HEALTH RISK ASSESSMENT OF AIR CONTAMINATION CAUSED BY POLYCYCLIC AROMATIC HYDROCARBONS FROM TRAFFIC

FRANTISEK BOZEK, VLADIMIR ADAMEC, JOSEF NAVRATIL, JOSEF KELLNER, ALENA BUMBOVA, JIRI DVORAK
Civil Protection Department
University of Defence
Kounicova 65, 662 10 Brno
CZECH REPUBLIC
frantisek.bozek@unob.cz http://www.vojenskaskola.cz

Abstract: - The concentrations of 17 types of polycyclic aromatic hydrocarbons in the air have been detected in the selected area of the town of Brno in the Czech Republic. The sampling area is characterized by heavy traffic. The detected concentrations of six PAHs (benzo[a]pyrene, indeno[1,2,3-cd]pyrene, benzo[k]fluoranthene, benz[a]anthracene, dibenz[a,h]anthracene and chrysene) with likely carcinogenic effects on people have been chosen for risk assessment. The risk results from the exposure of adults and children up to the age of six living near the road to the contaminated air.

Key-Words: - air contamination, exposure, excess lifetime cancer risk, health, risk assessment, polycyclic aromatic hydrocarbons (PAHs), traffic

1 Introduction

The high increase of traffic contributes to the increasingly higher environmental burden. Besides the changes in the morphology of landscape it causes negative impacts on wild fauna, increased noise burden, vibrations, contamination of hydrosphere and lithosphere and it contributes the most significantly to deteriorating the quality of troposphere.

The main pollutants are CO_2, CO, NO_x, N_2O, SO_2, O_3, Pb, Cd, Ni, Cr, platinum metals, volatile organic compounds, benzene, aldehydes and persistant pollutants in the form of polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls, dibenzo-p-dioxins, dibenzofurans, etc. The emitted pollutants have negative impacts not only on ecosystems, but also on human population, which suffers from serious health problems.

2 The Analysis of Current State

At present the attention is paid to suspended dust particles. They are dangerous, because they not only cause the irritation of eyes, but they are mainly capable of binding a number of toxic inorganic and organic substances to their surface. The PAHs belong to highly toxic pollutants, which are adsorbed on suspended particles.

The PAHs emissions are produced during the operation of motor vehicles as they are present in fuel. They are also produced from carbonaceous fragments when there is an excess of fuel and a lack of oxygen [1]. The temperature of combustion engines is a significant factor affecting the production of PAHs, because the low temperature prevents their decomposition. Part of PAHs with three to four aromatic circles is adsorbed on particles, another part is present in the form of vapour in the air. Heavy PAHs are mainly bound to the surface of solid particles through the adsorption and condensation of cooled emitted gases. Their share of gas phase rapidly decreases with the rising number of circles [2]. PAHs are bound from 56 - 89 % in the fraction of particles smaller than 3.0 μm [3], the inhalation of which causes serious health problems. The derivates with four to five cycles have the maximum genotoxicity. The occurrence of PAHs derivates is connected also with the transformational processes in the atmosphere and many of them are of higher carcinogenity, mutagenity and teratogenity then the original noxys [4].

The particles smaller than 2.5 μm ($PM_{2.5}$) are the most dangerous ones, because they are not caught by upper respiration tract. The epidemiologic studies show that these particles have not only short-term effects, but also chronic effects, mainly in the long-term exposure and even in the below-limit concentrations. The short term effects include mechanical damage of cornea, more difficult breathing, declined health especially among asthmatics, damage of lung tissue leading even to the fibrillation of lungs, etc. The long-term effects can be seen in the increased occurrence of bronchitis, cardiovascular and reproduction malfunctions and in

case of extreme exposure also cancer, primary of the respiratory organs [5, 6]. Long-term exposure to increased concentration of particles results in a shorter average life span [7], increased morbidity and mortality [8]. The risks of these troubles closely correlate with the concentrations of mainly $PM_{2.5}$ [6-8].

For the abovementioned reasons, it was decided to quantify the concentration of priority PAHs in the area affected by heavy traffic and assess the resulting genotoxic risks for the local population.

3 Problem Solution

3.1 Applied Methods and Devices

Two crossroads with high intensity of traffic were selected for the experiment in the town of Brno, Czech Republic. The average number of vehicles is $3.7x10^4$ per day there. LECKEL MVS6 sampling device produced by Sven Leckel, Germany, was approximately 3 m far from the crossroad.

