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Environmental exposure of small children to polycyclic aromatic hydrocarbons

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Abstract ***Objectives***: The aim of the study was to assess the intake (by various routes of exposure) of polycyclic aromatic hydrocarbons (PAH) by children living in a Czech city, and its effect on excretion of 1-hydroxypyrene (1-OHP) in summer and winter periods. ***Methods***: Four groups of children (3–6 years old) were chosen: (1) two groups from a kindergarten situated in the city center with a higher traffic density (''polluted'' area); (2) two groups from a kindergarten situated in a green zone of the same city (''non-polluted'' area). Food consumption was recorded in all children and PAH intake from foodstuffs was estimated. Ambient air samples were collected from the playground and inside the kindergartens. Soil samples were collected too. Morning and evening urine samples were collected during sampling days. ***Results***: In both seasons, the mean outdoor total PAH concentration (sum of 12 individual PAH) in the ''polluted'' area was approximately three-times higher than that in the ''non-polluted'' area. Indoor concentration in the ''polluted'' area was more than six-times higher than that in the ''non-polluted'' area in summer, and almost three-times higher in winter. The same trend was observed for pyrene and for the sum of carcinogenic PAH. The contribution to the total pyrene absorbed dose from food consumption was much more important than that from inhalation and from ingestion of soil dust. Significantly higher urinary concentrations of 1-OHP (evening samples) were found in children from the ''polluted'' kindergarten in both seasons. The number of significant relationships between 1-OHP and pyrene absorbed dose was weak. ***Conclusions***: Food seems to be the main source of total pyrene and total PAH intake in small children, even under relatively higher air PAH exposure in the city. Estimated pyrene ingestion from soil had a negligible contribution to the total pyrene absorbed dose. Urinary 1-OHP seems to be an uncertain (non-sensitive) marker of the environmental inhalation exposure to pyrene (PAH) if the pollution of air by pyrene (PAH) is not excessive and the pyrene (PAH) dose by this route is much less than by ingestion. Usefulness of the urinary 1-OHP as an indicator of overall environmental exposure to PAH needs further investigation.

Key words Polycyclic aromatic hydrocarbons · Children · Environmental exposure · 1-Hydroxypyrene

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Introduction

Polycyclic aromatic hydrocarbons (PAH) are now well-established as human health hazards. A number of these are known to have carcinogenic and mutagenic properties. Human exposure to PAH occurs principally by (1) direct inhalation of polluted air and tobacco smoke, (2) dietary intake of smoked and other foodstuffs and polluted water, and (3) dermal contact with soot, tars, and polluted soils (International Agency for Research on Cancer (IARC) 1983).

The assessment of environmental exposure to PAH requires the measurement of levels present in each

pathway of possible contact. The biological monitoring of PAH represents the best way to assess the internal dose of PAH. Among the many PAH, pyrene is emitted in fairly large amounts. Pyrene is contained not only in exhaust from heaters, but also in automotive exhaust, cigarette smoke, and soil, as well as in food (Jongeneelen, 1994). Pyrene is metabolized and excreted as 1-hydroxypyrene (1-OHP) in urine in an amount dependent on the dose of pyrene. It was suggested that 1-OHP is a useful marker in exposure evaluation to pyrene, thus to PAH in general, as well as in assessment of their health effects. Indeed, several industrial hygiene studies have shown that 1-OHP in urine is a valid biomarker for occupational exposure to PAH (Buchet et al. 1992; Jongeneelen et al. 1990; Zhao et al. 1990).

Currently, urinary 1-OHP as well as selected hydroxylated metabolites of phenanthrene are tested as a biomarkers for the assessment of low-level environmental exposure of general population (Gündel et al. 1996; Seiwert et al. 1998; Viau et al. 1995). Our latest reports (Gilbert and Viau 1997; Vyskocil et al. 1997, 2000) and reports by Kanoh et al. (1993), Jongeneelen (1994) and Zhao et al. (1992) suggest that levels of 1-OHP reflect the levels of environmental PAH exposure from different sources such as air, food and the environment of the home life. However, in our last studies (Vyskocil et al. 1997, 2000), it was shown that factors other than air pollution contribute more substantially to overall exposure to PAH, and it was speculated that diet could be one of the most important factors.

Up to now, only one study (Van Wijnen et al. 1996) followed up the intake of PAH in small children, who could be more exposed to PAH than adults. It is known that small children spend much of their time outdoors in close contact with soil. So, in children, dermal contact with soil polluted by PAH, ingestion of soil and inhalation of soil particles could be further routes for PAH intake, that are not so important in adults. PAH from vehicle exhausts and road runoff have been suggested as major sources of contamination of the roadside environment (Pathirana et al. 1994). This could present an important source of PAH exposure for children playing in the vicinity of roads.

The objective of our investigation was to assess the multi-pathway intake of PAH by small children living in a city. More specifically, it was designed to (1) determine concentrations of individual PAH in air and soil of a highly and a less polluted area of the city of Hradec Králové (Czech Republic) where they are exposed; (2) estimate intake of PAH from the diet on the basis of a detailed questionnaire; (3) identify among the multiple sources (air, soil, diet) relative magnitudes of exposure to PAH in children; (4) determine the effect of the PAH intake on 1-OHP concentrations in urine.

