Biological Monitoring of Polycyclic Aromatic Hydrocarbon Exposure in a Highly Polluted Area of Poland

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Air pollution in Poland and particularly in Silesia is among the worst in Europe. Many coal mines and coke oven plants are located in this area, representing a major source of carcinogenic polycyclic aromatic hydrocarbons (PAHs). We quantitated the PAH exposure level in air samples using personal sampling devices, collected urine samples from the same individuals, and measured 1-hydroxypyrene with high performance liquid chromatography. Samples were collected twice, once in February and once in September. Mean PAH level of samples collected at three different coke oven plants varied from 2.3 μg/m^3 to 12.3 μg/m^3; the lowest mean was in September. Mean levels of 0.15 μg/m^3 (September) and 0.44 μg/m^3 (February) were noted for the environmentally exposed group. Mean urinary 1-hydroxypyrene varied from 2.45 to 13.48 μmol/mol creatinine at the three coke oven plants. The corresponding variation between the three different environmentally exposed groups in Silesia was 0.41–1.54 μmol/mol creatinine. In the nonindustrialized area, the mean varied from 0.20 to 0.14 μmol/mol creatinine. Seasonal variation was found both at the coke oven plants and in the environmental exposed groups in Silesia. Both PAH levels and 1-hydroxypyrene varied seasonally among coke oven workers and the environmentally exposed group. Our study shows that PAH exposure in the industrialized area of Silesia is high compared to levels in Western Europe. 1-Hydroxypyrene excretion in environmentally exposed individuals in Poland is among the highest in Europe. *Key words*: air pollution, benzo[*a*]pyrene, biological monitoring, exposure monitoring, 1-hydroxypyrene, polycyclic aromatic hydrocarbons. *Environ Health Perspect* 103:838–843 (1995)

Silesia, a highly industrialized region of Poland, is one of the most polluted areas in Europe. The air pollution mainly comes from combustion of fossil fuels emitted by industrial plants and from burning of black coal for home heating during the winter. Assessment of polycyclic aromatic hydrocarbon (PAH) exposure in polluted regions is important for future epidemiological investigations. In addition, it is essential to compare several monitoring methods to validate these methods.

The standardized mortality rates (deaths per 100,000) for men who died from lung cancer in Silesia in 1990 was only slightly higher (73.4) than the average for Poland (71.1) (*1*). The highest mortality rates in Silesia were found in Swietochlowice (117.3) (*2*). The mortality rate from lung cancer was lower in the nonindustrialized region Biala Podlaska (63.1) (*1*). Monitoring data of air pollution in Silesia have shown that exposure to benzo[*a*]pyrene is high (*3*) compared to several European cities (*4*), but comparable to measurements in London in the 1950s (*5*).

Environmental PAH exposure in Silesia has been monitored by stationary samplers (*3*), and several studies have used diverse biomarkers of exposure to mutagenic and carcinogenic compounds (*6–9*). Exposure measurement is one of the key components in a dose–response assessment (*6*), and the sensitive urinary biomarker for PAH exposure, 1-hydroxypyrene (*10,11*), offers a good complement to standard ambient air monitoring. The literature contains little information on quantitative exposure data for individuals in exposed populations.

To investigate environmental exposure of individuals to PAHs in Silesia, air samples were collected by personal sampling devices. Urinary excretion of 1-hydroxypyrene was also analyzed to provide information on the amount of PAH absorbed.

