HUMAN HEALTH HAZARD OF POLYCYCLIC AROMATIC HYDROCARBON IN ROAD DUST IN HA NOI METROPOLIS

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ABSTRACT

This study investigates PAHs content in road dust of Hanoi metropolis, Vietnam. The samples were colected from the roads around city and analyzed by gas chromatography mass spectrometry (GC/MS). The total PAHs mass concentration ranges from 33.88 µg/kg to 5588,16 µg/kg, with the mean of 356,24 µg/kg in which HMW accounted up 70 % and LMW made up 30 %. The toxic equivalence factors (TEFs), mutagenic potency equivalent factors (MEFs) and the incremental lifetime cancer risk (ILCR) methodologies were applied to evaluate human exposure to carcinogenic PAHs sources. Carcinogenic equivalents (BaP-TEQ) and mutagenic equivalents (BaP-MEQ) were calculated from the potency relative to BaP (TEF) and BaP (MEF). The value of BaP-TEQ for Σ 8 PAHs varied from 1.13 µg/kg to 195.23 µg/kg with mean of 24.34 µg/kg, while the value of BaP-MEQ ranged 1.45 µg/kg to 123.15 µg/kg with mean of 19.96 µg/kg. Basing on ILCRs model, the total cancer risk for children and adults was up to 1.6×10^{-5} and 3.9×10^{-5}, posing a moderate potential cancer risk, respectively.

Key words: Heath risk, road dust, PAHs, ILCR, Hanoi metropolis

1. INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) are known as a group of organic compounds containing two or more fused aromatic rings. They can enter into the environment via pyrogenic and petrogenic sources [1, 2]. Pyrogenic sources are those where PAHs are generated by high temperature combustion of fossil (coal and petroleum) such as vehicle exhaust particles and biomass (burning of grass, and wood, bush fires), whereas petrogenic sources are derived from crude oil and its refined products (oil, petro, and diesel leaks, tyre particles, asphalts sealant) [2-4]. Ratios between low and high molecular weight PAHs as well as specific PAH isomers have been used to identify the sources of PAHs [2, 5]. Principle component analysis has been employed to quantify the source contribution of PAHs [6]. Due to their potential to bioaccumulation, persistence, and carcinogenic and mutagenic potencies, the United States

environmental protection agency (EPA) has identified several PAHs as significant pollutant due to deleterious effects on the environment and biological bodies and considered as priority pollutants to be controlled and routinely analyzed. Road dust which is deposited in two sides of road has a high potential source of PAHs generated from vehicles' activities as well as asphalt pavement and tire rubber constitute [7]. PAHs from car exhausts, coal emissions and tobacco smoke make up nearly all the carcinogenic PAHs [7]. In some recent years, road dust has been a significant source of PAHs in the environment media which has adverse effects on ecological function and human health via ingestion, dermal contact and inhalation [1]. The elevated concentration of PAHs in water, air and soil can be toxic to the aquatic organism, human [1, 7]. In Vietnam, anthropogenic problems associated with organic pollution of road dust such as PAH are more felt gloomy due to lack of database, inadequate pollution management. Especially in Hanoi, the rapid urbanization, population blooming as well as poor infrastructure system has been in great challenge due to ever-increasing motor-traffic density. A fast growing fleet of motor vehicles at the rate of 12 % - 15 % annually results in a most serious environmental burden and elevates road dust loadings [8]. However, very few studies are devoted to the occurrence, source and toxicity of PAHs in road dust from metropolis. Therefore, to evaluate the health risk of PAHs exposure via inhalation, dust intake and dermal contact is utmost necessary. The main objective of this study is to identify the PAHs concentration and possible source in Hanoi road dust. Also, to evaluate human hazard to carcinogenic PAHs exposure through multiply pathway. These results will provide an important insight to PAHs in Hanoi urban environment and is conductive to the scientific society, the local enterprises and policy makers of municipality.

