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Association of polycyclic aromatic hydrocarbons (PAHs) and lead co-exposure with child physical growth and development in an e-waste recycling town

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HIGHLIGHTS

- Higher levels of PAH and lead are found in the blood of children from Guiyu.
- IP is the most abundant congener among the 16 measured PAHs.
- Milk consumption might be a protect factor from PAH accumulation.
- PAH levels were negative associated with child height and chest circumference.
- High PAH levels in children from Guiyu comes from e-waste recycling activities.

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ABSTRACT

Informal e-waste recycling activities results in serious environmental pollution of PAHs. We evaluated the body burden of 16 PAH congeners and potential health risks for children. A total of 167 children from exposed and reference area entered this study. Child blood samples were collected; height, weight, head and chest circumferences were measured. Blood PAH and lead concentrations were determined. The blood median of total PAHs from the exposed group was significantly higher than the reference group (68.53 μg/L vs. 26.92 μg/L, $P < 0.01$). The major sources of Σ16-PAH and Σ7 carcinogenic-PAH were residence adjacent to e-waste workshop, paternal occupation related to e-waste recycling and house as a workshop. Inverse correlations were observed in the age and milk consumption with these two PAH groups, while a positive association was found between BMI and Σ7 carcinogenic-PAH, and between child height and blood lead. When divided into high and low exposure groups by Σ16-PAH, a significant negative association was found between body height and blood PAHs (β and 95%CI: −3.838, −6.469 to −1.206), while for weight and chest circumferences, negative associations were obtained only in the male subgroup before adjustment. After adjustment by sex, age, child milk products consumption per month and blood lead, child height was negatively associated with Σ16-PAH (β and 95%CI: −3.884, −6.736 to −1.033). Same trends were observed for child chest circumference (β and 95%CI: −1.147, −2.229 to −0.065). We suggest a negative association of PAHs and child height and chest circumference, while the correlation is more obvious in boys.

1. Introduction

Guiyu town, located in Guangdong province, south of China, is one of the largest e-waste destinations and recycling areas in the world, and has nearly 30-year history of unregulated e-waste disposal (Huo et al., 2007; Guo et al., 2012). In Guiyu, more than 6000 small-scale family-run workshops (nearly 60–80% of families in the town) and 160,000 workers are engaged in the business of e-waste dismantling and recycling. E-waste recycling in Guiyu is home-based and family-run with highly insufficient occupational hygienic conditions. Due to these informal activities, amount of chemicals including toxic heavy metals and persistent organic pollutants (POPs) such as lead, chromium, cadmium, polychlorinated biphenyls (PCBs), polybrominated diphenyl ethers (PBDEs) and polycyclic aromatic hydrocarbons (PAHs) are released to the

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environment, can pose a threat to the local people, especially to children (Wu et al., 2010, 2012; Chen et al., 2011; Zhang et al., 2011, 2014; Lin et al., 2013; Yang et al., 2013).

Remarkably, many studies have found that soil, water and air in Guiyu are highly polluted by PAHs (Alabi et al., 2012; Zhang et al., 2011). In Guiyu, the sum of 16 PAHs concentrations in total suspended particulates (TSP) and $PM_{2.5}$ were 148 and 102 ng m^{-3}, respectively. The monthly average levels of benzo[a]pyrene (B[a]P) in $PM_{2.5}$ and TSP were 8.85 and 15.4 ng m^{-3} and the highest levels reached 18.9 and 29.9 ng m^{-3}, respectively (Deng et al., 2006). B[a]P, as an indicator of carcinogenic risk, was 2-6 times higher than that in other Asian cities (WHO, 2000). Metabolites of B[a]P are mutagenic and highly carcinogenic, and it is listed as a Group 1 carcinogen by the IARC. Biomonitoring studies have showed that these PAHs contaminants mainly result from e-waste dismantling in Guiyu (Yu et al., 2006; Zhang et al., 2011; Leung et al., 2013). Workers and local residents are continually exposed to PAHs through inhalation, dietary ingestion, and dermal absorption. Once taken into the body, PAHs can accumulate in the fatty tissues of humans and pose a serious threat to the health of local residents (Moon et al., 2012). It is known that PAHs represent a class of toxicological compounds that could cause a variety of hazardous effects *in vivo* and *in vitro* even at low concentrations, leading to an increased risk of cancer, teratogenicity, and disruption of the endocrine system (Santodonato, 1997; Brody and Rudel, 2003; Ramirez et al., 2011; Tian et al., 2013; Yang et al., 2014). In particular, growing evidence supports the developmental toxicity from prenatal or early postnatal exposure to PAHs, may cause intrauterine growth retardation (IUGR) and lower birth weight (Choi et al., 2008, 2012; Jedrychowski et al., 2015; Perera et al., 2006). Before this study, we have investigated PAHs in umbilical cord blood samples collected from Guiyu. Our results showed that high levels of PAHs is correlated with a reduced fetal physical development and adverse birth outcomes (Guo et al., 2012), which were evidenced by altering the expression of insulin-like growth factor (Xu et al., 2013). Furthermore, a prospective cohort study of nonsmoking African–American and Dominican mother-newborn pairs residing in New York City has reported that prenatal exposure to airborne PAHs is significantly associated with lower birth weight, birth length, head circumference, and developmental delay at 3 years of age, and reduce IQ at 5 years of age (Perera et al., 2006, 2009; Edwards et al., 2010).