Materials and Methods

Study subjects and data collection. The occupationally exposed group consisted of 66 workers from three different plants located in Silesia, which is a center of coal-based industries in Poland. The coke ovens are denoted plants B, D, and E; plants B and D have been studied previously (*12*). Plants B and E have side-filling of coal, whereas plant D has batteries with side-filling and batteries with top-filling. In addition we studied two environmentally exposed groups, one consisting of individuals living in an industrialized area, Silesia, and the other consisting of individuals living in a nonindustrialized area, Biala Podlaska. The environmentally exposed group in Silesia consisted of three subgroups (Table 1). For each participant, we collected data on lifestyle factors including smoking and medical history, age, and workplace description. Urine samples were collected in polyethylene tubes before and after shift for the occupationally exposed group and during morning and afternoon in the environmentally exposed groups. PAH breathing-zone samples and urine samples were collected the same day. Air samples were collected with personal sampling devices in only one of three environmentally exposed groups in Silesia. We were not able to collect air samples with personal monitoring devices in Biala Podlaska. The measurements were taken twice, once in summer (September) and once in winter (February and March). For a summary of groups and sample collection data see Tables 1 and 2. Samples in Silesia were collected in 1992 and samples in Biala Podlaska were collected in 1993. Most of the coke oven workers live near the plant.

PAH exposure assessment. We quantitated 48 PAHs with molecular weight from 128 to 302. The sum of the following 12 compounds were used for the calculations unless otherwise noted: fluoranthene, pyrene, benz[*a*]anthracene, chrysene/triphenylene, benzo[*e*]pyrene, benzo[*a*]pyrene, indeno[1,2,3-*cd*]pyrene, dibenz[*a,h*]-anthracene, benzo[*ghi*]perylene, and benzofluoranthenes (two isomers). In our method, three-ring and lighter PAHs are incompletely collected and therefore left out in this selection. The 12 compounds here consist of four rings or more, as outlined in the National Institute of Occupational Safety and Health method for PAH determinations (*13*).

Particulate PAHs were sampled on

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Versapor-800 (Gelman Sciences, Ann Arbor, Michigan) filters with Casella AFC 123 (Casella, London, England) and DuPont S2500 (Du Pont, Largo, Florida) at 2 L air/min for 6–8 hr. The standard 25-mm sampling cassette Nuclepore filter (Pleasanton, California) was made of polyethene with carbon black to minimize the effect of static electricity.

The method for sample preparation was modeled after that of Bjørseth (*14*). The filters were extracted (ultrasonic) with cyclohexane after addition of internal standards. Polar compounds and PAHs were extracted from the cyclohexane into *N,N*-dimethylformamide with 3% water. The *N,N*-dimethylformamide was diluted with an equal volume of water and extracted with cyclohexane that was dried (Na_2SO_4) and concentrated.

We analyzed the extracts by gas chromatography on a 25-m Cp-sil-8 CB column (inner diameter 0.25 mm, film thickness 0.25 µm) programmed from 120–320°C, 6°C/min. Splitless injection with a flame ionization detector was used. Internal standards were 3,6-dimethylphenanthrene and ß,ß′-binaphthyl. These standards were used to determine recovery and relative response factors. Quantitation was accomplished with Turbochrome 3 integration software (PE Nelson, Cupertino, California).

Data from PAH measurements with high-volume stationary samplers in Silesia were from Cimander et al. (*15*). The same method of analysis was used for subjects in Biala Podlaska and analysis was performed by the same labortory.

Determination of 1-hydroxypyrene. We determined 1-hydroxypyrene in urine essentially as described by Jongeneelen et al. (*10*). The samples were analyzed in sets together with five spiked urine samples containing 0.010, 0.020, 0.040, 0.100, and 0.250 µmol 1-hydroxypyrene/L. The spiked urine samples were treated as unknowns and used as standards in the quantitative determination of 1-hydroxypyrene. 1-Hydroxypyrene in the urine samples was enzymatically deconjugated and then transferred to primed C18 Sep-Pak cartridges (Millipore, Milford, Massachusetts), washed with water, and eluted with 4 mL methanol. This sample prepurification was performed with a Millilab lab robot (Millipore, Milford, Massachusetts). A 20-µl aliquot was injected in an HPLC with a Novapack C18 column (Millipore, Milford, Massachusetts) and quantitatively determined with a fluorescence detector LC 240 (Perkin-Elmer Ltd, Beaconsfield, England) with excitation wavelength 242 nm and emission wavelength 388 nm. Quantitation was accomplished with Millennium integration software (Millipore, Milford, Massachusetts). All values were corrected based on the creatinine content (*16*).

