EnvironmentAsia
The international journal published by the Thai Society of Higher Education Institutes on Environment

Available online at www.tshe.org/EA
EnvironmentAsia 7(2) (2014) 142-151

Health Risk Assessment and DNA Damage of Volatile Organic Compounds in Car Painting Houses

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Abstract

Car painters who work near volatile organic compounds (VOCs) sources, including paints, solvents and painting processes may be exposed to highly elevated VOCs levels. This study investigates air samples from car painting houses in Thailand to evaluate the health risks following inhalation exposure. Personal air samplings were obtained at nine garages in Phitsanulok, Thailand from June to September 2012. The concentrations of benzene, toluene, ethylbenzene, xylenes, and styrene in the air workplaces were significantly higher than in a control group of office workers ($p<0.05$). Toluene, xylene and ethylbenzene were the most abundant species. However, all VOCs in these air samples were lower than TWA limit of Thailand and the OSHA standard. The lifetime cancer and non-cancer risks for the workers exposed to VOCs were also assessed. The average lifetime cancer risk was 41.0 (38.2-47.2) per million, which is in the acceptable risk. The average lifetime non-cancer risk, the HI, was 0.962 (0.643-1.397), which is well below the reference hazard level. Urine samples, collected after 8-h work periods which were analyzed for VOCs metabolites, including t,t muconic acid, hippuric acid, mandelic acid and m-hippuric acid, demonstrate that the average levels of metabolites in car painters and in controls were close. All VOCs metabolites in urine samples were lower than BEI of ACGIH standard. Blood samples, collected after 8-h work periods which were analyzed by single cell gel electrophoresis (comet) assay. The DNA damage, assessed by tail moment, demonstrates that the average of tail moment in car painters were significantly higher than in the controls ($p<0.05$).

Keywords: *volatile organic compounds; car painting houses; TWA; health risk assessment; DNA damage; tail moment*

1. Introduction

Volatile organic compounds (VOCs) emissions from paints, solvents and painting processes are important sources for VOCs emissions (Malherbe and Mandin, 2007; Na *et al.,* 2004; Vega *et al.,* 2000; Yuan *et al.,* 2010). The IARC reported that "there is sufficient evidence of carcinogenicity by occupational exposure as painters" and classified painting as an occupation that increases certain cancer risks (IARC, 1989; Lynge *et al.,* 1997) The production of oil-paints and the use of paint diluents or thinners are the main sources of organic solvents exposure; 50% of these synthesized organic solvents are employed by car painters and therefore represent an occupational health problem. Thinners are complex commercial organic solvents mixtures that contain: benzene, toluene, xylene, hexane, some alcohols and more than 50 different organic compounds with masses <1%. (Bogadi-Sare *et al.,* 1997)

Many studies have been conducted on occupational exposure to VOCs from painting emissions. (Hoyos-Giraldoa *et al.,* 2009; Malherbe and Mandin,2007; Srivastava, 2000; Uang *et al.,* 2006; Yuan *et al.,*2010). These studies reported that the workers were exposed to highly elevated VOCs levels. Workers can be exposed to contaminants by inhalation, ingestion, and dermal contact. Most of the toxicants assessed are VOCs that remain as gases when emitted into the air. Significant exposure to these volatile organic toxicants emitted into the air only occurs through the inhalation pathway (Cal/EPA, 2003), and has therefore gained the attention of researchers. Currently, benzene is classified as a human carcinogen, and 1-3 butadiene, chloroform, trichloroethylene and 1-4 dioxane are classified as possible human carcinogens (USEPA, 1998).

2. Materials and Methods

2.1. Sampling and analysis

This study was conducted in Phitsanulok province, Thailand from June to September 2012. The car painters included 45 males, all of whom had volunteered to take part in the study. They were 18-55 years old, and they spent about 8 hours per day outdoors at garages. Personal air samples, urine and blood were collected at nine garages, and in each garage five car painters were included. A total of 45 samples were thus collected. Five male office workers volunteered as controls for this study; these persons were not in direct contact with VOCs. Each worker was instructed to collect a sample set consisting of one personal air sample within the breathing zone, one spot urine sample and one blood sample after the work shift. In addition, each of them was asked to fill out a questionnaire containing information, such as age, work-shift, and work duration.