Until now, most studies have focused on the distribution of PAHs in air, foods, human urine samples and umbilical cord blood (Tang et al., 2008; Reinik et al., 2007; Jung et al., 2012; Al-Saleh et al., 2013; Ciecierska and Obiedzinski, 2013; Hofmann et al., 2013). Considering possible health risks due to PAHs exposure among children, the present study determine its exposure in children by estimating blood levels of 16 PAHs. Thus, the exposure from variable composition of PAH mixtures emitted from different environmental sources can be more closely monitored, and avoid the underestimating by any single compound or metabolite (Singh et al., 2008a). For these reasons, the measurement of blood PAH levels was carried out as a possible biomarker, especially for those higher molecular weight PAHs (HMWs), which has been proposed in Environmental Protection Agency (EPA, USA) priority list. The aim of this study was to investigate the associations between child growth development and the co-exposure of environmental PAHs and lead in an e-waste recycling area.

2. Materials and methods

2.1. Geographic location and site description

All children from two kindergartens, respectively located in Guiyu and Chendian were recruited for the routine health check-up in 2008. Children from Guiyu, one of the popular destinations of e-waste, served as exposed group. Meanwhile, the reference group was the neighboring town, Chendian, situated 9 miles southwest of Guiyu. The local residents make a living by textiles industry in majority. The population, lifestyle, traffic density, and socioeconomic status were very similar between these two areas (Liu et al., 2011).

2.2. Study population

A total of 295 children joined in the routine health check-up, over 50 percent of these participants ($N = 167$) entered this study. The eligibility criteria for the subjects in this study were as follows: children 3 to 7-years of age, healthy, born and living in Guiyu or Chendian. Because of insufficient amount of the blood samples collected, 95 children (66 boys, 29 girls) in Guiyu, and 72 children (42 boys, 30 girls) in Chendian were included in the measurement of PAHs. Venipuncture blood were taken by nurses, specimens were stored in tubes with heparin as anticoagulant. All samples were transported on ice to laboratory, and stored at −20 °C until analysis. The study protocol was approved by the Human Ethics Committee of Shantou University Medical College. All the guardians of participants gave their written informed consent after receiving detailed explanation and possible consequences about the survey before enrollment.

2.3. Data collection

A structured interview questionnaire was used to identify Environmental Tobacco Smoke (ETS) and vehicle exhaust as the potential sources of PAH exposure, as well as general demographic and health parameters. The questionnaires were completed by the parents or guardians, and 25 factors were included, such as dwelling, child behavior and hobbies, diet and nutrition; parent educational level and occupation, and social status. Parental occupations were classified by the connection with e-waste recycling, such as transporting, selecting, splitting, acid bath, and burning to recover metals.

The physical developmental indices contain body height, weight, and head and chest circumferences were measured simultaneously when collecting blood samples based on a previous published paper (Huo et al., 2007).

2.4. Blood lead measurement

Lead in whole blood was determined by graphite furnace atomic absorption spectrometry (GFAAS, ZEEnit 650, Germany). The methods were based on previously published papers (Liu et al., 2011, 2015).

2.5. PAH measurement

2.5.1. Chemicals

A standard mixture was purchased from Chiron, Norway, which contains 16 PAH congeners of the EPA priority list, namely naphthalene (Nap), acenaphthylene (Ace), acenaphthene (A), fluorene (Fl), anthracene (Ant), phenanthrene (Phe), fluoranthene (Fla), pyrene (Pyr), benzo[a]anthracene (BaA), chrysene (Chr), benzo[k]fluoranthene (BkF), benzo[b]fluoranthene (BbF), benzo[a]pyrene (BaP), dibenzo[a,h]anthracene (DA), indeno[1,2,3-c,d]pyrene (IP), and benzo[g,h,i]perylene (BP). Among them, 7 were carcinogenic PAHs: BaA, Chr, BkF, BbF, BaP, DA and IP. Acetonitrile, di-chloromethane (DCM) and n-hexane were all pesticide grade (TEDIA); all other chemicals used were analytical grade (Guangzhou, China); and Solid Phase Extraction (SPE) cartridges (Supelclean™, LC-18) used for cleanup were procured from

Supelco, USA. The working standard solution was made via mixing each stock solution into an amber-colored volumetric flask in absence of light exposure and stored at 4 °C.

