Estimating Individual-Level Exposure to Airborne Polycyclic Aromatic Hydrocarbons throughout the Gestational Period Based on Personal, Indoor, and Outdoor Monitoring

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Objectives: Current understanding on health effects of long-term polycyclic aromatic hydrocarbon (PAH) exposure is limited by lack of data on time-varying nature of the pollutants at an individual level. In a cohort of pregnant women in Krakow, Poland, we examined the contribution of temporal, spatial, and behavioral factors to prenatal exposure to airborne PAHs within each trimester and developed a predictive model of PAH exposure over the entire gestational period.

Methods: We monitored nonsmoking pregnant women (n = 341) for their personal exposure to pyrene and eight carcinogenic PAHs—benz[a]anthracene, chrysene/tribenzyrene, benz[k]fluoranthene, benzo[b]fluoranthene, benzo[a]pyrene [B(a)P], indeno[1,2,3-cd]pyrene, dibenz[a]anthracene, and benzo[g,h,i]perylene—during their second trimester for a consecutive 48-hr period. In a subset (n = 78), we monitored indoor and outdoor levels simultaneously with the personal monitoring during the second trimester with an identical monitor. The subset of women was also monitored for personal exposure for a 48-hr period during each trimester. We repeatedly administered a questionnaire on health history, lifestyle, and environment.

Results: The observed personal, indoor, and outdoor B(a)P levels we observed in Krakow far exceeded the recommended Swedish guideline value for B(a)P of 0.1 ng/m3. Based on simultaneously monitored levels, the outdoor PAH level alone accounts for 93% of total variability in personal exposure during the heating season. Living near the Krakow bus depot, a crossroad, and the city center and time spent outdoors or commuting were not associated with higher personal exposure. During the nonheating season only, a 1-hr increase in environmental tobacco smoke (ETS) exposure was associated with a 10–16% increase in personal exposure to the nine measured PAHs. A 1°C decrease in ambient temperature was associated with a 3–5% increase in exposure to benz[a]anthracene, benz[k]fluoranthene, and dibenz[a,h]anthracene, after accounting for the outdoor concentration. A random effects model demonstrated that mean personal exposure at a given gestational period depends on the season, residence location, and ETS.

Conclusion: Considering that most women reported spending <3 hr/day outdoors, most women in the study were exposed to outdoor-originating PAHs within the indoor setting. Cross-sectional, longitudinal monitoring supplemented with questionnaire data allowed development of a gestation-length model of individual-level exposure with high precision and validity. These results are generalizable to other nonsmoking pregnant women in similar exposure settings and support reduction of exposure to protect the developing fetus.

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2005). However, application of this method to other populations is limited because of dissimilar sources, meteorologic conditions, and other social and cultural factors. For example, personal exposure to the sum of eight carcinogenic PAHs (Σ8c-PAHs) in Krakow, Poland, varies by >10-fold across seasons (Choi et al. 2006). In addition, mean personal exposure to PM2.5 for women monitored during the heating season was significantly higher than for women monitored during the nonheating season (43.37 vs. 29.77 µg/m3, p < 0.001) (Jedrychowski et al. 2006).

Another important consideration in long-term exposure estimation is the particular health outcome of interest. There is considerable evidence that host susceptibility associated with age of exposure is critical in determining the severity of adverse outcomes (Dejmek et al. 2000; Sanyal et al. 1993; Wormley et al. 2004). For example, effects of B(a)P on embryo trophoblasts (Sanyal et al. 1993) have been shown to vary depending on the period of exposure as well as the exposed organ (Sanyal et al. 1993; Wormley et al. 2004). Varying fetal vulnerability across the gestational period is caused not only by the changes in rate of cell division, migration, differentiation, and apoptosis, but also by the immaturity of the detoxification mechanisms and DNA repair capabilities (Anderson et al. 2000; Selevan et al. 2000).

The present analysis is part of an ongoing prospective cohort study on prenatal and early childhood exposure to multiple toxicants on a number of developmental and health outcomes (Choi et al. 2006; Jedrychowski et al. 2004, 2005a, 2005b, 2006). Here, we focus on developing a gestation-length model of personal exposure to Σ8c-PAH among pregnant women enrolled in an ongoing study in Krakow. Because of their high mutual correlations, we assumed in our previous analyses that Σ8c-PAHs represents the combined fetotoxicity of the eight carcinogenic PAHs monitored in the present study. We examined the validity of this assumption by observing whether the personal exposure patterns to each of the eight individual PAHs are consistent with those for Σ8c-PAHs. The objectives of this analysis were a) to characterize PAH exposure based on simultaneous personal, home indoor, and home outdoor monitoring; b) to identify short-term risk factors of personal exposure; and c) to build a predictive model for mean individual-level exposure during each month of gestation, based on these results.

**Materials and Methods**

Details regarding subject enrollment and air monitoring methods have been previously published (Choi et al. 2006; Jedrychowski et al. 2004, 2006) and can also be found in our Supplemental Material (online at http://www.epihealthperspectives.org/sites/default/files/Supplemental%20material%202008%2010972.pdf). Briefly, we recruited nonsmoking pregnant women from the prenatal care clinics from areas with well-recorded ambient air pollution levels in the Srodmiescie–Old Podgorze area (city center) and the Krowodrzy–Nowa Huta–New Podgorze area (city outer area) (Jedrychowski et al. 2003). We chose the enrollment target sites based on 1996 air monitoring data from the U.S. Environmental Protection Agency (EPA), which showed that B(a)P levels were 2-fold higher in Srodmiescie (annual mean, 13.3 ng/m3; 22.4 ng/m3 in winter; 4.6 ng/m3 in summer) than in Krowodrzy (annual mean, 6 ng/m3; 11.2 ng/m3 in winter; 0.9 ng/m3 in summer). To capture seasonal variability in personal exposure, we recruited approximately equal proportions of the total cohort during each season (23% enrolled during December–February; 27% during March–May; 26% during June–August; and 24% during September–November).

**Personal interview.** A research staff member administered a questionnaire to the women in their homes during their late second trimester. The questionnaire elicited information on demographic, health history, socioeconomic profile, outdoor environment, housing characteristics, indoor exposure sources, and other lifestyle choices. We restricted our analysis of the questionnaire data to the factors that potentially contribute to the exposure to PAHs, including smoking history by household members and dietary intake of PAH-containing foods (Table 1). Because behavior patterns might change through the pregnancy course, we administered the questionnaires again during the third trimester.