2. MATERIAL AND METHOD

2.1. Sampling collection

Thirty-two road dust samples were taken from roads including roundabout, bus station, highway and residential areas in Hanoi in November 2015. Each dust sample of appropriately 500 g was collected from an area (50 m×50 m) at the asphalted pavement within the shoulder of the road using the clean plastic broom [4]. The sampling grid is shown in Fig. 1.

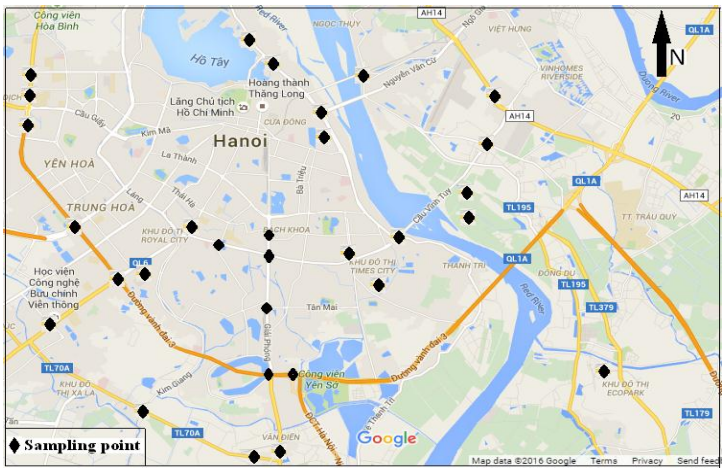

Figure 1. Sampling locations at Hanoi city.

2.2. PAH Analysis

The samples were sieved through a metal sieves with mesh of 60 µm. Then, a 5.00 g sample was extracted ultrasonically 3 times with 150ml dichloromethane. The extract was then concentrated under a rotary evaporator, the aliphatic (non-aromatic) hydrocarbons and PAH fractions were isolated by the standard silica-alumina column [9 - 16]. The extraction was removed into the

standard silica-alumina column filled with 10 g silicagel (60–100 mesh) on which it is covered by amorphous sodium sulfate. Then the aliphatic fraction was washed by hexane (50 ml) and was abandoned, while PAH fraction was washed by dichloromethane-hexane (1:1) (50 ml) and was kept and then re-concentrated with ultra-pure nitrogen to exactly 1 ml. The concentrated extracts were then analyzed for PAHs by a gas chromatography coupled to mass spectrometry (Agilent 6980N, U.S.A) equipped with a fused-silica DB-5MS capillary column (30 m × 0.32 mm i.d. 0.25 μm film thickness) [16 - 9]. The identification of individual PAHs was based on the comparison of retention times (chromatographic column) and mass spectra of PAHs in samples with those of PAH standards. Quantification of PAHs was performed in selected ion monitoring (SIM) mode.

2.3. Quality control

The GC/MS was calibrated with a diluted mixture of 16 standard PAHs: Nap, Ace, Acy, Flu, Phe, Ant, Fl, Pyr, BaA, Chry, BbF, BkF, BaP, IdP, DbA and BgP. Analysis of serial dilutions of the PAH standards showed that the detection limit for individual PAHs ranged from 50 ng/g to 7500 ng/g.

2.4. Health risk assessment from PAHs exposure

To quantify the toxicity or carcinogenic potency of PAHs relative to BaP, the toxic equivalent factors (BaP_{TEF}) and mutagenic equivalent factors (BaP_{MEF}) relating the carcinogenic mutagenic potency of individual PAH to BaP have been used [9-10, 12-11]. The BaP carcinogenic equivalent (BaP_{TEF}) and BaP mutagenic equivalent (BaP_{MEQ}) for the individual PAHs was calculated in Eq (1.2):

$$BaP_{TEQ} = \sum C_i \times BaP_{TEF} \quad (1)$$

$$BaP_{MEQ} = \sum C_i \times BaP_{MEF} \quad (2)$$

wwhere BaP_{TEF}, BaP_{MEF} is the cancer, mutagenic potency relative to BaP, respectively and Ci is the individual PAH concentration.

