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Estimation of Chronic Personal Exposure to Airborne Polycyclic Aromatic Hydrocarbons

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Abstract

Background—Polycyclic aromatic hydrocarbons (PAH) exposure from solid fuel burning represents an important public health issue for the majority of the global population. Yet, understanding of individual-level exposures remains limited.

Objectives—To develop regionally adaptable chronic personal exposure model to pro-carcinogenic PAH (c-PAH) for the population in Kraków, Poland.

Methods—We checked the assumption of spatial uniformity in eight c-PAH using the coefficients of divergence (COD), a marker of absolute concentration differences. Upon successful validation, we developed personal exposure models for eight pro-carcinogenic PAH by integrating individual-level data with area-level meteorological or pollutant data. We checked the resulting model for accuracy and precision against home outdoor monitoring data.

Results—During winter, COD of 0.1 for Kraków suggest overall spatial uniformity in the ambient concentration of the eight c-PAH. The three models that we developed were associated with index of agreement approximately equal to 0.9, root mean square error < 2.6 ng/m³, and 90th percentile of absolute difference 4 ng/m³ for the predicted and the observed concentrations for eight pro-carcinogenic PAH.

Conclusions—Inexpensive and logistically feasible information could be used to estimate chronic personal exposure to PAH profiles, in lieu of costly and labor-intensive personal air

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monitoring at wide scale. At the same time, thorough validation through direct personal monitoring and assumption checking are critical for successful model development.

Keywords

Polycyclic Aromatic Hydrocarbons; Coal Combustion; Exposure Misclassification; Coefficient of Divergence; Exposure Assessment; Household Energy

1. INTRODUCTION

Human exposure to PAH from incomplete combustion of solid fuel, including fossil fuel, is a persistent and poorly controlled global public health threat. This is particularly true for approximately half of the global population which relies on solid fuel burning (Torres-Duque et al., 2008). Airborne PAH is typically adsorbed on fine particulate matter (PM); inhalation remains an important route of exposure (WHO, 2010). Some PAH pose mutagenic and pro-carcinogenic risks (WHO, 2000a). Prenatal and early-postnatal PAH exposure have been associated with elevated risk of DNA damage at birth (Binkova et al., 2007; Jedrychowski et al., 2013; Perera et al., 2005; Rossner et al., 2013), immunodeficiency (Happo et al., 2008; Jeng et al., 2011), respiratory illnesses (WHO, 1987; WHO, 2000a; WHO, 2010), as well as neurodevelopmental impairments during childhood (Perera et al., 2009; Perera et al., 2006; Perera et al., 2012). Accordingly, accurate assessments of a person's exposure in his or her particular stage in the life-course are critical in order to understand more fully the health consequences of PAH.

The task of obtaining information on chronic exposure to airborne PAH at an individual level is limited by a number of factors, however (WHO, 2010). Firstly, PAH compounds form through complex generation mechanisms during combustion (e.g., vehicular or coal combustion) and non-combustion processes within indoor and outdoor settings (Baek et al., 1991; Lau et al., 1997; Li and Ro, 2000; Lung et al., 2003; Zhu et al., 1997). Thus, even relatively small urban and suburban regions are associated with a large spatial (Harrison et al., 2009) and diurnal variability (Arey et al., 1989) in ambient PAH concentrations. Human behavior patterns add additional uncertainty. Factors further influencing variability and hence the prediction of exposure include the individual's proximity to sources (both indoors and out), meteorological conditions, combustion condition and fuel type, and building-related factors such as infiltration, dilution, and removal, among others (Choi and Spengler, 2014; Dubowsky et al., 1999; Li and Ro, 2000; McBride et al., 1999). Secondly, monitoring approaches and laboratory analysis methodologies for PAH remain inconsistent and, overall, cost prohibitive. Thirdly, human exposure conditions in solid fuel-dependent countries are dissimilar from conditions in non-dependent countries. In Poland, where the present analysis takes place, the consumption of coal for home heating is one of the heaviest among the European Union nations (Lvovsky et al., 2000). Coal burning for residential heating in low-efficiency domestic boilers and stoves without any abatement technology is a major source of PAH (Junninen et al., 2009). An estimated >90% of ambient benzo[a]pyrene (B[a]P) and >50% of PM₁₀ are emitted by residential heating (Junninen et al., 2009), while traffic and industrial sources constitute a lesser proportion of city-wide PAH levels. Such practices are associated with dramatic seasonal variability in outdoor B[a]P concentration (range, 1–200

ng/m³) with little spatial variability (Junninen et al., 2009). Finally, outdoor concentrations as measured from the stationary monitoring stations may not accurately represent personal PAH exposure for the healthy and vulnerable segments (e.g., pregnant, young and elderly) of the population who spend the majority of their daily hours indoors (Payne-Sturges et al., 2004). Even when correlation coefficients are high, spatial heterogeneity in pollutant concentration may exist, thus leading to serious exposure misclassification (Bell et al., 2010).

