Aerosol and Air Quality Research

Assessing Lung and Skin Cancer Risks for Steel and Iron Manufacturing Industry Workers Exposed to Polycyclic Aromatic Hydrocarbons

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ABSTRACT

Conducting both inhalatory and dermal samplings are crucial for chemicals, such as polycyclic aromatic hydrocarbons (PAHs), which are known with chronic dermal and inhalatory health effects. The present study is the first one set out for conducting both PAH inhalatory and dermal exposure assessments simultaneously for electric arc furnace (EAF) workers in an iron and steelmaking industry. The results were further used to assess the health risks posed on workers. Finally, risk-based control strategies were suggested. Three similar exposure groups (SEGs) of the melting, ladling, and casting were selected for conducting personal samplings. A total of 15 and 120 samples were collected for assessing workers' inhalatory and dermal PAH exposures, respectively. Results show that the inhalatory gas-phase PAH exposure levels (2.17×10^4–1.52×10^5 ng m^{-3}) were consistently higher than that of particle-phase (2.73×10^3–6.76×10^3 ng m^{-3}) in all selected exposure groups. The mean inhalatory exposure concentrations of the melting and casting workers (2.84×10^4–3.32×10^4 ng m^{-3}) were higher, but ladling workers (1.58×10^5 ng m^{-3}) were lower, than the time-weighted average permissible exposure level (PEL-TWA) regulated in Taiwan (1.00×10^5 ng m^{-3}). The estimated lifetime excessive lung cancer risks for the latter (2.09×10^{-3}) were lower than that of the former (3.43×10^{-3}–8.84×10^{-3}), they were all higher than the significant risk (10^{-3}) defined by the US Supreme Court. The mean whole-body total PAH dermal exposure levels were 2.84×10^5–7.45×10^5 ng day^{-1}, and those body surfaces uncovered by clothes were found with higher exposure levels. Since the estimated lifetime excessive skin cancer risks (7.74×10^{-5}–1.62×10^{-4}) were lower than of the lung cancer risk, which indicates that the control of workers' inhalatory exposures is more important than that of dermal exposures. The installation of effective ventilation systems and the use of proper personal respiratory protection equipment are recommended for the abatement of risks posed on workers associated with PAH exposures.

Keywords: Polycyclic aromatic hydrocarbons (PAHs), Exposure assessment, Health risk assessment, Control strategy, Steel and iron manufacturing factory

1 INTRODUCTION

The electric arc furnace (EAF) is widely used in the steel and iron manufacturing industry producing ~1,600 million tons yr^{-1} steel worldwide (World Steel Association, 2017). The European Commission has also reported that pollutant emissions from EAFs have being increased constantly since 2001 (UNEP, 2001). The EAF is usually equipped with carbon electrodes to heat and melt the feedstock (i.e., the scrap metal) at the temperature ~1,500°C. Considering the impurity of raw materials and the involved high temperatures, emissions of polycyclic aromatic hydrocarbons
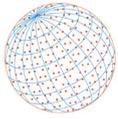
(PAHs) from the EAF production process can definitely be expected. Major PAH emission sources have been identified, including those originally containing in scraps, or formed during the process due to incomplete combustion of scrap organic matter, fuels, and additives (e.g., coal) (IPPC, 2001). Epidemiological studies have shown that long-term exposure to PAHs might result in workers with excessive lung and skin cancer risks, and the occurrence of the respiratory, skin, bladder and many other diseases (Boffetta et al., 1997; Houshutama et al., 2006; Rota et al., 2014). Some PAH compounds, particularly for those containing four or more aromatic rings, are known with carcinogenicity and mutagenicity (IARC, 2010; Boström et al., 2002; Tsai et al., 2002a; Nikolaou et al., 2009), which have been included in the European Union priority lists of pollutants (Fernandez-Gonzalez et al., 2007).

The severe impact associated with emissions of PAHs from EAFs in iron and steel industries to the ambient atmosphere have been intensively investigated (IPPC, 2001; Yang et al., 2002; Cetin et al., 2007; Bayram et al., 2008; Bozlaker et al., 2008; Kaya et al., 2012; Odabasi et al., 2009; Odabasi et al., 2010; Baek et al., 2010; Almaula et al., 2005; Gomes, 2008; Kim et al., 2013; Aydin et al., 2014). But it should be noted that concentrations of PAHs in workplace atmospheres of an iron and steel manufacturing factory are much higher than that of ambient atmosphere being affected. Particularly, the concentrations at the EAF related workplaces (including the scrap charging, melting, slag removing, steel tapping, casting, and ladling) have been found even higher than other workplaces (Baraniecka et al., 2010). Therefore, it can be expected that conducting PAH exposure and health-risk assessments for EAF workers would be of importance to the steel and iron manufacturing industry before initiating effective exposure control strategies.

Yet, it is true that a recent study has been conducted to assess inhalatory PAH exposure concentrations for EAF workers at different worksites (Aries et al., 2008). However, their dermal exposures and inherent health risks have never been assessed. Considering long-term exposures to PAHs might result in workers with excessive respiratory and dermal cancer risks (Diggs et al., 2012; Wells et al., 2010; ATSDR, 1995; Srogi, 2007; Kuo et al., 2003), the present study was set out for conducting both inhalatory and dermal exposure and health-risk assessments for EAF workers simultaneously. Then, health risks posed on workers were further assessed, and finally risk-based control strategies were proposed. The results obtained from this study would be beneficial to the steel and iron industry to initiate effective control strategies.

2 METHODS

2.1 Personal Inhalable and Dermal PAH Exposure Samplings

The EAF steelmaking process was chosen from an iron and steelmaking industry located in southern Taiwan. Based on workers' job-tasks, EAF workers could be classified into five categories of the scrap charging, melting (including refining), deslagging, steel tapping, ladling, and casting. Because both deslagging and steel tapping workers were not suitable for conducting both personal inhalatory and dermal samplings, samplings were only conducted on melting, ladling, and casting workers. For each category, five workers were randomly selected. For each selected worker, both personal inhalatory and dermal samplings were conducted simultaneously for one workshift (i.e., 8 hours) on one workday.

For personal PAH inhalable exposure samplings, the method modified from the National Institute for Occupational Safety and Health (NIOSH) method 5515 was adopted. The above method has also been used in our previous studies for conducting PAH inhalatory exposure assessments (Tsai et al., 2001a; Lin et al., 2008; Chen et al., 2008). The sampling train consists of a filter cassette (IOM personal inhalable aerosol sampler, SKC Inc., Catalog No. 225-70), followed by a split, one side of the split installs with a needle valve for the purpose of controlling the flow rate of a sorbent tube located at the other side of the split (washed XAD-2, 3.5 g/0.5 g). The sampling flow rate was specified at 2 L min^{-1} for the IOM personal inhalable aerosol sampler, and 0.2 L min^{-1} for the sorbent tube. Before sampling, all filters and sorbent tubes were cleaned and extracted with a solvent solution (mixture of n-hexane and dichloromethane, v:v = 1:1) for 24 hrs in a Soxhlet extractor. Particles collected by the IOM personal sampler were determined by using an electrical balance (Sartorius MP 8-6, ±0.01 mg), then sent to the laboratory, together with the sorbent tube, for PAHs analysis to determine the concentrations of both particle-phase and
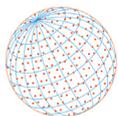
Table 1. Representative dermal sampling body sites and their corresponding body surface areas as recommended by U.S. EPA (1987), and the sampling spots that selected in this study.

