Genotoxic Risks for Population in Vicinity of Traffic Communication Caused by PAHs Emissions

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Abstract: - The paper deals with the assessment of risk to the population being exposed to the air contaminated by polycyclic aromatic hydrocarbons. The attention is paid to the population living in the vicinity of roads in a town agglomeration with heavy traffic. The concentrations of 17 types of polycyclic aromatic hydrocarbons have been detected in the air of selected areas with high frequencies of traffic. Gas chromatography with mass detector was used for the determination. The measured concentrations of individual pollutants with probable human carcinogenic, mutagenic and teratogenic effects were used for the assessment of a summary genotoxic risk. The risk assessment was carried out in compliance with the national methodology, respecting the standard operational procedure of U.S. EPA. It was discovered on the basis of developed appropriate exposure scenarios and the determined concentrations of contaminants that the summary genotoxic risk is getting near the unacceptable level especially in case of children to the age of six, people with the weakened immune system, asthmatics, and those with heart conditions. The outcomes acquired by the risk assessment within the contribution of individual pollutants correspond with the outcomes acquired with the help of toxicity equivalent.

Key-Words: - Air, contamination, excess lifetime cancer risk (ELCR), exposure scenarios, genotoxic risk, health, lifetime average daily dose (LADD), particulate matters (PM), polycyclic aromatic hydrocarbons (PAHs), traffic.

1 Introduction

The quality of atmosphere is much more affected by anthropogenic sources than natural sources of pollution in comparison with the previous periods. The mentioned ratio has been permanently increasing. Besides the fixed sources of pollution, which are mainly industrial and agricultural sources, producers of energy, entities disposing and treating wastes, areas of environmental burdens, etc, there are also mobile sources, represented mainly by the transport of goods and persons, contributing significantly (up to 37 %) to the increase of air emissions [1].

Emissions from various sources of pollution create qualitatively and quantitatively very complex mixture in the air. Its composition is the result of time and spatial distribution of inputs, their quantity, properties and interactions of present pollutants, as well as on meteorological and climatic conditions of a region. Long-distance transport of pollutants is a significant factor to be considered when studying pollution. The emitted pollutants may affect populations and damage eco-systems in a long distance from the source of pollution.

At present transport represents one of the most dynamically developing areas. Private car transport being used at the expense of public transport and permanently increasing share of road cargo transport in comparison to railway transport are significant trans-European problems from the viewpoint of sustainable development [2, 3].

Besides undisputable advantages connected with an intensive development of transport there are number of new negative phenomena. The most important phenomena are higher accident rate, loss of lives in traffic accidents, changed scenery and morphology of landscape, barriers for wild and migrating animals, reduction of bio-diversity, increased noise pollution, waste, vibrations, contamination of individual environmental elements caused by exhaust emissions, release of dangerous substances, use of spreading salt in roads in winter, etc. And last but not least an appropriation of land is an important factor, especially

the appropriation of agricultural land resources for the construction and re-construction of road and highway network [1].

The main pollutants from traffic are, besides the waste originated during and after the life cycle of vehicles and in accidents, mainly CO_2, CO, NO_x, N_2O, SO_2, O_3, Pb, Cd, Ni, Cr, platinum metals, volatile organic substances, CH_4, 1,3-butadiene, benzene, toluene, xylenes, phenols, aldehydes, ketones, tar, but also persistent organic pollutants in the form of polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls, dibenzo-p-dioxins, dibenzofurans, etc. [4, 5]. The emitted pollutants then secondarily and often non-quantifiably affect morbidity and mortality of population, quality and values of social assets and the functions of eco-system.

Significant decrease of air quality is apparently the most serious problem caused by the intensive development of traffic.

2 The Analysis of Current State

The air contamination significantly increases especially in city and industrial agglomerations as the volume of atmosphere is finite, the sources of pollution are most often of a regional character and self-cleaning and dispersing capacity is limited. The increased concentration of contaminants in the atmosphere is of an exponential character especially in the last decades and in city areas with heavy traffic they reach the values to which only a few organisms are able to adapt [6].

The exceeding of hygienic limits, which are characteristic for every pollutant, may cause a serious health impairment of population and disturb the function and balance of eco-systems [5]. Therefore the polluted air represents a serious environmental and health problem, the immediate solution of which is topical and necessary.