In spite of the need for accurate assessments, directly monitored personal PAH exposure data remain scarce because data collection is expensive and burdensome on the study participants (Harrison et al., 2009). Consequently, direct personal exposure assessments of PAH usually involve a brief monitoring period in a small subset of volunteers (Harrison et al., 2009; Perera et al., 2006). The relatively intrusive and cumbersome nature of the existing monitoring methods yields, at best, ‘snap-shot’ estimation of true personal exposure.

There is a need for method development to assess chronic human exposure to airborne PAH. Prior to our investigation, there has been only one study of personal PAH exposure measured by direct monitoring (Harrison et al., 2009). That study, conducted in 100 adults, yielded models with overall poor fit (range of R^2 , 0.084–0.418), particularly at the higher exposure range (Harrison et al., 2009). In contrast, here we analyze the largest number of simultaneous and direct monitoring measurements of personal exposure, indoor, and outdoor levels (344 women and 649 measurements) within a single study, collected to date (Choi et al., 2008a). Based on our earlier observation, pregnant women in Kraków, our target population of interest, were exposed to a sharp seasonal variation in infiltrated PAH (Choi et al., 2008a). Furthermore, PAH of outdoor origin were the predominant contributor to indoor and personal PAH exposure concentrations (Choi and Spengler, 2014). While our earlier predictive models demonstrated overall excellent fit and performance, they were highly dependent on direct monitoring of personal exposure (Choi et al., 2008a). Our present work was born of the recognition that models with wider application and adaptability require lower data collection cost and logistical imposition on the study participants. Consequently, the primary goal of our present analysis is method development for estimating chronic personal exposures to airborne PAH using outdoor-based data sources. Specifically, we aim to establish a benchmark for PAH hybrid modeling to which other future analyses can be compared. We utilize outdoor-based data sources to estimate an individual’s chronic exposure to airborne PAH. While an outdoor factor-driven model has the benefit of minimizing the logistical burden on study subjects, we also recognize that model validation with direct personal monitoring data remains a critical challenge (Jerrett et al., 2005). Therefore, our specific aims are: (i) to identify outdoor sources, building characteristics, as well as the demographic and behavioral traits of the study participants; (ii) to examine whether intraurban PAH distribution supports the development of ambient factor-driven model; and (iii) to develop an efficient model of chronic personal PAH exposure.

2. METHODS

Details regarding subject enrollment and air monitoring methods have been published (Choi et al., 2012; Edwards et al., 2010; Jedrychowski et al., 2004; Perera et al., 2003).

2.1. Study Site

Kraków, Poland, is located in southwestern Poland. Poland is one of the highest coal producing and consuming country within Europe, and coal combustion- related air pollution in Kraków remains heavy (Anderson, 1995; Junninen et al., 2009; Lvovsky et al., 2000; Nowicki, 1993). The three most prominent air pollution sources in Kraków include industrial and coal-fired power plants (Szafraniec and Jedrychowski, 2001), coal-burning domestic stoves with no or outdated abatement technologies (Junninen et al., 2009; Lvovsky et al., 2000), and automobile traffic (Szafraniec and Jedrychowski, 2001). The city sits within a basin and is surrounded by mountains (Bokwa, 2008). Such a topological feature is associated with atmospheric inversion during approximately 27% of the entire year (Bokwa, 2008). During the 1980s-1990s, overall large intraurban variability in sulfur dioxide (SO₂) and particulate matter with <10 µm in aerodynamic diameter (PM₁₀) were observed (Bokwa, 2008). For example, during 1996, the mean annual concentration of B[a]P was 2-fold higher in the city center, Srod miescie (annual mean 13.3 ng/m³; 22.4 ng/m³ in winter; 4.6 ng/m³ in summer) than the suburban Krowodrza (annual mean 6 ng/m³; 11.2 ng/m³ in winter; 0.9 ng/m³ in summer). However, since this period, the dramatic decline in ambient criteria pollutant concentration has been associated with reduction in intraurban variability in criteria pollutant levels (Bokwa, 2008).

2.2. Subject Enrollment

Briefly, the pregnant subjects were recruited from prenatal care clinics in the city center (Srod miescie–Old Podgorze) and in the outlying suburban areas (Krowodrza–Nowa Huta–New Podgorze). The city center and the suburban area were chosen as the target areas in order to capture putative intraurban variability in ambient PAH concentration. To reduce confounding, only young (ages 18–35 years) and healthy women with no known risks for adverse birth outcomes during their first trimester were eligible. The study was approved by the institutional review boards of Jagiellonian University in Kraków and Columbia Presbyterian Medical Center, New York City, US. Written informed consent was obtained from all study participants.