| U.S. EPA recommended sampling body sites	Corresponding body surface area* (cm^2)	Sampling spots selected in this study
Head/front	1300	Attached on the front hat
Head/back	650	Attached on the back hat
Neck/front	110	Attached on the left collar closed to neck/front area
Neck/back	150	Attached on the collar closed to neck/back area
Back	3550	Attached on the skin of the back
Chest/abdomen	3550	Attached on the skin of the chest
Upper arm	2910	Attached on the skin of the lower arm (Renamed as: Arm and hand)
Lower arm	1210	
Hand	820	
Upper leg	3820	Attached on the skin of shin (Renamed as: Leg and foot)
Lower leg	2380	
Foot	1310	

* For the standard man: body weight = 70 kg, height = 173 cm.

gas-phase PAHs, respectively. Here, PAHs collected by the front and rear section of the sorbent tube were analyzed separately to exam if it reached a breakthrough situation. If the gas-phase PAH concentration of the rear section is greater than 5% concentration of the front section, the sample is considered to be "breakthrough" and will be discarded. In the present study, no "breakthrough" was found in all collected sorbent tube samples. A total of 15 personal inhalable samples (including 15 gas-phase PAH samples and 15 particle-phase PAH samples) were collected from this study.

The surrogate skin sampling technique was adopted in the present study for conducting dermal exposure assessment. The method was first recognized as a standard method for assessing dermal pesticide exposures (WHO, 1986; U.S. EPA, 1987), but its use has been extended to assess occupational exposures to PAHs (Jongeneelen et al., 1988; Tsai et al., 2001b; Tsai et al., 2004) and dichlorobenzidene (London et al., 1989). A soft polypropylene with a surface area 100 cm^2 was used as the collecting medium (i.e., exposure pad). Table 1 shows the 12 sampling body sites and their corresponding surface areas (based on a standard man: height = 173 cm, body weight = 70 kg) for conducting dermal exposure assessment for each selected worker as recommended by U.S. EPA (1987). Subjected to workers' unwillingness, several body sites were merged into one site to reduce the sample number for each selected worker. Here, only one pad was placed on the skin of the lower arm to estimate the exposure of the three body sites of the upper arm, lower arm and hand (renamed as "arm and hand"). Similarly, one pad was attached on the skin of shin to estimate the total exposures of the upper leg, lower leg, and foot (renamed as "leg and foot") (also shown in Table 1). As a result, 120 pad samples (= 15 workers × 8 pad samples/worker) were obtained from this study. Before sampling, all pads were cleaned and extracted with a solvent solution (mixture of n-hexane and dichloromethane, v:v = 1:1) for 24 hrs in a Soxhlet extractor. Immediately after sampling, the pads were removed, packed in an aluminum foil, and stored at 4°C until analysis.

2.2 Sample Analyses

For PAH analyses, all extracts from the Soxhlet extractor for 24 hrs was concentrated, cleaned-up and re-concentrated to exactly 1.0mL or 0.5mL. PAH content was determined by using a gas chromatograph (GC) (Hewlett-Packard 5890A) with a mass selective detector (MSD) (Hewlett-Packard 5972) and a computer workstation. This GC/MS was equipped with a Hewlett-Packard capillary column (HP Ultra 2–50 m × 0.32 mm × 0.17 μm), an HP-7673A automatic sampler, injection volume 1 μL, splitless injection at 310°C, ion sources temperature at 310°C, oven temperature from 50°C to 100°C at 20°C min^{-1}; 100°C to 290°C at 3°C min^{-1}; and hold at 290°C for 40 min. The masses of primary and secondary ions of PAHs were determined using the scan mode for pure PAH standards. Qualification of PAHs was performed using the selected ion monitoring (SIM) mode (Tsai et al., 2002a; Tsai et al., 2001a; Chen et al., 2008; Jongeneelen et al., 1988; Tsai et al., 2001b; Tsai et al., 2004; Tsai et al., 2002b; Li et al., 2003; Lee et al., 2004; Lin et al., 2006; Chen et al., 2006).
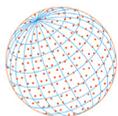
The concentrations of 22 PAH compounds were determined, including naphthalene (NaP), acenaphthylene (AcPy), acenaphthene (AcP), fluorene (Flu), phenanthrene (PA), anthracene (Ant), fluoranthene (FL), pyrene (Pyr), cyclopenta[c,d]pyrene (CYC), benz[a]anthracene (BaA), chrysene (CHR), benzo[b]fluoranthene (BbF), benzo[k]fluoranthene (BkF), benz[e]pyrene (BeP), benzo[a]pyrene (BaP), berylene (PER), indeno[1,2,3-cd]pyrene (IND), dibenz[a,h]anthracene (DBA), benzo[b]chrycene (BbC), benzo[ghi]perylene (BghiP), coronene (COR) and dibenzo[a,e]pyrene (DBP). Analysis of the serial dilution of PAH standards show that the limit of detection (LOD) of GC/MS was 0.093–1.51 ng. Five internal standards (Nap-d8, Acp-d10, PA-d10, CHR-d12, and PER-d12) were used to check the response factors and recovery efficiencies for PAHs analysis. The recovery efficiencies of 22 individual PAHs and these five internal standards were determined by processing a solution containing known PAH concentrations through the same experimental procedure that used for the analyzing samples. The recovery efficiency of PAHs varied between 0.806 and 0.925 in this study. The above values were used to adjust the observed concentration. The mean relative standard deviation (RSD) (%) of recovery efficiencies was 3.97% (range 1.34–10.4%). The blank tests for PAHs were accomplished by the same procedure as the recovery-efficiency tests without adding the known standard solution before extraction.

2.3 Data Analyses

2.3.1 Calculating personal inhalable PAH exposure concentration and PAH homologue distributions

In this study, the concentration of total inhalatory PAHs was defined as the summation of the gas phase and particle phase of the selected 22 PAH compounds. The log-normality of the exposure profile for each exposure group was examined by using the W-test (Gilbert, 1987). The arithmetic mean was used to describe the average concentration (Rappaport, 1991). The method of the minimum variance unbiased estimate (MVUE) was adopted to estimate the arithmetic mean (AM_{MVUE}) and its lower and upper one-side 95% confidence intervals ($LCL_{1,95\%}$ and $UCL_{1,95\%}$, respectively). Full calculating procedures were described in the study conducted by Attfield and Hewet (Attfield and Hewet, 1992). In addition, PAH contents were further sorted into three categories according to their molecular weights, including the low molecular weight (LMW-PAHs; containing two- and three-ringed PAHs), middle molecular weight (MMW-PAHs; containing four-ringed PAHs), and high molecular weight (HMW-PAHs; containing five-, six- and seven-ringed PAHs).