At present the attention is paid to suspended dust particulate matter (PM) [7]. 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 [5]. The polycyclic aromatic hydrocarbons (PAHs) belong to highly toxic pollutants, which are adsorbed on suspended particulates. PAHs belong to the group polycyclic aromatic compounds, which also include azaarenes, oxaarenes, thiaarenes and their derivatives, e.g. alkyl- or nitro- derivatives. These compounds originate from PAH by the substitution of carbon in a benzene cycles by the atoms of N, O, or S and may be identified and quantified in the PAHs fractions, because their physico-chemical properties are similar [8].

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 [9, 10]. They get into air as unburned remains of fuels and as the products of pyrosynthesis during the combustion of aliphatic hydrocarbons in engines. The temperature of combustion engines is a significant factor affecting the production of PAHs, because the low temperature prevents their re-decomposition. They are also part of road surfaces from which they are released by abrasion. The occurrence of PAHs derivatives is connected also with the transformation processes in the atmosphere and many of them are of higher carcinogenity, mutagenity and teratogenity then the original noxys [11].

Diesel engines of trucks are the main sources of emissions of low-molecular PAHs with mostly three benzene cycles in a molecule. On the other hand the gas engines of cars are the main sources of high-molecular PAHs with four and five benzene cycles in a molecule [8].

The content of PAHs bound to the surface of particulates is affected by their distribution between gas and solid phases. The distribution of PAHs between both phases is strongly dependent on the tension of vapours of a particular PAH, temperature and partially on the composition and origin of particulates. The adsorption of PAHs on solid phase is supported by lower tension of vapours, lower temperature and higher content of organic carbon in particulates. Therefore light PAHs are emitted from combustion process almost solely into gas phase. Part of PAHs with three to four aromatic cycles is adsorbed on particulates and another part is present in the form of vapour in the air. Heavy PAHs are mainly bound to the surface of solid particulates through the adsorption and condensation of cooled emitted gases. Their ratio of gas phase rapidly decreases with the rising number of cycles [12]. This fact is documented by Venkataraman and Friedlander, who proved, that high-molecular benzo[a]pyrene and benzo[ghi]perlene are bound mainly on particulates, even in summer season [13]. It was proved by the analysis of exhaust fumes that the emissions of PAHs can be reduced by catalytic converters [8].

PAHs are bound from 56 - 89 % in the fraction of particulates smaller than 3.0 μm [14]. Regarding the gradual increase of particulates in the combustion process (diesel engines) and the atmosphere, PAHs become parts of bigger fractions of particulates, too. The content of organic carbon has for this reason the bimodal distribution with a peak at the particulates smaller than 1μm and the second one at the particulates of 2-10 μm in size. The PAHs bound in a coarse fraction become mostly the part of road dust after some time [15].

The particulates 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 particulates 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 [5, 6]. 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, 16, 17, 18].

Long-term exposure to increased concentration of particulates results in a shorter average life span [19], increased morbidity and mortality [20]. The risks of these troubles closely correlate with the concentrations of mainly $PM_{2.5}$ [17, 18, 19, 20].

PAHs are absorbed together with particulates in the lungs and digestive tract and metabolized through polyfunctional system of oxydases. Experimental studies show that PAHs are suspected of having carcinogenic, mutagenic and teratogenic effects. Many of them are classified into 2A category according to the IARC classification [21] and into B2 category according to the U.S. EPA classification [22] as probable human carcinogens.

Not all PAHs and their derivatives show the abovementioned negative effects. Therefore the toxicity of individual PAHs was analyzed in relation to their chemical structures. It is generally true that the carcinogenity and mutagenity of PAHs increase with the increasing number of aromatic rings in the first instance. The PAHs with four to six aromatic rings in a molecule show the highest carcinogenity and mutagenity [23]. The negative effects gradually decrease in case of PAHs with higher number of aromatic cycles. This effect is probably caused by a relatively big size of a molecule and therefore lower intercellular mobility, so there is a lower probability of their bond to an acceptor. Mutual position of aromatic cycles, the presence of various substitution groups or heterocyclic elements and a spatial orientation of substitution groups significantly affect the metabolism and consequent negative effects of PAHs in an exposed organism [24].