**Personal PAH monitoring.** We measured personal exposure directly at the individual level by having each pregnant woman wear a backpack in all microenvironments during waking hours and place it at bedside at night, for a consecutive 48-hr period (see Supplemental Material online at http://www.epihealthperspectives.org/sites/default/files/Supplemental%20material%202008%2010972.pdf). Because we did not ask the subjects to keep a log of the time spent in each microenvironment, we interpreted personal exposures as the cumulative concentration over the 48-hr monitoring period. In a randomly assigned subset of 78 women, we conducted repeat personal monitoring for a 48-hr period during each trimester.

**Indoor and outdoor air monitoring.** In the subset of 78 women, we conducted indoor and outdoor PAH monitoring at the same time as the second-trimester personal monitoring. The indoor samplers were placed in a room where the woman spent most of her time (i.e., living room or near the kitchen). The sampler was placed atop furniture 0.5–2 m above the floor away from the heating sources, about 1 m away from the wall of the home or the apartment. All sampling units were checked and batteries replaced by staff approximately midway through the 48-hr period.

**Laboratory analysis of PAHs in air monitoring samples.** We removed the quartz filter and polyurethane foam (PUF) plug, spiked with p-terphenyl-d14 (as an extraction surrogate), and performed Soxhlet extraction (Corning Inc., Corning, NY, USA) with 6% diethyl ether in hexanes for 1 hr. Each extract was concentrated to a final volume of 1.0 mL and frozen at −12°C. We analyzed the air extracts by gas chromatography/mass spectrometry for pyrene and the eight carcinogenic PAHs—benz[a]anthracene [B(a)A], chrysene/ isochrysenes, benzo[k]fluoranthene [B(k)F], benzo[b]fluoranthene [B(b)F], indeno[1,2,3-c,d]pyrene, dibenz[a,h]anthracene [D(a,h)A], and benzo[g,h,i]perylene [B(g,h,i)P]—as described in U.S. EPA (1999). The recovery of the extraction surrogate, p-terphenyl-d14, was consistently > 60%, indicating satisfactory recovery of collected PAHs. The 48-hr matrix spikes showed that the procedure efficiently extracted all nine PAHs from the filter and PUF, with recovery ranges from 91% to 117% and recovery standard deviations from 18% to 31%. We did not adjust air concentrations for spike recoveries. Two laboratory technicians analyzed all PAH samples using the same technique. Measurement agreement of the duplicate PAH samples were > 90% over the 2-year exposure assessment period. The detection limit for each target PAH was 1.0 ± 0.2 ng/sample; 100% of the air samples were above the detection limit for all PAHs except for D(a,h)A (73% > detection limit). The study was reviewed and approved by the institutional review boards of Jagiellonian University and Columbia Presbyterian Medical Center. We obtained informed consent from all study participants.

**Statistical analyses.** We limited statistical analysis to the air samples with a high/ good quality assurance score (0 or 1): 489 (96%) personal air monitoring samples, 76 (97%) indoor, and 70 (91%) outdoor samples. Sixty-eight of 78 women completed all three serial personal monitorings and met the quality standard. The personal, indoor, and outdoor exposure levels of nine individual PAHs as well as Σ8c-PAHs (nanograms per cubic meter) were skewed (all p-values for Kolmogorov–Smirnov test < 0.001). After natural log (ln) transformation, the distribution of the personal air samples remained bimodal, with a low exposure range during June–August and a high exposure range during September–May, whereas indoor and outdoor measurements conformed to a normal distribution. When stratified by season, all distributions approximated the normal distribution (Kolmogorov–Smirnov tests.
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$P > 0.05$). The PAH concentrations below the detection limit were assigned half of the limit value. We assessed agreement between repeated personal monitoring and simultaneous personal, indoor, and outdoor monitoring using Spearman’s rank correlation coefficients. Our statistical models used personal measurements of ln-transformed values for all nine PAHs as well as the ln-transformed $28c$-PAH values as the outcomes. We developed a gestation-length model by hierarchically generalizing cross-sectional data of simultaneous personal, indoor, and outdoor monitoring data.

We first examined the personal exposure PAH levels as a function of the corresponding outdoor levels through ordinary least-squares regression. To apply this model to the remaining women who lacked outdoor and indoor monitoring data, we considered two approaches to estimate each woman’s outdoor concentration at a given gestational month. First, we estimated the outdoor concentration as the cohort’s outdoor mean level at a given month and year, ignoring the spatial variability. Whenever available, we used the actual data as the predictor of the personal exposure at a given gestational month. In the second approach, we considered the indicator variable for month and year of a given gestational month as a proxy for the outdoor concentration. In the resulting ordinary least-squares regression model of the personal exposure, we controlled for the effects of ETS exposure and residence in the city center. Given the result of this model, we considered repeated personal monitoring and indicator variables for month and year at a given gestational age as the main exposure variables in the random effects model. We used backward model selection strategy to reduce the number of fixed effect variables with selection criteria $\alpha = 0.10$. However, we retained some variables in the final model regardless of this criterion, based on our prior knowledge regarding outdoor sources in Krakow. The final model included the fixed time (month and year indicator of personal monitoring, trimester at the time of monitoring), fixed spatial (living in city center), fixed behavior (hours of ETS exposure at home), and fixed interaction (December 2001, January 2002, and December and city center, respectively) terms, as well as the subject-specific random deviation from the population mean. We examined the precision of the predicted mean exposure at a given gestational month using the “leave-one-out” cross-validation method. We calculated the relative cross-validation residual as (predicted personal exposure concentration – observed personal exposure concentration)/observed personal exposure concentration. We fit the model with one observation deleted from the data at each time and used that observation as the test