Residents are exposed to urban road dust through three main pathways: ingestion, inhalation and dermal contact with dust particles [1]. Evaluation of Incremental Lifetime Cancer Risk (ILCR) was carried basing the equations as follow:

$$ILCR_{ingestion} = \frac{\left\{BaP_{TEQ}\left\{CSF_{ing}\sqrt[3]{\frac{BW}{L}}\times IR_{soil}\times EF\times ED\right\}\right\}}{BW\times AT\times 10^{6}} \quad (3)$$

$$ILCR_{Dermal} = \frac{\left\{BaP_{TEQ}\left\{CSF_{derm}\sqrt[3]{\frac{BW}{L}}\times SA\times AF\times ABS\times EF\times ED\right\}\right\}}{BW\times AT\times 10^{6}} \quad (4)$$

$$ILCR_{inhalation} = \frac{\left\{BaP_{TEQ}\left\{CSF_{inh}\sqrt[3]{\frac{BW}{L}}\times IR_{air}\times EF\times ED\right\}\right\}}{BW\times AT\times PEF} \quad (5)$$

where: $ILCR_{ing}$, $ILCR_{derm}$, $ILCR_{inh}$ are the incremental lifetime cancer risk for ingestion, dermal and inhalation routes respectively. $CSF_{ingestion}$, $CSF_{iinhalation}$, SF_{dermal} is carcinogenic slope factors (7.3, 3.85, 25 $mg.kg^{-1} d^{-1}$, respectively [10-12, 12-11, 13]); BW is body weight (15 kg and 60 kg for child and adult, respectively [10-12, 11-14]); AT is the average life span (25550 days [11-14]); L is lifetime (70 years [10-12, 16-9]); EF is the exposure frequency (180 $d.year^{-1}$ [10-12, 11-14]); ED is the exposure duration (6 and 24 years for child and adult, respectively [10-12,

11-14]), IR_{air} is the inhalation rate (5 and 20 m^3/day for child and adult, respectively [11-14]), IR_{soil} is the soil intake rate (200 and mg.d^{-1} for child and adult, respectively [10-12, 11-14]); SA is the dermal surface exposure (2800 and 5700 cm^2 for child and adult, respectively [11-14]); AF is the dermal adherence factor (0.2 and 0.07 mg.cm^{-2} for child and adult, respectively [10-12]); ABS is the dermal adsorption fraction (0.13 [11]) and PEF is the soil dust produce factor (1.36×10^9 mg kg^{-1} [11-14]). The total risks were the sum of risks associated with all exposure routes.

2.6. Statistical analyses

Principle component analysis (PCA) can be used to reduce data and extract a smaller number of independent factors to find the relationship among observed variables. Classic statistical analyses were processed using IBM SPSS software version 20. A probability level of $P < 0.05$ was considered statistical significant.

3. RESULTS AND DISCUSSION

3.1. Total PAHs concentration

Different levels of 16 PAH congeners were detected in all Hanoi metropolis road dust samples. Table 1 represented the levels of PAHs in road dust. Total PAHs concentrations in road dust samples displayed a very wide range from 33.88 µg/kg to 5588,16 µg/kg, with the mean of 356,24 µg/kg. Maliszewska KordyBach (1996) divided contamination levels in to four categories according to the total PAHs: non-contaminated (< 200 µg/kg), slightly contaminated (200 - 600 µg/kg), contaminated (600-1000 µg/kg), and heavily contaminated (>1000 µg/kg) [13]. Using these criteria, all road dust samples were fallen in slightly contaminated. However, the categories of contamination levels varied largely among the sites consisting of roundabout, highway, bus station and residential road and shown in Table 2. The total PAHs concentrations in road dust at roundabout, highway, bus station and residential road ranged from 30.88 to 5588 µg/kg, 87.38 to 378.32 µg/kg, 55.89 to 288.88 µg/kg and 45.09 to 314.78 µg/kg, with the mean of 876.13 µg/kg, 215.38 µg/kg, 141.62 µg/kg and 121.69 µg/kg, respectively. Consequently, the roundabout was classified into contaminated level, following to highway with slightly contaminated, while residential and bus station revealed non-contaminated. The total PAHs concentrations in roundabouts were much higher than those of other traffic areas and decreased in order: roundabouts > highway > bus station > residential area. This may be due to the increased traffic density, multiple traffic sources, including a high fraction of diesel vehicles such as trucks and buses in highways. The low vehicle speeds around roundabouts may make the longer traveling time in the circulations due to traffic congestion increase traffic volumes, energy consumption and surface abrasion that can release higher loadings of PAHs [15]. The contribution in LMW (Low molecular weight) and HMW (high molecular weight) portion comparing to total PAH mass differentiated among sites. The order of distribution was followed by roundabout (78.85 %) > Residential areas (69.43 %) > Station (66.58 %) > highway (62.49 %). The HMW were prevail in PAH congeners in all sites that agreed the previous studies [3,15]. This may be due to the tendency of higher molecular weight compounds to adhere to road dust [15]. Also, the high fraction of HMW PAHs in total PAHs mass indicated that they mostly came from source of vehicle emission [3]. This result was similar with the speciation of PAHs in gasoline vehicle soots enriched with HMW PAHs, revealing a dominant influence of gasoline vehicle release [2]. The elevated concentration of HMW PAHs in solid surface can be understood that the HMW PAHs are likely associated with airborne particulate that undergo