2.1.1. Analysis of air samples

Air samples were collected by diffusion into a tube type diffusive sampler, made of stainless steel (Markes, Markes International Ltd., United Kingdom) packed with carbo pack B 60/80 mesh. The tube was attached to the clothes within 30 cm from the nose (breathing zone). The analyses were carried out essentially according to the Instruction Manual TO-17 (USEPA, 1999). The air samples were analyzed by GC-MS (QP5000, Shimudsu, Japan). Fig. 1 shows the chromatograms of an air sample containing VOCs. The quantitative analysis of the VOCs was performed using the calibration curves at six concentrations. The quality assurance/quality control (QA/ QC) program included laboratory and field blank samples. An external standard was analyzed daily.

2.1.2. Analysis of urine samples

Ten ml of each urine sample was collected in a plastic container, sealed and stored at -20°C until analysis. Under these conditions, samples can be stored for 2 months. The urine samples were analyzed by HPLC–UV(HP1260, Hewlett Packard, Germany) in order to determine concentrations of t,t muconic acid (at 254 nm), hippuric acid, mandelic acid and m-hippuric acid (at 210 nm). For this purpose we followed the methods by Boogard *et al.* (1996) and NIOSH, 2003 with a slight modification. All measured values were divided by the concentration of urinary creatinine, as analyzed by an analyzer for clinical chemistry, model Stardust MC15 (Diasys diagnostic system, Germany).

2.1.3. Analysis of blood samples

Five ml. of each whole blood samples was collected in a heparin vaccutainer tube and transported to the laboratory and processed for analysis immediately. The blood samples were analyzed DNA damage by comet assay in order to determine tail moment. For this purpose we followed the methods by Singh *et al.* (1988) with a slight modification. A total of 50 cells randomly chosen from each of the duplicated

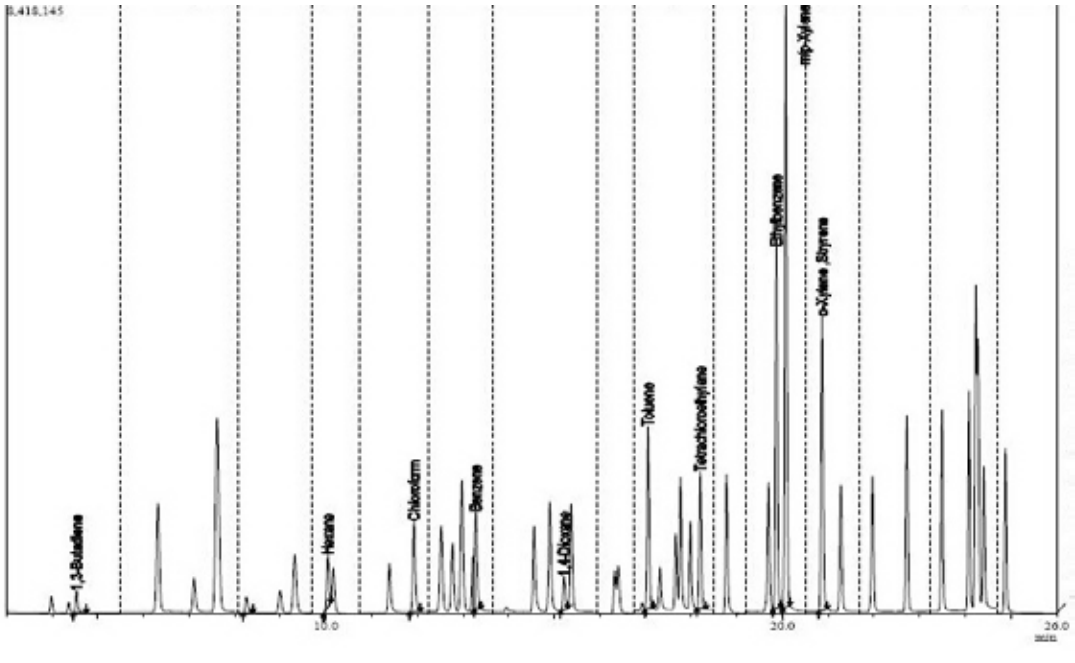


Figure 1. The chromatograms of the VOCs from GC-MS

slides were examined by fluorescence microscopy (Olympus BX61, USA). The DNA damage or DNA stand breaks was measured quantitatively using the comet software and expressed as tail moment.