Methods

Four groups of children (3–6 years old) living in the city of Hradec Králové were chosen. Two groups ($n = 15$ in summer and $n = 10$ in winter) were chosen from a kindergarten (identified as a "polluted area") situated near an important road (the central point of the playground was situated at a distance of 22 m from the road) with high traffic density (about 18,000 cars/day). Two groups ($n = 15$ in summer and $n = 17$ in winter) were chosen from a kindergarten (identified as a "non-polluted area") situated in a residential zone with low local traffic (about 80 cars/day). All children lived in the vicinity of the corresponding kindergartens, in houses situated on small streets with local circulation only. Informed consent was obtained from the parents of children prior to their inclusion in the study, and the Ethics Committee of Charles University Medical Faculty in Hradec Králové approved the work.

One day before, and during sampling periods, detailed food consumption was recorded in all subjects by parents and teachers. During the sampling period, the time that the children spent outside and inside the kindergarten was recorded. The parents answered several questions concerning the other possible sources of PAH exposure in children (smoking habits of parents in the home, heating of houses, cooking devices in houses, frequency of grilled, baked, smoked and roasted food and other possible sources of PAH exposure in the residential area).

Daily pyrene intake from foodstuffs was estimated using a method described in our previous publication (Chénier and Viau 1997). In brief, using literature data, we drew up a table where foods were divided into four classes according to their pyrene concentrations: low (< 1 μg/kg, taken as 0.5 μg/kg), medium (1–5 μg/kg, taken as 3 μg/kg), high (5–25 μg/kg), taken as 15 μg/kg) and very high (> 25 μg/kg, taken as 75 μg/kg). A description of every meal was recorded in a diary, and the pyrene content in all foods was calculated.

Ambient air samples were collected in the playgrounds (the sampler was placed in the center of the playground) and inside the kindergartens (the sampler was placed in the central corridor) for three consecutive days in summer 1997 (three samples per site) and winter 1998 (three samples per site). The collection of ambient air by stationary air samplers (duration of sampling = 8–14 h; air flow $= 20$ m^3/h) and its analysis for PAH were performed by the EPA TO-13 method (Environmental Protection Agency (EPA) 1988). The air samples were collected on quartz-fiber filters and polyurethane foams (PUF). Quartz-fiber filters were extracted with methylene chloride, and PUF plugs with 10% diethylether in hexane in a Soxhlet apparatus. The extracts were dried by being passed through the column containing anhydrous sodium sulphate, and were concentrated in a rotary evaporator. The concentrated extracts were cleaned by being passed through a silica gel column. Gradient high-performance liquid chromatography (HPLC) with fluorescence detection was used for qualitative and quantitative determination of individual PAH. In each sample the concentration of 12 selected PAH compounds (phenanthrene, anthracene, fluoranthene, pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, benzo[g,h,i]perylene, and indeno[1,2,3-cd]pyrene) was determined. Total PAH concentration was calculated as the sum of the concentrations of the 12 PAH.

Atmospheric conditions during summer collections were partially sunny. The temperatures ranged from 14.7 to 22.1 °C and the wind blew at 6–9 m/s. During winter collections we observed partially sunny conditions as well, with temperatures from –0.3 to –4.2 °C and with the wind from 6 to 14 m/s.

The urine samples were collected during the 3 days that the air samples were collected, in the morning and in the evening (before the child went to sleep). Urine samples were analyzed for 1-OHP by a gradient HPLC method with fluorescence detection, that was adapted in our laboratory (Bouchard et al. 1994). Urine creatinine was used to standardize the results.

Soil samples were taken from the upper soil layer (first 20 cm). Each representative sample of soil was prepared from a mixture of samples collected from five different parts of the playgrounds and sandpits in each kindergarten. In winter the children did not play in the sandpits. For this reason we did not collect the sand samples then. All soil samples were analyzed for PAH by a modified HPLC method according to Haak and Ham (1994). The samples were

extracted for 4 h in an ultrasonic bath with the mixture of hexane and dichloromethane (1:1). The extracts were dried (just to the dry condition) in a rotary evaporator and dissolved in the mobile phase. Gradient HPLC with fluorescence detection was used for qualitative and quantitative determination of individual PAH. In each sample the concentration of the 12 selected PAH compounds was determined.

The Kolmogorov-Smirnov normality test was applied to check the normality of the distributions. Correlation coefficients were used for the determination of relationships between urinary 1-OHP and absorbed doses of pyrene. Paired and unpaired *t*-tests, non-parametric Mann-Whitney unpaired test and Wilcoxon matched-pairs test were used to test the differences between the groups. The level of significance was taken as $P < 0.05$.

To assess the influence of independent variables (smokers at home, frequent consumption of grilled, baked, smoked and roasted food, local sources of outdoor exposure to PAH in residential area, pyrene intake from foodstuffs 1 day before and during the 3 sampling days, and pyrene intake from air during the 3 sampling days) on the excretion of 1-OHP, we applied the method of selection of the best regression equation. This method is based on generating all the subsets of potential independent variables and comparing corresponding coefficients of determination. All the coefficients of determination of all the sizes of subsets were calculated and the best ones stored. Then we could decide which variables were more important in the sense of their contribution to the coefficient of determination, and which were negligible from this point of view. The purpose was to select a reasonably small subset of potential independent variables, which could yield an acceptable coefficient of determination. A more detailed description of this approach can be found in the publication by Draper and Smith (1966).