"single hop" transport behavior committing more of the HMW PAHs to settle and accumulate in road surface close to emission sources [5].

Table 1. Occurrence of PAHs in road dust in Hanoi metropolis (n = 32) (µg/kg).

Compounds	Aromatic ring	TEF[3,13,16]	MEF[3,13,16]	Mean	Min	Max	Std.Dev
Naphthalene (Nap)	2	0.001		52.9	0.11	529.89	99.59
Acenaphthylene (Acy)	3	0.001		4.02	0.11	28.82	5.28
Acenaphthene (Ace)	3	0.001		4.01	0.11	27.79	6.66
Flourene (Fl)	3	0.001		6.05	0.06	34.76	6.78
Phenanthrene (Phe)	3	0.001		7.3	0.11	73.51	12.83
Anthracene (Ant)	3	0.01		5.76	0.12	63.63	11.05
Flouranthene (Flu)	4	0.001		33.09	9.41	181.05	32.20
Pyrene (Pyr)	4	0.001		44.88	0.14	259.15	48.33
Benzo(a) anthracene (BaA)	4	0.1	0.082	13.62	0.21	64.42	12.64
Chrysene (Chry)	4	0.01	0.017	141.36	0.11	4252.1	750.25
Benzo(b)flouranthene (BbF)	5	0.1	0.250	9.53	1.52	35.34	8.11
Benzo(k)flourathene (BkF)	5	0.1	0.11	8.0	0.11	52.36	11.83
Benzo(a) pyrene (BaP)	5	1	1	7.7	0.11	53.04	11.67
Dibezo(ah) anthracene (DbA)	5	1	0.29	12.02	0.11	127.93	26.36
Benzo(ghi) perylene (BgP)	6	0.01	0.190	6.68	0.11	66.19	13.51
Indeno (1,2,3-cd) pyrene (IdP)	6	0.01	0,310	2.31	0.11	7.23	2.27
ΣPAHs				356.24	30.8	5588.1	964.62
BaP-TEQ				24.34	1.13	195.23	40.58
BaP - MEQ				19.96	1.45	123.15	30.40

Table 2. PAH contamination levels in sites basing on Maliszewska-Kordybach (1996).

Position	Σ PAHs (µg/kg)	Contamination degree
Highway	215,38	Slight contaminated
Residential	121,69	No contaminated
Bus station	141,62	No contaminated
Roundabout	876,13	Contaminated