The air samples were collected daily in 7-day sampling campaigns in 5-week intervals. There were 84 samples collected in six sampling campaigns during approximately 8 months. The samples were collected for 24 hours with 55 m^3 air collection capacity. Quartz filters were chosen for the sampling and their weight was pre-determined with the accuracy of 0.05 mg. Two devices were placed at the collection point to verify the quantity of collected $PM_{2.5}$ particles.

The exposed filters were taken out of the sampling device, wrapped up into the double layer of aluminium foil and distributed into the laboratory. The samples were extracted by dichloromethane in the Fex IKA extract of the IKA Werke GmbH & CO KG company and then thickened in a cuvette by blowing off with nitrogen. Then the extract was purified with the help of liquid chromatography on an activated silica gel by rinsing with hexane and dichloromethane.

The quantification of PAH from the extracts was carried out with the help of gaseous chromatography in combination with spectrometry (GS/MS) in the device of SHIMADZU company. The extracts were stored into vials, which were moved to autosampler. Helium with the flow rat of 0.5 $m.s^{-1}$ was selected as a mobile phase. The temperature of injector was set to be 573 K. After gasification the sample was moved by the mobile phase into the heated EQUITY 5 column, diameter of 0.25 mm and length of 30 m. The stationary phase in the form of 0.25 μm thick film consisted of polar and nonpolar phases in 1:19 ratio. The GC was set according to the known time spectra of monitored PAHs. The quantification of individual PAHs was carried out with a mass spectrometer in SIM mode, i.e. with the use of three ions set for detecting 17 PAHs. Terphenyl was used as internal standard for the quantification.

The risk assessment method was used in compliance with the legal system of the Czech Republic [9] while observing the U.S. EPA method [10, 11].

3.2 Outcomes and Discussion

The outcomes of the average concentrations of monitored PAHs during a certain period of time are presented in Table 1. When the concentrations were below the detection limit, half of the value of detection limit was used, i.e. 0.015 $ng.m^{-3}$for PAHs.

Relatively high values of PAHs monitored at the end of November and the beginning of December are likely connected with a frequent occurrence of temperature inversions and worsened dispersion conditions. A significant cause of high concentration of PAHs in the air was probably also the contamination caused by household furnaces. The proof of that is the concentration ratio of benzo[a]pyrene and coronene $p = 3.69$ during the above mentioned period of time, while during the other sampling campaigns the ratio was $p < 1.00$. The increased production of PAHs could also be the result of cold starts of engines in winter time.

The PAHs suspected of having carcinogenic effects on people and classified in the B 2 group were selected for the risk assessment of inhalation exposure of inhabitants. No PAH is classified in A and B 1 groups yet [10, 12, 13].

The risk assessment was carried out according to the following formula (1):

$$ELCR = 1 - e^{(-LADD \times SF_I)} \quad (1)$$

where ELCR represents the excess lifetime cancer risk, i.e. the probability that the number of tumorous diseases exceeds the general average, LADD [$mg.kg^{-1}.den^{-1}$] represents the lifetime average daily dose and SF_i [$mg^{-1}.kg.den$] is the inhalation slope factor.

LADD was calculated with the help of equation (2) and the values of exposure factors for the inhalation exposure scenarios were taken from the national directives [9]. The values of inhalation slope factors SF_i for the monitored PAHs were adopted from the materials of U.S. EPA [13, 14]. The data are presented in the Table 2 together with the calculated values of $LADD_A$ for adults and the corresponding increase of $ECLR_A$ including the analogical values of $LADD_C$ and $ELCR_C$ for children up to the age of six.

$$LADD = \bar{c} \times IR \times ET \times EF \times ED \times BW^{-1} \times AT^{-1} \quad (2)$$

The equation (2) includes $\bar{c}$, which represents the average concentrations of particular PAHs [$mg.m^{-3}$]

during the monitored period of time, subtracted from Table 2 and calculated as an arithmetic average of measured concentrations during individual sampling campaigns. The other symbols in the equation (2) represent exposure factors. IR represents the intake rate ($IR_A = 0.83\ m^3.h^{-1}$ for adults and $IR_C = 0.5\ m^3.h^{-1}$ for children up to the age of six); ET = 21 $hrs.day^{-1}$ is the exposure time; and EF = 350 $day.year^{-1}$ is the exposure frequency. ED represents the exposure duration, the value of which is $ED_A = 70$ years for adults and $ED_C = 6$ years for children. BW is the average body weight (it was accepted that $BW_A = 70$ kg for adults and $BW_C = 15$ kg for children), AT is the averaging time, which was $AT_A = 25\,550$ days for adults and $AT_C = 2\,190$ days for children. It was assumed that the concentration of pollutants would remain approximately constant in that period of time.