Results

Air concentrations of pyrene, the sum of six carcinogenic PAH and the sum of 12 PAH compounds measured indoors and outdoors in two kindergartens is presented in Table 1. In both seasons, the mean outdoor total PAH concentration (sum of 12 individual PAH) in the ''polluted'' area was approximately three-times higher than that in the ''non-polluted'' area. Indoor concentration in the ''polluted'' area was more than six-times higher than that in the ''non-polluted'' area in summer, and almost three-times higher in winter. Similar tendencies were observed for pyrene and for the sum of six selected carcinogenic PAH.

Table 2 shows the PAH concentrations found in the surface soil sampled from the playgrounds and sandpits in both kindergartens. As can be seen from our summer results, the sum of 12 PAH and the sum of six carcinogenic PAH collected from the ''polluted'' kindergarten were roughly twice as high and more than four-times higher, respectively, than that in the ''non-polluted'' one. The concentrations of total PAH and carcinogenic PAH in sand samples were several times lower when compared with earth samples. During winter collection we found lower total PAH, selected carcinogenic PAH and pyrene concentration in the earth sam-

Table 1 Concentrations of polycyclic aromatic hydrocarbons (*PAH*) in air (arithmetic mean ± SD of three samples)

Place (season)	Total PAH[a] (ng/m^3)		Carcinogenic PAH[b] (ng/m^3)		Pyrene (ng/m^3)	
	Outdoor	Indoor	Outdoor	Indoor	Outdoor	Indoor
Kindergarten in polluted area (summer)	4.70 ± 0.36	18.17 ± 1.96	1.03 ± 0.15	3.40 ± 0.46	0.43 ± 0.06	2.53 ± 0.23
Kindergarten in non-polluted area (summer)	1.40 ± 0.26	2.73 ± 0.45	0.27 ± 0.12	0.37 ± 0.15	0.27 ± 0.06	0.47 ± 0.15
Kindergarten in polluted area (winter)	12.70 ± 3.70	5.20 ± 0.56	3.87 ± 1.10	1.47 ± 0.31	1.50 ± 1.13	0.63 ± 0.06
Kindergarten in non-polluted area (winter)	4.40 ± 1.35	2.07 ± 0.45	0.90 ± 0.50	0.45 ± 0.07	0.77 ± 0.21	0.33 ± 0.06

[a] Total of 12 PAH: phenanthrene, anthracene, fluoranthene, pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]-fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, benzo[g,h,i]-perylene, indeno[1,2,3-cd]pyrene

[b] Sum of six carcinogenic PAH: benz[a]anthracene, benzo[b]-fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, dibenz[a,h]-anthracene, indeno[1,2,3-cd]pyrene

Table 2 Polycyclic aromatic hydrocarbons (*PAH*) concentrations in soil and daily absorbed doses of pyrene. Analysis of representative samples of soil prepared from mixture of samples collected from five different places in each kindergarten

Place (season)	Total PAH[a]	Carcinogen PAH[b]		Pyrene	
	(ng/g)	(ng/g)	ng/g	ng/day[c]	ng/day[d]
Kindergarten in polluted area (summer)	122.0 (Earth) 29.9 (Sand)	53.1 (Earth) 5.1 (Sand)	4.6 (Earth) 0.6 (Sand)	0.210	0.026–0.210
Kindergarten in non-polluted area (summer)	73.8 (Earth) 14.1 (Sand)	13.0 (Earth) 0.5 (Sand)	6.4 (Earth) 0.5 (Sand)	0.279	0.035–0.279
Kindergarten in polluted area (winter)	109.1 (Earth)	42.7 (Earth)	3.7 (Earth)	0.292	0.037–0.292
Kindergarten in non-polluted area (winter)	38.9 (Earth)	4.1 (Earth)	3.9 (Earth)	0.311	0.039–0.311

[a] Total of 12 PAH: phenanthrene, anthracene, fluoranthene, pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]-fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, benzo[g,h,i]-perylene, indeno[1,2,3-cd]pyrene

[b] Sum of six carcinogenic PAH: benz[a]anthracene, benzo[b]-fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, dibenz[a,h]-anthracene, indeno[1,2,3-cd]pyrene

[c] Daily ingestion intake of pyrene from soil (assumed soil ingestion = 80 mg /day) (Paustenbach 1989). Assumed intake of 50% of the earth and 50% of the sand (in summer)

[d] Daily doses of ''soil'' pyrene absorbed from gastrointestinal tract (ingestion bioavailability of pyrene 12.5–100%), (CanTox 1991; Bouchard and Viau 1997)

ples from both kindergartens (when compared with the summer samples).

Absorbed mean daily doses of pyrene by inhalation and ingestion are presented in Tables 2, 3 and 4. If we assume that the children's intake of soil by ingestion is 80 mg/day (which is a conservative estimate, as the best estimate for children aged 2–6 years is about 40–50 mg/day) (Paustenbach, 1989) and the ingestion bioavailability of pyrene ranged from 12.5% (CanTox 1991) to 100% (Bouchard and Viau 1997), the daily calculated absorbed doses of pyrene from the soil varied between 0.026 and 0.311 ng. These doses corresponded roughly to 0.010% (bioavailability 12.5%) and 0.022% (bioavailability 100%) of the total absorbed doses, and were negligible in comparison with the doses from food ingestion and inhalation. Therefore, they were not taken into consideration in further calculations.

