(Revised MS: ENVINT-D-07-00727)

Assessing Inhalatory and Dermal Exposures and Their Resultant Health-Risks for Workers Exposed to Polycyclic Aromatic Hydrocarbons (PAHs) Contained in Oil Mists in a Fastener Manufacturing Industry

Mei-Ru Chen[1], Ying-Fang Wang[1], Perng-Jy Tsai[1,2]*, Wen-Jhy Lee[2,3]

[1]*Department of Environmental and Occupational Health, Medical College, National Cheng Kung University. 138, Sheng-Li Rd, Tainan 70428, Taiwan*

[2]*Sustainable Environment Research Center, National Cheng Kung University. 1, University Road, Tainan 70101, Taiwan*

[3]*Department of Environmental Engineering, National Cheng Kung University, 1 University Road, Tainan 70101, Taiwan*

***Corresponding author**: Perng-Jy Tsai, Department of Environmental and Occupational Health, Medical College, National Cheng Kung University. 138, Sheng-Li Road, Tainan 70428, Taiwan. Tel.: +886-6-2088390; Fax: +886-6-2752484; *E-mail address*: pjtsai@mail.ncku.edu.tw

Abstract

This study first assessed workers' inhalatory and dermal exposures to polycyclic aromatic hydrocarbon (PAH) contained in oil mists. Then, their resultant lung cancer and skin cancer risks were estimated. Finally, control strategies were initiated from the health risk management aspect. All threading workers in a fastener manufacturing plant were included. 16 inhalatory and 88 dermal PAH exposure samples were collected. Results show that the inhalatory gas phase total PAH exposure level (8.60×10^{4} ng/m^3) was much higher than that of particle phase (2.90×10^{3} ng/m^3). Workers' mean inhalatory exposure level (8.83×10^{4} ng/m^3) was lower, but its corresponding 1-sided upper 95% confidence level ($UCL_{1,95\%}=1.02\times10^{5}$ ng/m^3) was higher than the time-weighted average permissible exposure level (PEL-TWA) regulated in Taiwan for PAHs (1.00×10^{5} ng/m^3). The mean whole body total PAHs dermal exposure levels was 5.44×10^{6} ng/day and the top five exposed surface areas were lower arm, hand, upper arm, neck, and head/front. The estimated lifetime skin cancer risk (9.72×10^{-3}) was lower than that of lung cancer risk (1.64×10^{-2}), but both were higher than the significant risk level (10^{-3}) defined by the US Supreme Court in 1980. The installation of a local exhaust ventilation system at the threading machine should be considered as the first priority measurement because both lung and skin cancer risks can be reduced simultaneously. If the personal protection equipment would be adopted in the future, both respiratory protection equipment and protective clothing should be used simultaneously.

Keywords: Polycyclic aromatic hydrocarbons (PAHs); Oil mists; Exposure assessment; Health risk assessment; Fastener manufacturing industry

1. Introduction

Based on the Taiwan governmental statistics in 2002, there were ~1,270 fastener manufacturing plants and in total employed with ~37,000 employees in the whole country. The total fastener production rates increased from ~451,000 tons/yr in 1991 to ~1,269,000 tons/yr in 2003 accounting for ~14% world production. The manufacture of fasteners involves seven important industrial processes, including the wire drawing, forming, threading, cleaning, heat treatment, surface treatment, and packaging/shipping. Among them, mineral oil-based metalworking fluids (MWFs) are used in forming, threading, and heat treatment processes for cooling, lubricating, and corrosion inhibition purposes and hence might result in the emission of oil mist to the workplace atmosphere and the exposures of workers (Thornburg, 2000; Michalek et al., 2003). Considering mineral oils are produced by petroleum distillation processes, semi-volatile organic compounds, such as polycyclic aromatic hydrocarbons (PAHs), are expected to be contained in emitted oil mists. Epidemiological and animal studies have indicated that oil mist exposures might result in the laryngeal cancer (Russi et al., 1997), asthma (Robertson et al., 1988), bronchial hyper-responsiveness (Kennedy et al., 1999), lipoid pneumonia (Cullen et al., 1981), lung cancer (Kazerouni et al., 2000), and many other respiratory illnesses (Jarvholm et al., 1982; Massin et al., 1996). Particularly, workers exposed to high airborne concentrations of PAHs might result in excessive lung cancer rates (Verma et al., 1992; Moulin et al., 1993). In addition, intensive studies have also reported that all mineral oils are mutagenic to skin because of their inherent PAH contents (Bingham et al., 1969; Roy et al., 1988; Granella et al., 1995). However, it should be noted that only very limited studies have been conducted to address workers' dermal exposures to PAHs (Jongeneelen et al., 1988; Tsai et al., 2001a).