Statistical methods. Both urinary 1-hydroxypyrene and air measurements of PAH, pyrene, fluoranthene, and benzo[*a*]-pyrene were log normally distributed. Therefore, these data were log–transformed for *t*-tests and analysis of variance. In *t*-tests, the mean values were back-transformed, resulting in a geometric mean which was used in the Tables. The residuals after regression analysis gave the best fit to normal distribution when analyzed on log-transformed 1-hydroxy-pyrene and pyrene data. Coefficients for regression analysis were not back-transformed; therefore, the information in the coefficients are limited. For testing group differences with analysis of variance, Scheffe's method was used. The calculations were done with Statgraphics, version 5 (STSC, Rockville, Maryland).

Table 1. Characterization of the occupationally and environmentally exposed groups

Group	Mean age	Smokers (%)	*N*	Subtotals
Coke oven plants				
Plant B	45.9	41.2	17	
Plant D	46.4	76.5	17	
Plant E	36.7	71.9	32	66
Industrialized environment				
Gliwice	39.7	76.2	21	
Bytom	39.1	58.6	29	
Swietochlowice	54.3	18.8	16	66
Nonindustrialized environment				
Biala Podlaska	33.7	51.5	66	66

Results

In Silesia, environmental and occupational PAH levels were monitored both by personal carried sampling devices and stationary samplers (*15*). In Biala Podlaska, PAH levels were monitored only by stationary sampling. There were no unusual weather conditions during the sampling.

The concentrations of 48 PAHs were quantitated in each sample. The correlation coefficients between a selected number of these compounds were determined (Table 3), and there was good agreement between these variables. Therefore, in the following

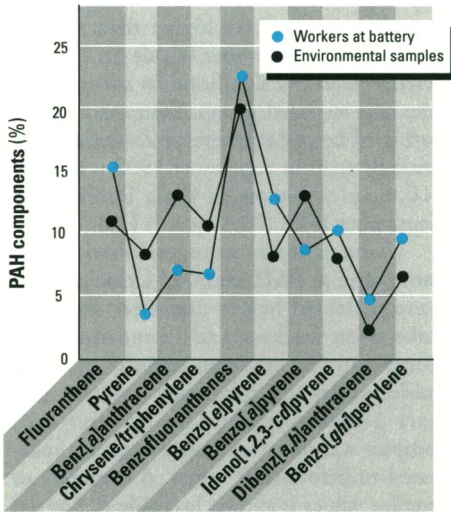

Figure 1. Relative proportion of 12 separated polycyclic aromatic hydrocarbon compounds. Chrysene and triphenylene and benzofluoranthenes were quantitated together.

Table 2. Number of samples collected from the participants in the study

	February			September		
	1-Hydroxypyrene[a]		PAH air	1-Hydroxypyrene[a]		PAH air
Location	Morning	Afternoon	measurements	Morning	Afternoon	measurements
Coke oven plants						
Plant B	16	16	13	13	11	6
Plant D	13	13	13	11	13	8
Plant E	32	31	28	19	18	19
Industrialized environment						
Gliwice	17	17	12	13	12	10
Bytom	29	29	—	15	14	—
Swietochlowice	16	14	—	7	6	—
Nonindustrialized environment						
Biala Podlaska	5	31	—	—	45	—

[a]Morning = before shift; afternoon = after shift.