After entering the organism PAHs may be metabolized not only on multifunctional alcohols, but also on epoxides, arene oxides and free radicals. Solely alcoholic products are capable of a subsequent excretion from organism. Epoxides, arene oxides and free radicals are highly reactive, they can bind and create covalent adducts with vitally important macromolecules, such as cellular proteins and DNA, or they can cause the peroxidation of membrane lipids. Although it has been proved that the reactions of PAHs metabolites with proteins prevail over conjugative reactions with nucleic acids, the probability of a final step leading to malignant transformation of cells is relatively high [25].

The exposure to PAHs mixture is a complex problem. The biological relations of human exposure to complex mixture of PAHs depend on the interaction of high, low and non-carcinogenic PAHs. The interaction between non-carcinogenic and carcinogenic PAHs was intensively researched on experimental animals. Low-carcinogenic or non-carcinogenic PAHs, such as benzo[e]pyrene, benzo[ghi]perylene, fluoranthene and pyrene, highly increase the occurrence of tumours when interact with benzo[a]pyrene. On the other hand other low-carcinogenic or non-carcinogenic PAHs, such as benzo[a]fluoranthene, chrysene, perylene, or the mixture of anthracene, chrysene and pyrene, reduce the carcinogenic activity of benzo[a]pyrene [24].

Analogical effects were observed also for mutagenity. Hermann proves that some PAHs with two or three unsubstituted benzene cycles, which do not show mutagenity, significantly increase the mutagenity of benzo[a]pyrene. On the contrary some considerably mutagenic PAHs in mixture with benzo[a]pyrene reduce and often even eliminate its mutagenity, already in lower concentrations [26].

The risk of tumour formation in breathing apparatus increases if some metals, e.g. Pb, Cd, Hg, and As, are adsorbed on particulates together with PAHs [27].

As far as non-carcinogenic effects of PAHs are concerned, the adverse haematological and dermal effects were described in case of experimental animals, but they were not monitored among people. Despite a wide distribution of PAHs in the bodies of experimental animals it appears that PAHs affect mainly certain target organs, mainly the organs of a lymphatic system and a haematogenesis [24].

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

3 Applied Methods and Devices

Two locations with high intensity of traffic and a different morphology of surrounding environment were selected for the experiment in the city of Brno, Czech Republic.

Location 1 was at the crossroads with the average number of vehicles 3.60×10^4 per day. Four story houses are built in the surrounding area and form a canyon at some areas of the street. LECKEL MVS6 middle volume sampling device produced by Sven Leckel, Germany, was approximately 3 m far from the crossroad. The sampling point is shown in Figure 1.

Location 2 was a widely open environment with a lot

of greenery and the average number of vehicles $3.45x10^4$ per day. The built-up area with housing and outlets is opposite the area where air sampling devices of the same type as at location 1 were located. The sampling devices were located approximately 25 m from the crossroad from which they were separated by the zone of full-grown, 8 m high, deciduous and coniferous trees as it is seen in Figure 2.

Fig. 1 Sampling Point, Location 1


Fig. 2 Sampling Point, Location 2

LECKEL MVS6 devices are constructed so that a flow rate may be regulated with the help of a temperature-compensated slit. The sampling was carried out for 24 hours and the sampling capacity was approx. 55 m^3 of air. Filters for absorbing the $PM_{2.5}$ particulates were inserted between teflon canals into a sampling head, which is part of the device.

There were two devices at each sampling point for parallel measurements of $PM_{2.5}$ concentrations. A filter from quartz fibre was installed in one device and filter from nitrocellulose was installed in the second one. The weight of the filters was set with an accuracy of 0.05 mg before installing them into the head. The $PM_{2.5}$ particulates trapped on the nitrocellulose filter were analyzed for heavy metals. The $PM_{2.5}$ particulates trapped on the quartz filter were analyzed for determining the concentration of PAHs.

The air samples were collected all day in 7-day sampling campaigns in 5-week intervals. There were 42 samples collected at each location in six sampling campaigns to detect average concentrations of PAHs.

The exposed filters were taken out of the sampling device, wrapped up into the double layer of aluminium foil and distributed into the laboratory for analysis. First the filters 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 mass spectrometry (GS/MS) in the device of SHIMADZU company. The extracts were stored into vials, which were moved to auto sampler. Helium with the flow rate 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 mm thick film consisted of polar and non-polar phases in 1:19 ratio. The gas chromatograph 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 health risks were calculated in compliance with the current legal regulations of the Czech Republic [28], which observes the method proposed by the U.S. EPA [22, 29].