In fastener manufacturing industries, workers are exposed to PAHs contained in oil mists via both inhalatory and dermal exposure routes. Therefore, the present study was set out first to assess workers' inhalatory and dermal PAH exposures. Then, their resultant lung cancer and skin cancer risks were estimated. Finally, control strategies were initiated from the health risk management

aspect.

2. Material and Methods

2.1 Personal inhalable and dermal PAH exposure samplings

For fastener manufacturing industries, the forming, threading, and heat treatment processes are involved in the use of mineral oil-based MWFs for cooling, lubricating, and corrosion inhibition purposes. Among them, the threading workers were found with the highest oil mist exposures in our previous study (Chen et al., 2007), and hence were selected for illustration. All threading workers were included to conduct personal samplings for assessing their inhalable and dermal PAH exposures.

The sampling method modified from the NIOSH method 5515 was used to assess workers' inhalatory PAH exposures. The sampling train consisted of a filter cassette (IOM personal inhalable aerosol sampler, SKC Inc., Catalog No. 225-70) and followed by a sorbent tube (polyurethane form (PUF) plug/3.5 g ??? (XAD-2) resins/PUF separation layer/0.5 g XAD-2 resin/ PUF plug). The sampling flow rate was specified at 2.0 L/min. 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. Oil mists collected by the IOM personal sampler were determined by using an electrical balance (Sartorius MP 8-6, ±0.01mg), then sent to the laboratory, together with the sorbent tube, for PAHs analysis to determine the concentrations of both particle phae PAHs and gas phase PAHs, respectively. A total of 16 personal inhalable samples (gas+particle phase PAH samples) were collected from this study.

The surrogate skin sampling technique was adopted in this study for dermal exposure assessment. The method was first recognized as a standard method for assessing dermal pesticide exposures (WHO, 1986; US EPA, 1987), but its use has been extended to assess occupational exposures to PAHs (Jongeneelen et al., 1988) 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 11 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 US EPA (US EPA, 1987). However, subjected to worker's willingness, 11 sampling spots closed to the above mentioned sampling body sites were chosen for each selected worker (also shown in Table 1). As a result, 88 pad samples 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 PAHs analyses, all collected samples (including the filter, sorbent tube, and exposure pad) were placed in a solvent solution (the mixture of *n*-hexane and dichloromethane, v:v = 1:1, respectively), and extracted in a Soxhlet extractor for 24 hrs. The extract was then 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; 100 °C to 290 °C at 3 °C /min; 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 (Jongeneelen et al., 1988; Tsai et al., 2001a; Tsai et al., 2001b; Tsai et al., 2002; Tsai et al., 2004; 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 0.786 and 0.935, and averaged 0.865 in this study. The above values were used to adjust the observed concentration. The mean relative standard deviation (RSD) (%) of recovery efficiencies was 5.13% (range 1.28-8.89%). The recovery efficiencies of five internal standards were between 0.791 and 0.986 and were fairly consistent. The blank tests for PAHs were accomplished by the same procedure as the recovery-efficiency tests without adding the known standard solution before extraction. Analysis of field blank, including filters and PUF/resin cartridges, showed no significant contaminant.

2.3 Data analysis

2.3.1 Calculating PAH homologue distributions

In this study, the concentration of total PAHs was defined as the sum of the concentrations of the selected 22 PAH compounds. 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 PAH exposure 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 (Chu et al., 1984; Thorslund et al., 1991; Nisbet et al., 1992). From these, the list of TEFs completed by Nisbet and LaGoy (Chu et al., 1984) 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 inhalable and dermal PAH exposure concentrations