Table 3. Correlation coefficients between selected PAHs (sum of 12) and total PAHs (sum of all measured), fluoranthene, pyrene, and benzo[*a*]pyrene

	Total PAHs	Fluoranthene	Pyrene	Benzo[*a*]pyrene
February (*N* = 66)				
Selected PAHs*	0.96	0.94	0.91	0.98
September (*N* = 43)				
Selected PAHs*	0.96	0.96	0.94	0.96

*$p < 0.00005$ for all values; analysis performed on log-transformed data, Pearson product moment.

analysis we used 12 PAHs (including chrysene/triphenylene and two isomers of benzofluoranthene; Fig. 1), as well as pyrene and benzo[*a*]pyrene alone. There were no obvious systematic differences between the profiles of PAH compounds. PAH exposure levels are shown in Table 4, and a box plot of the PAH exposure level are shown in Figure 2. The PAH levels were higher in samples from coke oven workers than from environmentally exposed subjects, but there was great variation in samples from the various coke oven plants. The difference between arithmetic mean and median values shows that the data have a skewed distribution. The PAH levels in samples collected in winter were higher than samples collected in the summer (Table 5). The difference was only significant in the environmentally exposed group. Stationary monitoring of benzo[*a*]pyrene in Zabrze (industrialized area) in September 1992 was lower (10.4 ng/m^3) than in Biala Podlaska in September 1993 (20.4 ng/m^3) (*15*).

Urine samples were collected at the same time as PAH samples. Urine samples were collected before and after work for coke oven workers and in morning and afternoon for environmentally exposed subjects. Among coke oven workers, urinary 1-hydroxypyrene in the after-shift samples was lower or nearly constant compared to before-shift values. A summary of average values is shown in Table 6. For the following analysis, we used the data from after-shift or afternoon samples.

To analyze for a possible association between urinary excretion in wintertime and summertime, we calculated the correlation coefficient between winter and summer samples. The correlation between urinary 1-hydroxypyrene from winter and summer samples in the coke oven workers was relatively high (0.72; $p>0.00005$), and the values for the environmentally exposed subjects were lower (0.53; $p = 0.003$).

In Silesia, we found a higher level of urinary 1-hydroxypyrene in samples collected in the winter compared to samples collected in the summer both from coke oven workers and environmentally exposed subjects, but it was only among the environmentally exposed subjects that this difference was significant. In Biala Podlaska, the nonindustrialized area, we found no such seasonal difference (Table 7). A seasonal effect was found in environmental samples from the industrialized areas of Gliwice and Bytom, but not from the industrialized area of Swietochlowice (Fig. 3). Workers at coke oven plants are exposed to higher PAH levels than the environmentally exposed individuals. There was a significantly ($p<0.005$) higher level of 1-hydroxypyrene in individuals in the combined environmental group from Silesia compared to the values found at Biala Podlaska both in winter and summer (Table 7). In Silesia, the 1-hydroxypyrene level was higher in samples from coke oven workers than in samples from environmentally exposed subjects; these differences were also significantly different ($p<0.05$) except for the difference between Gliwize and plant E (Table 7).

In the environmentally exposed groups, both from the industrialized Silesia region and the nonindustrialized Biala Podlaska region, there was a significantly higher excretion of 1-hydroxypyrene among smokers than among nonsmokers. Among coke oven workers we also found a higher level of 1-hydroxypyrene in smokers, but this difference was not significant (Table 8).

We analyzed the associations among urinary 1-hydroxypyrene and exposure to pyrene, smoking, and age in both the winter and summer samples. The simple regression data between urinary 1-hydroxypyrene and pyrene exposure are shown in Table 9, and multiple regression analyses are shown in Table 10. The coefficients cannot be directly evaluated because both 1-hydroxypyrene and pyrene values are log transformed. Comparing the squared correlation coefficient in simple and multiple regression shows the importance of smoking as an explanatory variable. All individuals with data on urinary 1-hydroxypyrene and pyrene exposure (environmental summer samples) were smokers; therefore a multiple regression analysis could not be done.