| Characteristic                        | Single personal measurement$^a$ [n = 266] | Personal/indoor/outdoor measurement$^b$ [n = 78] | p-Value |
|--------------------------------------|------------------------------------------|-------------------------------------------------|---------|
| Mother’s age (years)                 | 28.4 ± 18 (19–38)                        | 28.4 ± 18 (18–35)                                | 0.41    |
| Prepregnancy weight (kg)             | 58.04 ± 6.20 (40–90)                     | 59.94 ± 11.43 (43–118)                           | 0.11    |
| Mother’s height (cm)                 | 185.08 ± 5.54 (149–180)                  | 164.69 ± 5.92 (144–178)                          | 0.55    |
| Annual mean household income$^c$ [no. (%)] | 178 (67)                                | 54 (69)                                          | 0.72    |
| Low                                  | 13 (5)                                   | 3 (4)                                            |         |
| Medium                               | 4 (1)                                    | 0 (0)                                            |         |
| Refused/don’t know                   | 71 (27)                                  | 21 (27)                                          | 0.95    |
| Maternal education [no. (%)]         |                                          |                                                  |         |
| < High school                        | 28 (10)                                  | 9 (11)                                           |         |
| High school graduate                 | 71 (27)                                  | 20 (26)                                          |         |
| > High school                        | 167 (63)                                 | 49 (63)                                          |         |
| Occupation during current pregnancy [no. (%)] | 152 (57)                          | 45 (58)                                          | 0.68    |
| Office setting$^d$                    | 14 (5)                                   | 6 (8)                                            |         |
| Restaurant/factory                   | 100 (38)                                 | 27 (35)                                          |         |
| Other/unknown                        |                                          |                                                  |         |
| Residence area$^e$ [no. (%)]         |                                          |                                                  | 0.46    |
| Outer area (low pollution)           | 208 (78)                                 | 64 (82)                                          |         |
| City center (high pollution)         | 58 (22)                                  | 14 (18)                                          |         |
| Prior live birth [no. (%)]           | 96 (36)                                  | 30 (39)                                          | 0.70    |
| Currently married [no. (%)]          | 247 (83)                                 | 139 (84)                                         |         |
| Wine, beer, liquor glasses/day [no. (%)] | 3 (1)                                | 1 (1)                                            | 0.91    |
| Daily home ETS exposure [no. (%)]    |                                          |                                                  | 0.67    |
| ≥ 4 hr/day                           | 36 (14)                                  | 10 (13)                                          |         |
| > 5 hr/day                           | 9 (3)                                    | 4 (5)                                            |         |
| Non-smoker home                      | 222 (80)                                 | 64 (82)                                          |         |
| Time spent outdoor ≤ 3 hr/day [no. (%)] | 209 (79)                          | 64 (82)                                          | 0.50    |
| Time spent at home [no. (%)]         |                                          |                                                  | < 0.01  |
| < 3 hr/day                           | 26 (10)                                  | 8 (10)                                           |         |
| 4–10 hr/day                          | 218 (82)                                 | 53 (68)                                          |         |
| 10–16 hr/day                         | 53 (20)                                  | 6 (8)                                            |         |
| Time spent in transit [no. (%)]      |                                          |                                                  | 0.19    |
| < 1 hr/day                           | 7 (3)                                    | 4 (5)                                            |         |
| 1–2 hr/day                           | 221 (83)                                 | 68 (87)                                          |         |
| ≥ 3 hr/day                           | 38 (14)                                  | 6 (8)                                            |         |
| Amount of outdoor vehicular traffic [no. (%)] | 125 (47)                          | 56 (72)                                          | 0.12    |
| Light                                | 216 (81)                                 | 56 (72)                                          |         |
| Medium                               | 37 (14)                                  | 14 (18)                                          |         |
| Heavy                                | 13 (5)                                   | 9 (10)                                           |         |
| Live next door to outdoor sources [no. (%)]$^f$ | 152 (57)                          | 48 (62)                                          | 0.25    |
| Repair garage                        | 46 (17)                                  | 17 (22)                                          | 0.37    |
| Industrial plant                     | 11 (4)                                   | 4 (5)                                            | 0.71    |
| Bus depot                            | 27 (10)                                  | 8 (10)                                           | 0.98    |
| Incinerator                          | 2 (1)                                    | 0 (0)                                            | 0.44    |
| Fuel type$^g$ [no. (%)]              |                                          |                                                  | 0.16    |
| Gas                                  | 60 (22)                                  | 14 (18)                                          |         |
| Electricity                          | 26 (10)                                  | 15 (19)                                          |         |
| Coal                                 | 15 (6)                                   | 3 (4)                                            |         |
| Wood                                 | 4 (2)                                    | 0 (0)                                            |         |
| Town central heating                 | 152 (57)                                 | 48 (62)                                          |         |
| Heating method [no. (%)]             |                                          |                                                  | 0.25    |
| Radiator                             | 237 (89)                                 | 64 (82)                                          |         |
| Stove                                | 10 (4)                                   | 3 (4)                                            |         |
| Electric heater                      | 12 (5)                                   | 8 (10)                                           |         |
| Other                                | 7 (3)                                    | 3 (4)                                            |         |
| Cooking method [no. (%)]             |                                          |                                                  | 0.43    |
| Tram                                 | 47 (18)                                  | 12 (15)                                          |         |
| Bus                                  | 49 (18)                                  | 22 (28)                                          |         |
| Drive/use taxi                       | 97 (37)                                  | 26 (33)                                          |         |
| Walk/bike                            | 72 (27)                                  | 18 (23)                                          |         |
| Exhaust fan use [no. (%)]            |                                          |                                                  | 0.19    |
| Never/no fan                         | 132 (50)                                 | 45 (58)                                          |         |
| Sometimes                            | 50 (19)                                  | 8 (10)                                           |         |
| ≥ Half time                          | 94 (32)                                  | 25 (32)                                          |         |
| Burn candle at home [yes] [no. (%)]  | 177 (67)                                 | 45 (58)                                          | 0.15    |
| Burn incense at home [yes] [no. (%)] | 61 (23)                                  | 18 (23)                                          | 0.98    |

$^a$Subject was monitored only for personal exposure during late second to early third trimester. $^b$Subject was monitored for personal exposure for a 48-hr period during each trimester, in addition to indoor and outdoor monitoring using an identical instrument. $^c$Low, < 37,024 Poland zlotych (PLZ); medium, 37,024–74,048 PLZ; high > 74,048 PLZ. One Euro = 4.2009 PLZ in 2002. $^d$Subject was monitored for personal exposure only for 48-hr period during each trimester, in addition to indoor and outdoor monitoring using an identical instrument. $^e$Low, < 37,024 Poland zlotych (PLZ); medium, 37,024–74,048 PLZ; high > 74,048 PLZ. One Euro = 4.2009 PLZ in 2002. $^f$Multiple sources are identified for each person. Thus, sources were treated as a separate question. $^g$Eight women (3%) from singly monitored group did not report their fuel type.
as the pregnancy progressed, personal behavior patterns changed. Among 286 women who initially reported no ETS exposure during the first and second trimester, 22 (8%) reported 1–4 hr/day of exposure, and 3 (1%) reported 5–10 hr/day during the third trimester (7% increase). Although 90% \((n = 310)\) reported spending at least 5 hr/day at home during the second trimester, this increased to 98% \((n = 333)\) during the third trimester.