In this study, the total inhalatory PAH exposure was defined as the summation of the gas phase and particle phase PAH exposures. To estimate a worker's total dermal exposure level, the unit dermal surface area PAH exposure level for a given PAH species *i* on the body surface area *j* (UDC_{ij}; ng/100cm^2/day) was directly measured from this 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/100cm^2/day) can be determined by using equation 1:

$$UDE_{total,j} = \sum_{i=1}^{22} UDC_{ij} \qquad (1)$$

Dermal PAHs exposure levels for total PAHs at the body surface area *j* ($DE_{total,j}$; ng/day) can be determined by equation 2:

$$DE_{total,j} = \sum_{i=1}^{22} UDC_{ij} AD_j \qquad (2)$$

Where,

AD_j=the corresponding body surface area for the body surface area *j* (unit: 100cm^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) was determined by using equation 3:

$$DE_{i,whole} = \sum_{j=1}^{11} UDC_{ij} AD_j \quad (3)$$

Total dermal PAHs exposure levels on the whole body surface area ($DE_{total,whole}$; ng/day) was determined by using equation 4:

$$DE_{total,whole} = \sum_{i=1}^{22} \sum_{j=1}^{11} UDC_{ij} AD_j \quad (4)$$

2.3.4 Estimating personal inhalable and dermal PAH exposure profiles

In this study, 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-sided 95% confidence intervals ($LCL_{1,95\%}$ and $UCL_{1,95\%}$, respectively). Full calculating procedures were described in the study conducted by Attfield and Hewet (1992). The above method has also been recommended by the American Industrial Hygiene Association (AIHA) Exposure Assessment Strategies Committee for exposure data with various sample sizes and geometric standard deviations (GSDs) (Mulhausen and Damiano, 1998).

2.4 Heath-risk assessment

Regarding the lung cancer risk via the inhalation route, WHO suggested the unit risk of 8.7×10^{-2} $(\mu g/m^3)^{-1}$ for the lifetime (70 years) PAHs exposure, assuming one exposed to one unit BaP concentration (i.e., 1 $\mu g/m^3$) (WHO, 1987). It worth noting above unit risk was proposed to estimate the lung cancer risk caused by the lifetime exposure, therefore, it has been adopted by a recent study for assessing the lung cancer risks of general adults exposure to the ambient atmospheric PAHs (Zmirou et al., 2000). For occupational exposure, Pott (1985) established a relationship between BaP exposure and lung cancer risk based on a data bank provided by an epidemiological study conducted by Redmond et al (1976). He suggested a unit risk of 7.0×10^{-2} $(\mu g/m^3)^{-1}$ for a 25 years occupational PAHs exposure with an averaged BaP concentration of 1 $\mu g/m^3$. By using the same data bank, the US 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) (US 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.

To estimate the skin cancer risk posed by 25 years occupational dermal PAHs exposure, a unit risk 37.47(mg/kg bodyweight/day)$^{-1}$ 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 bodyweight/day (Hussain 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).

3. Results and Discussion

3.1 Personal inhalatory PAH exposures

In this study, we found that the mean individual and total PAH exposure concentrations of gas phase, particle phase and gas+particle phase for all selected threading workers were log-normally distributed ($p<0.001$, W-test). The above result suggests that the selected threading workers can be regarded as a similar exposure group (Mulhausen and Damiano, 1998). Table 3 shows the estimated mean and its corresponding $LCL_{1,95\%}$ and $UCL_{1,95\%}$ PAH exposure concentrations of gas phase, particle phase and gas+particle phase for threading workers. We found that the mean gas phase total PAH exposure level (8.60×10^4 ng/m^3) was much higher than that of particle phase (2.30×10^3 ng/m^3). By examining PAH homologue distributions for both gas-phase and particle-phase PAHs, it can be seen that the fractions of LMW-, MMW- and HMW-PAHs for the former were 97.2%, 2.5%, and 0.3%, and for the latter were 66.8%, 18.3%, and 14.9%, respectively. Gas-phase PAHs had a higher fraction in LMW-PAHs was because of their intrinsic

high volatility nature. To the contrary, particle-phase PAHs had a higher fraction in both MMW-PAHs and HMW-PAHs was simply because of their low volatility nature.

Table 3 also shows that gas and particle phase total PAH exposures account for 97.4% and 2.6% of workers' inhalatory (i.e., gas + particle phase) PAH exposures, respectively. But for total BaP_{eq} exposure levels, the gas phase (1.08×10^2 ng/m^3) was lower than that of particle phase (1.18×10^2 ng/m^3) accounting for 46.1% and 53.9% of inhalatory exposures (2.34×10^2 ng/m^3), respectively. The above results were because gas phase PAHs were dominated by PAHs with lower TEFs (i.e., LMW-PAHs; see Table 2), but particle-phase PAHs were dominated by PAHs with higher TEFs (i.e., MMW-PAHs and HMW-PAHs; see Table 2).