4 Outcomes and Discussion

The Table 1 shows the arithmetic averages of concentrations of individual PAHs at location 1 in individual sampling campaigns and at the same time the average concentration $\bar{c}$ for the whole monitored period. Analogical outcomes for location 2 can be seen in the Table 2. When the measured concentrations were below the limit of determination, half of the value of detection limit was used, i.e. 0.015 $ng.m^{-3}$ for PAHs.

The time dependence of concentrations of PAHs within the weekly sampling campaigns proved that Friday is the day of the highest air burden, likely due to the town residents leave for the countryside. The lowest air burden was monitored on Monday.

It results from the outcomes presented in table 1 and table 2 that the changes of average concentrations of individual PAHs in the sampling campaigns carried out during the monitored period are almost identical at both locations. It is also obvious that the average concentrations of individual PAHs at location 2 for the whole monitored period are approximately by 20 %

lower than at location 1, except for benzo[b]fluoranthene, dibenz[a,h]anthracene and coronene. The finding is in correlation with a lower intensity of traffic at location 2 (approx. by 4 %) and the morphology and character of landscape around the sampling points.

Table 1 Average concentrations of PAHs [ng.m^{-3}] at location 1

Pollutant	Average concentrations of PAHs [ng.m^{-3}] at location 1 in the sampling campaigns in 2005						
	April 4 - 10	May 23 - 29	Jun 27 - July 3	August 22 - 28	October 10 - 16	November 28 - December 4	Average $\bar{c}$ for the whole monitored period
naphtalene	4.110	2.159	2.330	3.842	0.618	0.932	2.332
acenaphthylene	0.234	0.074	0.038	0.067	0.039	0.271	0.121
acenaphthene	0.186	0.120	0.044	0.113	0.015	0.135	0.102
fluorene	0.465	0.477	0.350	0.513	0.067	0.345	0.370
fenanthrene	8.703	7.296	5.430	7.423	0.966	3.892	5.618
anthracene	0.443	0.388	0.343	0.471	0.107	0.429	0.364
fluoranthene	3.301	1.168	0.998	1.468	2.190	9.049	3.029
pyrene	5.195	1.518	1.229	2.864	3.912	10.031	4.125
benz[a]anthracene	0.652	0.397	0.093	0.173	0.969	5.258	1.257
chrysene	1.227	0.642	0.198	0.191	1.482	6.993	1.789
benzo[b]fluoranthene	0.501	0.537	0.073	0.091	0.421	4.136	0.960
benzo[k]fluoranthene	0.508	0.449	0.048	0.088	0.481	7.248	1.470
benzo[a]pyrene	0.836	0.621	0.101	0.200	0.410	8.321	1.748
indeno[1,2,3-cd]pyrene	0.855	0.379	0.561	0.184	0.524	3.877	1.063
dibenz[a,h]anthracene	0.680	0.266	0.015	0.015	0.065	0.175	0.203
benzo[ghi]perylene	1.395	0.903	0.192	0.215	0.334	2.530	0.928
coronene	1.658	1.253	0.313	0.527	0.411	2.258	1.070

Table 2 Average concentrations of PAHs [ng.m^{-3}] at location 2

Pollutant	Average concentrations of PAHs [ng.m^{-3}] at location 2 in the sampling campaigns in 2005						
	April 4 - 10	May 23 - 29	Jun 27 - July 3	August 22 - 28	October 10 - 16	November 28 - December 4	Average $\bar{c}$ for the whole monitored period
naphtalene	4.282	2.243	1.089	3.115	0.588	0.923	2.040
acenaphthylene	0.192	0.032	0.015	0.024	0.034	0.146	0.074
acenaphthene	0.184	0.103	0.054	0.056	0.015	0.111	0.087
fluorene	0.439	0.292	0.215	0.507	0.079	0.317	0.308
fenanthrene	8.922	4.595	3.255	6.745	0.697	3.689	4.651
anthracene	0.357	0.287	0.019	0.340	0.055	0.256	0.219
fluoranthene	3.061	0.726	0.560	0.902	1.587	8.321	2.526
pyrene	4.918	1.111	0.970	2.864	2.013	8.311	3.365
benz[a]anthracene	0.551	0.218	0.017	0.111	0.677	4.275	0.975
chrysene	1.157	0.371	0.016	0.059	1.077	6.458	1.523
benzo[b]fluoranthene	0.537	0.360	0.018	0.061	0.341	4.577	0.982
benzo[k]fluoranthene	0.952	0.260	0.024	0.048	0.354	5.780	1.236
benzo[a]pyrene	0.982	0.562	0.078	0.128	0.192	5.638	1.263
indeno[1,2,3-cd]pyrene	1.213	0.440	0.015	0.189	0.223	3.844	0.987
dibenz[a,h]anthracene	0.655	0.321	0.015	0.027	0.036	0.242	0.216
benzo[ghi]perylene	1.277	0.694	0.015	0.040	0.151	2.469	0.774
coronene	2.629	0.683	0.181	0.138	0.283	2.843	1.126