Considering that the type of occupation has been shown to be associated with personal exposure levels of aromatic hydrocarbons (Ibeg et al. 2001), we examined whether this is also true with personal PAH exposure in this cohort. We also examined whether the number of hours spent outdoors, at home, or during commuting were associated with personal PAH exposure. Although a higher proportion of women who worked in restaurants reported that they spent > 3 hr outdoors (50% during April–September, 25% during October–March), the personal exposure concentration during the second trimester was not associated with the number of hours spent outdoors, at home, or in transit \((p > 0.05)\), nor was the type of occupation associated with the personal exposure level \((p > 0.05)\).

**PAH concentrations based on personal, home indoor and home outdoor monitoring.** Table 2 shows the concentration distributions of the nine PAHs and \(\Sigma 8\)-PAH according to the monitoring type and seasons of monitoring [see also Supplemental Material, Table 2 (online at http://www.echonline.org/members/2008/10972/suppl.pdf)]. The nine PAHs and \(\Sigma 8\)-PAH for the personal, indoor, and outdoor levels \((\text{ng/m}^3)\) vary by more than 10-fold between winter (December–February) and summer (June–August). Mean personal exposure to \(\Sigma 8\)-PAH \((\text{ng/m}^3)\) during the winter is 60 ng/m³ higher than the mean during the summer \((p < 0.001)\). Pyrene is the most abundant PAH, accounting for 20–26% of the total concentration during each season. \(B(\beta)F\), \(B(\alpha)P\), \(B(\alpha)A\), and indeno\((1,2,3-cd)P\) are the next most abundant PAHs, and their concentrations are comparable in all seasons. Personal, indoor, and outdoor PAH levels are virtually identical during the June–August period (mean personal – indoor difference = 0.01 ± 0.93 ng/m³; mean personal – outdoor difference = 0.75 ± 1.92 ng/m³). In contrast, the mean personal exposure concentration is higher than the home indoor level and lower than the home outdoor level (mean personal – indoor difference = 10.97 ± 30.66 ng/m³; mean personal – outdoor difference = 29.23 ± 28.07 ng/m³) between December and March (Jedrychowski et al. 2007).

High crude correlation coefficients between the second- and third-trimester personal monitoring values reflect the short temporal gap between the monitoring periods (mean, 6 weeks; range, 5–10 weeks) (Table 3). In contrast, the longer gap between the first and second personal monitoring (mean, 19 weeks; range, 17–23 weeks) yields lower crude correlation coefficients for the nine PAHs. Simultaneously monitored personal, indoor, and outdoor PAHs were highly correlated (pairwise Spearman’s coefficients for the nine PAHs ≥ 0.84, \(p < 0.01)\).

To identify the risk factors of personal exposure, we summarized mean personal PAH concentrations according to the potential risk factors [Table 4; see also Supplemental Material, Table 3 (online at http://www.echonline.org/members/2008/10972/suppl.pdf)]. As expected, season was associated with large variation in personal exposure to the nine PAH levels and \(\Sigma 8\)-PAH \((\text{all } p\text{-values} < 0.01)\). Compared with the nonsmokers, women who reported ≥ 5 hr/days of ETS were, on average, exposed to 0.95–7.48 ng/m³ higher concentrations for the nine PAHs \((\text{all } p\text{-values} < 0.05)\). The mean difference for \(\Sigma 8\)-PAH for the same categories of women was 37 ng/m³ \((p = 0.003)\). Neither the fuel type for home heating nor the outdoor traffic intensity was associated with significantly higher personal exposure. Women who lived near an industrial plant had significantly higher personal exposure to \(B(\alpha)P\), \(1(1,2,3-cd)P\), and pyrene, as well as \(\Sigma 8\)-PAH \((p < 0.05)\). For the women who spent > 3 hr/day outdoors, mean personal exposure to \(\Sigma 8\)-PAH...
was significantly lower \((p = 0.011)\). However, contrary to our expectation, neither the duration of commute nor the time spent at home was associated with any change in personal exposure level. Despite no difference in commuting duration, women who commuted by tram had, on average, 13 ng/m\(^3\) higher personal exposure to \(8c\)-PAH compared with those who walked \((p < 0.01)\). In addition, those who used an exhaust fan during at least half of all cooking time were exposed to an 8 ng/m\(^3\) lower personal \(8c\)-PAH concentration compared with those who did not use a fan \((p = 0.017)\). To further clarify the role of using an exhaust fan, we repeated the comparison in season-stratified data, restricted to nonsmoker households. Although consistent exhaust fan users’ personal \(8c\)-PAH was 10 and 14 ng/m\(^3\).

### Table 2. Personal exposure, indoor, and outdoor levels (ng/m\(^3\)), according to the season at time of monitoring.

|            | March–May | June–August | September–November | December–February |
|------------|-----------|-------------|--------------------|-------------------|
|            | No. | GM | GSD | Min | Max | No. | GM | GSD | Min | Max | No. | GM | GSD | Min | Max | No. | GM | GSD | Min | Max |
| Indoor     | 21  | 3.74 | 0.16 | 55.39 | 16 | 0.56 | 1.71 | 0.22 | 1.86 | 17 | 2.71 | 2.97 | 0.31 | 24.39 | 14 | 14.43 | 2.57 | 24.95 | 38.01 |
| Outdoor    | 18  | 0.65 | 0.02 | 8.88 | 16 | 0.45 | 0.46 | 0.19 | 0.99 | 16 | 0.39 | 0.54 | 0.20 | 0.82 | 16 | 1.22 | 0.70 | 1.67 | 3.43 |
|            | 86  | 2.27 | 0.15 | 28.72 | 81 | 0.45 | 0.56 | 0.20 | 0.70 | 82 | 0.39 | 0.53 | 0.19 | 0.70 | 82 | 1.16 | 0.70 | 1.67 | 3.43 |
|            | 20  | 1.38 | 0.03 | 19.75 | 18 | 0.56 | 0.37 | 0.25 | 0.59 | 19 | 0.57 | 0.46 | 0.25 | 0.59 | 19 | 1.29 | 0.70 | 1.67 | 3.43 |

Abbreviations: GM, geometric mean; GSD, geometric standard deviation; Max, maximum; Min, minimum.