The present study also shows that workers' mean inhalatory exposure level (=8.83×10^4 ng/m^3) was lower than the time-weighted average permissible exposure level (PEL-TWA) regulated in Taiwan for PAHs (=1.00×10^5 ng/m^3). However, it also can be seen that the corresponding $UCL_{1,95\%}$ (1.02×10^5 ng/m^3) was slightly higher than the above PEL-TWA indicating that PAH exposures of threading workers might not be negligible. In our previous study, we found that the mean oil mist exposure concentration of threading workers (=2.11mg/m^3) was much lower than the current PEL-TWA for oil mists (=5.0 mg/m^3) (Chen et al., 2007). Based on the results obtained from the present study, it clearly indicates the inadequacy in assessing workers' inhalatory exposures if only oil mist concentrations were measured.

3.2 Personal dermal PAH exposures

Table 4 shows both the total PAHs and total BaP_{eq} dermal exposure levels for the whole body surface area ($DE_{total,whole}$) and each individual body surface ($UDE_{total,j}$). Results show that the mean $DE_{total,whole}$ in total PAHs and total BaP_{eq} were 5.44×10^6 and 3.71×10^5 ng/day, respectively. Among the estimated mean $UDE_{total,j}$ (range= 1.34×10^4–7.38×10^4 ng/100cm^2/day), the top five exposed surface areas were lower arm, hand, upper arm, neck, and head/front with exposure levels of 7.38×10^4, 5.41×10^4, 3.24×10^4, 2.48×10^4, and 2.33×10^4 ng/100cm^2, respectively. Clearly, the above high exposure levels were mainly because their surface areas were uncovered

(or partly uncovered) by clothes, and they are closer to the emission source than other body surface areas. Here, it should be noted that the exposure level of hand was lower than that of upper arm was because the latter was covered by cotton gloves. On the other hand, we found that those six surface areas covered by clothes (i.e., head/back, chest/abdomen, back, upper leg, lower leg, and foot) had lower exposure levels (range= 1.34×10^{4}–2.29×10^{4} ng/100cm^2) than the above five surface areas. The same trend can also be seen in dermal total-BaP_{eq} exposure levels for each individual body surface (Table 4).

In this study we also found that the fractions of LMW-, MMW-, and HMW-PAHs in $DE_{total,whole}$ (=70.3%, 21.4%, and 8.3%, respectively; data not shown in Table 4) was quite similar to that of inhalatory particle phase PAH exposures (=68.8%, 18.3%, and 14.9%, respectively; Table 3), but was very different from that of inhalatory gas phase (=97.2%, 2.5%, and 0.3%, respectively; Table 3) and gas +particle phase PAHs (96.4%, 2.9%, 0.7%, respectively; Table 3). The above results suggest airborne particle phase PAHs was the main contributor for dermal PAH exposures. Considering those body surface areas uncovered (or partly uncovered) by clothes had higher exposure levels, it implies that the impaction of PAHs-containing oil mists on the uncovered body surface areas might play the important role on workers' dermal exposures.

3.3 Health-risk assessments for both inhalatory and dermal PAH exposures

In this study, workers' total BaP_{eq} levels were used to estimate the mean, and its corresponding $LCL_{1,95\%}$ and $UCL_{1,95\%}$ lifetime lung cancer risks for the studied exposure group. As shown in Table 5, the estimated mean lifetime lung cancer risk (1.64×10^{-2}) was contributed more by particle phase PAH exposures (8.81×10^{-3}) than by gas phase PAH exposures (7.55×10^{-3}). However, it should be noted that in this study particle phase PAHs was vigorously extracted by using a solvent solution (i.e., a mixture of n-hexane and dichloromethane), which could be very different from that could be extracted in the respiratory system. Particularly, it is expected that the health risk associated with particle phase PAH exposures could be overestimated. At this stage, the elution rate of particle-phase PAHs deposited in lung still remains unknown which warrants

the need for further investigation in the future.

