.8±2.7	
S/T(%)	30±11.4		24.3±10.7		42.0±30.5		30.7±15.9	
crust/T(%)	43.1±25.3		51.8±21.0		33.1±18.4		44.4±21.9	
salt/T(%)	20±7.2		18.3±9.4		17.2±8.5		18.3±7.4	

Note. [a]P<0.05: Compared with different sampling points.

element, with average EFs ranging from 1207.8 to 522.5 for the whole data set, followed by Cd, which had EF's of 673.4 and 840.5 in PM_{10} and $PM_{2.5}$, respectively from the highway site.

The samples of the Orchard site had lower EFs for elements in soil dust such as Ca, Fe, Al than the Highway site, indicating that PM from road dust had a greater effect on the Highway site. The concentrations of all elements measured except Cu in the Highway site samples were much higher than in the Orchard site samples. A similar trend was observed for the EFs except that of Cd. The heavy metals had higher EFs and was thus more enriched in $PM_{2.5}$ compared to PM_{10}. $PM_{2.5}$ may cause more serious damage to human health because of their larger surface areas than PM_{10}. The EF of salt elements in the four types of samples had no significant difference.

When comparing samples from the two sites(Table 4), most of the concentration of OC、EC in PM_{10} from the orchard site were lower than those from the Highway site PM_{10}, which were 86.6%and 66.7% of that in samples from the Highway site. However, the concentration of OC in the Highway site $PM_{2.5}$ was lower than that in the orchard site $PM_{2.5}$.

Table 4. Concentrations of OC EC and TC in PM_{10} and $PM_{2.5}$ (μg/m^3)

Groups	OC Concentration	EC Concentration	TC Concentration	OC/EC
Highway site $PM_{2.5}$	0.023±0.019	0.005±0.002	0.028±0.011	4.6±2.7
Highway site PM_{10}	0.038±0.013	0.009±0.004	0.047±0.025	4.2±2.5
Orchard site $PM_{2.5}$	0.025±0.018	0.005±0.003	0.031±0.017	5.0±3.4
Orchard site PM_{10}	0.033±0.014	0.006±0.002	0.039±0.024	5.0±2.9[a]

Note. [a]P<0.05 compared with different sample site at the same size particles.

As shown in Table 4, the OC/EC in PM_{10} and $PM_{2.5}$ samples from the Highway site were 4.2 and 4.6 respectively, while they were both 5.0 in samples from the Orchard site.

Concentrations of IL-6, IL-1, and TNF-α in Lung Homogenate

Table 5 showed the levels of proinflammatory cytokines measured as indicators of inflammation in the lung homogenate of particulate-exposed rats. The levels of TNF-α, IL-6, and IL-1 in four exposed groups were significantly higher than those in the control group (P<0.05). The levels of TNF-α and IL-1 were also significantly higher in the $PM_{2.5}$ groups compared with those in the PM_{10} groups (P<0.05). However, the concentrations of these inflammatory indicators were not significantly different when comparing the same particle size at different sampling sites (P>0.05).

Table 5. Concentrations of IL-1, IL-6, TNF-α in lung homogenate (pg/mg) ($\bar{x} \pm s$)

Groups	*n*	TNF-α	IL-6	IL-1
Control	6	1.34±0.10	2.69±0.09	3.23±0.27
Highway site $PM_{2.5}$	6	1.84±0.16[ab]	3.63±0.33[a]	5.62±0.47[ab]
Highway site PM_{10}	5	1.49±0.13	3.54±0.19[a]	4.58±0.33[a]
Orchard site $PM_{2.5}$	6	1.73±0.14[ab]	3.48±0.21[a]	5.64±0.65[ab]
Orchard site PM_{10}	6	1.52±0.11[a]	3.33±0.14[a]	4.70±0.21[a]

Note. [a]P<0.05 compared with control; [b]P<0.05 compared with different size particles from the same sampling site.

Concentrations of LDH, T-AOC, TP in BALF

The LDH, T-AOC, and TP activities induced by $PM_{2.5}$ and PM_{10} in BAL fluid were measured as indicators of oxidative damage. The levels of LDH, T-AOC, and TP in the rats exposed to particulate matter significantly increased or decreased in comparison with the control group (P<0.05). The levels of LDH and TP induced by the Highway site source $PM_{2.5}$ were significantly higher than the levels induced by Highway site source PM_{10} (P<0.05). The TP levels in the rats exposed to particulate matter were also significantly higher than those in the control rates, indicating that the body produced inflammatory responses to the particulate (Table 6).