*Season is determined at the time of monitoring, so season for those persons with serial monitoring refers to different calendar periods.
Lower during spring ($p < 0.05$) and winter ($p > 0.10$), respectively, the size of the reduction was $< 3 \text{ng/m}^3$ during summer and fall (both $p$-values $> 0.10$).

Short-term predictors of personal exposure to pyrene and $1,2,3$-c-PAH. We considered the following variables as potential risk factors of personal PAH exposure: residence in the city center, ambient temperature, wind speed, ETS exposure, frequency of exhaust fan use, residence near an industrial plant, commute by tram, apartment height (floor), home heating fuel of coal or wood, time spent outdoors (hour/day), and simultaneously monitored outdoor concentration. Table 5 shows personal exposure models for each PAH and includes only those variables that met our

| Table 3. Crude Spearman correlation coefficients of repeated personal measurements and contemporaneous personal/indoors/outdoor measurements ($n = 68$). |
|---------------------------------------------------------------|
| **PAH** | **Across repeated personal monitoring** | **Simultaneous personal/indoors/outdoor monitoring** |
| | **2nd vs. 1st monitoring** | **2nd vs. 3rd monitoring** | **2nd personal vs. indoor** | **2nd personal vs. outdoor** | **2nd indoor vs. outdoor** |
| B(a)A | 0.19 | 0.54 | 0.96 | 0.98 | 0.97 |
| B(b)F | 0.08 | 0.48 | 0.97 | 0.97 | 0.97 |
| B(k)F | 0.05 | 0.62 | 0.95 | 0.96 | 0.96 |
| B(a)/t)P | 0.15 | 0.44 | 0.95 | 0.97 | 0.97 |
| B(p)P | 0.10 | 0.57 | 0.96 | 0.96 | 0.97 |
| Chrysene | 0.13 | 0.49 | 0.96 | 0.96 | 0.96 |
| D(a)/A | 0.23 | 0.41 | 0.95 | 0.96 | 0.96 |
| I(1,2,3-c)P | 0.19 | 0.43 | 0.96 | 0.97 | 0.98 |
| Pyrene | 0.11 | 0.53 | 0.86 | 0.84 | 0.53 |
| $1,2,3$-PAH | 0.12 | 0.51 | 0.96 | 0.98 | 0.97 |
| Days between monitoring | 88 ± 6 | 47 ± 7 | — | — | — |

| Abbreviations: Max, maximum; Min, minimum. |
| * $p < 0.01$ for all values, based on pairwise Spearman rank correlation. |

| Table 4. Personal exposure concentration (geometric mean and 95% confidence interval) to individual PAHs (ng/m$^3$) for the pregnant women. |
| **Factor** | **B(a)A** | **B(b)F** | **B(k)F** | **B(a)/t)P** | **B(p)P** | **Chrysene** | **D(a)/A** | **I(1,2,3-c)P** | **Pyrene** |
| **Season** | **April–September** ($n = 160$) | | | | | | | | |
| | (0.73–0.92) | (1.4–1.76) | (0.47–0.59) | (0.96–1.07) | (0.73–0.95) | (0.79–0.98) | (0.15–0.19) | (0.33–0.18) | (0.22–0.42) |
| **October–March** ($n = 181$) | 6.65$^*$ | 10.12$^*$ | 3.21$^*$ | 5.45$^*$ | 7.32$^*$ | 5.61$^*$ | 1.24$^*$ | 6.86$^*$ | 9.71$^*$ |
| | (5.8–7.61) | (8.92–11.47) | (2.38–6.85) | (4.83–6.13) | (6.4–8.41) | (4.92–6.39) | (0.91–1.41) | (6.08–7.75) | (8.55–11.02) |
| **Residence** | | | | | | | | | |
| | — | — | — | — | — | — | — | — | — |
| **Outdoor traffic** | | | | | | | | | |
| | — | — | — | — | — | — | — | — | — |
| **ETS/h/day** | | | | | | | | | |
| | — | — | — | — | — | — | — | — | — |
| **Coal/wood heating** | | | | | | | | | |
| | — | — | — | — | — | — | — | — | — |
| **Outdoor temperature** | | | | | | | | | |
| | — | — | — | — | — | — | — | — | — |
| **Industrial plant** | | | | | | | | | |
| | — | — | — | — | — | — | — | — | — |
| **Bus depot** | | | | | | | | | |
| | — | — | — | — | — | — | — | — | — |
| **Total outdoor temperature** | | | | | | | | | |
| | — | — | — | — | — | — | — | — | — |
| **Commute time** | | | | | | | | | |
| | — | — | — | — | — | — | — | — | — |

*Season was determined at the time of monitoring, so for those persons with serial monitoring refers to different calendar periods. $^*$ $p < 0.05$, $^{**} p < 0.01$.
forward selection criterion (type 1 error = 0.10). During the heating season, a 1-ln-unit increase in the outdoor concentration of the individual PAHs and Σ8c-PAH was associated with a 91–100% increase in personal exposure. The efficiency of the outdoor concentration as a predictor precluded other variables. The negative γ-intercept for most PAHs, although not significant, indicates that home concentrations were lower than the corresponding outdoor levels during the heating season.

In contrast, during the nonheating season, a 1-ln-unit increase in the outdoor concentration was associated with a 58–89% increase in personal exposure to the individual carcinogenic PAHs, despite the higher reported likelihood of window ventilation. Only during the nonheating season, ETS and ambient temperature significantly increase personal exposure to B(\(a\))F, B(\(b\),\(h\),\(i\))P, B(\(a\))P, chrysene, 1/1,2,3-cdP, and Σ8c-PAH. A 1-hr increase in ETS exposure was associated with a 10–16% increase in personal exposure to these PAHs. Similarly, a 1°C decrease in ambient temperature was associated with a 3–5% increase in exposure for B(\(a\))A, B(\(d\))F, and D(\(a\),\(b\))A, after accounting for the outdoor concentration. The behavior of pyrene is unique, probably due to its low molecular weight. The distribution pattern and statistical association of the eight PAHs are consistent overall with those for Σ8c-PAH, demonstrating that Σ8c-PAH is an appropriate proxy of exposure to the eight individual carcinogenic PAHs.