2.3.2 Calculating BaP equivalent concentrations

Because BaP has been known to be the most carcinogenic PAH compound, the carcinogenic potency of each collected sample was also determined in terms of its BaP equivalent concentration (BaP_{eq}). The carcinogenic potency of the total PAHs exposures could then be estimated as the sum of each individual BaP_{eq}. To calculate the BaP_{eq} for each individual PAH species, it requires the use of its toxic equivalent factor (TEF) for the given species relative to BaP carcinogenic potency. To date, only a few proposals for TEFs are available (Nisbet and LaGoy, 1992; Kalberlah et al., 1995; Muller, 1997). From these, the list of TEFs completed by Nisbet and LaGoy (1992) and Malcolm and Dobson (1994) were adopted in this study (Table 2), as these have been demonstrated to be a better reflection of the actual state of knowledge on the toxic potency of each individual PAH species relative to BaP (Petry et al., 1996).

2.3.3 Calculating personal dermal PAH exposure concentrations

In the present study, the unit dermal surface area PAH exposure level for total PAHs (i.e., the sum of 22 PAH species) for a given body surface area j ($UDE_{total,j}$; ng (100 cm^2 day)$^{-1}$) can be determined using Eq. (1):

$$UDE_{total,j} = UDC_{1,j} + UDC_{2,j} + \ldots + UDC_{22,j} \tag{1}$$

where, $UDC_{i,j}$ = the unit dermal surface area PAH exposure level for a specific PAH species i (22 species in total, i.e., $I = 1$–22) on the body surface area j (8 surface area in total, unit: ng (100 cm^2 day)$^{-1}$, directly obtained from the analyzed pad sample result).
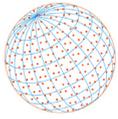
Table 2. PAHs and their toxic equivalent factors (TEFs) suggested by Nisbet and LaGoy (1992), Malcolm and Dobson (1994), Kalberlah *et al.* (1995), Muller (1997), and those adopted by the present study.

Compound	Nisbet and LaGoy (1992)	Malcolm and Dobson (1994)	Kalberlah *et al.* (1995)	Muller (1997)	TEFs used in this study
Naphthalene (Nap)	0.001	0.001	–	–	0.001
Acenaphthylene (AcPy)	0.001	0.001	0.01	–	0.001
Acenaphthene (Acp)	0.001	0.001	0.001	–	0.001
Fluorene (Flu)	0.001	0.001	0	–	0.001
Phenanthrene (PA)	0.001	0.001	0	0.00064	0.001
Anthracene (Ant)	0.01	0.01	0.01	–	0.01
Fluoranthene (FL)	0.001	0.001	0.01	–	0.001
Pyrene (Pyr)	0.001	0.001	0.001	0	0.001
Benzo(a)anthracene (BaA)	0.1	0.1	0.1	–	0.1
Chrysene (CHR)	0.01	0.01	0.01	0.026	0.01
Cyclopenta(c,d)pyrene (CYC)	–	0.1	–	0.012	0.1
Benzo(b)fluoranthene (BbF)	0.1	0.1	0.1	0.11	0.1
Benzo(k)fluoranthene (BkF)	0.1	0.1	0.1	0.037	0.1
Benzo(e)pyrene (BeP)	–	0.01	–	0	0.01
Benzo(a)pyrene (BaP)	1.0	1.0	1.0	1.0	1.0
Perylene (PER)	–	0.001	–	–	0.001
Indeno(1,2,3,-cd)pyrene (IND)	0.1	0.1	0.1	–	0.1
Dibenzo(a,h)anthracene (DBA)	1.0	1.0	1.0	0.89	1.0
Benzo(b)chrycene (BbC)	–	–	–	–	–*
Benzo(ghi)perylene (BghiP)	0.01	0.01	0.01	–	0.01
Coronene (COR)	–	0.001	–	–	0.001
Dibenzo[a,e]pyrene (DBP)	–	–	–	–	–*

* No TEF has been suggested.

Dermal PAHs exposure levels for total PAHs at the body surface area j ($DE_{total,j}$; 8 body surface area in total, i.e., j = 1–8, unit: ng day^{-1}) can be determined by Eq. (2):

$$DE_{total,j} = UDE_{total,j}\, AD_j \tag{2}$$

where, AD_j = the corresponding body surface area for the specific body surface area j (8 body surface area in total, i.e., j = 1–8, unit: 100 cm^2) of a standard man (Table 1).

Dermal PAH exposure levels for species i on the whole body surface area ($DE_{i,whole}$; ng day^{-1}) was determined by Eq. (3):

$$DE_{i,whole} = UDC_{i,1}\, AD_1 + UDC_{i,2}\, AD_2 + \ldots + UDC_{i,8}\, AD_8 \tag{3}$$

Total dermal PAHs exposure levels on the whole body surface area ($DE_{total,whole}$; ng day^{-1}) was determined using Eq. (4):

$$DE_{total,whole} = DE_{1,whole} + DE_{2,whole} + \ldots + DE_{22,whole} \tag{4}$$

2.3.4 Heath-risk assessment

Regarding the excessive lung cancer risk associated with PAH exposures via the inhalation route, WHO suggested the unit risk of 8.7×10^{-2} (μg m^{-3})$^{-1}$ for the lifetime (70 years) PAHs exposure, assuming one exposed to one unit BaP concentration (i.e., 1 μg m^{-3}) (WHO, 1987). It worth noting above unit risk was proposed to estimate the lung cancer risk caused by lifetime exposure, therefore, it has been adopted by a recent study for assessing the lung cancer risks of general adult exposure to the ambient atmospheric PAHs (Zmirou *et al.*, 2000). For occupational exposure, Pott established a relationship between BaP exposure and lung cancer risk (Pott, 1985), based on a data bank provided by an epidemiological study conducted by Redmond *et al.* (1976).
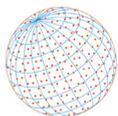
He suggested the unit risk of 7.0×10^{-2} $(\mu g\ m^{-3})^{-1}$ for a 25 years occupational PAHs exposure with the averaged BaP concentration of $1\ \mu g\ m^{-3}$. By using the same data bank, the U.S. EPA, however, suggested a different unit risk of 6.4×10^{-4} $(\mu g\ m^{-3})^{-1}$ for PAHs exposure based on its total PAH content (expressed as the benzene soluble fractions) (U.S. EPA, 1984). Since a recent study has indicated BaP could be a better indicator than total PAH content on characterizing the carcinogenic potency of PAHs (Petry *et al.*, 1996), the unit risk suggested by Pott (1985) was used in this study. In addition, to directly adopt the above unit risk, the period of 25 years occupational exposure was used in estimating workers' lung cancer risks accordingly. In the present study, the excessive lung cancer risk (LCR) associated with inhalatory PAH exposures was calculate based on the followings:

$$LCR = C_{inh\text{-}Total\text{-}BaPeq} \times 7.0 \times 10^{-2} \qquad (5)$$

where, $C_{inh\text{-}Total\text{-}BaPeq}$: the estimated the arithmetic mean (AM_{MVUE}) and its lower and upper one-side 95% confidence intervals ($LCL_{1,95\%}$ and $UCL_{1,95\%}$, respectively) inhalatory total-BaPeq concentration (unit: $\mu g\ m^{-3}$) for each selected group of workers.