Table 5 also shows the estimated mean (and its corresponding $LCL_{1,95\%}$ and $UCL_{1,95\%}$) lifetime skin cancer risk for the selected threading workers. To date, the desorption rate of PAHs for oil mists deposited on the skin surface still remains unknown. However, US EPA (1992) has suggested a maximum of 20% of PAHs that adsorbed onto soil could be desorbed onto the skin in 24 hours. The above value was adopted in this study. Assuming the body weight of the standard man (=70 kg) is representative to all threading workers and all of them will be exposed to oil mists for 25 years (5 days per week, 50 weeks per year) during an average life span of 70 years. The resultant mean and its corresponding $LCL_{1,95\%}$ and $UCL_{1,95\%}$ lifetime dermal total BaP_{eq} exposure levels for the threading workers were 2.59×10^{-1}, 1.77×10^{-1}, and 4.48×10^{-1} μg/kg/day, respectively. The unit risk (37.47×10^{-3} (μg/kg /day)$^{-1}$) suggested by Hussain et al. (1998) was adopted in this study to estimate the lifetime skin cancer risk. The estimated mean and its corresponding $LCL_{1,95\%}$ and $UCL_{1,95\%}$ lifetime skin cancer risks were 9.72×10^{-3}, 6.64×10^{-3}, and 1.68×10^{-2}, respectively. Although the resultant mean lifetime skin cancer risks (9.72×10^{-3}) was slightly lower than that of the mean lifetime lung cancer risk (1.64×10^{-2}), both are higher than the significant risk level (10^{-3}) defined by the US Supreme Court in 1980. Therefore, it is recommended that measurements should be taken in order to reduce both inhalatory and dermal exposures.

Considering PAHs contained oil mists contributed to not only inhalatory but also dermal exposures, the installation of local exhaust ventilation systems at the emission source (i.e., the threading machine) should be considered as the first priority measurement because both lung and skin cancer risks can be reduced simultaneously. On the other hand, if the personal protection equipment will be adopted for exposure abatement purpose, both respiratory protection equipment and protective clothing should be used simultaneously from the health risk management aspect.

4. Conclusions

We found that the inhalatory gas-phase total PAHs exposure level was much higher than that

of particle-phase. By combining both gas- and particle-phase total PAHs, workers' mean inhalatory exposure level was lower, but its corresponding $UCL_{1,95\%}$ was slightly higher than the PEL-TWA for PAHs. On the other hand, in our previous study we found that workers' mean oil mist exposure concentration was much lower than the current PEL-TWA for oil mist. The present study clearly indicates the inadequacy in assessing workers' inhalatory exposures if only oil mist concentrations were measured. For workers' dermal exposures, the top five exposed surface areas were lower arm, hand, upper arm, neck, and head/front mainly because their surface areas were uncovered (or partly uncovered) by clothes. The estimated lifetime lung cancer risk caused by inhalatory PAH exposures was higher than that of skin cancer risks caused by dermal exposures. However, both cancer risks are higher than the significant risk level defined by the US Supreme Court in 1980. Therefore, it is recommended that measurements should be taken in order to reduce both inhalatory and dermal exposures. The installation of local exhaust ventilation systems at the emission source should be considered as the first priority measurement because both lung and skin cancer risks can be reduced simultaneously. If the personal protection equipment would be adopted in the future, both respiratory protection equipment and protective clothing should be used simultaneously from the health risk management aspect.

Acknowledgment

The authors wish to thank National Science Council in Taiwan for funding this research project.

References

Attfield MD, Hewett P. Exact expressions for the bias and variance of estimators of the mean of a lognormal distribution. Am Ind Hyg Asso J 1992;53:423–435.

Bingham E, Horton AW. Environmental carcinogenesis experimental observations related to occupational cancer. Adv Biol Skin 1969;7:183–193.

Chen YC, Lee WJ, Uang SN, Lee SH, Tsai PJ. Characteristics of Polycyclic Aromatic Hydrocarbon (PAH) Emissions from a UH-1H Helicopter Engine and Its Impact on the

Ambient Environment. Atm Environ 2006;40:7589–7597.

Chen MR, Tsai PJ, Chang CC, Shih TS, Lee WJ, Liao PC. Particle size distributions of Oil Mists in Workplace Atmospheres and Their Exposure Concentrations to Workers in a Fastener Manufacturing Industry. J Haz Mater 2007;146:393–398.

Chu M, Chen C. Evaluation and estimation of potential carcinogenic risks of polynuclear aromatic hydrocarbons. Paper presented at the symposium on polycyclic aromatic hydrocarbons in the workplace. Pacific Rim Risk Conference, Honolulu; 1984.

Cullen MR, Balmes JR, Robins JM, Smith G.JW. Lipoid pneumonia caused by oil mist exposure from a steel rolling tandem mill. Am J Ind Med 1981;2:51–58.