Because all children lived in the vicinity of the kindergartens, for calculation of the pyrene exposure dose by inhalation it was assumed that the air pyrene concentrations during their stay at home were similar to the outdoor pyrene concentrations of the corresponding kindergarten. The indoors home concentrations of pyrene were assumed to be the same as the outdoor concentrations, because only families of non-smokers were selected for our study. No other typical sources (wood/coal heating and cooking, high-density traffic, high frequency of grilled and roasted meals) of indoor pollution by PAH were identified in these families. The children spent on average 4 h (summer) or 2 h (winter) outside during their stay in kindergarten. The pulmonary ventilation of ten children during different activities was measured using pulse frequency and spiro-ergometry in a parallel study (manuscript in preparation). The pulmonary ventilation was on average 10.1 l/min during sleep, 12.3 l/min during indoor activity and 13.6 l/min during outdoor activity. Assuming an inhalation bioavailability of pyrene of 84.2% reported by CanTox (1991), the absorbed mean daily doses of pyrene by inhalation were only 0.4–15% and 0.3–8 % of the total absorbed dose in the "polluted" and "non-polluted" kindergarten, respectively. Food represented the main source of exposure to pyrene: 85–99.6% and 92–99.7% in the "polluted" and "non-polluted" kindergarten, respectively (Tables 3 and 4).

When we used the summer data from all 3 days of observation for statistical comparison of both kindergartens (Table 3) we found significantly higher intake of pyrene by inhalation ($P < 0.001$) and ingestion ($P < 0.05$) in the "polluted" kindergarten. The winter comparison of kindergartens (Table 4) revealed a significantly higher intake of pyrene by inhalation ($P < 0.001$) in the "polluted" kindergarten too.

Table 3 Absorbed mean daily doses of pyrene (summer)

Place	Day of sampling	Food consumption[a]			Air inhalation[b]		Total dose ng/day
		Number of samples	ng/day	% of total dose	ng/day	% of total dose	
			Geometric mean (range)		Geometric mean (range)		
Kindergarten in polluted area	0	15	231 (115–838)	–	–	–	–
			1,848 (920–6,704)	–			
	1	15	235 (124–807)	96	9.2 (7.5–12.5)	4	244
			1,880 (992–6,456)	99.5		0.5	1,857
	2	15	271 (145–954)	96	9.5 (6.9–11.2)	4	281
			2168 (1,160–7,632)	99.6		0.4	2,178
	3	15	219 (145–421)	95	12.4 (10.1–14.9)	5	231
			1,752 (1,160–3,368)	99.3		0.7	1,764
	1–3	45	241 (124–954)[c]	96	10.3 (6.9–14.9)[d,e]	4	251
			1,928 (992–7,632)[c]	99.5		0.5	1,938
Kindergarten in non-polluted area	0	15	195 (124–349)	–	–	–	–
			1,560 (992–2,792)	–			
	1	15	203 (135–341)	98	4.2 (3.5–4.3)	2	207
			1,624 (1,080–2,728)	99.7		0.3	1,628
	2	15	193 (154–255)	97	4.9 (4.2–5.4)	3	198
			1,544 (1,232–2,040)	99.7		0.3	1,549
	3	15	170 (106–472)	98	3.5 (2.8–4.6)	2	174
			1,360 (848–3,776)	99.7		0.3	1,364
	1–3	45	188 (106–472)	98	4.2 (2.8–5.4)[f]	2	192
			1,504 (848–3,776)	99.7		0.3	1,508

[a] Daily doses of pyrene absorbed from gastrointestinal tract; ingestion bioavailability of pyrene ranged (probably due to particulate as well as gaseous form of pyrene) from 12.5% (first line) to 100% (second line), (CanTox 1991; Bouchard and Viau 1997)

[b] Daily doses of pyrene absorbed from respiratory tract (inhalation bioavailability of pyrene = 84.2%) (CanTox 1991)

[c] $P < 0.05$ (vs food consumption days 1–3 in the non-polluted kindergarten)

[d] $P < 0.001$ (vs air inhalation days 1–3 in the non-polluted kindergarten)

[e] $P < 0.001$ (vs air inhalation days 1–3 in the winter polluted kindergarten, Table 4)

[f] $P < 0.001$ (vs air inhalation days 1–3 in the winter non-polluted kindergarten, Table 4)

Table 4 Absorbed mean daily doses of pyrene (winter) (*SN* small number of samples)