2.5.2. Preparation of sample

The preparation, cleanup, and analysis of specimen were based on a previously published method (Guo et al., 2012), with minor modifications. Blood samples (2 mL) were saponified with 0.4 M NaOH in EtOH–H_2O (9:1, v/v) at 60 °C for 30 min, then extracted three times by shaking for 10 min with n-hexane in an amber colored, capped conical flask. The pooled extract was evaporated to 0.5 mL under a gentle nitrogen steam and kept in the refrigerator until clean-up, in order to avoid loss of lighter PAHs by volatilization. Solid phase extraction SPE cartridges (LC-18, Supelco) were used to remove impurities and co-extracted lipids, in avoid of the interfering with subsequent separation and identification. Before sample application, SPE cartridges were washed with Milli-Q ultrapure water and activated with a mixture of n-hexane and dichloromethane (1:1; v/v). Then samples were loaded on the cartridges and extracted by aspiration through the cartridge, resting on a manifold vacuum station with a pressure regulator set at a flow rate of less than 2 mL/min. Finally, the sorbent was washed with 10 mL of water, dried, and then eluted with a mixture of n-hexane: dichloromethane (1:1). Extracts were concentrated to 100 μL by evaporation with a gentle stream of nitrogen. Samples were transferred to glass inserts in amber GC vials in the dichloromethane phase for gas chromatography/mass spectrometry (7890A-5975C Agilent Technologies) analysis; electron-ionization was used in ion monitoring mode. Auto-splitless injection of a DB-5 MS capillary column (30 m × 0.25 mm i.d., 0.25 μm film thickness, J&W Scientific) was used, with helium (1 mL min^{-1}) as the carrier gas. The temperatures of ion source, quadrupole, interface, and injector were kept at 230, 150, and 280 °C, respectively. Retention times for PAH congeners were established with the standard solutions. Oven temperature was ramped from 50 °C (1 min) to 200 °C (0 min) at 25 °C/min and from 200 °C to 316 °C at 8 °C/min.

2.5.3. Quality control

For every sequence of eight samples, a solvent blank and a procedural blank were added to ensure that the samples and the analysis processes were free of contamination. The limit of detection (LOD) of the targeted compounds was defined as three times of the signal-to-noise (S/N) ratio and ranged from 0.01 to 0.08 μg/L for PAH congeners. Peaks were quantified only if S/N was >3, the ratio between two monitored ions was within 15% of the standard value, and the concentration of analytical samples was at least twice the blank sample. Therefore, the average of procedural blank value was subtracted from each sample. Recovery experiments were conducted to check the analytical quality control; six blood samples in duplicate were spiked with the mixed PAH standards at 5, 10, and 20 μg/L. The recoveries of the spiked blanks for carcinogenic PAHs varied from 78% to 94%. Sixteen-point calibration curves were constructed for the quantification and good linearity ($r^2 > 0.99$) was obtained.

2.6. Statistical analysis

The data of PAHs and lead were expressed as medians with 25th–75th percentiles because of their non-normal distribution, and non-parametric tests were employed for statistical comparison. We defined Σ16-PAH as the sum of sixteen individual PAH congener concentrations. Similarly, Σ7 carcinogenic-PAH was the sum of seven individual carcinogenic PAH congener concentrations. The individual PAH contributions to the Σ16-PAH and Σ7 carcinogenic-PAH were calculated based on molar concentration. Potential associations between the logarithm of PAH concentrations and child physical indices were identified by Spearman correlation analysis and multiple linear regression analysis. The significance tests were calculated for categorical data by the χ^2 test. All the statistical analyses were conducted with SPSS statistical software version 13.0 (SPSS Inc., Chicago, IL) and Microsoft Excel and $P < 0.05$ was considered statistically significant.

3. Results

3.1. General characteristics of children

The gender distribution, body height and weight, and head and chest circumferences were not significantly different between the exposed and reference group (all $P > 0.05$). No difference of age between two groups were found, but the mean BMI in exposed group was significantly higher than that in the reference group ($P = 0.009$) (Table 1).

Among the 167 participants, only 4 mothers and 9 fathers had completed a college education, while no significant difference in parent education was noted between two groups ($P > 0.05$). The smoking status of family member and average monthly household income were also not significantly different.

3.2. PAH concentrations in the blood of children

The results about sixteen PAH congeners analyzed showed that the medians of Σ16-PAH, Σ7 carcinogenic-PAH and most individual PAH congener in the blood samples from e-waste recycling area were significantly higher than that from the reference area as shown in Table 2 ($P < 0.01$ or $P < 0.05$). The predominant PAH

Table 1
Demographic characteristics of the children investigated.