The lowest concentrations of PAHs were monitored in summer months, which may be explained by more favourable dispersion and meteorological conditions (stronger wind, solar activity) and a lower intensity of traffic due to holidays.

On the contrary relatively high concentrations of PAHs in the air 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 air contamination caused by household furnaces. The proof of that is the concentration ratio of benzo[a]pyrene and coronene $p > 1.00$ in this period, while during the other sampling campaigns the ratio at both monitored locations was $p < 1.00$. The higher ratio $p_1 = 3.69$ at location 1 compared to $p_2 = 1.98$ at location 2 is evidently caused by apartment houses built in the area, which are partially heated by solid fuels (coal). The increased production of PAHs contributing to air burden 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 category were solely selected for the risk assessment of inhalation exposure of citizens. No PAH is classified in A and B 1 categories yet [22, 30]. 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 [28]. The values of inhalation slope factors SF_i for the monitored PAHs were adopted from the materials of U.S. EPA [30, 32] and were published by experts also in other papers [31]. The inhalation factors of directives for individual PAHs are presented in Table 3 for location 1 and Table 4 for location 2 together with the calculated values of $LADD_A$ for adults and the corresponding increase of risk $ECLR_A$ including the analogical values of $LADD_C$ and $ELCR_C$ for children 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 all monitored period of time, subtracted from Table 1 or 2. 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 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, the value of 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 social risk, i.e. for the group of more than 100 threatened people, is considered to be $ELCR \leq 10^{-6}$, which represents the increase of cancer by one case in the group of million of people [33]. Therefore it may be stated on the basis of the calculated values of $ELCR_A$ a $ELCR_C$ presented in table 3 and table 4 that, with the exception of benzo[a]pyrene, the risk resulting from the exposure to air contaminated by individual PAHs is acceptable both for the population of adults and children to the age of six living near the roads. The concentration of benzo[a]pyrene at both locations represent certain risk for adults, which is still tolerable, while the risk for children is already significant.

If the solely additive effects of individual carcinogens of PAHs are considered while the synergic effects are neglected, then 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):

$$ELCR_S = \sum_{i=1}^{n} ELCR_i \quad (3)$$

where $ELCR_i$ is the excess lifetime cancer risk due to i-PAH; n is the number of PAHs belonging to B2 category, while $i \in \langle 1; n \rangle \wedge i \in N$ 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 table 3 and table 4 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 $ELCR_{SA1} \approx 3.3 \times 10^{-6}$ for adults and $ELCR_{SC1} \approx 9.4 \times 10^{-6}$ for children to the age of six at location 1. The conditions at location 2 are rather more favourable especially due to the morphology of landscape and the values are $ELCR_{SA2} \approx 2.6 \times 10^{-6}$ and $ELCR_{SC2} \approx 7.3 \times 10^{-6}$. It is clear from the above mentioned values of $ELCR_S$ that the summary genotoxic risk especially for children to the age of six almost reaches the level of unacceptability.

Table 3 Average concentrations of monitored PAHs for the assessed period of time, SF_i values, calculated $LADD_A$, $LADD_C$ values and the excess lifetime cancer risk over the general average $ELCR_A$ for adults and $ELCR_C$ for children at location 1

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[b]fluoranthene	9.598x10^{-7}	6.1x10^{-1}	2.292x10^{-7}	6.442x10^{-7}	1.398x10^{-7}	3.930x10^{-7}
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	2.027x10^{-7}	6.1	4.840x10^{-8}	1.361x10^{-7}	2.952x10^{-7}	8.302x10^{-7}

Table 4 Average concentrations of monitored PAHs for the assessed period of time, SF_i values, calculated $LADD_A$, $LADD_C$ values and the excess lifetime cancer risk over the general average $ELCR_A$ for adults and $ELCR_C$ for children at location 2