Elovaara E, Heikkila P, Pyy L, Mutanen P, Riihimaki V. Significance of dermal and respiratory uptake in creosote workers: exposure to polycyclic aromatic hydrocarbons and nrinary excretion of 1-hydroxylpyrene. Occup Environ Med 1995;52:196–203.

Gilbert RO. Statistical Methods for Environmental Pollution Monitoring. New York: Van Nostrand Reinhold, 1987.

Granella M, Clonfero E. The mutagenic activity and polycyclic aromatic hydrocarbon compound content of mineral oils. Int Arch Occup Environ Health 1991;63:149–153.

Hussain M, Rae J, Gilman A, Kauss P. Lifetime health-risk assessment from exposure of recreational users to polycyclic aromatic hydrocarbons. Arc Environ Contam Tox 1998;35:527–531.

Jarvholm B, Bake B, Lavenius B, Thiringer G., Vokmann R. Respiratory symptoms and lung function in oil mist-exposed workers. J Occup Med 1982;24:473–479.

Jongeneelen FJ, Scheepers PJF, Groenendijk A, Van Aerts LA, Anzion RBM, Bos RP, Veestra SJ. Airborne concentrations, skin contamination, and urinary metabolite excretion of polycyclic aromativ hydrocarbons among paving workers exposed to coal tar derived road tars. Am Ind Hyg Asso J 1988;49:600–607.

Kazerouni N, Thomas TL, Petralia SA, Hayes RB. Mortality among workers exposed to cutting

oil mist: update of previous reports. Am J Ind Med 2000;38:410–416.

Kennedy SM, Chan YM, Teschke K, Karlen B. Change in airway responsiveness among apprentices exposed to metalworking fluids. Am J Respir Crit Care Med 1999;159:87–93.

Krewski D, Gaylor D, Szyzkowicz M. A model-free approach to low-dose extrapolation. Environ Health Perspect 1991;90:279–285.

Lee WJ, Chao WH, Shih ML, Tsai CH, Chen TCH, Tsai PJ. Emissions of Polycyclic Aromatic Hydrocarbons from Batch Hot Mix Asphalt Plants. Environ Sci Technol 2004;38: 5274–5280.

Li CT, Lin YC, Lee WJ, Tsai PJ. Emissions of Polycyclic Aromatic Hydrocarbons and Their Carcinogenic Potencies from Cooking Sources to the Atmosphere of the Urban Area. Environ Health Perspect 2003;111:483–487.

Lin YC, Lee WJ, Li HW, Chen CB, Fang GC, Tsai PJ. Impact of Using Fishing Boat Fuel with High Poly-Aromatic Content on the Emission of Polycyclic Aromatic Hydrocarbons from the Diesel Engine. Atm Environ 2006;40:1601–1609.

London MA, Boiano JM, Lee SA. Exposure assessment 3.3'-dicholor0benzidine (DCB) at two chemical plants. App Ind Hyg 1989;4:101–104.

Massin N, Bohadana AB, Wild P, Goutet P, Kirstetter H, Toamain JP. Airway responsiveness, respiratory symptoms, and exposures to soluble oil mist in mechanical workers. Occup Environ Med 1996;53:748–752.

Michalek DJ, Hii WWS, Sun JS, Gunter KL. Experimental and analytical efforts to characterize cutting fluid mist formation and behavior in machining. Appl Occup Envir Hyg 2003;18:842–854.

Moulin JJ, Wild P, Fourmier-Betz B, Mur JM, Smagghe G.. Mortality from lung cancer and cardiovascular disease among stainless steel-steel producing workers. Cancer Causes Control 1993;4:75–81.

Mulhausen JR, Damiano J. A Stategy for Assessing and Managing Occupational Exposures, Fairfax: American Industrial Hygiene Association (AIHA), 1998.

Nisbet C, LaGoy P. Toxic equivalency factors (TEFs) for polycyclic aromatic hydrocarbons (PAHs). Reg Tox Pharmocol 1992;16:290–300.

Petry T, Schmid P, Schlatter C. The use of toxic equivalency factors in assessing occupational and environmental health risk associated with exposure to airborne mixtures of polycyclic aromatic hydrocarbons (PAHs). Chemosphere 1996;32:639–648.

Pott F. Pyrolyseagase, PAH und lungenkrebrisiko-daten und bewertung. Staub-Reinhaltung dedr Luft 1985;45:368–379.