### Table 5. Risk factors of personal exposure to PAHs, based on simultaneously monitored levels and other putative predictors.

| Outcome: | Heating season (October–March) | | | Nonheating season (April–September) | | |
|---|---|---|---|---|---|---|
| Personal exposure to PAH | Slope | Heating season (October–March) | R² | ΔR² | Slope | Nonheating season (April–September) | R² | ΔR² |
| ln(\(B(\alpha)A\)) | −0.30 | −0.62 to 0.03 | 0.21 | −0.19 to 0.60 | 0.66 | 0.55 to 0.78 | 0.914 | 0.914 |
| Outdoor ln(\(B(\alpha)A\)) | 0.92 | 0.79 to 1.05 | 0.897 | 0.897 | −0.03 | −0.05 to 0.00 | 0.929 | 0.015 |
| Temperature (°C) | −0.23 | −0.52 to 0.06 | −0.23 | −0.36 to −0.09 | 0.82 | 0.72 to 0.92 | 0.917 | 0.917 |
| Outdoor ln(\(B(\alpha)F\)) | 0.97 | 0.85 to 1.08 | 0.931 | 0.931 | 0.10 | 0.01 to 0.20 | 0.933 | 0.016 |
| ETS (hr) | −0.17 | −0.40 to 0.07 | 0.33 | −0.13 to 0.80 | 0.58 | 0.42 to 0.74 | 0.826 | 0.826 |
| ln(\(B(g,h,i)P\)) | −0.11 | −0.37 to 0.14 | 0.22 | −0.16 to 0.60 | 0.78 | 0.67 to 0.89 | 0.909 | 0.909 |
| Outdoor ln(\(B(g,h,i)P\)) | 0.98 | 0.86 to 1.11 | 0.928 | 0.928 | −0.03 | −0.05 to 0.00 | 0.936 | 0.027 |
| Temperature (°C) | 0.02 | 0.00 to 0.04 | 0.941 | 0.012 | 0.12 | 0.04 to 0.21 | 0.950 | 0.015 |
| ETS (hr) | −0.05 | −0.09 to −0.02 | 0.881 | 0.055 |
| Apt height (floor) | 0.17 | 0.00 to 0.35 | 0.960 | 0.010 |
| ln(\(B(\alpha)P\)) | −0.05 | −0.31 to 0.20 | −0.13 | −0.26 to 0.01 | 0.89 | 0.77 to 1.01 | 0.904 | 0.904 |
| Outdoor ln(\(B(\alpha)P\)) | 0.96 | 0.84 to 1.08 | 0.921 | 0.921 | 0.14 | 0.02 to 0.26 | 0.925 | 0.021 |
| ln(Chrysene) | −0.54 | −0.83 to −0.25 | −0.42 | −0.54 to −0.31 | 0.74 | 0.63 to 0.84 | 0.885 | 0.885 |
| Outdoor ln(Chrysene) | 0.99 | 0.87 to 1.12 | 0.917 | 0.917 | 0.13 | 0.03 to 0.22 | 0.915 | 0.030 |
| ln(D(\(a,H)A\)) | −0.14 | −0.24 to −0.04 | 0.04 | −0.45 to 0.53 | 0.80 | 0.62 to 0.98 | 0.874 | 0.674 |
| ln(\(1,2,3-c,dP\)) | −0.27 | −0.58 to 0.04 | −0.23 | −0.39 to −0.08 | 0.85 | 0.75 to 0.95 | 0.906 | 0.906 |
| Outdoor ln(\(1,2,3-c,dP\)) | 1.03 | 0.90 to 1.17 | 0.914 | 0.914 | 0.16 | 0.06 to 0.25 | 0.930 | 0.024 |
| ETS (hr) | 0.07 | 0.00 to 0.14 | 0.928 | 0.015 | 0.26 | 0.06 to 0.46 | 0.950 | 0.020 |
| Apt height (floor) | 0.02 | 0.00 to 0.04 | 0.940 | 0.012 |
| ln(Pyrene) | −0.30 | −0.74 to 0.14 | 0.08 | −0.14 to 0.31 | 0.60 | 0.45 to 0.75 | 0.733 | 0.733 |
| Commute by tram | 0.87 | 0.72 to 1.02 | 0.865 | 0.865 | 0.29 | 0.01 to 0.57 | 0.783 | 0.049 |
| ln(8c-PAH) | −0.41 | −1.01 to 0.19 | 0.52 | −0.02 to 1.09 | 0.75 | 0.64 to 0.86 | 0.921 | 0.921 |
| Outdoor ln(8c-PAH) | 1.02 | 0.88 to 1.16 | 0.925 | 0.925 | −0.02 | −0.05 to 0.00 | 0.939 | 0.017 |
| Temperature (°C) | 0.02 | −0.00 to 0.04 | 0.934 | 0.009 | 0.10 | 0.01 to 0.19 | 0.949 | 0.010 |
| ETS (hr) | 0.16 | −0.02 to 0.34 | 0.957 | 0.008 |

Abbreviations: Apt, apartment; CI, confidence interval. We forward-selected the model in stepwise model selection strategy, with α = 0.10, from a pool of potential predictors: outdoor concentration of given PAH, residence location (city center = 1, outer area = 0), ambient temperature, wind speed, ETS exposure, frequency of exhaust fan use, live near an industrial plant, commute by tram, apartment height (floor), home heating with fuel of coal or wood, time spent outdoors (hr/day).
and year ($R^2 = 0.74$) or indicator variable of given month and year ($R^2 = 0.73$) was lower than that for the reference group whose personal exposure level we simultaneously monitored with the outdoor level ($R^2 = 0.95$), it is still considerably high (see Figure 2).

Additionally, we estimated individual exposure to $\Sigma 8c$-PAH at a given gestational month as a function of the variables shown in Table 6. Compared with the initial model [Supplemental Material, Table 4 (online at http://www.ehponline.org/members/2008/10972/suppl.pdf)], we achieved a modest gain in model fit in the reduced model (Table 6). Compared with the second trimester, the exposure level of each subject during the third trimester is significantly lower, whereas that during the first trimester is not, adjusted for other covariates. A 1-hr increase in ETS exposure at home was associated with a 7% increase in personal exposure at a given gestational month. Because we suspect that there was an inversion of pollution in the city center during the winter of 2001–2002, we decided to account for the spatiotemporal interactions (December 2001, January 2002, and December and city center, respectively) despite the fact that our model failed to detect a significant increase during these periods.

The range of predicted personal exposure to $\Sigma 8c$-PAH was 3.42–151.41 ng/m$^3$. In the random effects model (Table 6), all predictors are binary except for ETS exposure. As a result, the predicted personal exposures have wider range compared with the range of the predicted variables. The relative cross-validation residuals are clustered randomly around 0, indicating that our final model achieves good prediction (Figure 3). We also compared the prediction ability of our final model with a naïve model based only on trimester and ETS exposure. The estimated prediction error of our final model is 0.81, whereas that of the naïve model is 1.57, which demonstrates a much better prediction ability of our final model compared with the naïve model.