Place	Day of sampling	Food consumption[a]			Air inhalation[b]		Total dose ng/day
		Number of samples	ng/day	% of total dose	ng/day	% of total dose	
			Geometric mean (range)		Geometric mean (range)		
Kindergarten in polluted area	0	3	SN	SN	–	–	–
	1	10	235 (146–570)	96	10.8(10.5–11.4)	4	246
			1880 (1168–4560)	99.4		0.6	1891
	2	10	218 (163–437)	95	11.6 (10.1–12.4)	5	230
			1744 (1304–3496)	99.3		0.7	1756
	3	10	169 (119–231)	85	29.5 (11.1–35.8)	15	199
			1352 (952–1 848)	97.9		2.1	1382
	1–3	30	205 (146–570)	93	15.5 (10.1–35.8)[c]	7	221
			1640 (1168–4560)	99.1		0.9	1656
Kindergarten in non-polluted area	0	16	169 (83–284)	–	–	–	–
			1352 (664–2272)	–			
	1	17	155 (85–221)	92	12.8 (10.4–14.4)	8	168
			1240 (680–1768)	99		1	1253
	2	17	184 (103–408)	95	9.0 (8.0–10.1)	5	193
			1472 (824–3264)	99.4		0.6	1481
	3	17	193 (122–299)	96	8.0 (7.0–11.5)	4	201
			1544 (976–2392)	99.5		0.5	1552
	1–3	51	177 (83–408)	95	9.7 (7.0–14.4)	5	187
			1416 (664–3264)	99.3		0.7	1426

[a] Daily doses of pyrene absorbed from gastrointestinal tract; ingestion bioavailability of pyrene ranged (probably due to particulate as well as gaseous form of pyrene) from 12.5% (first line) to 100% (second line), (CanTox 1991; Bouchard and Viau 1997)

[b] Daily doses of pyrene absorbed from respiratory tract (inhalation bioavailability of pyrene = 84.2%), (CanTox 1991)

[c] $P < 0.001$ (vs air inhalation days 1–3 in the non-polluted kindergarten)

When we compared the winter and summer observations, in winter we found significantly higher inhalation intakes of pyrene ($P < 0.001$) in both kindergartens. No significant differences were found between summer and winter intakes of pyrene by ingestion (Tables 3 and 4).

The comparison of 1-OHP concentrations between morning and evening urine samples revealed significant differences only in the ''non-polluted'' kindergarten during summer collection. These differences were observed on the 2nd ($P < 0.05$) and the 3rd ($P < 0.05$) day of collection (Table 5).

In both seasons, after comparing the data from all 3 days of observation between kindergartens, we found significantly higher concentrations of 1-OHP ($P < 0.05$) only in the evening samples from children in the ''polluted'' kindergarten. On the other hand, no significant differences were found when the summer and winter 1-OHP data at the same kindergarten were compared (Tables 5 and 6).

The relationships between urinary 1-OHP and absorbed doses of pyrene were tested for each day and for each kindergarten in the following groups: (1) absorbed pyrene doses by inhalation versus 1-OHP in urine samples collected in the evening of the air sampling day or in the next morning; (2) absorbed pyrene doses by ingestion versus 1-OHP in urine samples collected as described above; (3) total absorbed doses versus 1-OHP in urine samples collected as described above.

From 20 comparisons (Table 7) we found only two significant correlation ($P < 0.05$) between the daily dose of pyrene from inhalation and the concentration of 1-OHP in the evening samples of urine. Correlation coefficients equaled 0.6041 and 0.6552.

From 24 comparisons (Table 7) we found six significant correlation ($P < 0.05$–0.001) between the daily dose of pyrene from food consumption and the concentration of 1-OHP in the samples of urine. Correlation coefficients varied from 0.5396 to 0.9108.

From 20 comparisons (Table 7) we found six significant correlation ($P < 0.05$–0.001) between the daily total pyrene dose and the concentration of 1-OHP in the samples of urine. Correlation coefficients varied from 0.5383 to 0.9097.

As a result of the method of generating all possible subsets of independent variables (smokers at home, frequent consumption of grilled, baked, smoked and roasted food, local sources of outdoor exposure to PAH in residential area, pyrene intake from foodstuffs and pyrene intake from the air), almost all the coefficients of determination were low. There was one exception only, which occurred when we assumed the 1-OHP from the morning of the 3rd day to be a dependent variable. The coefficient of determination (CD) was as high as 83% when the 2nd-day ingestion dose of pyrene was picked as an independent variable.

Discussion

As can be seen from Table 1, the mean outdoor total PAH concentrations in the ''polluted'' kindergarten

Table 5 1-Hydroxypyrene (*1-OHP*) concentrations in urine (summer)

Place	Day of sampling	1-Hydroxypyrene (morning)		1-Hydroxypyrene (evening)	
		Number of samples	μmol/mol creatinine	Number of samples	μmol/mol creatinine
			Geometric mean (range)		Geometric mean (range)
Kindergarten in polluted area	1	15	0.106 (0.067–0.243)	14	0.115 (0.058–0.495)
	2	15	0.085 (0.026–0.709)	9	0.121 (0.055–0.265)
	3	12	0.089 (0.022–1.426)	10	0.091 (0.021–0.242)
	1–3	42	0.093 (0.022–1.426)	33	0.108 (0.021–0.495)[a]
Kindergarten in non-polluted area	1	15	0.089 (0.044–0.133)	9	0.092 (0.039–0.237)
	2	13	0.094 (0.021–0.281)	11	0.054 (0.016–0.118)[b]
	3	14	0.056 (0.018–0.098)	10	0.080 (0.046–0.151)[b]
	1–3	42	0.078 (0.018–0.281)	30	0.074 (0.016–0.237)

[a] $P < 0.05$ (vs evening concentration of 1-OHP days 1–3 in the non-polluted kindergarten)
[b] $P < 0.05$ (vs morning concentrations of 1-OHP)

Table 6 1-hydroxypyrene (*1-OHP*) concentrations in urine (winter)