Rappaport SM. Assessment of long-term exposures to toxic substances in air. Ann Occup Hyg 1991;35:61–121.

Redmond C, Strobino B, Cypess R. Cancer experience among coke by-product workers. Ann NY Acad Sci 1976;217:102–115.

Robertson AA, Weir DC, Burge PS. Occupational asthma due to oil mists. Thorax 1988;43: 200–205.

Roy TA, Johnson SW, Blackburn GR, Merkerer CR. Correlation of mutagenic and dermal carcinogenic activities of mineral oils with polycyclic aromatic hydrocarbon compound content. Appl Toxicol 1988;10:466–476.

Russi M, Dubrow R, Flannery JT, Cullen MR, Mayne ST. Occupational exposure to machining fluids and laryngeal cancer risk: contrasting results using two separate control groups. Am J Ind Med 1997;31:166–171.

Thornburg D. Leith, Mist generation during metal machining. J Trib 2000;122:544–549.

Thorslund T, Farrer D. Development of relative potency estimated for PAHs and hydrocarbon combustion product fractions compared to benzo(a)pyrene and their use in carcinogenic risk assessments. US Environmental Protection Administration (EPA), Washington DC; 1991.

Tsai PJ, Hsieh HY, Lee WJ, Lai SO. Health-risk assessment for workers exposed to polycyclic aromatic hydrocarbons in a carbon black manufacturing industry. Sci Total Environ 2001;278:137–150.

Tsai PJ, Shieh HY, Hsieh LT, Lee WJ. The Fate of PAHs in the Carbon Black Manufacturing Process. Atmospheric Environment 2001;35:3495–3501.

Tsai PJ, Shih TS, Chen SL, Lee WJ, Lai GH, Liou SH. Assessing the Contents of Polycyclic Aromatic Hydrocarbons in the Tollbooths of a Highway Toll Station via Direct and Indirect Approaches. Environ Sci Technol 2002;36:4748–4753.

Tsai PJ, Shih TS, Chen SL, Lee WJ, Lai GH, Liou SH. Assessing and Predicting the Exposures of Polycyclic Aromatic Hydrocarbons (PAHs) and Their Carcinogenic Potencies from Vehicle Engine Exhausts to Highway Toll Station Workers. Atm Environ 2004;38:333–343.

US EPA. Health effects assessments for polycyclic aromatic hydrocarbons (PAHs). US Environmental Protection Agency (EPA), Environmental Criteria and Assessment Office, EPA 549/1-86-013, Cincinnati; 1984.

US EPA. Pesticide assessment guideline, subdivision U, applicator exposure monitoring. US Environmental Protection Administration (EPA), U.S. Department of Commerce, National Technical Information Service, Springfield; 1987.

US EPA. Dermal exposure assessment: principles and applications. US Environmental Protection Administration (EPA), EPA/600/8-91/011B, Washington DC; 1992.

Verma DK, Julian JA, Roberts RS, Muir DC, Jadon N, Shaw DS. Polycyclic aromatic hydrocarbons (PAHs): a possible cause cause of lung cancer mortality among nickel/copper smelter and refinery workers. Am Ind Hyg Assoc J 1992;53:317–324.

WHO. World Health Organization field surveys of exposure to pesticide protocol. Tox Letter 1986;33:223–235.

WHO. Air quality guideline for Europe. WHO European Series, WHO Regional Bureau, Copenhagen; 1987.

Zmirou D, Masclet P, Boudet C, Dor F, Dëchenaux J. Personal exposure to atmospheric polycyclic aromatic hydrocarbons in a general adult popolation and lung cancer risk assessment. J Occup Environ Health 2000;42:121-125.

Table Captions

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

Table 2 PAHs and their toxic equivalent factors (TEFs) suggested by Nisbet and LaGoy in 1992

Table 3 Exposure concentrations of 22 selected PAH compounds, LMW-PAHs, MMW-PAHs, HMW-PAHs, total PAHs and total BaPeq for threading workers

Table 4 Unit total PAHs and total BaP_{eq} exposure concentrations for the 11 selected body surface areas ($UDE_{total,j}$; ng/100cm^2/day) and total PAHs and total BaP_{eq} exposure concentrations for the whole body surface area ($DE_{total,whole}$; ng/day)

Table 5 Estimated cancer risk associated with both inhalatory and dermal PAH exposures for threading workers