Characteristic[a]	Exposed group ($N = 95$)	Reference group ($N = 72$)		P value
Age (years)	4.64 ± 1.40	4.80 ± 1.00	$t = -1.921$	0.051
Sex [n (%)]			$\chi^2 = 1.772$	0.183
Male	65 (68.4)	42 (58.3)		
Female	30 (31.6)	30 (41.8)		
BMI	15.3 ± 1.1	14.8 ± 1.2	$t = 2.632$	0.009
Height (cm)	102.4 ± 8.1	103.2 ± 6.7	$t = -0.603$	0.547
Weight (kg)	16.1 ± 2.7	15.8 ± 2.3	$t = 0.763$	0.446
Head circumferences (cm)	49.1 ± 1.5	48.9 ± 1.7	$t = 0.758$	0.449
Chest circumferences (cm)	51.4 ± 3.3	50.6 ± 2.4	$t = 1.658$	0.099
Mother's education (years) [N (%)]			$\chi^2 = 1.936$	0.380
⩽6	35 (36.8)	30 (41.7)		
7–9	44 (46.3)	32 (44.4)		
>10	16 (16.9)	10 (13.9)		
Father's education (years) [N (%)]			$\chi^2 = 1.274$	0.529
⩽6	26 (27.4)	13 (18.1)		
7–9	55 (58.0)	41 (56.9)		
>10	14 (14.6)	18 (25.0)		
Environmental tobacco smoke [N (%)]			$\chi^2 = 0.009$	0.926
No	30 (25.7)	19 (25.0)		
Yes	65 (74.3)	53 (75.0)		
Average household income[b] [N (%)]			$\chi^2 = 0.248$	0.884
1000–1500	30 (31.5)	17 (23.6)		
1501–2000	22 (23.2)	19 (26.4)		
>2000	43 (45.3)	36 (50.0)		

[a] Values are mean ± SD or percent.
[b] Chinese Yuan per month.

congeners were three-, five-, and six-ring compounds, with IP occupying the highest percentage, followed by BP, BbkF, and Phe. B[a]P was 0.04 μg/L in the blood samples from the exposed area, while 0.02 μg/L in reference group ($P < 0.01$). Children in exposed group also had higher blood lead levels than that in reference group ($P < 0.01$). As shown in Table 3, PAH concentrations differed in the two groups by child's sex and age ($P < 0.01$ or $P < 0.05$). Both the medians of Σ16-PAH and Σ7 carcinogenic-PAH concentrations in each subgroup in children from Guiyu were higher than that in Chendian (all $P < 0.05$), with an exception of Σ7 carcinogenic-PAH in children over 6 years old. Same differences were observed for blood lead levels between the subgroups from e-waste exposed and reference area.

3.3. Factors contributing to blood PAH and lead concentrations in children blood

Among the factors investigated, the concentrations of Σ16-PAH and Σ7 carcinogenic-PAH were correlated with child's age, dwelling environment, milk consumption, blood lead concentrations, paternal involvement in e-waste recycling and duration of work ($P < 0.01$ or $P < 0.05$), and so were blood lead levels (Table 4). Blood lead level was negatively correlated with child height ($R_s = -0.158$, $P < 0.05$). We found that the concentrations of certain PAH congeners (BbkF, BaP, IP, Phe, Ant) were higher in the children who lived adjacent to workshops than the others in exposure area (Fig. 1). PAH concentrations were also higher in the child whose father was engaged in e-waste recycling than those whose father did not, especially for carcinogenic PAH congeners such as Chr, BbkF, BaP, and IP (Fig. 2).

3.4. PAH concentrations in blood and physical indices of children

To determine the relationships between Σ16-PAH and physical indexes, we divided all the observed subjects into high and low exposure group according to the Σ16-PAH concentrations in children blood (using the 75th percentile as the cutoff), following a linear regression model analysis. Table 5 presents the unadjusted and adjusted values and 95% confidence intervals for these models on estimating the differences in child physical growth and development. When evaluated by the unadjusted β, height, head and chest circumference of the boys in high Σ16-PAH exposure group were significantly lower than that in the low exposure group. After adjusted by sex, age, child milk products consumption per month and blood lead, child height was negatively associated with Σ16-PAH (β and 95%CI: −3.884, −6.736 to −1.033). Same trends were observed for child chest circumference (β and 95%CI: −1.147, −2.229 to −0.065).