2.3. Interview

During the late 2nd trimester, a research worker administered an in-depth health, lifestyle, and environmental (HLE) questionnaire to the pregnant women in their homes. The questionnaire elicited information on demographic and socioeconomic status; outdoor environment (live in the same building or next door to a dry-cleaning shop, an industrial plant, car repair shop, restaurant, bus depot, crossroad, or incinerator); quality of the indoor environment (intensity of outdoor traffic, types of industrial activity near home, frequency of opening the window in warmer months, domestic heating method (by hot water pipe radiator, coal stove, electric heater, or forced hot air vents), fuel types (coal stove at home, coal stove in the basement, gas, wood, being supplied with town central heating, or electricity, and amount of vehicular traffic outside the room in which the mothers spent the majority of time at home); active and passive smoking history (number persons who smoke cigarettes, cigars, pipes or marijuana, respectively, at home or at work, as well as hours of exposure per day and months of exposure during pregnancy); dietary intake of PAH-

containing foods (frequency of eating smoked, grilled, blackened items, respectively); and other daily activity patterns (burning candles at home, number of hours spent indoors and outdoors and in mass transit, respectively, as well as mode of transportation during pregnancy and cooking method including use of daily use of exhaust fan over cooking stove). The questions regarding and dietary PAH intake frequency were asked only during the second and third trimester.

2.4. Personal, Indoor, and Outdoor PAH Monitoring

Those who met all the eligibility criteria and agreed to participate were randomly assigned to simple or complex air monitoring subgroups (shown in Table 1). The women in the simple monitoring group were monitored only once during the second trimester for personal exposure ($n = 266$). Those in the complex plan were repeatedly monitored for their personal exposure once every trimester ($n = 78$, final total measurements (j) = 229 after dropping five samples for poor quality), as well as their home indoor ($j = 77$), and outdoor ($j = 77$) levels of PAH and $PM_{2.5}$. Indoor/outdoor monitoring was conducted only once per woman concurrently with her second trimester personal monitoring campaign. The air monitoring campaign was conducted throughout the year between November 2000 and January 2003 (Choi et al., 2008a). Further details can be found in the Supporting Information section.

A set of three identical monitoring devices was used to measure personal, indoor, and outdoor concentrations of eight pro-carcinogenic PAH and pyrene. For personal exposure monitoring, each subject wore a backpack for a consecutive 48-hr period of 785 days (Choi et al., 2008a) in all microenvironments during waking hours. At night time, the subject placed the backpack adjacent to her bed. The backpack was fitted with a lightweight URG-2000-25 Personal Air Sampler (URG, Chapel Hill, NC, USA). The sampling pump (BGI, Waltham, MA, USA) with split flow inlet drew in the air continuously at 2 L/min. Before each monitoring, flow rates were determined using DC-Lite Flow Meter (BIOS International Corporation) which was calibrated using GilibratorTM2 (Gilian®, SENSIDYNE Inc.) standards. The impactor inlet was fastened to the top of the shoulder strap to collect air sample near the woman's breathing zone. The pump flow was split two ways to simultaneously collect particles $2.5 \mu m$ in aerodynamic diameter ($PM_{2.5}$) and PAH. $PM_{2.5}$ was collected on pre-cleaned Personal Exposure Monitor Sampler (PEMS) with teflon filter 37 mm in diameter, PAH were collected on quartz microfiber filter (Palliflex Tissuquartz 2500 QAS, 25 mm in diameter), and semivolatile vapors and aerosols were collected on a polyurethane foam (PUF) plug backup (Kinney et al., 2002). Flow rate after 24-hour and 48-hour of monitoring were determined by DC-Lite Flow Meter and two rotameters, which were calibrated using a Bubble meter. Average airflow was $1.99 \pm 0.04 m^3/minute$, reflecting an equal split of the flow to the personal exposure monitor (PEM) filter and PUF filter, respectively.

As described elsewhere (Choi et al., 2008a; Choi et al., 2008b; Choi and Spengler, 2014), the home indoor and the home outdoor PAH monitoring were conducted concurrently with the personal exposure monitoring. For indoor monitoring, the backpack was placed in a room where the woman spent most of her time at home (i.e. living room, bedroom or near the kitchen). The sampler was placed atop a furniture 0.5–2 m above the floor away from the

heating source or the window. For the outdoor measurement, the backpack was attached to the balcony rails or other appropriate locations, about one meter away from the wall of the home or the apartment. All air monitoring data were assigned a Quality Assurance (QA) score (0–3) for flow rate, flow time, and completeness of documentation (Kinney et al., 2002).

2.5. Laboratory Analysis of PAH in Air Monitoring Samples

After sample collection, the samples were stored at 18 °C. They were shipped to the laboratory within the first 60 days and extracted within 14 days after arrival. Gas chromatography/mass spectrometry (GC/MS) was used to analyze for pyrene and the eight carcinogenic PAH—benz[a]anthracene (B[a]A), Chrysene/isoChrysene, benzo[b]fluoranthene (B[b]F), benzo[k]fluoranthene (B[k]F), B[a]P, 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). No or slight loss (< 20% of pyrene, Chrysene, and benz[a]anthracene) is observed in duplicate spiked sample blanks which simulated the storage and transport conditions between Kraków, New York City, and San Antonio spanning 90 and 107 days (Camann and Whyatt, 2001). Further details can be found in the Supporting Information section.