To estimate the excessive skin cancer risk posed by occupational dermal PAHs exposure, assuming the body weight of the standard man (= 70 kg) is representative to all selected workers and all of them will be exposed to PAHs for 25 years (5 days per week, 50 weeks per year) during an average life span of 70 years, a unit risk 37.47 $(mg\ (kg\ bodyweight\ day^{-1})^{-1})^{-1}$ suitable for low-dose exposures (such as environmental and occupational exposures) was adopted in this study, assuming personal averaged BaP exposure level was $1\ mg\ kg^{-1}$ bodyweight day^{-1} (Hussian *et al.*, 1998). The use of the above unit risk in the present study is because the above value was obtained by extrapolating the lifetime risks from high doses to low doses by using the Model-Free Extrapolation (MFX) computer model, assuming a linear dose-response curve at low doses (Krewski *et al.*, 1991). In the present study, the excessive skin cancer risk (SCR) associated with dermal PAH exposures was calculate based on the followings:

$$SCR = (C_{derTotal\text{-}BaPeq} \times 37.47)/70 \qquad (6)$$

where, $C_{der\text{-}Total\text{-}BaPeq}$: the estimated the arithmetic mean (AM_{MVUE}) and its lower and upper one-side 95% confidence intervals ($LCL_{1,95\%}$ and $UCL_{1,95\%}$, respectively) dermal total-BaPeq concentration (mg day^{-1}) for each selected group of workers.

3 RESULTS AND DISCUSSION

3.1 Personal Inhalatory PAH Exposures

Tables 3, 4, and 5 show the estimated arithmetic mean (AM_{MVUE}), its corresponding $LCL_{1,95\%}$ and $UCL_{1,95\%}$ PAH exposure concentrations, and arithmetic mean BaP equivalent concentration (BaP_{eq}) of gas phase, particle phase and gas + particle phase for the melting, ladling, and casting workers, respectively. We found that the mean individual and total PAH exposure concentrations of the gas-phase, particle-phase and total (i.e., gas-phase + particle-phase) for each group of workers were consistently in a log-normally distributed form ($p < 0.001$, W-test). The above result suggests that workers of each group can be regarded as a similar exposure group (SEG) (Mulhausen and Damiano, 1998).

For melting workers (Table 3), it can be seen that the gas-phase PAHs exposure level ($2.17 \times 10^4\ ng\ m^{-3}$) was much higher than that of particle-phase ($6.76 \times 10^3\ ng\ m^{-3}$). The above results obviously indicate that workers' inhalatory total PAH exposures were dominated by the gas-phase PAHs (accounting for ~76.4% of the total). By examining PAH homologue distributions for both gas-phase and particle-phase PAHs, we found that the fractions of LMW-, MMW- and HMW-PAHs for the former were 95.3%, 3.6%, and 1.1%, and for the latter were 78.7%, 9.5%, and 11.9%, respectively. Though the fraction of both MMW- and HMW-PAHs for the latter were much higher than that of the former, both gas-phase and particle-phase PAHs were dominated by the LMW-PAHs. The above result can also be used to explain why the total PAHs was dominated by LMW-PAHs. The same trend also can be seen in inhalatory PAH exposures for both ladling and casting worker (see Tables 4 and 5, respectively). In principle, the results obtained from the present
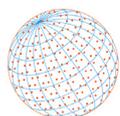
Table 3. Mean inhalatory exposure concentrations of 22 selected PAH compounds, LMW-PAHs, MMW-PAHs, HMW-PAHs, total PAHs and total BaP_{eq} and their corresponding 1-sided lower and upper 95% confidence levels ($LCL_{1,95\%}$ and $UCL_{1,95\%}$) for melting workers (n = 5) (ng m^{-3}).

PAH compounds	Gas phase			Particle phase			Gas phase+Particle phase		
	AM_{MVUE}	$LCL_{1,95\%}$	$UCL_{1,95\%}$	AM_{MVUE}	$LCL_{1,95\%}$	$UCL_{1,95\%}$	AM_{MVUE}	$LCL_{1,95\%}$	$UCL_{1,95\%}$
Nap	1.91×10^4	1.57×10^4	2.49×10^4	3.79×10^3	3.06×10^3	5.15×10^3	2.28×10^4	1.94×10^4	2.82×10^4
AcPy	3.17×10^2	2.41×10^2	4.98×10^2	7.22×10^2	5.82×10^2	9.86×10^2	1.04×10^3	9.48×10^2	1.15×10^3
Acp	1.83×10^2	1.52×10^2	2.34×10^2	3.88×10^2	3.12×10^2	5.31×10^2	5.70×10^2	4.98×10^2	6.72×10^2
Flu	1.80×10^2	1.50×10^2	2.31×10^2	1.84×10^2	1.49×10^2	2.46×10^2	3.63×10^2	3.26×10^2	4.12×10^2
PA	7.75×10^1	5.98×10^1	1.17×10^2	5.77×10^1	4.76×10^1	7.51×10^1	1.35×10^2	1.14×10^2	1.69×10^2
Ant	8.68×10^2	6.79×10^2	1.27×10^3	1.75×10^2	1.44×10^2	2.31×10^2	1.04×10^3	8.47×10^2	1.40×10^3
FL	4.19×10^2	3.44×10^2	5.47×10^2	1.73×10^2	1.40×10^2	2.31×10^2	5.90×10^2	5.13×10^2	7.04×10^2
Pyr	3.04×10^2	2.52×10^2	3.92×10^2	3.07×10^2	2.49×10^2	4.13×10^2	6.10×10^2	5.42×10^2	7.03×10^2
BaA	3.79×10^1	2.67×10^1	7.56×10^1	8.98×10^1	7.36×10^1	1.18×10^2	1.27×10^2	1.04×10^2	1.69×10^2
CHR	1.90×10^1	1.53×10^1	2.59×10^1	7.04×10^1	5.56×10^1	1.00×10^2	8.93×10^1	7.35×10^1	1.17×10^2
CYC	6.35×10^1	4.83×10^1	9.96×10^1	1.33×10^2	1.06×10^2	1.85×10^2	1.92×10^2	1.61×10^2	2.58×10^2
BbF	6.14×10^1	5.06×10^1	8.03×10^1	2.33×10^2	1.87×10^2	3.21×10^2	2.94×10^2	2.48×10^2	3.67×10^2
BkF	7.38	5.70	1.11×10^1	2.47×10^1	1.87×10^1	3.95×10^1	3.20×10^1	2.51×10^1	4.67×10^1
BeP	4.63×10^1	3.41×10^1	8.00×10^1	1.19×10^2	9.47×10^1	1.65×10^2	1.65×10^2	1.33×10^2	2.22×10^2
BaP	6.43	4.41	1.42×10^1	2.32×10^1	1.75×10^1	3.73×10^1	2.96×10^1	2.24×10^1	4.69×10^1
PER	4.64	3.74	6.31	2.08×10^1	1.55×10^1	3.43×10^1	2.54×10^1	2.05×10^1	3.45×10^1
IND	9.14	7.58	1.18×10^1	7.14×10^1	5.77×10^1	9.70×10^1	8.05×10^1	6.76×10^1	1.01×10^2
DBA	4.12	3.39	5.38	1.32×10^1	1.07×10^1	1.79×10^1	1.74×10^1	1.51×10^1	2.07×10^1
BbC	9.27	7.69	1.20×10^1	2.86×10^1	2.17×10^1	4.48×10^1	3.78×10^1	3.12×10^1	4.91×10^1
Bghip	1.77×10^1	1.17×10^1	4.52×10^1	1.23×10^2	1.00×10^2	1.63×10^2	1.40×10^2	1.25×10^2	1.62×10^2
COR	1.56×10^1	1.30×10^1	2.00×10^1	1.36×10^1	1.10×10^1	1.84×10^1	2.92×10^1	2.70×10^1	3.19×10^1
DBP	ND	ND	ND	ND	ND	ND	ND	ND	ND
LMW-PAHs	2.07×10^4	1.70×10^4	2.73×10^4	5.32×10^3	4.29×10^3	7.21×10^3	2.60×10^4	2.21×10^4	3.20×10^4
MMW-PAHs	7.79×10^2	6.38×10^2	1.04×10^3	6.40×10^2	5.19×10^2	8.63×10^2	1.42×10^3	1.23×10^3	1.69×10^3
HMW-PAHs	2.46×10^2	1.90×10^2	3.86×10^2	8.03×10^2	6.41×10^2	1.12×10^3	1.05×10^3	8.76×10^2	1.34×10^3
Total PAHs	2.17×10^4	1.78×10^4	2.87×10^4	6.76×10^3	5.45×10^3	9.20×10^3	2.84×10^4	2.42×10^4	3.51×10^4
Total BaP_{eq}	5.86×10^1	4.60×10^1	8.85×10^1	1.02×10^2	8.11×10^1	1.46×10^2	1.61×10^2	1.32×10^2	2.13×10^2