**Discussion**

Investigating health risks of PAH exposure, such as cancer or developmental effects, presents a number of challenges, the greatest of which is the lack of reliable and comprehensive human exposure data (Bostrom et al. 2002). Thus, the PAH monitoring scheme presented here with cross-sectional (simultaneous personal/indoor/outdoor monitoring) and a longitudinal component (repeated personal monitoring), supplemented by an in-depth questionnaire, provides airborne PAH exposure information for a nonoccupationally exposed population [Table 2; see also Supplemental Material, Table 2 (online at http://www.ehponline.org/members/2008/10972/suppl.pdf)].

Our first objective—to descriptively characterize the role of the home indoor and home outdoor setting on personal exposure of pregnant women—resulted in the following two conclusions: First, mean personal exposure during a given month and year is intermediate between indoor and outdoor levels during October–March. During June–August, mean personal exposure is almost identical to both

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**Figure 2.** Estimation of personal (ln) $\Sigma 8c$-PAH based on mean outdoor level (A) or indicator variable for given month and year (B). (A) Personal mean (ln)$\Sigma 8c$-PAH at a given calendar month and year $= \alpha + \beta \times$ (group’s mean outdoor level during given month and year) $+ \text{SE}$. (B) Personal mean (ln)$\Sigma 8c$-PAH at a given calendar month and year $= \alpha + \beta \times$ (indicator variable for given month and year) $+ \text{SE}$.

**Figure 3.** Predicted $\Sigma 8c$-PAH exposure at sixth gestational month versus the observed $\Sigma 8c$-PAH at same gestational period.

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**Table 6.** Random-effects model* of mean personal exposure to (ln)$\Sigma 8c$-PAH at a given gestational month.

| Factor                        | Slope    | SE       | $p$-Value |
|-------------------------------|----------|----------|-----------|
| $\gamma$-Intercept            | 2.3909   | 0.2580   | $<0.0001$ |
| Gestational age               |          |          |           |
| Second trimester              | Reference|          |           |
| First trimester               | -0.1467  | 0.1081   | 0.1756    |
| Third trimester               | -0.4220  | 0.1072   | 0.0001    |
| Household member behavior     |          |          |           |
| ETS at home (hr)              | 0.0677   | 0.0293   | 0.0214    |
| Residence location            |          |          |           |
| Outer area                    | Reference|          |           |
| City center                   | 0.0707   | 0.0915   | 0.4402    |
| Personal monitoring period    |          |          |           |
| May–August                    | Reference|          |           |
| September                     | 0.4418   | 0.1210   | 0.0003    |
| October                       | 1.3573   | 0.1259   | $<0.0001$ |
| November                      | 1.7547   | 0.1587   | $<0.0001$ |
| December                      | 1.8279   | 0.4982   | 0.0003    |
| January                       | 2.1199   | 0.1999   | $<0.0001$ |
| February                      | 2.0851   | 0.1327   | $<0.0001$ |
| March                         | 1.6439   | 0.1265   | $<0.0001$ |
| April                         | 1.2639   | 0.1241   | $<0.0001$ |
| Year 2000                     | Reference|          |           |
| Year 2001                     | -0.5826  | 0.2235   | 0.0096    |
| Year 2002                     | -0.8216  | 0.2245   | 0.0003    |
| Interaction terms             |          |          |           |
| December 2001                 | 0.0366   | 0.3192   | 0.9087    |
| January 2002                  | 0.3416   | 0.2468   | 0.1674    |
| December and city center      | 0.1959   | 0.3840   | 0.6774    |

*The variables listed here are fixed factors, and we considered the person effect a random factor. We did not consider temperature and wind speed as confounders because of high missing values.
the indoor and the outdoor level. A strong season-dependent rise in indoor PAH concentration, combined with the fact that most of the women spent most of their time at home, suggests that the home indoor environment offers little protection from outdoor PAHs, particularly during the heating season.

Second, the observed indoor and outdoor concentrations of B(a)P during spring/fall are comparable to the levels observed in other developed and developing countries. For example, the ambient B(a)P level in a heavily trafficked street in Copenhagen, Denmark, was 4.4 ± 1.2 ng/m³ in 1992 (Nielsen et al. 1996). Mean personal B(a)P exposure for traffic police officers in Beijing, China, was 51.9 ± 84.2 ng/m³ during winter (Liu et al. 2007). The mean indoor level in nonsmoker households during autumn in Hangzhou, China, was 4 ng/m³ (Liu et al. 2001). The mean ambient B(a)P level during the period of coal-fired power plant operation in Tongliang, China, was 16.8 ± 20.1 ng/m³ (Chow et al. 2006). In tropical cities such as Bangkok, Thailand, the median ambient level was 8.08 ng/m³ (Ruchirawat et al. 2002). At the same time, the Krakow B(a)P levels are higher than those in Los Angeles, California, (0.065 ng/m³), Houston, Texas, (0.025 ng/m³), and Elizabeth, New Jersey (0.14 ng/m³) (Naumova et al. 2002); Beauharnois, Quebec, Canada (0.177 ng/m³) (Sanderson and Farant 2004); Zagreb, Croatia (winter mean, 5.12 ± 3.46 ng/m³) (Sisovic et al. 2002), Grenoble, France (summer mean, 0.07 ± 0.02 ng/m³; winter mean, 1.02 ± 0.87 ng/m³) (Nielsen et al. 1996; Tao et al. 2006); and New York City (0.49 ± 0.65 ng/m³) (Choi et al. 2006). However, such international comparisons have limited health inferential value, not only because of the differences in PAH profiles, monitoring methodologies, monitoring durations, laboratory techniques, and instrument measurement errors, but also because of uncertainties in personal behavior choices and time spent in various microenvironmental settings. In the United States, the 10-min average PAH concentrations (nanograms per cubic meter) in various microenvironments, including food courts and buses, have been shown to have large inherent variability (Levy et al. 2002). Thus, short-term acute exposures in certain microenvironments, even in cities with documented low ambient PAH concentrations, might affect health outcomes, particularly in individuals with enhanced predisposition (Levy et al. 2002). Our present observation of lower exposure among the women who stay outdoors for longer hours is consistent with previous time–activity patterns of exposure to PM₂.₅ (Moschandreas and Saksea 2002). That is, microenvironments other than home are likely to provide greater protection among the women who report a higher proportion of daily hours in the nonhome setting.