3.2. Identification of the sources PAHs

Principal component analysis (PCA) is powerful tool for distinguishing pollution sources. According to the initial Eigen values, two principle components had Eigen values greater than 1 and cumulatively represented 86.25 % of data variance. The 16 PAH congeners were separated into two distinct components on the factor loading plot (Figure 2). The first component included BaA, Flu, Pyr, Fl, Phe, Ant, Acy, Chry, Ace and Nap describing 61,27 % of total variance of data while the second component included BbF, BkF, BaP, DbA, BgP and IdP accounting up 24.97 %. The first component consisted of both light molecular weight (2-4 rings) and high molecular weight PAHs (4-6 rings) suggesting mixed sources, in which Nap, Ace, Acy, Fl, Phe, Flu, Pyr and Ant could be considered as combined component of petroleum and cooking source [2.7], while Chry, BaA were derived from coal combustion and vehicle emission [5]. The

second component was heavily weighted by BbF, BkF, BaP, DbA, BgP and IdP which were consistent with emission characteristics of PAHs from coal combustion and vehicle [4] while BbF was consistent with emission of diesel [5]. It could be included that primary sources of PAHs in Hanoi Metropolis road dust are vehicle exhausts, coal combustion and petroleum components and secondary source is the deposits coming from long-range atmospheric transport.

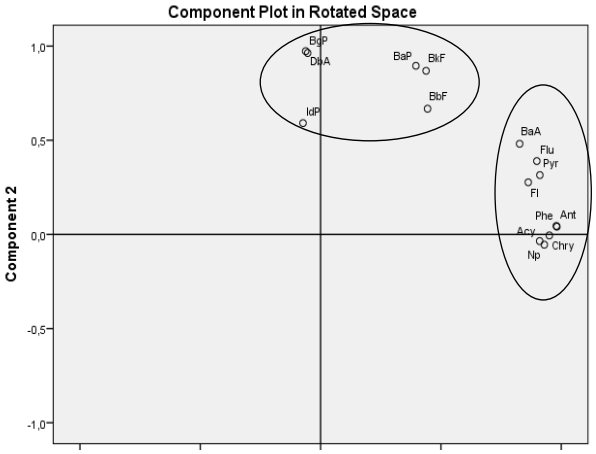

Figure 2. PCA of road dust.

3.3. Mutagenic and carcinogenic equivalent of PAhs and their contribution

The theoretical mutagenic and carcinogenic equivalents of PAH were calculated by multiphying the concentrations in road dust by appropriate mutagenic and carcinogenic potencies relative to BaP and the total values were expressed as BaP-MEQ and BaP-TEQ, respectively which gave the best information of PAH toxicity and were calculated by equation 1, 2 [10, 11]. The results showed that total BaP-equivalent concentration ranged from 1.23 µg/kg to 195.63 µg/kg, averaging 24.55 µg/kg. While BaP carcinogenic equivalent (BaP_{TEQ}) varied from 1.13 µg/kg to 195.23 µg/kg with mean of 24.34 µg/kg, BaP mutagenic equivalent (BaP_{MEQ}) were from 1.45 µg/kg to 123.15 µg/kg with mean of 19.96 µg/kg. The contribution of mutagenic and carcinogenic potencies was shown in Figure 3.

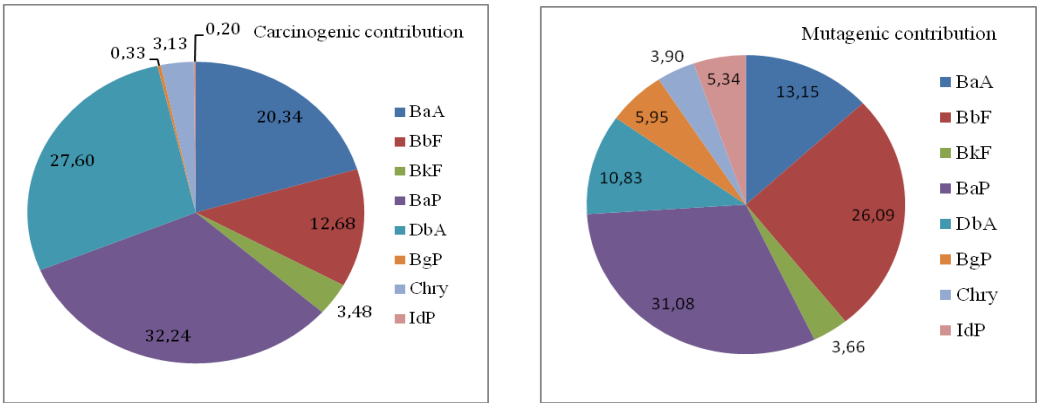

Figure 3: contribution of individual PAH to mutagenic and carcinogenic potencies.