study are not so surprising. For example, a higher fraction in LMW-PAHs for the gas-phase PAHs was mainly because of the intrinsic higher volatility nature of LMW-PAHs as in comparison with both MMW- and HMW-PAHs. To the contrary, because both MMW-PAHs and HMW-PAHs are less volatile and hence their fractions in particle-phase PAHs were higher than those of gas-phase PAHs. The above findings agree well with Yang *et al.* (2002) which shows that LMW-PAHs are the predominant species sampled from the stacks in steel and iron plant with an EAC.

However, it should be noted that the contributions of the gas-phase PAHs to the total PAH exposures for melting workers (76.4%) was much lower than those of ladling and casting workers (= 96.5% and 91.8%, respectively). Based on our field observation, though temperatures involved in the melting process was much higher that of ladling and casting, the larger amount of gas-phase PAHs were directly exhausted to the installed air pollution control devices because of the upward thermal draft force and the drawing force caused by the fan. On the other hand, lower involved temperatures were involved in both ladling and casting processes, though less gas-phase PAHs were generated, most of them were discharged directly to the workplace atmosphere. As a result, the contributions of the gas-phase PAHs to the total PAH exposures for ladling and casting workers could be higher than that of melting workers.

Table 3 also shows the estimated total BaP_{eq} exposure level for melting workers. It can be seen that the gas-phase total BaP_{eq} exposure level (5.86×10^1 ng m^{-3}) was lower than that of particle phase (1.02×10^2 ng m^{-3}) (accounting for ~36.4% and ~63.6% of total inhalatory exposures (1.61×10^2 ng m^{-3}), respectively). The above result was mainly because gas-phase PAHs were dominated by PAHs with lower TEFs (i.e., LMW-PAHs), but particle-phase PAHs had higher fractions
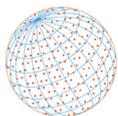
Table 4. Mean inhalatory exposure concentrations of 22 selected PAH compounds, LMW-PAHs, MMW-PAHs, HMW-PAHs, total PAHs and total BaP$_{eq}$ and their corresponding 1-sided lower and upper 95% confidence levels (LCL$_{1,95\%}$ and UCL$_{1,95\%}$) for ladling workers (n = 5) (ng m^{-3}).

PAH compounds	Gas phase			Particle phase			Gas phase + Particle phase		
	AM$_{MVUE}$	LCL$_{1,95\%}$	UCL$_{1,95\%}$	AM$_{MVUE}$	LCL$_{1,95\%}$	UCL$_{1,95\%}$	AM$_{MVUE}$	LCL$_{1,95\%}$	UCL$_{1,95\%}$
Nap	1.38×10^5	1.21×10^5	1.61×10^5	3.54×10^3	2.82×10^3	4.95×10^3	1.41×10^5	1.25×10^5	1.64×10^5
AcPy	3.29×10^3	2.87×10^3	3.89×10^3	6.57×10^2	5.25×10^2	9.11×10^2	3.94×10^3	3.60×10^3	4.38×10^3
Acp	1.66×10^3	1.44×10^3	1.99×10^3	3.92×10^2	3.11×10^2	5.51×10^2	2.05×10^3	1.88×10^3	2.27×10^3
Flu	1.61×10^3	1.39×10^3	1.93×10^3	1.91×10^2	1.53×10^2	2.64×10^2	1.80×10^3	1.61×10^3	2.06×10^3
PA	3.36×10^2	2.90×10^2	4.05×10^2	3.74×10^1	3.03×10^1	5.07×10^1	3.74×10^2	3.28×10^2	4.38×10^2
Ant	5.19×10^3	4.57×10^3	6.06×10^3	1.66×10^2	1.32×10^2	2.32×10^2	5.36×10^3	4.75×10^3	6.18×10^3
FL	8.53×10^2	7.42×10^2	1.01×10^3	4.59×10^1	3.66×10^1	6.37×10^1	8.99×10^2	7.94×10^2	1.04×10^3
Pyr	7.40×10^2	6.25×10^2	9.22×10^2	7.76×10^1	5.99×10^1	1.17×10^2	8.17×10^2	7.16×10^2	9.61×10^2
BaA	5.51×10^1	4.54×10^1	7.20×10^1	4.98×10^1	3.86×10^1	7.40×10^1	1.05×10^2	8.89×10^1	1.30×10^2
CHR	6.92×10^1	5.66×10^1	9.16×10^1	6.74×10^1	5.42×10^1	9.26×10^1	1.36×10^2	1.27×10^2	1.48×10^2
CYC	5.57×10^1	4.78×10^1	6.77×10^1	1.06×10^1	8.53	1.43×10^1	6.63×10^1	5.81×10^1	7.78×10^1
BbF	4.78×10^1	3.96×10^1	6.18×10^1	4.82×10^1	3.49×10^1	8.76×10^1	9.58×10^1	7.99×10^1	1.22×10^2
BkF	4.59	3.77×10^1	6.00×10^1	3.11×10^1	2.57×10^1	4.03×10^1	7.70×10^1	6.65×10^1	9.27×10^1
BeP	7.36×10^1	5.91×10^1	1.01×10^2	4.38×10^1	3.38×10^1	6.63×10^1	1.17×10^2	1.10×10^2	1.26×10^2
BaP	4.79×10^1	3.99×10^1	6.14×10^1	2.61×10^1	2.08×10^1	3.63×10^1	7.40×10^1	6.35×10^1	8.96×10^1
PER	5.69×10^1	5.01×10^1	6.66×10^1	1.41×10^1	1.13×10^1	1.94×10^1	7.11×10^1	6.47×10^1	7.91×10^1
IND	4.33×10^1	3.11×10^1	8.10×10^1	2.47×10^1	1.86×10^1	3.94×10^1	6.80×10^1	5.10×10^1	1.11×10^2
DBA	7.87×10^1	6.50×10^1	1.02×10^2	1.32×10^1	1.08×10^1	1.73×10^1	9.18×10^1	7.80×10^1	1.13×10^2
BbC	4.28×10^1	3.40×10^1	6.01×10^1	1.05×10^1	8.43	1.43×10^1	5.32×10^1	4.36×10^1	7.02×10^1
Bghip	6.63×10^1	5.55×10^1	8.39×10^1	1.49×10^1	1.10×10^1	2.51×10^1	8.11×10^1	7.36×10^1	9.07×10^1
COR	3.71×10^1	3.25×10^1	4.34×10^1	2.05	1.59	3.05	3.91×10^1	3.46×10^1	4.53×10^1
DBP	3.33×10^1	2.89×10^1	3.99×10^1	2.00	1.65	2.60	3.53×10^1	3.10×10^1	4.15×10^1
LMW-PAHs	1.50×10^5	1.32×10^5	1.76×10^5	4.98×10^3	3.97×10^3	6.96×10^3	1.55×10^5	1.37×10^5	1.79×10^5
MMW-PAHs	1.72×10^3	1.47×10^3	2.10×10^3	2.41×10^2	1.89×10^2	3.47×10^2	1.96×10^3	1.73×10^3	2.28×10^3
HMW-PAHs	6.29×10^2	5.21×10^2	8.29×10^2	2.41×10^2	1.87×10^2	3.66×10^2	8.70×10^2	7.54×10^2	1.06×10^3
Total PAHs	1.52×10^5	1.34×10^5	1.79×10^5	5.46×10^3	4.35×10^3	7.67×10^3	1.58×10^5	1.40×10^5	1.83×10^5
Total BaP$_{eq}$	3.52×10^2	3.01×10^2	4.33×10^2	6.35×10^1	5.05×10^1	9.02×10^1	4.15×10^2	3.61×10^2	4.97×10^2