Pollutant	$\bar{c}$ [mg.m^{-3}]	SF_i [kg.day.mg^{-1}]	$LADD_A$ [mg.kg^{-1}.day^{-1}]	$LADD_C$ [mg.kg^{-1}.day^{-1}]	$ELCR_A$	$ELCR_C$
benz[a]anthracene	9.748x10^{-7}	6.1x10^{-1}	2.328x10^{-7}	6.543x10^{-7}	1.420x10^{-7}	3.991x10^{-7}
chrysene	1.523x10^{-6}	6.1x10^{-3}	3.636x10^{-7}	1.022x10^{-6}	2.218x10^{-9}	6.234x10^{-9}
benzo[b]fluoranthene	9.823.10^{-7}	6.1x10^{-1}	2.345x10^{-7}	6.594x10^{-7}	1.430x10^{-7}	4.022x10^{-7}
benzo[k]fluoranthene	1.236x10^{-6}	6.1x10^{-2}	2.951x10^{-7}	8.296x10^{-7}	1.800x10^{-8}	5.061x10^{-8}
benzo[a]pyrene	1.263x10^{-6}	6.1	3.016x10^{-7}	8.478x10^{-7}	1.840x10^{-6}	5.172x10^{-6}
indeno[1,2,3-cd]pyrene	9.873x10^{-7}	6.1x10^{-1}	2.357x10^{-7}	6.627x10^{-7}	1.438x10^{-7}	4.042x10^{-7}
dibenz[a,h]anthracene	2.160x10^{-7}	6.1	5.157x10^{-8}	1.450x10^{-7}	3.146x10^{-7}	8.845x10^{-7}

Assuming the accumulation of genotoxic risk $ELCR_{SC}$ for the population of children to the age of six and $ELCR_{SA}$ for adults the increase of excess lifetime cancer risk $ELCR_{SS}$ is given by the formula (4):

$$ELCR_{SS} = ELCR_{SC} + ELCR_{SA} \quad (4)$$

$ELCR_{SS1} \approx 1.3x10^{-5}$ for location 1 and $ELCR_{SS2} \approx 9.9x10^{-6}$ for location 2. The assessed values indicate that the summary social risk already exceeded the tolerance limit given by the interval $ELCR_{SS} \in (10^{-6}; 10^{-5}\rangle$ at location 1 and it almost reaches this limit at location 2.

Similar outcomes regarding the contribution of individual PAHs to total toxicity have been achieved by calculating the toxicity equivalent quotient (TEQ) when knowing the factors of equivalent toxicity and the average concentration of the monitored PAHs. The relationship is expressed by the following formula (5):

$$TEQ = \bar{c} \times TEF \quad (5)$$

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 and LaGoy [34] were applied in the calculation, because they show higher values for benzo[k]fluoranthene and dibenz[a,h]anthracene, chrysene in relation to the analogous data of U.S. EPA used by Peters et al. [35]. The TES for the other PAHs of B2 category are the same in both author´s teams. Provided that individual PAHs have additive health effects the TEQ total toxicity equivalent may be calculated according to the following formula (6):

$$TEQ_S = \sum_{i=1}^{q} TEQ_i \quad (6)$$

where TEQ_i represent the toxicity equivalency factors of individual PAHs, $i \in \langle 1; q\rangle \wedge i \in N$ and q represent the number of PAHs with the probable human carcinogenic effects, in this case included into B2 category according to U.S. EPA classification.

The acquired outcomes are shown in the Table 5. 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 (see the values of $ELCR_A$ and $ELCR_C$ in tables 3 and 4). It is necessary to accept that a number of uncertainties exist in the above presented process of risk assessment. However, it can globally be assumed that the quantified values of risks are rather overestimated. It is necessary to mention mainly the following significant uncertainties:

Table 5 The average concentrations of monitored PAHs [mg.m^{-3}] for the assessed period of time, TEF values, calculated TEQ values for individual PAHs, and toxicity equivalent quotient summary TEQ_S