2.2. Health risk assessment

The health risk assessment focused on chronic exposure to compounds that may cause cancer or other toxic effects, rather than on acute toxicity. The main exposure route of interest was inhalation. The inhalation intake was calculated by averaging daily intake over the exposure period. The carcinogenic and noncarcinogenic intakes of VOCs for car painters were calculated as:

$$I = (C \times ET \times EF \times ED) / AT$$

where I is the inhalation intake ($\mu g/m^3$), C is the concentration of the compound in the personal air sample ($\mu g/m^3$), ET is the exposure time (hr/day), EF is the exposure frequency (days/year), ED is the exposure duration (years), and AT is an average lifetime (years). Inhalation exposure is always related to exposure frequency, duration, and quantity (dose) and activity pattern. To simplify the exposure and risk assessment, several assumptions regarding individual exposures were made based on the questionnaire data and professional judgment. Exposure to VOCs was based on the average 8 hrs (full shift) time weighted average (TWA) concentration, the summation throughout the workday of the product of the concentrations, and the time periods for the concentration encountered in each time interval, and was averaged over an 8 hrs standard workday. The inhaled compounds were assumed to be totally absorbed for risk calculations in some studies (Hoddinott and Lee, 2000; Muller *et al.,* 2003). Table 1 summarizes the exposure and risk assessment factors.

Risk characterization requires combining the estimated exposure concentrations with toxicity data to provide a quantitative estimate of the potential health impacts. In this assessment, risk estimates for VOCs with a cancer endpoint were expressed in terms of the probability of developing cancer from a lifetime of continuous exposure to VOCs. The lifetime cancer risk was estimated using the equation

$$\text{cancer risk} = I\ (\mu g/m^3) \times \text{cancer unit risk factors}\ (\mu g/m^3)$$

The non-cancer risk is expressed in terms of the hazard quotient (HQ), which is the estimated ground level concentration divided by the reference exposure level (REL) for a single substance and a particular endpoint. The REL is an exposure level at, or below which, no non-cancer adverse health effect is anticipated to occur in a human population exposed for a specific duration (Cal/EPA, 2005). The non-cancer health impacts were expressed as the hazard index (HI), which is determined by calculating the HQ for a compounds and summing all of the HQ at a specific location.

$$HQ = I\ (\mu g/m^3) / RELs\ (\mu g/m^3)$$

$$HI = HQ_1 + HQ_2 + HQ_3 + ... + HQ_n$$

For a given airborne toxic compound, exposures below the reference level ($HI \leq 1$) are unlikely to be associated with adverse health effects (Cal/EPA, 2003). The potential for adverse effects increases as exposures further exceed the reference dose. Table 2 summarizes the toxicity values for compounds of concern.

3. Results

3.1. Characterization and exposure assessment

The measurements of VOCs in the personal air sampling of car painters, and in the controls (office workers) are summarized in Table 3 and Fig. 2. The average concentrations of VOCs found were in decreasing order: toluene, xylene, ethylbenzene, hexane, benzene,

Table 1. The exposure and risk assessment factors

Exposure settings	Value	Source of exposure settings
Exposure time	8 h /day	Questionnaires
Exposure frequency	300 day /year	Questionnaires
Exposure duration: carcinogenic	11 year	Questionnaires
Exposure duration: noncarcinogenic	11 year	Questionnaires
Average life time: carcinogenic	70 year	Cal/EPA, 2005
Average life time: noncarcinogenic	70 year	Cal/EPA, 2005

Reference: Cal/EPA, 2005

Table 2. The toxicity values for compounds of concern

Compound	RELs ($\mu g/m^3$)	Cancer Unit Risk Factors $(\mu g/m^3)^{-1}$	USEPA Class
benzene	6.0E+01	2.9E-05	A
toluene	3.0E+02		D
ethylbenzene	2.0E+03		D
xylene	7.0E+02		D
styrene	9.0E+02		-
1,3-butadiene	2.0E+01	1.7E-04	B2
hexane	7.0E+03		D
chloroform	3.0E+02	5.3E-06	B2
1,4-dioxane	3.0E+03	7.7E-06	B2

RELs=Chronic Inhalation Reference Expose Levels
Reference: Cal/EPA, 2005

styrene, 1,3-butadiene, chloroform, and 1,4-dioxane. At all garages, the carpainters' air samples displayed significantly ($p<0.05$) higher concentrations of benzene, toluene, ethylbenzene, xylene, and styrene than in the air samples from the controls.