Table 4. PAH exposure level from personal sampling devices at three coke oven plants and at Gliwice, an industrialized area

			Exposure (μg/m^3), arithmetic mean (SD)		
Time	Place	*N*	PAHs	Pyrene	Benzo[*a*]pyrene
February	Plant B	13	7.4 (7.1)	0.52 (0.48)	1.02 (1.06)
September	Plant B	6	2.3 (4.8)	0.15 (0.34)	0.31 (0.65)
February	Plant D	13	12.3 (28.3)	1.46 (4.13)	1.33 (2.8)
September	Plant D	8	11.4 (18.9)	0.72 (1.26)	1.51 (2.56)
February	Plant E	28	6.9 (13.4)	0.49 (1.16)	0.93 (1.92)
September	Plant E	19	5.3 (10.3)	0.41 (0.83)	0.72 (1.50)
February	Gliwice	12	0.44 (0.22)	0.012 (0.010)	0.041 (0.032)
September	Gliwice	10	0.15 (0.24)	0.009 (0.009)	0.009 (0.024)

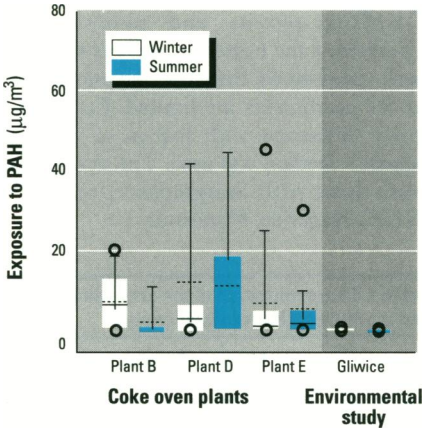

Figure 2. Box plot of PAH (sum of the 12 compounds listed in text and shown in Fig. 1) exposure in samples collected with personal monitors from coke oven workers and environmentally exposed subjects. All groups were sampled in winter and summer. Dotted line is arithmetic mean, solid line is median. The box encompasses 25th and 75th percentiles; whiskers extends to 10th and 90th percentiles. Values falling outside this range are indicated by circles.

Table 5. Seasonal variations in PAH levels

	Geometric mean (μg/m^3)		
Sample type	Winter (*N*)	Summer (*N*)	*p*-value[a]
Environmental (Gliwice)	0.40 (12)	0.06 (10)	<0.001
Occupational (sum of 3 coke oven plants)	2.08 (54)	1.38 (33)	0.33

[a]Student's *t*-test for difference between summer and winter samples, unpaired. Data log-transformed.

Discussion

Using two independent monitoring methods, we found higher exposure to PAHs in an industrialized region compared to a nonindustrialized region. Exposure via the environment in the industrialized region was high, although exposure was lower than for occupationally exposed coke oven workers. Urinary 1-hydroxypyrene depended both on pyrene concentration in the air and smoking habits.

The distribution of individual PAHs differs from previous data on PAHs collected from stationary samplers in the same area (*3,7*) although the major compounds like fluoranthene, benzo[*bjk*]fluoranthenes, pyrene, and benzo[*a*]pyrene are the most highly concentrated in all these analyses. The average concentration of benzo[*a*]pyrene in Gliwice in September (0.041 μg/m^3; Table 4) is comparable to measurements in central London in the period

Table 6. Mean values (arithmetic) of urinary 1-hydroxypyrene before and after shift[a]

		Urinary 1-hydroxypyrene (μmol/mol creatinine)					
		Before shift (morning)			After shift (afternoon)		
Place	Time	Mean	SD	*N*	Mean	SD	*N*
Coke ovens							
Plant B	Winter	8.12	8.62	16	11.59	10.37	16
Plant B	Summer	8.67	10.55	13	4.69	4.71	11
Plant D	Winter	15.82	22.78	13	10.86	8.06	13
Plant D	Summer	14.64	14.72	13	13.48	9.91	11
Plant E	Winter	4.21	4.11	32	4.14	6.00	31
Plant E	Summer	2.35	2.25	19	2.45	1.94	18
Environmental exposure[a]							
Gliwice	Winter	1.25	0.93	17	1.54	1.20	17
Gliwice	Summer	0.90	0.60	13	0.84	0.57	12
Bytom	Winter	0.77	0.57	29	0.76	0.57	29
Bytom	Summer	0.32	0.24	15	0.38	0.21	14
Swietochlowice	Winter	0.47	0.24	16	0.36	0.17	14
Swietochlowice	Summer	0.44	0.28	7	0.41	0.36	6
Biala Podlaska	Winter	0.19	0.15	5	0.21	0.14	31
Biala Podlaska	Summer	—	—	—	0.27	0.20	45