4. Discussion

4.1. Factors related to PAH and children development

In this study, we found family-style workshops may largely contribute to elevate the PAH concentrations in child blood. Moreover, the strong negative correlation between milk products consumption and the PAH concentrations, suggests that milk is also important in preventing PAH toxicity, as it is important aspect in preventing heavy metal toxicity (Mishra et al., 2012). A positive correlation between the concentrations of PAH and lead in children blood samples ($P < 0.001$), indicates PAH and lead are co-existed during the informal e-waste recycling processes. After adjusted its confounding effects on child development, a negative association with borderline statistical significance was observed.

4.2. Contributions of PAH congeners

Among the PAHs we detected, more kinds of congeners were found when compared with other studies reported in child blood of Lucknow, India (Singh et al., 2008a,b), including harmful

Table 2
Comparison of 16 USEPA PAHs and lead in children between exposed group and reference group.

Blood contaminants	Exposed group ($N = 95$) Median (25th–75th)		Reference group ($N = 72$) Median (25th–75th)	
USEPA[a] PAHs (μg/L)				
Two rings*		0.63 (0.42–0.95)		0.51 (0.43–0.66)
Nap*	0.63 (0.42–0.95)		0.51 (0.43–0.66)	
Three rings**		2.89 (1.39–4.94)		1.48 (0.91–2.07)
Ace	0.07 (0.04–0.85)		0.50 (0.03–0.78)	
A**	0.18 (0.10–0.34)		0.11 (0.06–0.17)	
Fl	0.81 (0.04–1.75)		0.45 (0.32–0.74)	
Ant**	0.09 (0.04–0.12)		0.02 (0.01–0.03)	
Phe**	1.42 (0.59–2.40)		0.49 (0.04–0.66)	
Four rings**		0.91 (0.68–1.28)		0.60 (0.46–0.86)
Fla**	0.07 (0.05–0.11)		0.06 (0.04–0.08)	
Pyr	0.11 (0.02–0.24)		0.21 (0.04–0.32)	
BaA**	0.26 (0.14–0.45)		0.15 (0.10–0.24)	
Chr**	0.35 (0.26–0.52)		0.20 (0.12–0.26)	
Five rings**		1.92 (1.47–2.49)		1.21 (0.98–1.47)
BbkF**	1.78 (1.32–2.33)		1.12 (0.90–1.37)	
BaP**	0.04 (0.02–0.07)		0.02 (0.01–0.03)	
DA**	0.08 (0.06–0.10)		0.07 (0.05–0.08)	
Six rings**		60.92 (37.81–76.55)		22.76 (16.78–28.04)
IP**	58.00 (34.44–67.14)		19.49 (14.49–23.22)	
BP	4.06 (ND-7.73)		2.27 (ND-6.39)	
∑16-PAHs**		68.53 (46.74–82.12)		26.92 (20.86–32.09)
∑7 carcinogenic-PAHs**		60.27 (37.81–70.43)		21.30 (15.78–25.08)
Blood lead level (μg/dL)**	13.89 (9.47–18.28)		8.55 (6.37–11.39)	

[a] USEPA, United States Environment protection Agency B2 classification: chrysene, benzo[a]anthracene, benzo[k]fluoranthene, benzo[b]fluoranthene, benzo[a]pyrene, indeno[1, 2, 3-c, d]pyrene, dibenz[a, h]anthracene. ND: not detected under the detection limit.
* $P < 0.05$.
** $P < 0.01$.

Table 3
Comparison of ∑16-PAH, ∑7 carcinogenic-PAH and lead concentrations stratified by age and gender.

Blood contaminant	N		∑16-PAH[a] (μg/L)		∑7 carcinogenic-PAH[a] (μg/L)		Blood lead level[a] (μg/dL)	
	E	R	Expose[b]	Reference	Expose[b]	Reference	Expose[b]	Reference
Sex								
Male	66	42	71.00 (42.31–83.29)**	28.07 (21.79–33.14)	62.87 (35.3–71.04) **	22.0 (16.95–25.41)	14.50 (9.82–18.56)**	8.87 (8.44–11.36)
Female	29	30	65.62 (55.24–74.94)**	23.88 (19.90–29.89)	56.16 (47.0–67.59)**	19.65 (14.28–24.57)	13.50 (8.97–16.01)**	8.34 (6.40–11.49)
Age (y)								
3	19	7	78.95 (49.97–82.81)**	29.31 (26.54–34.05)	66.33 (45.47–73.33)**	23.99 (22.4–28.02)	14.80 (8.79–21.84)	10.32 (8.37–14.25)
4	34	17	69.50 (58.17–89.37)**	26.90 (23.1–32.38)	61.46 (45.04–70.82)**	21.93 (18.26–23.09)	14.91 (10.56–19.24)**	8.42 (6.64–12.71)
5	30	29	67.34 (44.89–79.01)**	23.67 (19.9–30.54)	59.19 (39.38–68.48)**	19.33 (13.62–25.05)	12.36 (8.34–18.01)**	8.31 (5.75–11.21)
6–7	12	19	48.23 (21.49–69.69)*	28.39 (22.2–34.79)	38.59 (5.53–64.14)	19.79 (16.8–24.09)	11.92 (9.83–15.46)*	8.93 (6.62–11.05)

E: e-waste exposed group in Guiyu; R: reference group in Chendian.
[a] Median (25th–75th).
[b] Compared the e-waste exposed group with the reference group.
* $P < 0.05$.
** $P < 0.01$.