3.2. Health risk assessment

Health risk for car painters was assessed using personal air sampling data. In this study, benzene, 1,3-butadiene, chloroform, and

Table 3. The volatile organic compounds (VOCs) concentrations (μg/m3) in the personal air samples from car painters (n=5/ garage), and from the controls in 8 hrs worktime.

garage	benzene		toluene		ethylbenzene		xylene		styrene	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	6.4*	0.4	5803.1*	669.1	1127.6*	299.1	3006.5*	205.5	3.4*	1.1
2	4.0*	0.4	5384.1*	2808.7	560.6*	138.2	1485.5*	277.2	4.9*	0.9
3	6.3*	0.9	8210.2*	1712.2	1057.1*	396.1	2052.8*	365.6	7.6*	0.5
4	6.6*	2.0	8454.1*	1661.7	1224.7*	268.3	2314.9*	807.5	5.5*	1.2
5	4.0*	1.2	4367.1*	1160.4	533.6*	80.4	1480.9*	290.7	2.2*	0.7
6	7.5*	1.9	4691.5*	994.5	623.7*	155.3	2063.4*	367.3	7.1*	0.6
7	6.8*	1.2	4521.3*	857.7	1171.7*	310.3	3155.6*	1974.5	6.6*	1.0
8	6.1*	0.8	5189.0*	1842.8	575.3*	94.4	1738.0*	530.4	2.2*	0.8
9	8.4*	0.9	3683.2*	326.0	594.1*	47.3	1373.7*	72.4	1.9*	0.5
Mean 9 garages	6.2	1.8	5589.3	2102.0	829.8	356.2	2074.6	932.2	4.6	2.3
Min	2.6		3295.5		449.2		1251.0		1.2	
Max	9.5		9979.7		1515.2		5834.0		8.1	
controls	0.8*	0.6	11.1*	2.5	0.0*	0.0	1.4*	1.9	0.0*	0.0
Min (controls)	0.1		7.8		0.0		0.0		0.0	
Max (controls)	1.7		13.9		0.0		3.7		0.0	

*significantly different ($p<0.05$)

Table 3. (Continued) The volatile organic compounds (VOCs) concentrations (µg/m3) and SD in the personal air sampling of car painters (n=5 / garage), and controls in 8 hrs worktime.

garage	1,3-butadiene		hexane		Chloroform		1,4-dioxane	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	4.3	0.2	17.9*	5.0	0.0	0.0	0.0	0.0
2	4.7	0.2	16.5*	8.9	0.0	0.0	0.0	0.0
3	4.3	0.5	7.1	1.3	0.0	0.0	0.0	0.0
4	4.6	0.5	6.8	1.8	0.0	0.0	0.0	0.0
5	4.5	0.7	7.4	1.8	0.0	0.0	0.0	0.0
6	4.6	0.9	21.1*	4.2	0.0	0.0	0.0	0.0
7	4.3	0.5	13.6*	4.1	0.0	0.0	0.0	0.0
8	4.5	0.5	18.6*	5.9	0.0	0.0	0.0	0.0
9	5.0	0.6	13.6*	7.0	0.0	0.0	0.0	0.0
Mean 9 garages	4.5	0.6	13.6	6.9	0.0	0.0	0.0	0.0
Min	3.8		5.1		0.0		0.0	
Max	5.8		27.0		0.0		0.0	
controls	4.3	0.5	3.6*	1.1	0.0	0.0	0.0	0.0
Min (controls)	3.7		2.5		0.0		0.0	
Max (controls)	4.9		5.0		0.0		0.0	

*significantly different ($p<0.05$)