Our second objective was to identify behavioral and environmental risk factors of personal PAH exposure during a given trimester using direct air monitoring and questionnaire data. Season-dependent outdoor sources are the most important risk factors of Σ8c-PAH exposure for the women in the study (Tables 4 and 5). This confirms a prior observation that coal burning for heating and industry is the main outdoor source of the ambient PAHs in Krakow (Jedrychowski et al. 2003). During the heating season, a 1-in-unit increase in the outdoor concentration of the individual carcinogenic PAHs was associated with a 90–99% increase in the corresponding personal level. Outdoor (ln)Σ8c-PAH was associated with a 100% increase in the simultaneously monitored personal exposure level. The efficiency of the outdoor concentration as a predictor is high: The outdoor level alone accounts for 86–93% of total variability in personal exposure to each carcinogenic PAH. Contrary to our expectation, neither the indicators of spatial variability, including living near the bus depot, crossroad, or in the city center, nor the personal behavior choices, including time spent outdoors or commuting, were associated with higher personal exposure, after accounting for the ambient concentration. Considering that > 80% of the cohort spent < 3 hr/day outdoors, our analysis suggests that most women were exposed to outdoor-originating PAHs in the indoor setting. Although we did not assess the reliability for reporting time duration in specific locations, overall the time spent outdoors was strongly negatively correlated (Pearson’s correlation coefficient = −0.886, p < 0.001) with a derived total time spent in the indoor microenvironment (24 hr minus hours outdoors minus hours in transit). This provides some assurance that self-estimated time spent in general categories is reliable.

A 1°C decrease in ambient temperature was associated with a 2% increase in personal (ln)Σ8c-PAH exposure during the April–September period, after accounting for the outdoor level. Although rigorous examination of the relationship between the concentration, ambient temperature, and pollutant chemistry is beyond the scope of this analysis, this suggests that the ambient temperature drop during early fall contributes to the increase in personal exposure concentration, independent of heating-related PAH generation. Also, only during the nonheating season, a 1-hr increase in home ETS exposure was associated with a 10% increase (95% confidence interval, 1–19%) in personal (ln)Σ8c-PAH.

We examined the within-Krakow generalizability of our cohort’s exposure data by fitting the group’s mean outdoor concentration on personal PAH concentration for the remaining women (n = 266) who lack outdoor measurement (Figure 2A). Overall, a high fit based on the outdoor concentration mean (R² = 0.74) or the indicator variable for given month/year during 2001–2002 period (R² = 0.73) demonstrates that either of these variables could be used to estimate mean personal exposure level for nonsmoking Krakow citizens. Currently, the Polish Environmental Protection Ministry does not conduct routine PAH monitoring in Krakow.

On the other hand, we developed a model of long-term individual levels of exposure (Table 6) specifically for the present cohort. We do not recommended application of the model to other segments of the Krakow population for several reasons. The women in the present study are not representative of the general Polish population. They are non-smokers with high educational attainment and have no preexisting medical conditions. In addition, behavior choices of pregnant women are likely to be different from those of the general Krakow population. Furthermore, a combined contribution of Krakow’s heating sources (i.e., coal burning), industrial activity, geographic terrain, and meteorologic conditions limit the generalizability of the model for populations in other locations. Accordingly, the subset analysis (Table 5, Figure 2) to the entire cohort (Table 6) demonstrates that although short-term indoor levels reflect personal exposure levels more closely, PAH exposure for the duration of pregnancy is significantly influenced by the outdoor PAH level. This is consistent with prior observations of a strong influence of ambient PM concentrations on personal PM exposure levels (Kinney et al. 2006). The mean individual level of exposure was significantly lower during the third trimester compared with the second trimester, controlling for the season, year, and ETS (Table 6). This implies that single 48-hr monitoring during the second trimester is not representative of exposure during the third trimester. Accordingly, we will examine intrauterine growth restriction based on the predicted exposure during all gestational months. During the data collection phase, we suspected that there was an air pollution inversion in the city center during early December. Thus, we retained second-order interaction terms, December 2001, January 2002, and December city center, to account for a brief period of acute exposure.

Because we did not monitor the PAH levels in microenvironments other than the home indoor, we cannot directly determine the relative proportion of total 48-hr personal PAH exposure associated with other microenvironments such as work, personal vehicle, or tram. Other limitations of the analysis include lack of information on building structure, ventilation frequency, and air exchange rate.
Although coal-fired municipal furnaces and industrial activity are most likely to be the main source of the outdoor PAHs (Lovsky et al. 2000), we could not determine the exact extent and duration of these sources during our air-monitoring campaign. Thus, we could not directly measure the relationship between specific sources and the personal exposure levels. Furthermore, we conceptualized season’s effect on PAH concentration as a proxy of ambient temperature (i.e., low temperature is related to personal exposure to PAHs through coal-fired furnaces). Season might have affected the exposure profile for each woman through other mechanisms, such as inversion. Also, we did not directly measure timed activity patterns for each person. Thus, the cumulative exposure over a 48-h period could not be partitioned according to various microenvironments.

The U.K. government’s Expert Panel on Air Quality Standards has recommended an annual average standard for \( B(a)P \) of 0.25 ng/m\(^3\) (Dimashki et al. 2001). Although a similar recommendation does not exist for the United States, the Swedish guideline for \( B(a)P \) exposure is 0.1 ng/m\(^3\) (Bostrom et al. 2002). The personal, indoor, and outdoor \( B(a)P \) levels we observed here far exceeded the recommended level in all seasons and thus are of concern.

In summary, most women in the present cohort are exposed to outdoor-derived PAHs within the indoor setting. That is, indoor concentration is a better predictor of short-term (i.e., 48-h-period) personal exposure; however, long-term personal exposure is largely determined by the outdoor PAH concentration. Mean personal exposure at a given gestational month depends on the season at a given gestational age. Unique demographic attributes of the present cohort and environmental conditions of Krakow mean that personal exposure assessment through a cross-sectional and longitudinal monitoring scheme, supplemented by questionnaire, are critical for a comprehensive understanding of individual-level exposure. Furthermore, the overwhelming influence of the outdoor level on personal exposure means that general Krakow population exposure can be estimated with fairly high precision based on the outdoor mean concentration at given month/year. However, because of the specific behavior patterns of pregnant women, the estimated individual level of exposure based on a random effects model (Table 6) may not be generalizable to the unmeasured Krakow population.

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