Table 4
Spearman correlations (R_s) of Σ16-PAH, Σ7 carcinogenic-PAH, blood lead levels and factors about children and potential sources of exposure.

	Σ16-PAH	Σ7 carcinogenic-PAH	Blood lead level
Location of child residence in Guiyu	0.688**	0.651**	0.486**
Residence adjacent to e-waste workshop	0.305**	0.279**	0.229**
Age	−0.267**	−0.290**	−0.205**
Father's engagement in work related to e-waste	0.321**	0.285**	0.317**
Time that father is engaged in work related to e-waste	0.324**	0.289**	0.347**
House as a family workshop	0.213*	0.211*	0.233**
Child milk consumption per month	−0.176*	−0.215*	0.129
Blood lead level	0.374**	0.327**	–
BMI	0.150	0.164*	0.080
Height	−0.105	−0.104	−0.158*

BMI: body mass index (kg/m^2); R_s: Spearman correlation coefficient.
$N = 167$; compared with the reference group.
* $P < 0.05$.
** $P < 0.01$.

carcinogens, chrysene and indeno [1,2,3-c,d]pyrene. In our study, the Σ16-PAHs (68.53 μg/L) and non-carcinogenic PAHs (8.26 μg/L) were much lower than the results reported in Indian children, with 145.90 μg/L of Σ13-PAH and 113.55 μg/L of non-carcinogenic PAH. These different exposure doses may be attributed to the different sources that PAHs derived, and thus exhibited distinct difference in the compositions of these two studies. In our study, Σ7 carcinogenic-PAH contributed the most of the Σ16-PAH, and higher than the reference group, like the results in our previous studies for cord blood from the residents of e-waste processing (Wu et al., 2010; Xu et al., 2013). While in other studies, where non-carcinogenic PAH is the major, are mainly derived from other sources, such as cooking, burning fuel, and vehicular traffic.

The distribution patterns are similar to the previous studies about environmental atmosphere samples in Guiyu, in which five-ring and six-ring PAHs are accounted for 73% of total PAHs, and similar in the main parts of Σ16-PAH, such as: IP, BP, BbkF, Phe and Chr (Wong et al., 2007).

Noteworthy, Benzo[g,h,i]perylene (BP) is very high in both area, this phenomenon may account for their higher molecular weight, as well as HMWs had a higher fraction in particulate PAHs simply because of their low volatility (Maskaoui and Hu, 2009).

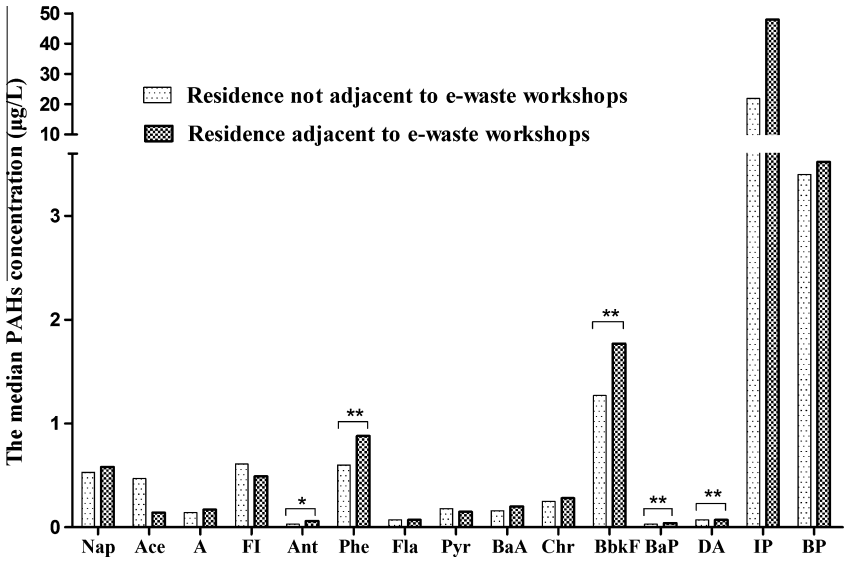

Fig. 1. Blood PAHs in children with different residence.