[a]For environmental samples, morning and afternoon values are given.

Table 7. Seasonal variations of urinary 1-hydroxypyrene

	Geometric mean (μmol/mol creatinine)		
Sample type	Winter (*N*)	Summer (*N*)	*p*-value[a]
Environmental (Gliwice, Bytom, and Swietochlowice)	0.63 (60)	0.39 (32)	0.02
Occupational	3.81 (60)	3.19 (40)	0.62
Environmental (Biala Podlaska)	0.17 (31)	0.22 (45)	0.15
p-value[b]	<0.005	<0.005	

[a]Unpaired Student's *t*-test of log-transformed data.
[b]*p*-Value for all comparisons in winter and summer groups.

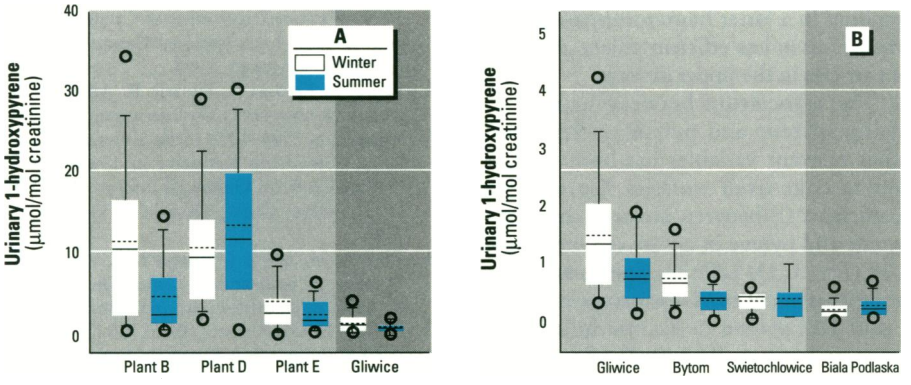

Figure 3. Box plot of urinary 1-hydroxypyrene levels in samples collected in Silesia and Biala Podlaska. Samples were collected in winter and summer. (A) Data from all coke oven plants (plants B, D, and E) and Gliwice, the most polluted area. (B) Data from all regions in the environmental study: industrialized areas: Gliwice, Bytom, and Swietochloweice, and Biala Podlaska, a nonindustrial area. All groups were sampled in winter and summer. Dotted line is arithmetic mean, solid line is median. The box encompasses 25th and 75th percentiles; whiskers extends to 10th and 90th percentiles. Values falling outside this range are indicated by circles.

1949–1951 (0.046 μg/m^3) (*5,17*), but these concentrations are not directly comparable because sampling procedures and quantitation methods differ. In a recent report, Brown et al.(*18*) studied lung cancer mortality in Poland and the significance of pollution, occupational exposure, and social factors as causation of disease.

An average of 0.8 m^3 of air was sampled by the filters; this volume is minimal for precisely measuring low PAH concentrations (i.e., environmental samples), but the volume is acceptable for occupational samples from coke oven plants. Low sampling volume can explain part of the deviations in our PAH profile and that of Chorazy et al. (*3*). But it is not unusual to find small differences in the composition of samples collected by portable samplers and those collected by stationary samplers. The advantage of personal sampling is that it reflects individual exposure at home and at work. For the occupationally exposed subjects, a difference between results from portable and the stationary samplers is likely because emissions from coke oven plants are only one of several PAH sources in the area.