2.6. Statistical Analyses

PAH compounds were natural-log (ln) transformed in order to achieve normal distributions (Kolmogorov-Smirnov tests > 0.05) and homoscedasticity. The exposure unit of interest was a one ln unit increase in concentration of each PAH compound. The season was defined as summer (June through August), transitional (April, May, September, and October), and winter (November through March) according to the dates of direct monitoring. The relevance of predictor variables were examined using Mann–Whitney U-test or the Kruskal–Wallis test depending on the number of categories for the independent variables at $\alpha = 0.05$ level of significance. There were no PAH concentrations below the detection limit. All extreme and outlying values were double-checked for accuracy in measurement. Upon positive verification, all extreme and outlying values were retained in the data. Considering high mutual correlations of the eight pro-carcinogenic PAH, the composite sum of their concentration ($\Sigma 8$ c-PAH) was considered to examine the importance of subjects' lifestyle choices and behavior pattern on $\Sigma 8$ c-PAH. In the total cohort ($n=344$), descriptive analysis was conducted to identify behavior patterns predictive of an elevated personal exposure to $\Sigma 8$ c-PAH (Table S1). Considering largely bimodal distribution of $\Sigma 8$ c-PAH during winter and summer, we stratified the data into summer, transitional, and winter. During each given season, the distribution of $\Sigma 8$ c-PAH were non-parametrically compared between two categories of independent variables using Mann-Whitney's U-test at $\alpha = 0.05$ level of significance (shown in Table S1).

2.6.1. Spatial Uniformity of Home Outdoor PAH—The distribution of B[a]P adsorbed on ambient $PM_{2.5}$ is homogeneous within Kraków during the winter of 2005 (Junninen et al., 2009). We examined the consistency of this pattern in our 2001–2002 air monitoring data. Overall spatial uniformity in PAH concentration was examined through Pearson's correlation coefficient (r) on ln-scale and coefficient of divergence (COD) on

arithmetic scale (Wilson et al., 2006). As home outdoor measurement reflects an integrated mean over a three-day monitoring period (12, 24, and 12 hrs on days 1, 2, and 3), we attributed it to all three-calendar days in order to obtain 225 days of estimates. Weekly COD was estimated by stratifying the measurements per week irrespective of the home location. The COD of spatial uniformity between home outdoor locations was estimated as:

$$COD_{fh} = \sqrt{\frac{1}{n} \sum_{n=1}^n \left(\frac{X_{if} - X_{ih}}{X_{if} + X_{ih}} \right)^2} \quad \text{eq. [1]}$$

where X_{if} and X_{ih} represent the 48-h average PAH concentration for sampling month i at homes f and h , and where n is the number of observations. COD value is self-normalizing. Thus, a COD value near zero denotes spatial uniformity while the values near one indicates substantial heterogeneity in concentration. A cut-off value for heterogeneous distribution was set as COD = 20% (Wilson et al., 2005).

2.6.2. Personal Exposure Models—Our earlier analysis showed that the subgroup of women ($n=78$) with a complex air monitoring scheme is demographically representative of the larger group with simple monitoring scheme ($n=266$) (Choi et al., 2008a). The subgroup's personal exposure characteristics were also very similar to the singly monitored women (Choi et al., 2008a). Based on this observation, we divided the data according to the training and validation dataset as shown in Table 1. All models were developed on the training dataset and the results of the models checked on validation data. All independent variables were forward selected if the probability of the given variable in the model showed $F < 0.05$ and removed if the probability of the model showed $F > 0.10$. The final set of predictors was chosen for models I, II, and III, respectively, on the training dataset. In addition, “leave-one-out” cross-validation was also performed. The model performance was compared on the validation dataset by considering efficiency and root mean square error (RMSE) “goodness-of-fit” value.

Model I: In the training dataset ($n=78$), the dependent variable, $\ln(PAH)_{ijp}$, represents 48-hour integrated concentration of personal exposure for subject i to PAH compound j during gestational month p . Based on a lack of association between lifestyle, self-reported sources, and time spent in various setting with personal exposure to $\Sigma 8$ c-PAH (Table S1), the independent variables included only secondhand smoke (SHS), as well as the calendar period of interest (i.e. first to last month of pregnancy period).

$$\ln(PAH)_{ijp} = \alpha + \beta \times SHS_{ijp} + \sum_{1}^9 \theta_9(month)_{ijp} + \varepsilon_{ijp} \quad \text{eq. [2]}$$

The calendar period for each gestational month was coded as an indicator variable (e.g. if the first gestational month correspond to January, then January coded as 1; else = 0) with July as the reference month. The coefficient, α , represents the y-intercept; β and θ , respectively represent the slopes for the SHS and the months of gestation. A random error, ε_{ijp} , was assumed to have normal distribution with a mean of zero. Forward step-wise

selection modeling strategy was used with an inclusion criterion of an F-statistic p-value < 0.05 for each considered variable.