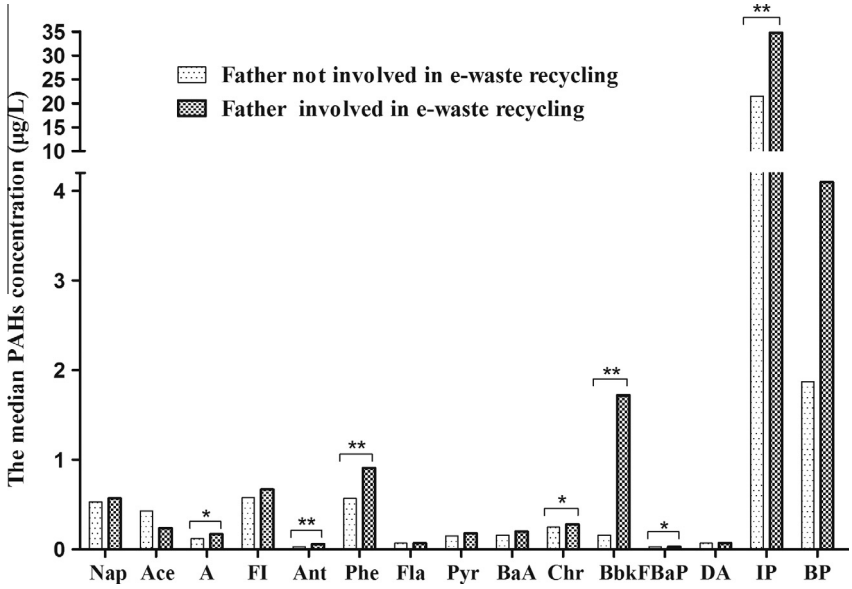

Fig. 2. Blood PAHs in children with different paternal occupation.

Table 5
Correlation of blood PAH with child physical growth and development by linear regression analysis (high exposure vs. low exposure).[a]

Physical growth	β and 95%CI of general model	β and 95%CI of adjusted model
Height (cm)		
Male	−5.043 (−8.116 to −1.971)**	−4.233[b] (−7.768 to −0.698)*
Female	−2.383 (−7.467 to 2.700)	−4.587[b] (−10.286 to 1.113)
Total	−3.838 (−6.469 to −1.206)**	−3.884[c] (−6.736 to −1.033)**
Weight (kg)		
Male	−1.297 (−2.389 to −0.206)*	−1.100[b] (−2.389 to 0.189)
Female	−0.324 (−1.849 to 1.195)	0.461[b] (−2.312 to 1.390)
Total	−0.860 (−1.747 to 0.026)	−0.897[c] (−1.892 to 0.099)
Head circumference (cm)		
Male	−0.610 (−1.264 to 0.044)	−0.387[b] (−1.171 to 0.398)
Female	−0.254 (−1.275 to 0.767)	−0.477[b] (−1.808 to 0.855)
Total	−0.350 (−0.924 to 0.225)	−0.4312[c] (−1.071 to 0.211)
Chest circumference (cm)		
Male	−1.660 (−2.957 to −0.363)*	−1.512[b] (−2.972 to 0.053)
Female	−0.379 (−1.901 to 1.143)	−0.703[b] (−2.592 to 1.186)
Total	−0.999 (−2.060 to 0.062)	−1.147[c] (−2.229 to −0.065)*

[a] High exposure was defined as the fourth quartile of Σ16-PAH ($N = 42$); low exposure was defined as all others (quartiles 1, 2, 3) ($N = 125$).
[b] Adjusted for the average monthly household income, child milk products consumption per month and blood lead level.
[c] Adjusted for sex, the average monthly household income, child milk products consumption per month, environmental tobacco smoking (ETS) and blood lead level.
* $P < 0.05$.
** $P < 0.01$.

Furthermore, relatively high concentrations of Σ16-PAH (26.92 μg/L) in the reference group, and similar PAH profiles with the exposed group were found. This may due to the following reasons: rather short distance between two adjacent places and pollutants in textile industry. It is possible that PAHs in Guiyu may spread to Chendian by dust, river, and air, especially those adsorbed in fine and ultrafine particles (PM_1, $PM_{2.5}$) which can be transferred in long distances, including 9 miles (Dvorská et al., 2012). Additionally, PAHs may be emitted in textiles industry (Ning et al., 2014), by which people in Chendian make a living.

4.3. Gender and age effects of PAHs in children blood samples

The medians of Σ16-PAH and Σ7 carcinogenic-PAH in the boys of Guiyu were higher than that in the girls. The similarity of the structure of the aromatic hydrocarbon receptor and estrogen receptor were reported (Berge et al., 2004), so females may have greater capacity to metabolize PAHs than males. Dietary exposure, metabolism, or other factors also may contribute to the higher levels of PAH in boys. Thus, we suggest that more investigations on the mechanism of gender effect in PAH exposure are needed. Moreover, a main reason for the younger children tended to have higher PAH concentrations in the e-waste exposure group, probably lying in their greater surface-area-to-body-weight ratio than older children (Cohen Hubal et al., 2000).