for PAHs with higher TEFs (i.e., MMW- and HMW-PAHs). Here, it should be noted that a different trend can be seen in total BaP$_{eq}$ exposures for both ladle and casting workers (Tables 4 and 5). For both groups of workers, their gas-phase total BaP$_{eq}$ exposure levels (= 3.52×10^2 ng m^{-3} and 6.75×10^1 ng m^{-3}, respectively) was higher than that of particle-phase (= 6.35×10^1 ng m^{-3} and 3.07×10^1 ng m^{-3}, respectively). Obviously, the above results can be explained by the fraction of the gas-phase PAHs in total PAH exposures for both ladling and casting workers was much higher than those of melting workers. However, the above results are quite different from those found in other studies, such as the carbon black manufacturing (Tsai *et al.*, 2001), and sintering (Lin *et al.*, 2008). It might because both ladling and casting involve the cooling process, which lead to less MMW- and HMW-PAH emissions, as in comparison with the high temperature involved melting process. Considering the mechanisms associated with the formation of PAHs is complicated, further investigations are needed in the future.

The present study also shows that mean inhalatory exposure levels for melting and casting workers (= 2.84×10^4 ng m^{-3} and 3.32×10^4 ng m^{-3}) were lower than the time-weighted average permissible exposure level (PEL-TWA) regulated in Taiwan for PAHs (= 1.00×10^5 ng m^{-3}). However, both the mean exposure level (1.58×10^5 ng m^{-3}) and its corresponding LCL$_{1,95\%}$ (1.40×10^5 ng m^{-3}) for ladle workers were higher than the above limit value. The above results clearly indicates the need for the abatement of inhalatory PAH exposures for ladling workers.

3.2 Personal Dermal PAH Exposures

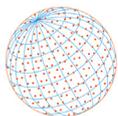Table 6 shows both the total PAHs and total BaP$_{eq}$ dermal exposure levels for the whole body

Table 5. Mean inhalatory exposure concentrations of 22 selected PAH compounds, LMW-PAHs, MMW-PAHs, HMW-PAHs, total PAHs and total BaP_{eq} and their corresponding 1-sided lower and upper 95% confidence levels ($LCL_{1,95\%}$ and $UCL_{1,95\%}$) for casting workers (n = 5) (ng m^{-3}).

PAH compounds	Gas phase			Particle phase			Gas phase+Particle phase		
	AM_{MVUE}	$LCL_{1,95\%}$	$UCL_{1,95\%}$	AM_{MVUE}	$LCL_{1,95\%}$	$UCL_{1,95\%}$	AM_{MVUE}	$LCL_{1,95\%}$	$UCL_{1,95\%}$
Nap	2.76×10^4	2.52×10^4	3.07×10^4	1.77×10^3	1.60×10^3	1.99×10^3	2.93×10^4	2.69×10^4	3.24×10^4
AcPy	6.63×10^2	6.08×10^2	7.30×10^2	3.28×10^2	2.97×10^2	3.68×10^2	9.81×10^2	9.20×10^2	1.05×10^3
Acp	3.36×10^2	3.10×10^2	3.68×10^2	1.96×10^2	1.77×10^2	2.20×10^2	5.26×10^2	4.94×10^2	5.62×10^2
Flu	3.26×10^2	3.00×10^2	3.57×10^2	9.55×10^1	8.63×10^1	1.07×10^2	4.18×10^2	3.91×10^2	4.49×10^2
PA	6.23×10^1	5.63×10^1	6.99×10^1	1.93×10^1	1.75×10^1	2.17×10^1	8.10×10^1	7.48×10^1	8.86×10^1
Ant	1.03×10^3	9.38×10^2	1.15×10^3	8.29×10^1	7.49×10^1	9.31×10^1	1.11×10^3	1.02×10^3	1.23×10^3
FL	1.74×10^2	1.59×10^2	1.92×10^2	2.29×10^1	2.07×10^1	2.57×10^1	1.96×10^2	1.81×10^2	2.14×10^2
Pyr	1.51×10^2	1.40×10^2	1.65×10^2	3.90×10^1	3.48×10^1	4.44×10^1	1.89×10^2	1.76×10^2	2.04×10^2
BaA	3.29	2.76	4.13	2.50×10^1	2.24×10^1	2.84×10^1	2.73×10^1	2.52×10^1	2.99×10^1
CHR	1.45×10^1	1.34×10^1	1.59×10^1	3.36×10^1	3.04×10^1	3.78×10^1	4.72×10^1	4.42×10^1	5.07×10^1
CYC	1.17×10^1	1.07×10^1	1.30×10^1	5.19	4.32	6.59	1.70×10^1	1.59×10^1	1.81×10^1
BbF	7.61	5.91	1.11×10^1	2.86×10^1	2.60×10^1	3.18×10^1	3.53×10^1	3.25×10^1	3.88×10^1
BkF	9.61	7.60	1.35×10^1	3.75	3.07	4.93	1.34×10^1	1.10×10^1	1.75×10^1
BeP	1.51×10^1	1.36×10^1	1.70×10^1	2.33×10^1	2.10×10^1	2.61×10^1	3.76×10^1	3.51×10^1	4.05×10^1
BaP	9.00	7.74	1.08×10^1	1.27×10^1	1.15×10^1	1.43×10^1	2.14×10^1	1.98×10^1	2.33×10^1
PER	1.25×10^1	1.17×10^1	1.35×10^1	7.24	6.47	8.25	1.96×10^1	1.83×10^1	2.11×10^1
IND	7.47	5.85	1.07×10^1	1.24×10^1	1.10×10^1	1.45×10^1	1.94×10^1	1.69×10^1	2.28×10^1
DBA	1.44×10^1	1.28×10^1	1.65×10^1	6.48	5.53	7.89	2.09×10^1	1.92×10^1	2.28×10^1
BbC	6.10	4.56	9.76	4.95	4.43	5.63	1.10×10^1	9.43	1.32×10^1
Bghip	1.36×10^1	1.25×10^1	1.48×10^1	9.45	8.42	1.08×10^1	2.29×10^1	2.15×10^1	2.45×10^1
COR	4.81	3.87	6.50	0.99	0.79	1.35	5.83	4.87	7.36
DBP	0.618	0.533	0.743	0.780	0.695	0.887	1.38	1.30	1.48
LMW-PAHs	3.01×10^4	2.74×10^4	3.34×10^4	2.49×10^3	2.25×10^3	2.80×10^3	3.25×10^4	2.98×10^4	3.58×10^4
MMW-PAHs	3.43×10^2	3.15×10^2	3.77×10^2	1.21×10^2	1.08×10^2	1.36×10^2	4.59×10^2	4.26×10^2	4.98×10^2
HMW-PAHs	1.13×10^2	9.74×10^1	1.38×10^2	1.16×10^2	1.03×10^2	1.33×10^3	2.26×10^2	2.06×10^2	2.52×10^2
Total PAHs	3.05×10^4	2.78×10^4	3.39×10^4	2.73×10^3	2.46×10^3	3.06×10^3	3.32×10^4	3.04×10^4	3.65×10^4
Total BaP_{eq}	6.75×10^1	6.03×10^1	7.73×10^1	3.07×10^1	2.73×10^1	3.52×10^1	9.75×10^1	8.94×10^1	1.07×10^2