Place	Day of sampling	1-Hydroxypyrene (morning)		1-Hydroxypyrene (evening)	
		Number of samples	μmol/mol creatinine	Number of samples	μmol/mol creatinine
			Geometric mean (range)		Geometric mean (range)
Kindergarten in polluted area	1	7	0.083 (0.030–0.133)	9	0.087 (0.051–0.137)
	2	10	0.088 (0.039–0.178)	9	0.116 (0.043–0.340)
	3	10	0.105 (0.023–0.142)	6	0.107 (0.048–0.228)
	1–3	27	0.092 (0.023–0.178)	24	0.103 (0.043–0.340)[a]
Kindergarten in non-polluted area	1	16	0.069 (0.010–0.186)	16	0.072 (0.030–0.246)
	2	17	0.075 (0.028–0.152)	16	0.067 (0.016–0.283)
	3	16	0.081 (0.021–0.762)	14	0.050 (0.020–0.132)
	1–3	50	0.075 (0.010–0.762)	46	0.062 (0.016–0.283)

[a] $P < 0.05$ (vs evening concentration of 1-OHP days 1–3 in the non-polluted kindergarten)

were roughly three-times higher than that in the ''non-polluted'' one during summer and winter. The same trend was observed for pyrene and for the sum of six carcinogenic PAH. Analyzed carcinogenic PAH in the ''polluted'' kindergarten represented about 22% of total outdoor PAH concentration in the summer, and about 30% in the winter. In the ''non-polluted'' kindergarten they represented about 19 and 20% in summer and winter, respectively. The mean total outdoor PAH concentrations which we found in the ''polluted'' kindergarten were approximately 15- and 5-times lower in summer and winter, respectively, than those which were found in the most polluted regions of the Czech Republic – Prague (National Institute of Health (NIH) 1997) and Teplice (Czech Ecological Institute (CEU) 1999). Our summer data were also roughly 15-times lower too than the total PAH concentration in one of the most polluted cities in Western Europe, London (Halsall 1994).

Indoor concentration in the ''polluted'' area was more than six-times higher than that in the ''non-polluted'' area (18.2 vs 2.7 ng/m^3) in summer and almost three-times higher (5.2 vs 2.1 ng/m^3) in winter. In summer we found in the ''polluted'' kindergarten an almost four-times higher total indoor PAH concentration than total PAH outdoor concentration. In the ''non-polluted'' kindergarten in summer we found an almost two-times higher indoor concentration, too. In winter, the indoor concentrations in both kindergartens were lower than outdoor ones, but in the case of the ''polluted'' kindergarten, the indoor concentration was still relatively high. The winter indoor concentration observed in the ''non-polluted'' kindergarten was similar to that observed in summer. The same trend was found for the sum of six analyzed carcinogenic PAH and for pyrene. The influence of ambient air quality on indoor PAH concentrations was readily observed by other authors, especially during wintertime when the outdoor concentrations are generally higher (Waldman et al. 1991). Van Rooij et al. (1994) allege, for example, a ratio of 2:1 between outdoor and indoor air PAH in rooms that are not polluted by tobacco smoke. However, we have to take into consideration that the ratio strongly depends on several factors, e.g. the air conditions, industries, density of traffic, type of cars, indoor sources etc. From this point of view, the higher total indoor PAH concentrations, especially in the case of the ''polluted'' kindergarten in summer, are not so surprising.

Table 7 The survey of relationships (correlation coefficients) between 1-hydroxypyrene (*1-OHP*) and pyrene absorbed doses (*r* correlation coefficients, *n* number of samples)