While contributions of individual PAHs to mutagenic potencies was in the order of BaP (31.08 %) > BbF (26.09 %) > BaA (13.15 %) > DbA (10,83 %) > BgP (5.95 %) > IdP (5.34 %) > BkF (3.66 %) > Chry (3.9 %), Those to carcinogenic potencies was in the order of BaP (32.24 %) > DbA (27.6 %) > BaA (20.34 %) > BbF (12.68 %) > BkF (3.48 %) > Chry (3.13 %) > BgP (0.33 %) > IdP (0.2 %). The findings revealed that the contributions of PAH congeners in mutagenic and carcinogenic potencies were similar. While BaP, BbF, BaA and DbA are the major mutagenic and carcinogenic contributors in Vietnamese road dust while BaP and B(b+k)F; IdP + BgP and BaP are predominant in mutagenic and carcinogenic potencies in Korea [15].

3.4. Health risk assessment

This study has identified the potential carcinogenic risk due to the exposure of human (child and adult) to PAHs in road dust samples. The Incremental lifetime cancer risk model (ILCR) has been applied to assess the potential risk of cancer induction in exposed human to environmental toxicants [1, 14, 16]. Carcinogenic potencies relative to BaP, carcinogenic slope factor (CSF) and probabilistic risk assessment framework were applied to estimate the cancer risk to human who exposed road dust though three pathways. The carcinogenic total risk was calculated from the summation of individual risks through each route of exposure. Exposure carcinogenic risk is calculated using equation 3-5.

The cancer risk levels for children via ingestion, inhalation and dermal contacts were 7×10^{-6}, 1×10^{-9}, and 9×10^{-6}, respectively, while these for adults were 1.4×10^{-5}, 1×10^{-8}, and 2.5×10^{-5}. Cancer risks of inhalation of children and adults were low, almost 10^3 times lower than from ingestion and dermal contact. Therefore, the inhalation of resuspended particles through mouth and nose were almost negligible when compared with other pathway. Ingestion and dermal contact appeared to be predominant exposure route for both children and adult. These findings were consistent with previous studies in China and Nigeria [3, 16]. Cancer risk between 10^{-6} and 10^{-4} indicate moderate health risk, while greater than 10^{-4} suggests high potential health risk [14]. The total cancer risk is the sum of risks incurred from exposure routes of ingestion, dermal contact and inhalation. In this study, the total cancer risk for adult and children were up 3.9×10^{-5} and 1.6×10^{-5}, respectively, which indicates a moderate potential cancer risk. Cancer risk via derma contact and digestion for adults was relatively higher than children due to larger exposure area and longer exposure duration, respectively. However, the children can be vulnerable due to high physical contacts (hand to mouth) with road dust from out-door activities. In addition, considering the smaller body weight, the PAH intake of a child were believed to be greater than that of an adult. Thus, the hazard health risk for children exposed to PAHs in road dust is thought to be greater than that of adult. The findings obtained in this study also raised the concern over the potential effect of ambient air contaminated with PAHs on the occurrence of common respiratory and skin diseases related to urban air pollution in Hanoi urban areas [8] and such effects need immediate attention.

4. CONCLUSION

Polycyclic aromatic hydrocarbons are pervasive in the road dust of Hanoi metropolis. The road dust samples were classified into slightly contaminated by PAHs for overall sampling sites according to Maliszewska-KordyBach (1996). However, there was stringent different contamination level among sites including roundabout, highway, bus station and residential areas in which the roundabout and highway were classified into contaminated level and slightly contaminated, respectively, residential and bus station presented non-contaminated. The vehicle exhausts, coal combustion and petroleum components were primary sources of PAH accumulated in road dust in Hanoi metropolis. Basing on TEQ and MEQ calculation, BaP, BbF, BaA and DbA are good makers in assessing PAHs related with health risks. The adopted approach in this study is very useful in determining the overall toxicity of road dust as well as evaluating specific chemical components in the environment samples. It is suggesting that mitigation programs related to PAHs in Hanoi should focus on controlling exhausts emission and coal combustion. The estimated integrated lifetime cancer risks associated with exposure to road dust indicated that ingestion and dermal contact are prevailing exposure to adult and children which fell in moderate potential cancer risk.