Table 6. Concentrations of LDH, T-AOC, and TP in BALF ($\bar{x} \pm s$)

Groups	*n*	LDH (U/L)	T-AOC (U/mL)	TP (g/L)
Control	6	41.33±5.84	0.89±0.15	0.18±0.02
Highway site $PM_{2.5}$	6	66.50±11.25[a,b]	0.55±0.08[a]	0.39±0.04[a,b]
Highway site PM_{10}	5	53.82±11.75	0.63±0.15[a]	0.32±0.05[a]
Orchard site $PM_{2.5}$	6	64.12±8.43[a]	0.53±0.17[a]	0.40±0.05[a]
Orchard site PM_{10}	5	55.31±11.23[a]	0.65±0.07[a]	0.37±0.04[a]

Note. [a]P<0.05 compared with control; [b]P<0.05 compared with different size particles at the same sample site.

Histopathological Evaluation

The histopathological study of the lung tissue of rats revealed that pulmonary exposure to particles produced persistent and progressive lung inflammatory responses. Figure 3 shows that the inflammation induced by traffic-related PM in the lung tissue of the experiment groups was greater than the inflammation observed in the control group. Exposure to $PM_{2.5}$ caused an overall change in the alveolar architecture with the exception of focal collections of alveolar macrophages laden with particles. Lung tissue thickening as a prelude to the development of fibrosis was also evident and progressive (Figure 3B, D), but these effects were absent in the lung tissue from the control group. The lung tissue from rats exposed to PM_{10} showed mild to moderate alveolar and interstitial inflammation, with inflamed cells predominately inside the edema area, while no inflamed cells were found in the area of normal alveolar tissue in the lungs of the control group (Figure 3C, E).

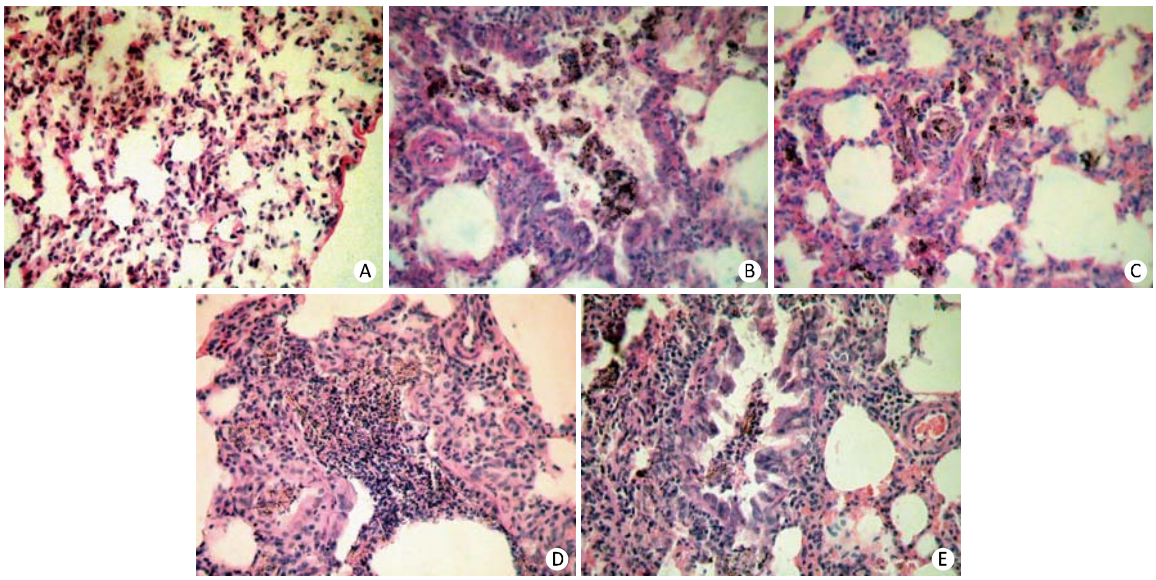

Figure 3. Light micrographs of lung tissue from rats exposed to PM. Magnifications varied from 3.3×10 to 3.3×40. (A) Control; (B) Highway site $PM_{2.5}$; (C) Highway site PM_{10}; (D) Orchard site $PM_{2.5}$; (E) Orchard site PM_{10}.