Table 8. Effect of smoking habit on urinary 1-hydroxypyrene

	Geometric mean (μmol/mol creatinine)		
Sample type	Nonsmokers (*N*)	Smokers (*N*)	*p*-value[a]
Environmental, Silesia			
Winter samples	0.38 (29)	1.01 (31)	<0.0005
Summer samples	0.19 (12)	0.59 (20)	0.0023
Occupational, Silesia			
Winter samples	3.25 (21)	4.15 (39)	0.57
Summer samples	2.19 (15)	3.99 (25)	0.12
Environmental, Biala Podlaska			
Winter samples	0.13 (19)	0.25 (12)	0.01
Summer samples	0.14 (18)	0.30 (27)	<0.0001

[a]Unpaired Student's *t*-test of log-transformed data.

The differences in PAH concentrations of samples collected at the various plants can partly be explained by plant design like top-filling and side-filling, production capacity, and other technical factors. PAHs in air had been measured previously at plants B and D (*12*), and the highest benzo[*a*]pyrene levels were found in plant D. Plant D was the only plant which had batteries with top-filling of coal. The concentration of PAHs in air and levels of urinary 1-hydroxypyrene were highest in the winter samples. The seasonal difference was greatest among environmental samples. Two factors are important in explaining this result. During the winter, more coal and coke are used for heating, and an inversion layer forms, increasing local air pollution in cold days with light winds. Seasonal variations and high PAH levels during the winter have also been recorded in the UK in the 1950s and 1960s (*17,19*).

To study seasonal effects on PAHs in air and on 1-hydroxypyrene in urine, we used an unpaired *t*-test. Some subjects gave samples during both winter and summer. However, it is not likely that this overlap would affect our conclusion of a seasonal effect. In the cohort, there were too few samples to perform a reliable paired *t*-test. Seasonal variations have also been found in levels of PAH–DNA adducts (*8*) and sister chromatid exchanges in lymphocytes collected from the same volunteers who participated in this study (Pendzich J, unpublished data).

The urinary 1-hydroxypyrene concentrations vary in the same way as the pyrene

Table 9. Simple regression analysis of 1-hydroxypyrene association to pyrene exposure[a]

Group	Time	N	Intercept	Regression coefficient	p for coefficient	R^2
Occupational	Winter	54	2.02	0.26	0.002	0.18
Occupational	Summer	29	1.70	0.26	0.03	0.16
Environmental	Winter	11	-1.88	-0.41	0.10	0.27
Environmental	Summer	9	1.30	0.32	0.29	0.16

[a]Both 1-hydroxypyrene and pyrene values are log-transformed. Coefficients and intercepts are not back-transformed.

Table 10. Multiple regression analysis of 1-hydroxypyrene association to pyrene exposure, smoking, and age[a]

Group	Regression coefficient	p	R^2 (adjusted)
Occupational, winter (N = 54)			0.26
Intercept	0.89	0.07	
Pyrene	0.25	0.001	
Smoking	0.63	0.02	
Age	0.01	0.12	
Occupational, summer (N = 29)			0.07
Intercept	1.94	0.06	
Pyrene	0.24	0.07	
Smoking	0.12	0.80	
Age	-0.01	0.67	
Environmental, winter (N = 11)			0.73
Intercept	-3.94	0.01	
Pyrene	-0.74	0.01	
Smoking	0.91	0.01	
Age	0.01	0.46	

[a]Both 1-hydroxypyrene and pyrene are log transformed. Coefficients and intercepts are not back-transformed.

measurements. For instance, workers at plant D had the highest PAH exposure and the highest levels of 1-hydroxypyrene in urine. PAH concentrations in samples from coke oven plants are high compared to data from other coke oven plants published recently (*20,21*).