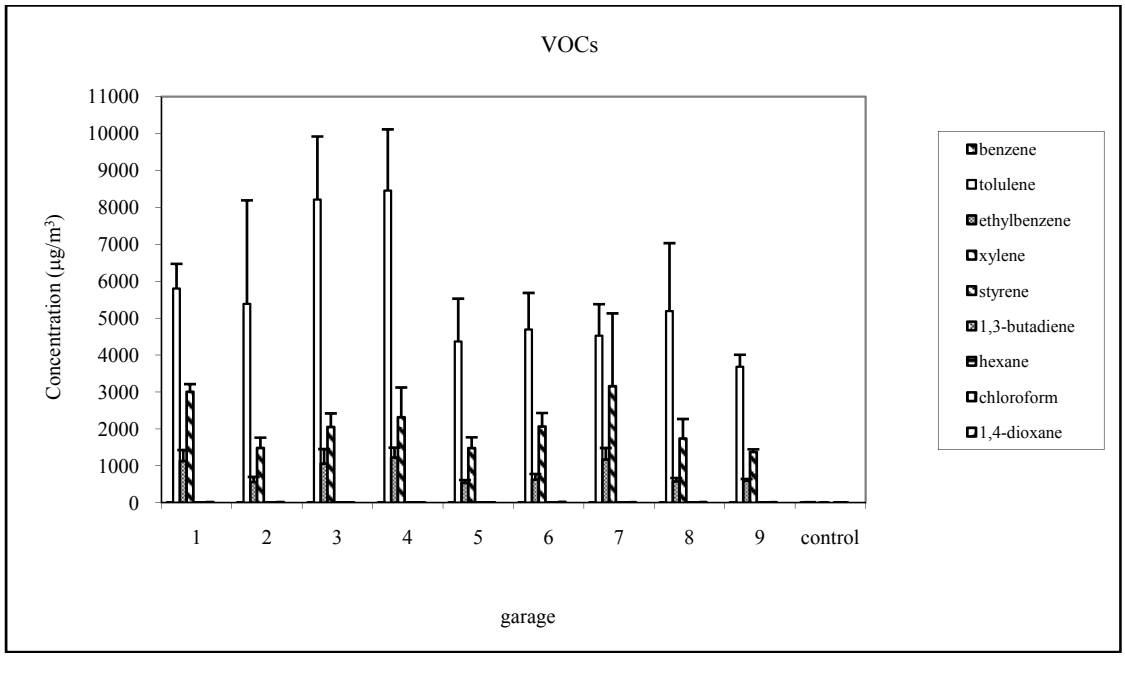


Figure 2. The volatile organic compounds (VOCs) concentrations (µg/m^3) in the personal air sampling of car painters, and controls in 8 hrs worktime.

Table 4. The average lifetime cancer risk assessment of car painters (per million)

garage	1	2	3	4	5	6	7	8	9	mean	control
Cancer Risk											
benzene	8.0	5.0	7.8	8.2	5.0	9.4	8.5	7.6	10.5	7.8	1.0
1,3-butadiene	31.7	34.6	31.5	33.5	33.3	33.8	31.6	32.8	36.6	33.3	31.1
chloroform	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1,4-dioxane	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total	39.6	39.6	39.3	41.7	38.2	43.1	40.1	40.4	47.2	41.0	32.2

1,4-dioxane were assessed as human carcinogens (USEPA, 1998). The average lifetime cancer risk was 41 per million as shown in Table 4. Risks less than 1 in a million are typically considered to be well below a level of concern, and risks above 100 per million are typically considered sufficient for action or intervention to take place. In the US, EPA final national contingency plan for Superfund site remediation, the EPA codified a range of acceptable risks (i.e., 1×10^{-4} -1×10^{-6}) as a basis for the cleanup and protection of human health at Superfund sites (USEPA, 1990).

The average lifetime non-cancer risk, the HI, was 0.962, as shown in Table 5, Exposure to compounds with HI ≤1.0 is considered not likely to result in adverse non-cancer health effects over a lifetime of exposure. If a HI > 1.0, then some possibilities exist that non-cancer effects may occur, although an HI > 1.0 does not indicate a definite effect.

3.3. Biological Exposure Indices: BEI

This study assessed the car painters' exposure to benzene, toluene, ethylbenzene and xylene, by determining the biological exposure indices (BEI) for t,t muconic acid, hippuric acid, mandelic acid and m-hippuric acid in urine. The results are shown in Table 6. Biological monitoring data demonstrate that the average level of metabolites in car painters and controls were not significantly different from the control group ($p>0.05$).

3.4. DNA damage

This study assessed DNA damage of the car painters exposure to VOCs by comet assay. The results are summarized in Table 7 and Fig. 3. The DNA damage was assessed by tail moment. In general tail moment is defined as the product of the tail length and the fraction of total DNA in the tail.