REFERENCES

1. Changsheng Q., Bing L., Haisui W.- Multi-pathway assessment of human health risk posed polycyclic aromatic hydrocacbon, Environ Geochem Health **37** (2015) 1-15.
2. Yunker M. B. Robie W. M, Roxanne V, Mitchell H - PAHs in the Fraser River Basin: a critical appraisal of PAH ratios as indicators of PAH source and composition. Organic Geochemistry **33** (2002) 489-525.
3. Peng C, Chen W, Liao X, Wang M, Zhiyun Q, Jiao W, Yang B. - Polycyclic aromatic hydrocarbons in urban soils of Bejing: Status, Sources, distribution and potential risk. Environmental Pollution **159** (2011) 802-808.
4. Ruchaya B. Michio M. Gullaya - Sources of polycyclic aromatic hydrocacbons in street dust in a tropical Asian mega city, Bangkok, Thailand. Scien total Environ. **384** (2007) 420-432.
5. Simcik M. F, Eisenreich S. J. Lioy P. J. - Source Apportionment and Source/Sink Relationships of PAHs in the Coastal Atmosphere of Chicago and Lake Michigan. Atmospheric Environment **33** (1999) 5071-5078.
6. Loganathan. P, Vigneswaran S, Kanda J. – Road deposited sediments: a critical review of their characteristics, source apportionment and management. Cri. Rev. Environ. Sci. Technol. **43** (2013) 1315-1348.
7. Khalili N. R. Scheff P. A. Holsen T - PAH Source Fingerprints for Coke Ovens, Diesel and Gasoline Engines, Highway Tunnels, and Wood Combustion Emissions. Atmospheric Environment **9** (1995) 533-542.
8. Hung NT. - Urban air quality modeling and management in Hanoi Vietnam, Ph.D thesis. National Environmental Research Institute, Aarhus University, 2010.
9. H. Guoa, S.C. Leea, K.F. Hoa, X.M. Wangb, S.C. Zouc. - Particle-associated polycyclic aromatic hydrocarbons in urban air of Hong Kong, Atmospheric Environment **37** (2003) 5307-5317.
10. Nisbet, I.C and LaGoy, P.K. - Toxic equivalency factors (TEFs) for polycyclic aromatic hydrocarbons (PAHs). Regul. Toxicol. Pharmacol**. 16** (1992) 290-300.
11. Knafla A. Phillipps K.A, Petrovic R.W, Richardson M.- Development of a dermal cancer slope factor for Benzo[a]pyrene. Regul Toxic and Pharm **45** (2006) 159-168.
12. US, EPA. Exposure Factors Handbook: 2011 Edition, Office of Research and Development, Washington, DC, 2011.
13. Maliszewska-Kordybach B. - Polycyclic aromatic hydrocarbons in agricultural soils in Poland. Preliminary proposals for criteria to evaluate the level of soil contaminantion Appl Geochem **11** (1996) 121–127.
14. US EPA Supplemental guidance for developing soil screening levels for superfund sites. OSWER, 2002, pp.9355.4-24.
15. Lee B.K and Trang T.T.D - Toxicity and source assignment of polycyclic aromatic hydrocarbons in road dust from urban residential and industrial areas in a typical industrial city in Korea, J Mater Cycles Waste Manag **13** (2011) 34-42.
16. Olawoyin R , Larry G. R, Oladapo T. O. - Eco-toxicological and epidemiological assessment of human exposure to polycyclic aromatic hydrocarbons in the Niger Delta, Nigeria. Res Art Toxic & Environ Health Science **4** (2012) 173-185.