Pollutant	TEF	Location 1			Location 2		
		$\bar{c}$ [ng.m^{-3}]	TEQ [ng.m^{-3}]	TEQ_S [ng.m^{-3}]	$\bar{c}$ [ng.m^{-3}]	TEQ [ng.m^{-3}]	TEQ_S [ng.m^{-3}]
benz[a]anthracene	0.100	1.257	$1.257x10^{-1}$		$9.748x10^{-1}$	$9.748x10^{-2}$	
chrysene	0.010	1.789	$1.789x10^{-2}$		1.523	$1.523x10^{-2}$	
benzo[b]fluoranthene	0.100	$9.598x10^{-1}$	$9.598x10^{-2}$		$9.823x10^{-1}$	$9.823x10^{-2}$	
benzo[k]fluoranthene	0.100	1.470	$1.470x10^{-1}$	**3.255**	1.236	$1.236x10^{-1}$	**2.776**
benzo[a]pyrene	1.000	1.748	1.748		1.263	1.263	
indeno[1,2,3-cd]pyrene	0.100	1.063	$1.063x10^{-1}$		$9.873x10^{-1}$	$9.873x10^{-2}$	
dibenz[a,h]anthracene	5.000*	$2.027x10^{-1}$	1.014		$2.160x10^{-1}$	1.080	

* in low concentrations

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 traffic supports the progressive time dependence of average concentrations. On the other hand the implementation of adequate countermeasures and a technical progress support the 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 [32, 36] mentioned in literature. The highest discovered values of SF_i [30, 31, 32] 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 1.5 m above the road (not on floor levels of buildings) 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 (immunosuppression, allergic persons, those with heart conditions) for whom even higher risk may be expected than in case of children to the age of six. At the same time the inhalation of PAHs from other sources was not considered, e.g. smoking, food (smoked, fried and grilled meat) and contaminated surface and, possibly, drinking water. The oral and dermal intakes of PAHs have not been considered, either.

e) It is also necessary to mention uncertainties connected with possible synergic, additive and inhibition effects of individual PAHs. The additive principle of the PAHs mixture effect on the population health risk was applied in the contribution, which is in compliance with the findings of the study. It proves rather additive effects of individual components on the creation of DNA adducts [37]. The subject issue still has many ambiguities as other authors furnish evidence of mutual inhibition [38, 39] or, on the contrary, synergic effects [40, 41]. It seems that the real character effect will rather be influenced by the composition of PAHs mixture and partially also by the concentration ratios of components being present [24, 26].

f) Other uncertainties of risk assessment are connected with possible interactions of individual PAHs with other contaminants, especially metals, with which they are $PM_{2.5}$ particulates-bound [1]. Synergic, inhibition and additive effects of these mixtures depend on the composition and concentration of present components [27]. There also may be a significant effect of mutual interactions of PAHs and their mixtures with the products of their transformation processes in the atmosphere. It is a wide range of hydroxy-, oxo-, nitro-, sulpho- and other derivatives of PAHs, the origin of which is the result of reaction of PAHs with O_3, O_2, SO_2, NO_x, etc. [11, 42]. There are even more uncertainties in the kinetics, mechanisms of actions and impacts of such complex mixtures, both in gaseous and aerosols forms, on living organisms. These uncertainties have mostly not been explained even for individual contaminants yet. The acceptance of these factors when assessing the genotoxic risk would require a number of autonomous pieces of work for each particular

mixture. Therefore these facts were not incorporated into the process of our risk assessment.

5 Conclusion

It can be stated from the analysis of risk resulting from the exposure of inhabitants to the air polluted by $PM_{2.5}$ particulates, which are contaminated by the adsorbed PAHs, that the most serious problems are in case of contamination caused by benzo[a]pyrene and then by dibenz[a,h]anthracene. The contributions of the other PAHs with probable carcinogenic effects to the total risk for people are irrelevant or negligible. This fact was paralelly proved by using the toxicity equivalent quotient. The findings proved that the most threatened groups of inhabitants living in cities near roads with the increased intensity of traffic are children to the age of six, people with the weakened immune system, asthmatics and those with heart conditions.

The total risk exceeds the current standards by more than three times for the population of adults and nine times for children. The conditions are slightly more favourable at an open location with the same intensity of traffic. The summary excess lifetime cancer risk already exceeded the tolerance limit in the densely built-up location ($ELCR_{SS} \approx 1.3x10^{-5}$) and it almost reaches this limit at the open location ($ELCR_{SS} \approx 9.9x10^{-6}$).

Despite the fact that the assessed values of risks are connected with a number of uncertainties and are probably overestimated, it is preferable, especially in relation to a dynamically developing traffic, 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, no entry of cars to city centres, promotion and education of inhabitants in the abovementioned areas, and a principally more effective use of preventive function in territorial planning.

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