The results about the PAH distribution in subgroups of all ages show a noticeable difference, except for Σ7 carcinogenic-PAH of the subgroup aged 6–7 subgroup, which has no obviously difference between the exposure and reference groups. This phenomenon may be due to the small sample size of child in 6–7 years old children from Guiyu.

4.4. Potential source of PAHs

The relatively high abundance of PAHs in Guiyu is due to the continuous emission of PAHs from the open burning of e-waste

(Wilcke et al., 2005). In the e-waste exposed area we studied, crude family-run recycling processes are mainly composed of manual processing methods, including sorting, firing, incineration, and acidic wash (Lin et al., 2013). These operations are usually carried out with little or no personal protection equipment or pollution control measures. Especially, fly-ash particulates laden with PAHs and other toxic chemicals might be emitted when melting the circuit boards on a coal grill to obtain useful metals. Previous studies conducted in Guiyu have reported high concentrations of PAHs in the air, sediment, and soil (Yu et al., 2006; Zhang et al., 2011; Leung et al., 2013). This process may be similar as the formations of most PAHs during incomplete combustion, and are ultimately deposited in soil (Haritash and Kaushik, 2009). High concentrations of environmental PAHs contribute to the uptake by humans via inhalation, skin contact, and gastrointestinal tract entry (Ramesh et al., 2004). Besides, children spend more time at outdoor, resulting in a continuous contact with contaminated soil and air. Furthermore, children have an exceptional vulnerability to environmental hazards, which are disproportionately susceptible when compared with adults (Carpenter and Bushkin-Bedient, 2013; Leith Sly and Carpenter, 2012).

4.5. Developmental effects of childhood PAH exposure

PAHs are known as carcinogens and mutagens, and adversely affect human reproductive and neural development systems (Jamin et al., 2013). Thus, the exposure of children may threaten their health, such as delay of their physical growth and development. Long-term and low-dose exposure to PAHs has been confirmed to be associated with a wide variety of effects, including irritability, mood instability, short- and long-term memory loss, and the lack of concentration in children (Dahlgren et al., 2003). Our results indicated that PAHs exposure may affect child physic growth especially height and chest circumference.

Although the precise mechanisms by which PAHs affect child development are not confirmed, some mechanisms including endocrine disruption (Westman et al., 2013), binding to human growth factor receptors and aryl hydrocarbon (Ah) receptor to induce P450 enzymes are suggested (Shimada et al., 2013). There are evidences that PAHs may act through disrupting estrogen-mediated pathways (Sievers et al., 2013). Phe and Fla show anti-androgenic effects and 1-naphthol and 2-naphthol may act as thyroid hormone receptor antagonists (Sun et al., 2008). Otherwise, PAH causing DNA damage may also result in the activation of apoptotic pathways, interference of transcription, DNA replication, or protein synthesis (Gurbani et al., 2013; Kuang et al., 2013; Wei et al., 2010).

One of the limitations of our study is that our samples were collected from two kindergartens' children instead of a random population. Others are the confounding effects from contaminants of cadmium, chromium, PBDEs and PCBs, owing to their co-existence in the environment and human body. There may exist other factors which also affect the associations between PAHs and child growth development, such as demographic factors and toxic chemicals, which we did not collect any information of them. Furthermore, birth weight is also of importance when evaluating influence of environment pollutants on child growth development. However, we did not take into account of the child birth weight and other anthropometric measures at birth, because we could not get the exactly original data on birth outcomes for these data were obtained according to their guardians memories rather than by checking their birth certificate, as some child birth certificates were missing. Thus, we gave up using these data for not all of them were accurate.

However, our study can still help in understanding PAH exposure and child physical growth development. Exposure to high concentrations of PAH may have a negative impact on child health to a certain extent, but the biological effects and potential risks still need to be investigated.

The finding about the high concentrations of PAHs in child blood at e-waste recycling areas in a developing country highlights the need for improving the workplace hygiene. In addition, precautionary measures and eliminate or minimize the adverse effects of PAHs on children are also very important because of their special vulnerability to environmental toxicants, that predispose the enhanced susceptibility to diseases in adulthood (Leith Sly and Carpenter, 2012).

5. Conclusions

We conclude that the present study indicates that children who reside in informal e-waste recycling areas are exposed to higher concentration of PAHs than who in the reference area, and PAHs may adversely correlate with child height and chest circumference.

Competing interests

The authors have declared that no competing interests exist.

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Further reading

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