Model II: Our earlier analyses showed that PAH of outdoor origin are the predominant contributor to personal exposure (Choi et al., 2008a; Choi and Spengler, 2014). Furthermore, proxy variables, such as home heating demand in degrees and wind speed, significantly predicted the ambient PAH concentrations (shown in Table S2). Specifically, one °C increase in demand for heating is associated with 14–16% increase in ambient (ln) PAH for eight PAH (Table S2). In addition, one meter/second reduction in wind speed was associated with ~50% increase in ambient PAH concentration. Thus, we postulate that HDD, mean wind speed, and self-reported duration of SHS represent an efficient set of proxy for personal PAH exposure concentrations. HDD denotes a temperature range in which household heating is required for thermal comfort of the residents (Silverberg et al., 2013). The HDD range of interest represents ambient temperature of 18 °C and below, calculated by subtracting spatially averaged ambient temperature of given day from 18 °C (Silverberg et al., 2013). Based on our observed ambient temperature range of interest (–16 to 18 °C), the corresponding heating demand ranged 34 to 0 °C. Accordingly, we posit that personal exposure for subject i to PAH compound j on day o is a linear function of the factors shown below:

$$\ln(PAH)_{ijo} = \phi + \eta_1(HDD)_o + \eta_2(wind\ speed)_o + \eta_3(SHS)_{ij} + \varepsilon_{ij} \quad \text{eq. [3]}$$

The coefficient ϕ represents the y-intercept; η_1 , η_2 , and η_3 represent slopes for HDD, wind speed, and SHS, respectively.

Model III: Considering the importance of the outdoor sources, we posit that ambient PAH explains the largest variability in personal exposure regardless of the person's location:

$$\ln(PAH)_{ijo} = \mu + \sigma(\overline{PAH}_{outdoor})_{jo} + \sigma(SHS)_{ij} + \varepsilon_{ij} \quad \text{eq. [4]}$$

That is, the term, $\ln(PAH)_{ijo}$ denotes the subject i 's personal exposure to PAH compound j on day o . $\ln(PAH)_{ijo}$ is postulated as a linear function of spatially averaged home outdoor PAH concentration on a given day o , and self-reported daily mean duration of SHS exposure. Variation of the above model was defined by partitioning, $(\overline{PAH}_{outdoor})_{jo}$ term as within-home and non-home fraction,

$$(\overline{PAH}_{outdoor})_{jo} = (\overline{PAH}_{outdoor})_{jo}((f_h \times \phi_{inf}) + (1 - f_h)) \quad \text{eq. [5]}$$

That is, f_h , represents the daily time fraction that the subject spends at home and ϕ_{inf} , represents the infiltrated fraction of the outdoor concentration. Total daily non-home time fraction is calculated as $(1 - f_h)$. It is calculated as $(1 - \text{sum of hours at occupational setting, commute, and other unspecified microenvironment})$ divided by 24 hrs.

2.7. Validation of the predicted PAH exposure

For the validation analyses, we examined the accuracy of the models by comparing four parameter sizes on the validation dataset (n=266). First, index of agreement (IOA) was calculated as a ratio of mean square error to “potential error” (Willmott, 1981).

$$IOA = 1 - \frac{\sum_i (O_i - P_i)^2}{\sum_i (|O_i - \bar{O}| + |P_i - \bar{O}|)^2} \quad \text{Eq. [6]}$$

P_i denotes predicted concentrations for subject i and O_i denotes observed concentration. IOA ranges between 0 and 1, where 0 indicates no agreement and 1 indicates perfect agreement. Briefly, the IOA estimates the predictive performance of the model for estimating the variation around the observed mean (Willmott, 1981). A cut-off value of $IOA > 0.5$ was considered acceptable (Willmott, 1981).

Second, the model error was estimated using Root Mean Square Error (RMSE) (Willmott, 1981) as shown below,

$$RMSE = \sqrt{\frac{1}{n} \sum_{n=1}^n (P_i - O_i)^2} \quad \text{Eq. [7]}$$

Third, Pearson’s correlation coefficients were estimated between observed versus predicted personal exposure using models I, II, and III, respectively. Pearson’s coefficients were used as indices of collinearity between the observed versus modeled values.

Finally, we also examined the distribution of the difference between observed and modeled absolute concentrations. We considered a model successful if the median error (50th percentile) of the absolute difference for B[a]P was within 100% of the standard value (WHO, 2000b; WHO, 2010). For the cross validation, the relative residual was calculated as (predicted personal exposure concentration – observed personal exposure concentration)/observed personal exposure concentration. One observation was deleted from the data at each model-fitting and used in the test sample to estimate the prediction error. The result of “Leave-One-Out” Cross-Validation for predicted personal exposure to B[a]P using Model 2 is shown (Figure S2). All statistical analyses, including the figure generation, were conducted in SPSS version 22.0 (SPSS Inc., Chicago, IL, USA).