The acceptable level of risk is considered to be $ELCR \leq 10^{-6}$, which represents the increase of cancer by one in the group of million of people [10]. Therefore it may be stated that with the exception of benz[a]pyrene the risk resulting from the exposure to PAHs near the crossroads is acceptable both for the population of adults and children. In case of benz[a]pyrene the risk for adults is exceeded roughly 2.5 times and is still tolerable, while the risk for children is already significant.

Provided that individual carcinogens have additive effects, the summary increase of probability that the occurrence of tumorous diseases exceeds the general average ($ELCR_S$) may be defined by the following formula (3):

$$ELCR_S = \sum_{i=1}^{q} ELCR_i \qquad (3)$$

where $ELCR_i$ is the excess lifetime cancer risk due to i-PAH; and n is the number of PAHs with $n \in N$ and N is a symbol for the set of natural numbers. It can be easily ascertained with the help of equation (3) and the values of $ELCR_A$ or $ELCR_C$ for individual contaminants from the Table 2 that the increased probability of occurrence of tumorous diseases above the general average due to the impact of PAHs suspected of having the carcinogenic effects is $ELCR_{SA} = 3.203x10^{-6}$ for adults and $ELCR_{SC} = 9.003x10^{-6}$ for children up to the age of six.

Similar outcomes regarding the contribution of individual PAHs to total toxicity have been achieved by calculating the toxicity equivalent (TEQ) when knowing the factors of equivalent toxicity and the average concentration of the monitored PAHs. The relationship is expressed by the following formula (4).

$$TEQ = \bar{c} \times TEF \qquad (4)$$

where $\bar{c}$ [$ng.m^{-3}$] is the average concentration of the contaminants during the monitored period of time and TEF are the toxicity equivalency factors. The TEF according to Nisbet a LaGoy [15] were applied in the calculation, because they show higher values in relation to the analogous data of U.S. EPA [16] in case of chrysene, benzo[k]fluoranthene and dibenz[a,h]anthracene. Provided that individual PAHs

Table 1 The average concentration of PAHs [$ng.m^{-3}$] in individual sampling campaigns

Pollutant	**Average concentration of PAHs [$ng.m^{-3}$] in 2005 sampling campaigns**					
	April 4 - 10	May 23 - 29	June 27 - July 3	August 22 - 28	October 10 - 16	November 28 - December 4
naphthalene	4.110	2.159	2.330	3.842	0.618	0.932
acenaphthylene	0.234	0.074	0.038	0.067	0.039	0.271
acenaphthene	0.186	0.120	0.044	0.113	0.015	0.135
fluorene	0.465	0.477	0.350	0.513	0.067	0.345
phenanthrene	8.703	7.296	5.430	7.423	0.966	3.892
anthracene	0.443	0.388	0.343	0.471	0.107	0.429
fluoranthene	3.301	1.168	0.998	1.468	2.190	9.049
pyrene	5.195	1.518	1.229	2.864	3.912	10.031
benz[a]anthracene	0.652	0.397	0.093	0.173	0.969	5.258
chrysene	1.227	0.642	0.198	0.191	1.482	6.993
benzo[b]fluoranthene	0.501	0.537	0.073	0.091	0.421	4.136
benzo[k]fluoranthene	0.508	0.449	0.048	0.088	0.481	7.248
benzo[a]pyrene	0.836	0.621	0.101	0.200	0.410	8.321
indeno[1,2,3-cd]pyrene	0.855	0.379	0.561	0.184	0.524	3.877
dibenz[a,h]anthracene	0.680	0.266	0.015	0.015	0.065	0.175
benzo[ghi]perylene	1.395	0.903	0.192	0.215	0.334	2.530
coronene	1.658	1.253	0.313	0.527	0.411	2.258

Table 2 The average concentrations of monitored PAHs [mg.m^{-3}] for the assessed period of time, SFi values, calculated values of $LADD_A$, $LADD_C$ and the increase of probability that the number of tumorous diseases exceeds the general average among adults ($ELCR_A$) and among children ($ELCR_C$)