Relationships	"Polluted" kindergarten (summer)	"Non-polluted" kindergarten (summer)	"Polluted" kindergarten (winter)	"Non-polluted" kindergarten (winter)
First day inhalation doses vs 1st day evening 1-OHP	$n = 14$ $r = -0.1017$	$n = 9$ $r = 0.2668$	$n = 9$ $r = -0.6158$	$n = 16$ $r = 0.4322$
First day inhalation doses vs 2nd day morning 1-OHP	$n = 15$ $r = 0.0133$	$n = 13$ $r = 0.3073$	$n = 10$ $r = 0.2599$	$n = 17$ $r = -0.0505$
Second day inhalation doses vs 2nd day evening 1-OHP	$n = 9$ $r = -0.5564$	$n = 11$ $r = -0.2244$	$n = 9$ $r = 0.6552$ $P < 0.05$	$n = 16$ $r = 0.2436$
Second day inhalation doses vs 3rd day morning 1-OHP	$n = 12$ $r = 0.2244$	$n = 14$ $r = 0.0265$	$n = 10$ $r = 0.0617$	$n = 16$ $r = 0.4366$
Third day inhalation doses vs 3rd day evening 1-OHP	$n = 10$ $r = 0.6041$ $P < 0.05$	$n = 10$ $r = 0.0256$	$n = 6$ $r = -0.0346$	$n = 14$ $r = 0.2632$
Ingestion doses before 1st day vs 1st day morning 1-OHP	$n = 15$ $r = -0.1923$	$n = 15$ $r = -0.0812$	–	$n = 16$ $r = -0.1322$
First day ingestion doses vs 1st day evening 1-OHP	$n = 14$ $r = 0.5396$ $P < 0.05$	$n = 9$ $r = -0.0316$	$n = 9$ $r = -0.6842$ $P < 0.05$	$n = 16$ $r = -0.1088$
First day ingestion doses vs 2nd day morning 1-OHP	$n = 15$ $r = 0.7358$ $P < 0.01$	$n = 13$ $r = -0.0806$	$n = 10$ $r = 0.2756$	$n = 17$ $r = 0.3267$
Second day ingestion doses vs 2nd day evening 1-OHP	$n = 9$ $r = 0.1184$	$n = 11$ $r = 0.3431$	$n = 9$ $r = 0.8456$ $P < 0.01$	$n = 16$ $r = -0.1881$
Second day ingestion doses vs 3rd day morning 1-OHP	$n = 12$ $r = 0.9108$ $P < 0.001$	$n = 14$ $r = 0.6590$ $P < 0.01$	$n = 10$ $r = -0.2149$	$n = 16$ $r = 0.0996$
Third day ingestion doses vs 3rd day evening 1-OHP	$n = 10$ $r = 0.1642$	$n = 10$ $r = -0.2603$	$n = 6$ $r = 0.2401$	$n = 14$ $r = -0.1321$
First day total doses vs 1st day evening 1-OHP	$n = 14$ $r = 0.5383$ $P < 0.05$	$n = 9$ $r = -0.0311$	$n = 9$ $r = -0.6847$ $P < 0.05$	$n = 16$ $r = -0.1016$
First day total doses vs 2nd day morning 1-OHP	$n = 15$ $r = 0.7348$ $P < 0.01$	$n = 13$ $r = 0.0812$	$n = 10$ $r = 0.2759$	$n = 17$ $r = 0.3278$
Second day total doses vs 2nd day evening 1-OHP	$n = 9$ $r = 0.1175$	$n = 11$ $r = 0.0812$	$n = 9$ $r = 0.8465$ $P < 0.001$	$n = 16$ $r = -0.1809$
Second day total doses vs 3rd day morning 1-OHP	$n = 12$ $r = 0.9097$ $P < 0.001$	$n = 14$ $r = 0.6127$ $P < 0.05$	$n = 10$ $r = -0.2143$	$n = 16$ $r = 0.0956$
Third day total doses vs 3rd day evening OHP	$n = 10$ $r = 0.1731$	$n = 10$ $r = -0.2598$	$n = 6$ $r = 0.2230$	$n = 14$ $r = -0.1290$

It must be noted that the kitchen was situated in the basement of the "polluted" kindergarten. As seen from the recorded menu, a higher number of baked and roasted meals were prepared in that kitchen during summer observation days. Because of poor local ventilation, preparation of these meals could markedly contribute to the contamination of indoor air by PAH. Another situation was found in winter, when the amount of baked and roasted meals prepared in the "polluted" kindergarten was comparable with the amount prepared in the "non-polluted" kindergarten. In addition, it must be mentioned that gas heating and gas water heaters were installed in both kindergartens, too.

The total PAH concentrations in the earth and sand samples collected from the "polluted" kindergarten in summer were roughly twice as high as those collected from the "non-polluted" one (Table 2). The adsorption of liposoluble PAH on the surface of soil particles depends on their mobility and on the content of organic matter in soil (Agency for Toxic Substances and Disease Registry (ATSDR) 1995). In agreement with this fact, in both kindergartens we found a several-times higher content of total PAH in the earth samples than in the sand samples. As can be seen in Table 2, the earth samples from both kindergartens contained higher concentrations of selected carcinogenic PAH too. Because of relatively low daily doses of pyrene from the soil, it can be concluded, that in our study, this type of pyrene exposure presented only negligible contribution to the children's total pyrene intake (Tables 2, 3 and 4). Chuang et al. (1997) measured soil PAH concentrations near houses in North Carolina (USA). Samples taken in a city near heavy traffic, and in a rural area, contained 30–410 and 18–53 ng/g pyrene, respectively.

Tables 3 and 4 suggest that food consumption represents the main source of PAH exposure for children, even in large cities that are not heavily polluted by PAH from industrial sources. Butler et al. (1993) measured personal exposure to benzo[a]pyrene in 15 adults living in a town (population of 17,000) in New Jersey (USA). Also in his study, food ingestion was clearly the predominant exposure pathway. Compared with dietary exposures, the proportional contribution of inhalation exposures during late summer was about 5%, which corresponds well with our results. Also Van Rooij et al. (1994) demonstrated in the study on adult volunteers in the Netherlands that the inhalation of ambient air is relatively unimportant for total pyrene intake, accounting for less than 1%. Lodovici et al. (1995) compared total dietary and inhalation PAH intake for an adult population living in a town of medium size in central Italy (the average of the sum of 15 PAH was 27 ng/m^3). The intake by inhalation was 11% of the total PAH intake. All these results confirm that food is the major source of exposure of humans to PAH in areas which are not highly polluted by PAH.

As can be seen from Tables 3 and 4, the contribution from outdoor and indoor inhalation exposure to the total pyrene dose was limited, in spite of the fact that we found significantly higher inhalation intake of pyrene in the "polluted" kindergarten. We are aware that our PAH intake estimates from food were prone to large uncertainties, and should not be overinterpreted. However, it must be noted that our intake results are comparable with dietary intakes reported by other authors. Vaessen et al. (1988) and Van Rooij et al. (1994) calculated oral pyrene intake per capita in the Netherlands as being of 1,600 ng/day and 1,899 ng/day, respectively. In the USA, Chuang et al. (1997) estimated pyrene intake in children (age not reported) from low-income families varying between 24 and 1,480 ng/day.