3. RESULTS AND DISCUSSION

3.1. Testing Underlying Assumptions of the Three Models

3.1.1. Individual lifestyle choices and behavior pattern—An improved understanding of the relationship between time-apportioned behaviors and other personal choices is critical for an optimal model development of the personal exposure to PAH (Jerrett et al., 2005). Yet, the relationship between the individual’s daily time activity pattern, lifestyle choices and his/her personal exposure are understudied aspects of personal exposure characterization (Jerrett et al., 2005). Within the present cohort, no behavioral

pattern of the pregnant women was associated with significant variability in their personal exposure to $\Sigma 8$ c-PAH (Table S1). Specifically, neither the occupation category nor the duration of daily work hours during the pregnancy period was significantly associated with an elevated personal exposure to $\Sigma 8$ c-PAH, not only during the winter and transition seasons, but also during the summer. The summer mean personal exposure concentrations to B[a]P (ng/m³) of the present cohort (0.54 ± 1.74 ng/m³) in the present Kraków cohort is comparable to those in other western European (0.21 ± 3.06 ng/m³) populations (Harrison et al., 2009). Thus, they were not considered in all subsequent analysis.

3.1.2. Intraurban Uniformity of PAH Concentrations—In order to develop our personal exposure models, we examined whether ambient PAH were distributed overall uniformly during each season. Spatial uniformity of ambient PAH concentration embodies an important assumption underlying our models because we posit that proxy variables of ambient concentration are sufficient for personal exposure prediction. Other researchers have noted homogeneous distribution of ambient B[a]P adsorbed on PM_{2.5} across Kraków during winter of 2005 (Junninen et al., 2009).

As shown in Table 2, a maximum of two homes were simultaneously monitored during each given week due to the limited number of monitors. Outdoor mean PAH concentrations were often >10-times higher during the winter (November—March) compared to the summer for the eight PAH (Tables 2). In spite of such a difference in the mean concentration, both summer and winter COD values for the eight pro-carcinogenic PAH were very low (i.e., 0.14, except for B[k]F) with high spatial Pearson's r (i.e. > 0.80). In contrast, during the transition season, ambient PAH CODs were more variable for the eight PAH (range 0.28–0.30) with overall lower spatial r values (range 0.45–0.59).

COD represents a more biologically relevant estimate of spatial homogeneity of ambient PAH, compared to spatial correlation coefficients (Bell et al., 2010). Even when spatial correlation coefficients are high during both winter and summer, absolute values could differ considerably across the region of concern (Bell et al., 2010). For example, B[a]P concentration difference of 10 ng/m³ during the winter were markedly different from those at 5 ng/m³ concentration difference during the transition season. Thus, lower outdoor B[a]P COD in Kraków during winter (0.13) than that during the transition period (0.28) imply greater model accuracy during the high exposure period.

3.1.3. Ambient PAH Concentration and Infiltration efficiency—Ambient meteorological variables (i.e., HDD and citywide mean wind speed) were efficient and robust predictors of the home outdoor PAH concentrations (adjusted-R² range, 0.66–0.72) (Table S2). That is, one °C increase in the demand for heating was associated on average with a 14–16% increase in ambient (ln) PAH. In addition, one meter/second reduction in wind speed was associated with ~50% increase in ambient (ln) PAH.

In addition, the estimated infiltration efficiencies for eight PAH (range, 80–90%) in the non-smoking households in Kraków (Choi et al., 2008a; Choi and Spengler, 2014) were higher than those in other geographic locations such as the homes near a smelter in Quebec, Canada (range, 20–50%) (Sanderson and Farant, 2000).

3.2. Models of Personal Exposure to Eight PAH

Table S3–S5 show the fitness of the models I, II, and III on the training dataset. Within model I, the calendar period coinciding with the person's monitoring period explained 70% variability in personal exposure for each PAH compound. In particular, the indicator variables for January, December, and February, in particular, predicted an approximate 110–200% increase in personal exposure for the eight carcinogenic PAH compared to July (i.e. reference period). Indicators for the transitional season (November and March, respectively) were associated with an approximate 70–130% increase in personal exposure for each PAH compared to the reference period.

According to model II, a single °C unit increase in citywide demand for heating associated with 10% increase in personal exposure to each PAH (Table S4). In addition, a single unit (m/s) reduction in citywide mean wind speed was associated with 30–40% increase in personal exposure to each PAH. HDD alone accounted for 59–65% of total variability in personal exposure for the eight pro-carcinogenic PAH.