Table 6. Mean unit surface area total PAHs and total BaPeq exposure concentrations for the 8 selected body surface areas ($UDE_{total,j}$) and total PAHs and total BaPeq exposure concentrations for the whole body surface area ($DE_{total, whole}$) of the three selected groups of workers.

Body surface	Melting (n = 5)		Ladling (n = 5)		Casting (n = 5)	
	Total PAHs	Total BaP_{eq}	Total PAHs	Total BaP_{eq}	Total PAHs	Total BaP_{eq}
$UDE_{total,j}$ (ng (cm^2 day)$^{-1}$)						
Head/front	27.4	4.25	27.0	4.18	23.8	3.59
Head/back	27.8	4.28	25.6	4.23	22.4	3.34
Neck/front	29.4	4.24	32.0	5.06	23.9	3.24
Neck/back	29.3	4.37	29.6	4.47	21.3	3.07
Chest/abdomen	25.8	4.22	31.8	5.37	22.2	3.54
Back	26.0	4.06	31.0	4.98	20.6	3.27
Arm and hand	31.8	4.67	31.5	4.68	23.0	3.19
Leg and foot	26.8	3.99	29.5	4.73	19.4	2.81
$DE_{total, whole}$ (ng day^{-1})	7.45×10^5	8.86×10^4	3.64×10^5	6.24×10^4	2.84×10^5	4.22×10^4

surface area ($DE_{total,whole}$) and each individual body surface ($UDE_{total,j}$). Results show that the mean $DE_{total,whole}$ in total PAHs were 7.45×10^5, 3.64×10^5, and 2.84×10^5 ng day^{-1} for melting, ladle, and casting workers, respectively, and in total BaP_{eq} were 8.86×10^4, 6.24×10^4, and 4.22×10^4 ng day^{-1}, respectively. The highest exposure level was found in melting workers could because
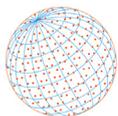
they involved in directly handling residual slag to notch. As a result, in addition to being exposed to diffused particle-phase PAHs (affecting neck/back exposures), both direct dermal contact and particle inertial effect could be another two important mechanisms for increasing their dermal exposures on both arm and hand, and neck/front regions. The above inference was supported by the fact that the body surface areas of melting workers with less covered by clothes, such as the arm and hand, neck/front, and neck/back region, were the top three exposed body surfaces. The second highest exposure level was found for ladling workers with the top three exposed body surfaces of the neck/front, chest/abdomen, and arm and hand. This was possibly due to ladle workers were associated with directly facing melted metal during pouring process, therefore, all these front body regions were much easily to be contaminated. For casting workers, the higher exposed body surfaces were neck/front and head/front which might because bending gesture was involved in the casting process. As a result, particle gravitational deposition could be the most important mechanism for affecting their dermal exposures in these two regions. Since dermal exposures were mainly affected by the particle-phase PAHs, therefore, the above trend can also be seen in dermal total BaP_{eq} exposure levels for each individual body surface (Table 6).

In this study, we also found that the fractions of LMW-, MMW-, and HMW-PAHs in $DE_{total,whole}$ respectively for melting workers were 23.0%, 12.1%, and 64.8%, for ladle workers were 29.1%, 20.1%, and 50.8%, and for casting workers were 27.4%, 18.7%, and 53.9% (data not shown in Table 6). The above fractions were quite comparable with the homologue distributions of particle-phase PAHs found for the three investigated groups of workers (see Tables 3–5). The results further confirm that airborne particle-phase PAHs was the main contributor for dermal exposures.

3.3 Health-risks Associated with Inhalatory and Dermal PAH Exposures

Table 7 shows the estimated mean, and its corresponding $LCL_{1,95\%}$ and $UCL_{1,95\%}$ lifetime excessive lung cancer risks (LCRs) for the three studied exposure groups according to their inhalatory total BaP_{eq} levels. Their mean cancer risks (and its corresponding $LCL_{1,95\%}$ and $UCL_{1,95\%}$) were found to be 3.43×10^{-3} (2.71×10^{-3} and 4.53×10^{-3}), 8.84×10^{-3} (7.47×10^{-3} and 1.02×10^{-2}), and 2.09×10^{-3} (1.86×10^{-3} and 2.39×10^{-3}), for melting, ladle, and casting workers, respectively. In principle, the range for the estimated LCR for each selected group of workers is quite narrow. The above results further confirms that each selected group of workers can be regarded as a similar exposure group (SEG). Here, it should be noted that all these values are consistently higher than the significant occupational cancer risk level (10^{-3}) defined by the US Supreme Court indicating that the inhalatory exposure levels for all three selected exposure groups are unacceptable (Rodrics *et al.*, 1987).

Table 7 also shows the estimated mean, and its corresponding $LCL_{1,95\%}$ and $UCL_{1,95\%}$ lifetime excessive skin cancer risks (SCRs) for the three studied exposure groups according to their dermal total BaP_{eq} levels. To date, the absorption rate for PAHs deposited on the skin surface still remains unknown. However, U.S. EPA (1992) has suggested a maximum absorption rate of 20% for PAHs for PAHs-containing soil in 24 hrs (U.S. EPA, 1992). The above value was adopted in this study. The resultant lifetime excessive skin cancer risks (and its corresponding $LCL_{1,95\%}$ and $UCL_{1,95\%}$) were found to be 1.62×10^{-4} (1.07×10^{-4} and 4.10×10^{-4}), 1.14×10^{-4} (1.05×10^{-4} and 1.26×10^{-4}), and 7.74×10^{-5} (6.39×10^{-5} and 9.98×10^{-5}) for smelting, ladle, and molding workers, respectively.