The 1-OHP elimination process could be described by a one-compartment model, with a half-life of 9.8 h (Brzeznicki et al. 1997). If we assume a balance between absorption and elimination of pyrene due to long-term low-level exposure from the environmental setting, it seems that the morning urine samples better reflect the changes of pyrene intake from "home air" and evening meals, while the evening urine samples better reflect pyrene intake from "kindergarten air" and morning and afternoon meals. The comparison of our data concerning excretion of 1-OHP during both seasons revealed only two significant differences ($P < 0.05$) between morning and evening 1-OHP concentrations (Tables 5 and 6). These results suggest that the contribution of exposure to pyrene (by all routes of entry) during the stay in the kindergarten was not significantly different from that resulting from exposure outside kindergarten, even for children going to the "polluted" kindergarten.

When we compared 3-day means from both kindergartens, in both seasons we found significantly higher concentrations of 1-OHP ($P < 0.05$) in the evening samples of urine from children in the "polluted" kindergarten. These data corresponded well with the calculated doses of pyrene from the air and food (Tables 5 and 6).

Two studies about exposure of children to PAH were performed in polluted areas. Kanoh et al. (1993) measured urinary 1-OHP levels in elementary school children who lived along arterial roads in areas of Tokyo. Children in the highly polluted area showed higher urinary 1-OHP than children in the less polluted area. Unfortunately, the levels of PAH in the air, food or soil were not measured to estimate the contribution of each source to total PAH intake. In a second study, Jongeneelen (1994) reported increased 1-OHP concentrations in 8 to 9-year-old children from a highly polluted region in Poland (Upper Silesia). In comparison with a reference group of adults from Holland, their median levels were nearly twice as high. Again, no data on concentration of PAH in the environment were provided.

Seiwert et al. (1998) presented a nation-wide cross-sectional study conducted repeatedly to assess the exposure of a German population to various pollutants including PAH. About 700 children (6–14 years of age) took part in the study. The geometric mean of their urinary 1-OHP levels ranged from 179 ng/l in West Germany to 449 ng/l in East Germany. The geometric mean of our urinary 1-OHP values in children varied between 148 and 259 ng/l (0.05 and 0.12 μmol/mol creatinine). In a study performed among Dutch children (3–6 years-old), Van Wijnen et al. (1996) found that the geometric means of 1-OHP varied between 0.18 and 0.25 μmol/mol creatinine.

In general, we can say that the strength of significant relationships between 1-OHP and pyrene absorbed doses was poor (Table 7). Van Wijnen et al. (1996) found no relationship between environmental exposure to PAH and urinary 1-OHP. These authors concluded, that "the possible ambient environment-related differences in exposure to PAH were probably too small to be detected in the intake of PAH by the daily diet". However it must be noted that the PAH intake estimates from food are prone to large uncertainties (Chénier and Viau 1997). Our estimates were based on the mean assumed PAH concentrations in food, acquired from diaries data and it is known that the PAH concentrations can vary largely in food with higher PAH content (for example in grilled, baked or roasted food) depending on food preparation conditions (Larsson et al. 1983).

It was shown that local sources of outdoor exposure, smokers at home and cooking with wood/coal could increase the urinary level of 1-OHP (Seiwert et al. 1998). As resulted from the method of generating all possible subsets of independent variables, only the CD for 1-OHP from day 3 morning was as high as 83% when the second day ingestion dose of pyrene was picked as an independent variable. We are unable to explain this phenomenon from the data alone, because in other similar cases the corresponding CDs were as low as 7%. When we calculated only smokers at home, frequent eating of smoked, grilled and roasted food and

local sources of outdoor exposure to PAH in residential area, all possible subsets of independent variables did not increase the CD in each case substantially. In most cases these variables were not among the first few in the regression model that we built up. Therefore, in this case, we assume that it should not be used as a primary independent variable that can affect the excretion of 1-OHP.

The reported data suggest that, under the described conditions, the 1-OHP might have limited applications as an indicator of pyrene ingestion or total dose of pyrene. Low levels of environmental exposure to PAH and uncertainty in environmental dose assessment might be the cause of the low correlation observed in this study. Also, small sample sizes contributed to inconsistencies in our estimates.

In conclusion, we found that food seems to be the main source of total pyrene and total PAH intakes in small children, even in urban PAH air exposure conditions. Even in small children who are in close daily contact with soil (polluted only by air deposition from the traffic and other non-industrial sources), the contribution of soil ingestion to the total PAH absorbed dose is negligible. We assume that the usefulness of urinary 1-OHP, as an indicator of the environmental exposure to PAH (if the pollution of air by PAH is not large), needs further research. Further studies are necessary to explain the relationship between dietary pyrene (as a component of PAH) intake and urinary 1-OHP excretion.

It must be noted that a parallel study conducted on multi-pathway intake of PAH by small children living in a city has been performed in Canada (Montreal) (Vyskocil et al. 2000). Results from that study also suggest that food might be the major source of PAH exposure and that urinary 1-OHP is only weakly correlated to estimates of multi-pathway exposure to these pollutants.

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