Pollutant	$\bar{c}$ [mg.m^{-3}]	**SF_i** [mg^{-1}.kg.day]	**$LADD_A$** [mg.kg^{-1}.day^{-1}]	**$LADD_C$** [mg.kg^{-1}.day^{-1}]	**$ELCR_A$**	**$ELCR_C$**
benz[a]anthracene	1.257x10^{-6}	6.1x10^{-1}	3.001x10^{-7}	8.437x10^{-7}	1.831x10^{-7}	5.147x10^{-7}
chrysene	1.789x10^{-6}	6.1x10^{-3}	4.272x10^{-7}	1.201x10^{-6}	2.606x10^{-9}	7.326x10^{-9}
benzo[k]fluoranthene	1.470x10^{-6}	6.1x10^{-2}	3.510x10^{-7}	9.867x10^{-7}	2.141x10^{-8}	6.019x10^{-8}
benzo[a]pyrene	1.748x10^{-6}	6.1	4.174x10^{-7}	1.173x10^{-6}	2.546x10^{-6}	7.155x10^{-6}
indeno[1,2,3-cd]pyrene	1.063x10^{-6}	6.1x10^{-1}	2.538x10^{-7}	7.135x10^{-7}	1.548x10^{-7}	4.352x10^{-7}
dibenz[a,h]anthracene	2.027.10^{-7}	6.1	4.840x10^{-8}	1.361x10^{-7}	2.952x10^{-7}	8.302x10^{-7}

have additive health effects the TEQ total toxicity equivalent may be calculated according to the following formula (5).

$$TEQ_S = \sum_{i=1}^{q} TEQ_i \qquad (5)$$

where TEQ_i represent the toxicity equivalency factors of individual PAHs and $q \in N$ is the number of included PAHs with the assumed carcinogenic effects, in this case of B 2 group.

The acquired outcomes are shown in the Table 3. It is obvious that regarding the relative contribution of individual PAHs to the total toxicity the outcomes are relatively identical with the outcomes acquired by the risk assessment.

It is necessary to accept that a number of uncertainties exist in the above presented process of risk assessment. However, it can be assumed that the quantified values of risks are rather overestimated. It is necessary to mention mainly the following significant uncertainties:

a) It cannot be expected that the quantified average concentrations of $\bar{c}$ PAHs will remain constant for the period of AT = 70 years. The permanent increase of transport supports the progressive time dependence of average concentrations. On the other hand the implementation of adequate countermeasures and a technical progress support decrease of average concentration in time. The question is which of these tendencies will prevail.
b) It has not been precisely known what effects PAHs have in human body yet. Thus the dose-effect relationship has not been precisely known either. Therefore there are significantly different values of inhalation slope factors [17, 18] mentioned in literature. The highest discovered values of SF_i [13, 14] were used in our assessment.
c) The worst options were considered in the construction of scenarios. In the risk assessment it was not considered that there are different concentrations of PAHs during day and night, in the inner and outer environment, different distances from the road, and the mobility of population, etc. Therefore the real average concentrations to which the population is exposed will apparently be considerably lower. The calculation used the concentrations of PAHs detected 3 m from the road and the relatively high values of exposure factors ET = 21 hrs.day^{-1} and ET = 350 days.year^{-1}. The average body weight of adults BW_A will probably be higher than the applied 70 kg.
d) On the other hand there were not considered the scenarios for sensitive people (allergic persons) and the inhalation of PAHs from other sources, e.g. smoking, food (smoked meat) and contaminated

Table 3 Average concentrations of monitored PAHs for the assessed period of time, TEF values, calculated TEQ values for individual PAHs, and TEQS

Pollutant	$\bar{c}$ [ng.m^{-3}]	**TEF**	**TEQ** [ng.m^{-3}]	**TEQ_S** [ng.m^{-3}]
benz[a]anthracene	1.257	0.100	1.257x10^{-1}	**3.159**
chrysene	1.789	0.010	1.789x10^{-2}	
benzo[k]fluoranthene	1.470	0.100	1.470x10^{-1}	
benzo[a]pyrene	1.748	1.000	1.748	
indeno[1,2,3-cd]pyrene	1.063	0.100	1.063x10^{-1}	
dibenz[a,h]anthracene	2.027.10^{-1}	5.000	1.014	

water. The dermal intake of PAHs has not been included either.

e) Possible synergic effects of PAHs among themselves and with other contaminants also have not to be considered.

4 Conclusion

It can be stated from the analysis of risk resulting from the exposure of inhabitants to the air polluted by $PM_{2.5}$ particles, which are contaminated by the adsorbed PAHs, that the most threatened are people living in cities near roads with the increased intensity of traffic. The people with the weakened immune system, asthmatics, cardiacs and children up to the age of six are the most threatened in these areas.

The total risk exceeds the current standards more than three times for the population of adults and nine times for children. Despite the fact that the stated values of risks are probably overestimated, it seems to be sensible to accept the principle of preliminary precaution and implement effective countermeasures. The countermeasures may include the introduction of more efficient catalytic converters, the improvement of technical conditions of vehicles and town transport infrastructure, the use of preventive function of territorial planning, no entry of cars to city centres, promotion and education of inhabitants in the above mentioned areas, etc.

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