Table 5. The average lifetime non cancer risk assessment of car painters

garage	1	2	3	4	5	6	7	8	9	mean	control
Non Cancer Risk (HQ)											
benzene	0.005	0.003	0.004	0.005	0.003	0.005	0.005	0.004	0.006	0.004	0.001
toluene	0.833	0.773	1.178	1.213	0.627	0.673	0.649	0.745	0.529	0.802	0.002
ethylbenzene	0.024	0.012	0.023	0.026	0.011	0.013	0.025	0.012	0.013	0.018	0.000
xylene	0.185	0.091	0.126	0.142	0.091	0.127	0.194	0.107	0.084	0.128	0.000
styrene	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
1,3-butadiene	0.009	0.010	0.009	0.010	0.010	0.010	0.009	0.010	0.011	0.010	0.009
hexane	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
chloroform	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
1,4-dioxane	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
HI	1.056	0.889	1.341	1.397	0.742	0.829	0.883	0.878	0.643	0.962	0.011

HQ = Hazard quotient is defined as the ratio of estimated exposure of an individual to the reference dose
HI = Hazard index is determined by calculating HQ for a compound and summing all of the HQ at a specific location

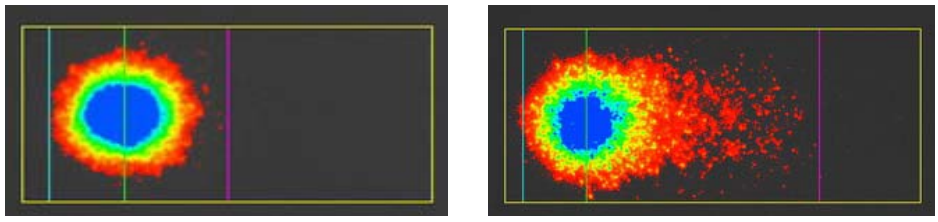
Table 6. The average level of urine metabolites from car painters and control

garage	benzene (t,t-muconic acid) (μg/g creatinine)		toluene (hippuric acid) (g/g creatinine)		ethylbenzene (madelic acid) (g/g creatinine)		xylene (m-hippuric acid) (g/g creatinine)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	89.23	29.24	0.16	0.05	0.00	0.00	0.01	0.01
2	78.89	16.85	0.21	0.13	0.00	0.00	0.00	0.00
3	102.96	37.84	0.22	0.12	0.00	0.00	0.00	0.00
4	87.19	22.00	0.20	0.12	0.01	0.01	0.00	0.01
5	86.07	25.08	0.31	0.31	0.00	0.00	0.00	0.00
6	97.96	23.42	0.15	0.07	0.00	0.01	0.00	0.01
7	94.92	16.53	0.15	0.06	0.00	0.00	0.00	0.00
8	87.25	20.41	0.25	0.16	0.01	0.00	0.01	0.01
9	101.19	24.21	0.13	0.13	0.01	0.01	0.00	0.00
Mean 9 garages	91.74	23.63	0.20	0.14	0.00	0.00	0.00	0.01
Min	55.48		0.01		0.00		0.00	
Max	135.00		0.74		0.01		0.02	
controls	56.80	12.86	0.06	0.06	0.00	0.00	0.00	0.00
Min (controls)	45.34	45.34	0.00		0.00		0.00	
Max (controls)	76.53	76.53	0.12		0.00		0.00	

Table 7. The average level of tail moment from car painters and controls

garage	tail moment	
	Mean	SD
1	7.69*	3.55
2	6.85*	1.17
3	4.02	0.54
4	7.08*	2.31
5	4.66	1.07
6	4.94*	2.00
7	5.23*	0.61
8	3.31	0.84
9	4.54*	1.33
Mean 9 garage	5.37	2.13
Min	2.48	
Max	11.93	
control	2.27*	0.40
Min	1.60	
Max	2.58	

*significantly different ($p<0.05$)



(a) (b)

Figure 3. The tail moment from (a) controls and (b) car painters

tail moment=(tail.mean-head mean)x tail%DNA

The average tail moment of all garage workers was significantly different from the controls ($p<0.05$) and higher than the control about 2 times.

4. Discussion

This study demonstrated that car painters were exposed to higher VOCs levels than control workers who were not direct contact with VOCs. Toluene, xylene and ethylbenzene were found to be the most abundant species in emission from the painting processes (5589.3, 2074.6 and 829.8 μg/m^3, respectively). However all VOCs in personal air samples were lower than TWA limit of the Thailand and the OSHA standards (OSHA, 1997).