Model III was associated with an ability to explain 74–80% of the total variability in personal exposure to each of eight pro-carcinogenic PAH (Table S5). In contrast, time-fractionated Model III did not yield notably greater precision (i.e. as judged using an adjusted- R^2) or higher accuracy (using regression coefficients) compared to the unfractionated Model III (Table S5). As a result, we chose unfractionated model III in all subsequent comparisons of model performances.

3.3. Performance of Models I, II, and III

Table 3 and Figures 1, 2, and 3 compare the performances of the modeled concentrations against the observed concentrations. In particular, Figure 1 plots mean modeled concentrations for all eight pro-carcinogenic PAH and pyrene against observed concentrations for the same PAH using three models. All eight particle-bound PAH were associated with precisely predicted concentration. D[ah]A and B[k]F, two PAH of the lowest mean observed concentration, demonstrated particularly precise and accurate predicted concentration. However, pyrene, a semi-volatile and most abundant PAH, was associated with the poorest fit using model I. Figure 2 compares the distributions of the residuals of the predicted B[a]P using the three models against the observed B[a]P concentration. The predicted B[a]P concentration based on Model II was associated with the lowest median residuals at higher exposure range. For example, during winter, the median residuals ranged between 1.68 ng/m³ (model II) and 2.04 ng/m³ (model III). In addition, Figure 3 shows the observed and modeled concentration of B[a]P in terms of median, interquartile, and 5th-to-95th percentile range. Model I and model III assigned a single value during given month. Thus, there was little variability in predicted concentrations. In contrast, model II used a fine scale of exposure (one °C) and averaging time (24-hour window).

As shown in Table 3, both the range and the standard deviations (SD) of the modeled concentrations tended to be narrower than the observed ranges and SDs for all nine PAH.

This reflects the inherent limitations of the models I and III, which assigned a mean value over a month or week of the averaging time (Figure 2 and S1).

Overall, all three models exceeded the criteria of successful fit to the data (based on IOA, RMSE, 50th percentile of the difference, and Pearson's *r*). However, closer comparison of the model fitness demonstrated overall superiority of the models I and II over model III (Table 4). Specifically, IOAs of eight pro-carcinogenic PAH based on model I were 0.90, while the IOAs based on model III ranged from 0.85–0.93 for the same PAH (Table 4). RMSE values, a measure of model error, tended to be lowest for model II and highest for model III. In addition, 90th percentile value for the absolute concentration difference between observed and predicted concentration were lowest for those based on model II, and highest for those based on model III. In contrast, Pearson's correlation coefficients were lowest for the predicted concentrations using model III, compared to the results based on other models (Table 4).

HDD-based Model II offers high efficiency (i.e., two predictor variables) without considerable loss in model precision. It is also important to note that one °C increase in HDD (and same unit reduction in ambient temperature) not only imply increased emissions from the sources but also meteorological conditions for retention and/or secondary generations within the ambient environment. Furthermore, the model assumes a linear relationship, whereas the true relationship might be more complex. In addition, the applicability of model II is bounded within the temporal period with ambient temperature 18 °C. Only during the period of heating demand in Kraków was a single unit increase in HDD associated with ~15% increase in outdoor PAH concentration (Table S3), as well as a 10% increase in personal exposure to PAH (Table S5). Our observation of a strong relationship between HDD and ambient airborne PAH supports the importance of meteorological conditions in personal exposure modeling. Investigations within other European cities also note the relevance of ambient temperature, relative humidity, and the height of the boundary layer as predictors of ambient PAH concentration (Barrado et al., 2013; Callén et al., 2013). High ambient PAH concentrations during the winter were determined not only by local sources (Junninen et al., 2009; Lvovsky et al., 2000) but also by lower photodegradation of environmental PAH (Reisen and Arey, 2005) and typical meteorological episodes of low wind speed and temperature inversion (Junninen et al., 2009). Previous studies in or near Kraków also demonstrated the importance of coal burning to ambient PAH concentration (Junninen et al., 2009; Rogula-Kozłowska et al., 2013).

3.4. Strengths and Limitations

Strengths of the present analysis include large sample sizes for direct personal, indoor, and outdoor monitoring (*j* = 649), which yielded temporally and spatially comprehensive information over a two-year period. The relatively large size of personal exposure monitoring data in the validation dataset (*j* = 266) provided critical validation source. A number of other studies have shown that accurate estimation of chronic human exposure hybrid modeling approach (i.e. direct personal monitoring, as well as information on multiple sources, microenvironments, and human behavior patterns) represent the optimal method to date (Aquilina et al., 2010a; Aquilina et al., 2010b; Delgado-Saborit et al., 2009a;

Delgado-Saborit et al., 2009b; Harrison et al., 2009). A recent review of methods by Jerrett et al. (2005) suggest validation analysis to be a critical step in model development (Jerrett et al., 2005). To the best of our knowledge, the three models within the present analysis demonstrate overall superior performances compared to the model results from earlier analyses. For example, the personal exposure model for the population of London, United Kingdom, accounts for ~35% of the total variance in personal exposure (Harrison et al., 2009). As the B[a]P levels in NYC and London rarely exceed 1 ng/m³ (the UK Standard) regardless of the season (Harrison et al., 2009), complexity of the sources as well as the local meteorological conditions might have contributed to such observation.