DISCUSSION

The objective of this study was to investigate the physicochemical characteristics and the potential toxicity of atmospheric particulates in summer time of 2006 in Beijing.

The total concentrations of 130 PAHs in both PM_{10} and $PM_{2.5}$ at the Highway site, which was affected by direct traffic emissions, was much higher than those at the Orchard site, indicating that PAH pollution was more closely related to a traffic source. The high molecular weight PAH homolog concentrations (4-6 rings PAHs), which are primarily associated with combustion of petroleum fuels, were higher than those of the lower molecular weight PAHs in all experimental samples, especially those collected at the Highway site. This suggests that combustion of petroleum based fuels can be a potential source of PAH in atmospheric PM. The particulates from the Highway site area were considered to be more carcinogenic and hence more toxic, as shown by the significantly higher concentrations of BghiP, BkF and BbF observed at this site compared to those at the Orchard site.

The elemental concentrations and EFs were determined for both $PM_{2.5}$ and PM_{10} samples to compare the adsorption capacities of $PM_{2.5}$ and PM_{10}. The EFs were calculated to elucidate the contributions to the particulate elements from sources other than natural crust. The EFs from all sample groups were higher than 10 for As, Cd, Cu, Zn, S, and Pb. The primary anthropogenic sources of these elements include vehicle exhausts and industrial emissions. The EF values of PM_{10} were measured to be lower than those of $PM_{2.5}$, indicating

that fine particles absorbed these toxic elements more easily than PM_{10} probably due to the larger surface area of the fine particles.

For samples with the same particle size, the Orchard site had lower EFs for Cd, Pb, Zn, and Cu than the Highway site, indicating the effects of traffic emissions on the elemental composition of PM. Elements such as S and As are enriched mainly as a result of coal combustion and also due to, most likely, the fact that a district heating boiler house is present near the Highway site[28]. The results from this study could suggest that coal-burning might be another source of PM in this area.

Most of the concentration of OC, EC in PM_{10} at the Orchard site were lower than those at the highway site, indicating that vehicle exhaust was the main source of particle pollution[29]. The OC/EC in both PM_{10} and $PM_{2.5}$ fractions at two sites were all higher than 4.0, indicating that OC were more enriched in samples compared with EC, and there might be secondary pollution. The structure of EC were honeycomb, which had great adsorption capacity to enrich many toxic pollutants, and promote the conversion of pollutants (eg. SO_2 to sulfate). On the other hand, the particle of OC could also adsorb many toxic pollutants such as PAHs, and the concentration of PAHs had great correlation about the toxicity of PM.

ELISA was used to determine the concentrations of TNF-α, IL-6, and IL-1 in the lung homogenate of rats exposed to PM. Cytokines, classified as pro-imflammatory (TNF-α, IL-6, and IL-1) and anti-inflammatory (IL-10, IL-4, and IL-13), play an important role in regulating immunity. In the lung homogenates, the levels of the proinflammatory factors, TNF-α, IL-6, and IL-1, induced by $PM_{2.5}$ and PM_{10} from the two sampling sites were all significantly higher than those of the control group. The results revealed that both $PM_{2.5}$ and PM_{10} could cause an inflammatory response in lung tissue, while $PM_{2.5}$ could be more cytotoxic than PM_{10}.

The levels of LDH, T-AOC and TP in the BALF were measured and used as indicators of oxidative damage in the lungs of particle-exposed rats. The levels of LDH and TP in the BALF were higher in rats exposed to samples from the Highway site compared with those from the Orchard site. PM, especially traffic-related $PM_{2.5}$ could lead to oxidative damage in alveolar cells and micrangium. The decreases in the T-AOC values in the rats exposed to PM suggested that the balance between oxidation and anti-oxidation was also damaged by exposure to PM. The difference in the response of LDH and TP to both PM_{10} and $PM_{2.5}$ from the orchard site was very small, suggesting a less dramatic response in a less polluted environment.

CONCLUSION

The concentrations of trace elements and PAHs in particulates from the Highway site were higher than those of the particulates from the Orchard site during summer time of 2006 in Beijing, China. The level of biochemical parameters and inflammatory cytokines in rats exposed to the traffic-related PM indicate that oxidative damage has occurred, as the antioxidant capacity decreases and the amount of lipid peroxidation products increases. The toxicity of PM is related to the particle size and proximity to the traffic source with greater toxicity associated with fine particles close to the traffic source.

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