The 1-hydroxypyrene levels in urine of individuals from the nonindustrialized region were consistently lower than levels in individuals from industrialized Silesia. Urinary 1-hydroxypyrene levels in nonoccupationally exposed individuals have been published previously (*22*). Median values among smokers varied from 0.26 to 0.51 µmol 1-hydroxypyrene/mol creatinine in studies conducted in Western Europe and 0.76 µmol/mol creatinine in inhabitants of Beijing. The median values (Fig. 3B) are higher than 0.5 in Bytom winter samples and in Gliwice winter and summer samples. These samples were from smokers and nonsmokers. To our knowledge there is no similar environmental study of urinary 1-hydroxypyrene in a highly industrialized area in Western Europe. But in similar studies in China, urinary 1-hydroxypyrene among residents of cities (*23,24*) are comparable to values found in Silesia. In Silesia, several coke oven plants are located in a relatively small area and coal and coke are widely used for domestic heating.

Smoking contributes to excretion of 1-hydroxypyrene. Smoking 20 cigarettes a day increases the excretion of this metabolite by approximately 0.30 µmol/mol creatinine (*25*). In some studies of occupationally exposed individuals, the difference in urinary 1-hydroxypyrene between smokers and nonsmokers was greater than expected (*26*), indicating that smoking has an effect on uptake, excretion, or metabolism of 1-hydroxypyrene. In our data (Table 8), the difference in urinary 1-hydroxypyrene between smokers and nonsmokers was 0.9 and 1.8 µmol/mol creatine for coke oven workers in winter and summer samples, respectively. The difference in urinary 1-hydroxypyrene between smokers and nonsmokers is also elevated in environmentally exposed subjects from Silesia. Smokers may have induced P450 enzymes (*27,28*), resulting in a faster biotransformation, and smokers have less efficient ciliary clearance of particles in the upper airways.

The association between urinary 1-hydroxypyrene and pyrene or PAH and other relevant variables has been studied among coke oven workers. Correlation coefficients (r) between urinary 1-hydroxypyrene and pyrene in air are reported to be 0.58 (R^2 = 0.33) (*29*); the Spearman correlation coefficient for 1-hydroxypyrene after a shift and pyrene in air multiplied with exposure hours is 0.15–0.40 (R^2 = 0.02–0.16) (*20*), and the correlation coefficient between urinary 1-hydroxypyrene and PAH in air is 0.50–0.55 (R^2 = 0.25–0.30), before and after shift (*21*).

One explanation for these relatively low correlation coefficients may be uptake through the skin (*21*). In a study of creosote workers where skin uptake was estimated, R^2 was 0.79 in a multiple regression analysis (*22*). Multiple regression analysis of data from coke oven plants have given R^2 from 0.31 to 0.34 with variables including PAHs in air and thiocyanate in urine and plant type (coke oven or electrode plant) (*21*) and 0.44 with variables of pyrene in air, exposure hours, use of airstream helmet, and smoking habit (20). In a study of nonoccupationally exposed smokers, a high partial R^2 of 0.66 was found for PAHs from cigarette smoke, and the authors could explain 73% of the variability of 1-hydroxypyrene (*25*). In our study all regression coefficients were positive except for winter samples in environmentally exposed subjects. The number of samples in the environmental study are low, and for simple regression analysis the coefficients were not significant. Based on earlier studies and on our own data, one can conclude that pyrene in air is not a very strong predictor of excretion of 1-hydroxypyrene.

This study, together with previous studies, shows that Silesia is a region with high PAH exposure. These investigations have produced important data for current and future epidemiological investigation in this area. The interindividual variation in both PAH exposure and urinary 1-hydroxypyrene clearly shows the advantage of personal monitoring, either by portable sampling devices for air measurements or biological monitoring. There is a need for future exposure studies in this region, and urinary 1-hydroxypyrene is a biomarker well suited for monitoring of occupational and environmental PAH exposure.

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