Table 7. Estimated lifetime excessive cancer risks associated with both inhalatory and dermal PAH exposures for the three selected SEGs.

| Type of exposure	Melting			Ladling			Casting		
	Mean	$LCL_{1,95\%}$	$UCL_{1,95\%}$	Mean	$LCL_{1,95\%}$	$UCL_{1,95\%}$	Mean	$LCL_{1,95\%}$	$UCL_{1,95\%}$
Inhalatory exposures									
Gas phase PAHs	1.25×10^{-3}	9.79×10^{-4}	1.88×10^{-3}	7.49×10^{-3}	6.40×10^{-3}	9.21×10^{-3}	1.44×10^{-3}	1.28×10^{-3}	1.64×10^{-3}
Particle phase PAHs	2.18×10^{-3}	1.73×10^{-3}	3.10×10^{-3}	1.35×10^{-3}	1.07×10^{-3}	1.92×10^{-3}	6.52×10^{-4}	5.80×10^{-4}	7.50×10^{-4}
Total PAHs	3.43×10^{-3}	2.71×10^{-3}	4.53×10^{-3}	8.84×10^{-3}	7.47×10^{-3}	1.02×10^{-2}	2.09×10^{-3}	1.86×10^{-3}	2.39×10^{-3}
Dermal exposures									
Total PAHs	1.62×10^{-4}	1.07×10^{-4}	4.10×10^{-4}	1.14×10^{-4}	1.05×10^{-4}	1.26×10^{-4}	7.74×10^{-5}	6.39×10^{-5}	9.98×10^{-5}
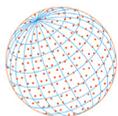
Table 8. Estimated lifetime excessive cancer risks (including lung and dermal) associated with PAH exposure to workers in different industries.

Exposure group	PAH lifetime cancer risk		Reference
	lung	dermal	
Carbon black manufacturing	[a] 4.35×10^{-2}–5.42×10^{-2}	1.13×10^{-3}–1.56×10^{-3}	Tsai *et al.*, 2001
Sintering	[a] 3.18×10^{-5}–4.98×10^{-5}	—	Lin *et al.*, 2008
	[b] 5.3×10^{-7}–1.5×10^{-5}	—	Jackson *et al.*, 2012
Fastener manufacturing	[a] 1.64×10^{-2}	9.72×10^{-3}	Chen *et al.*, 2008
Foundry	[a] 9.06×10^{-4}–1.09×10^{-3}	—	Liu *et al.*, 2010
EAF steelmaking	[b] 1.6×10^{-5}	—	Aries *et al.*, 2008
Basic oxygen steelmaking	[b] 2.4×10^{-6}–7.3×10^{-6}	—	Jackson *et al.*, 2012
Electric Arc Furnace Steel and Iron Manufacturing	2.09×10^{-3}–8.84×10^{-3}	7.74×10^{-5}–1.62×10^{-4}	This study

[a] The unit risk of 7×10^{-5} (ng m^{-3})$^{-1}$ was adopted and a worker's occupational exposure period was assumed to be 25 years.
[b] An inhalation potency factor of 3.9 (mg kg^{-1} day^{-1})$^{-1}$ for Bap was adopted and a worker's occupational exposure period was assumed to be 40 years.

Obviously, the estimated lifetime excessive skin cancer risks were less than the corresponding lung cancer risks (2.09×10^{-3}–8.84×10^{-3}). Particularly, all resultant skin cancer risks were less than the significant level 10^{-3}. However, it also should be noted that lifetime excessive skin cancer risks obtained from this study were estimated based on the maximum absorption rate of 20% that originally suggested for PAHs adsorbed by soil. Therefore, the above rate used in this study required further investigation in the future. Finally, the range for the estimated SCR for each selected group of workers is also quite narrow. Again, the above results further indicate that each selected group of workers can be considered as a similar exposure group (SEG).

Table 8 lists the estimated lifetime excessive cancer risks (including lung and dermal) associated with PAH exposures to workers in different industries for the comparison purpose. It can be seen that the excessive lung cancer risks found in this study (2.09×10^{-3}–8.84×10^{-3}) were higher than those in foundry (9.06×10^{-4}–1.09×10^{-3}) and sinter plant (3.18×10^{-5}–4.98×10^{-5}). In particular, the present study found that the estimated excessive lung cancer risks were higher than the corresponding excessive skin cancer risks. The same trend can also be found in both industries with estimated excessive skin cancer risks (i.e., the carbon black and fastener manufacturing industries). Since only two studies estimated excessive skin cancer risks, if the above trend is applicable to other industries warrants the needs for further investigation.

3.4 Control Strategies

Since the estimated lifetime excessive skin cancer risks (7.74×10^{-5}–1.62×10^{-4}) were lower than the obtained lung cancer risk (2.09×10^{-3}–8.84×10^{-3}), particularly also lower than the significant level 10^{-3}, which indicates that the control of workers' inhalatory exposures is more important than that of dermal exposures from the health risk assessment aspect. In the present study, control measurements for reducing workers' inhalatory PAH exposures are recommended and prioritized, which includes the change of manufacturing procesess for reducing PAH generation (the first prioritized control measurement), the installation of local exhaust ventilation systems to prevent from PAH emissions (the second prioritized control measurement), the installation of general exhaust ventilation systems to dilute workplace PAH concentrations (the third prioritized control measurement), and providing personal respiratory protection equipment (PPE) for reducing workers' inhalatory PAH exposures (the last prioritized control measurement). If the last resort was taken, the selected PPE should be accompanied with a proper cooling system to reduce thermal stress posed on workers.

4 CONCLUSIONS

We found that the inhalatory gas-phase PAH exposure level was much higher than that of particle-phase. The above results suggest that workers' inhalatory PAH expsoures were dominated by the gas-phase. By combining both gas- and particle-phase PAHs, only ladling workers' mean
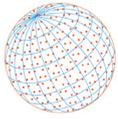
inhalatory total PAH exposure levels were higher than the PEL-TWA. Apparently, inhalatory PAH exposures for both casting and ladling workers were not so significant. But from the health-risk management aspect, we found that the estimated lifetime excessive lung cancer risk for the three selected exposure groups were all higher than the significant risk level defined by the US Supreme Court in 1980. As examining workers' dermal exposures, we found that the levels for the melting workers were higher than that of ladling and casting workers. The estimated lifetime excessive skin cancer risk caused by dermal PAH exposures was lower than that of inhalatory cancer risks caused by inhalatory PAH exposures. Since all estimated lifetime excessive skin cancer risk were lower than the significant risk level, it is concluded that the control of workers' inhalatory exposures would be more important than that of dermal exposures for EAF workers. The installation of effective ventilation systems and the use proper personal respiratory protection equipments would be beneficial to the industry for reducing health risks posed on workers.

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DISCLAIMER

Reference to any companies or specific commercial products does not constitute.

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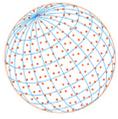
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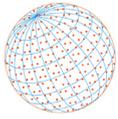
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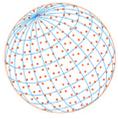
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