This result was similar to that of a Chinese study, which found that the concentrations of xylene, toluene, ethylbenzene (42.0, 25.7 and 11.1%, respectively) were the main species of VOCs emission from automobile paint applications. (Yuan *et al.,* 2010). This result was agreement with Taiwan study, which found that toluene and xylene in aircraft paint spraying workers were the most abundant species (40,318.7 and 2,171.2 μg/m^3, respectively) (Uang *et al.,* 2006).

The health risk assessment in this study shows that the lifetime cancer risk of car painters was below 100 per million, which is an acceptable risk 1,3-butadiene was the most important cause of cancer risk. The average lifetime non-cancer risk was below1. It was below the reference value. Toluene was the most important cause of non-cancer risk. Risk was not a health concern. It may be speculated that the car painters in the Thai car painting houses also worked in an open environment with good ventilation. However when they were employed for a longer periods of time, the health risks were raised.

The cancer risk of car painters in this study (41per million) was found to be lower than in a study of gas service station workers in Thailand (213 per million), while the non-cancer risk in this study (HI=0.962) was found to be higher than in the study of gas service station workers in Thailand (HI=0.157) (Yimrungruang *et al.,* 2008). It may be speculated that the differences are due to differences in the species of VOCs in the different VOCs sources.

The urinary levels of t,t muconic acid of car painters in this study (91.7 μg/g creatinine) was close to that of gasoline pump maintenance workers in the Italian study (92.0 μg/g creatinine) (Fracasso *et al.,* 2010), and traffic policemen (82.0 μg/l) (Fustinoni *et al.,* 2005). But it was lower than in the Italian study of gas service station workers (171μg/g creatinine) (Carrieri et al., 2005) and the South Korea study of industry workers (966 μg/g creatinine) (Kang *et al.,* 2005). However, all urine metabolites were lower than BEIs of ACGIH standard. (ACGIH, 2005; ACGIH, 2013)

The DNA damage (tail moment) in this study was in agreement with a Brazilian study, which found that the tail length in the car painters were significantly higher than in the controls (Martino-Roth *et al.,* 2003) and an Italian study, which found that tail moments in petrochemical industry operators were significant higher than in the controls (Fracasso *et al.,* 2010). Car painters exposed to VOCs were associated with chromosome damage and possible target tissues, and a higher cancer risk that may be influenced by dosage, time of exposure and susceptible genotypes of xenobiotic -metabolism and DNA repair genes (Hoyos-Giraldoa *et al.,* 2009).

The VOCs exposures in car painting houses would be influenced by paints and solvents (Na *et al.,* 2004; Vega et al., 2000), meteorological conditions, and ventilation (Jo and Song, 2001). It can be noted that the most car painters worked without using protective devices such as masks or gloves. The car painters should reconsider the use of protective devices. The results of these studies may be used as regulatory tools for improving heath monitoring and preventing cancer risk.

Moreover, there should be research and development leading to a decrease in the amount of benzene and other hazardous substances in paints and solvents. Studies about other dimensions of health risk from VOCs should be conducted.

5. Conclusion

Car painters who work near VOCs sources, such as paints and solvents, were exposed to higher VOCs levels than control persons who were not direct contact with VOCs. Toluene, xylene and ethylbenzene were found to be the most abundant species in emission from the painting processes. However, all VOCs in personal air samples were below the TWA limit of the Thailand and the OSHA standard. The health risk assessment shows that the cancer risk was below 100 per million which is an acceptable risks, and for the non-cancer risk, the HI was below 1. It was below the reference value. The urinary metabolites, including t,t muconic acid, hippuric acid, mandelic acid and m-hippuric acid in car painters and in controls were close and lower than the BEIs of ACGIH standard. The DNA damage measurements demonstrate that tail moment in car painters and in controls were significantly higher than in a control.

Acknowledgements

This research work is supported in part by the grant from the Center of Excellence on Environmental Health and Toxicology (EHT), Graduate School of Environmental Science, Faculty of Science, Burapha University, Chonburi. Technical assistances from the Environmental Research and Training Center, Department of Environmental Quality Promotion and Bureau of Occupational and Environmental Diseases, Department of Diseases Control are greatly acknowledged.

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Received 12 May 2014
Accepted 15 June 2014

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