Our analyses have several limitations. Firstly, models require validation with up-to-date data to retain their relevance to true population exposure. Unfortunately, no follow-up study has been conducted since 2003 in Kraków to yield more recent data on personal, indoor, and outdoor monitoring data. Thus, direct validation of temporal robustness of our models is not possible. However, more recent measurements of outdoor B[a]P and PM₁₀ by Junninen et al. (2008) during January–February of 2005 suggest temporal persistence of the trend. In our study, the geometric mean outdoor B[a]P concentration during the winter was 9.89 ± 2.36 ng/m³ (range, 1.91–33.12) based on our monitoring (Choi et al., 2008a). In contrast, the arithmetic mean concentration of B[a]P level during the winter of 2005 ranged from 20–56 ng/m³ (Junninen et al., 2009). The only data available on decadal trend between 2000 and 2010 is that of PM₁₀ (manuscript under review). Seasonal variations with elevated winter PM₁₀ levels were clearly observable while there was no substantial reduction of PM₁₀ in the 2000–2010 period. Absence of multiple-year trend in B[a]P and PM₁₀ suggest that our model based on the year 2000–2003 period of monitoring is likely to be temporally robust at least until 2005.

Secondly, due to budgetary constraints, we did not measure other microenvironmental exposures to PAH (e.g., occupation, commute). Therefore, non-home exposure misclassification in our analyses might have contributed to our model uncertainty due to our lack of workplace and other outdoor activity monitoring. More refined estimated daily hour fraction in various microenvironmental settings is not available because we did not ask women to keep a detailed activity log during the personal monitoring campaign. Daily total time at home was validated as 24 hrs — (total hrs away from home) against estimated total hours at home (i.e., hrs awake at home + 7 estimated hrs for sleep). The two variables are highly correlated with mean intraclass correlation coefficient = 0.675 in 75% of the entire cohort. We could not directly examine the role of cooking method, food type, fat content of the food and the quantity of food being cooked. The fat content of the food item, temperature of cooking, as well as the cooking method could influence the PAH emission (Moret and Conte, 2000; Srogi, 2007; Zhu and Wang, 2003).

Thirdly, our questionnaire was not designed to obtain precise information about the characteristics of the residential building, such as the effectiveness of the exhaust fan. We could not examine how the building characteristics influence air exchange rate, particle infiltration, and particle decay rate. In order to clarify whether these unknown outdoor sources influenced personal exposure, we examined the deleted residual against non-home daily hours. There was no apparent pattern between daily hours spent outside of home with

the size of the residual. Finally, as our personal exposure models were developed on rather homogeneous cohort of women, the application is limited to other Kraków residents with similar behavioral and residential traits.

4. CONCLUSION

Our study demonstrates that several feasible proxy variables can predict chronic personal exposure to PAH profiles, in lieu of costly and labor-intensive personal air monitoring. In particular, heating demand in degrees Celsius-based model yielded high efficiency, low model error, and high index of agreement for eight pro-carcinogenic PAH. The region-specific personal exposure model should consider intraurban as well as the short-term temporal variations in toxicants of concern, meteorological conditions, as well as an epidemiologically meaningful validation standard based on individual-level monitoring.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Observed versus modeled mean concentrations for nine PAH using three models. Each shape represents the mean predicted concentration for given PAH. White diagonal line denotes modeled value equaling observed value.

Figure 2. Observed versus the residuals of the modeled concentrations for B[a]P using the three models. The diagonal line denotes perfectly predicted concentration with 0 residual value.

Figure 3.

Observed versus modeled mean concentrations for nine PAH using three models. White diagonal line denotes modeled value equaling observed value.

Table 1

Number of air samples included based on monitoring scheme of 344 women.

Validation (n=266)		Training (n=78)	
Monitoring Type			
Period	Personal ^a	Personal ^a	Outdoor ^c
First trimester		76	
Second trimester	266	77	77
Third trimester		74	
Total measurements			649

^a Each subject wore the personal monitor.

^b The monitor was placed in the room where the women spent most time at home (i.e. living room, bedroom or near the kitchen).

^c Monitor was fastened at home outdoor locations such as balcony railing or other appropriate locations, about one meter away from the wall of the home or the apartment.

Table 2

Home outdoor PAH central tendency statistics, outdoor spatial correlations, and coefficient of divergence.

		f^b	Home A ^a			Home B ^a			Mean COD	r^c
			Mean $\pm$ SD	Min, Max	Mean $\pm$ SD	Min, Max	Mean $\pm$ SD	Min, Max		
B[a]A	Summer ^d	4	0.5 $\pm$ 0.2	0.3, 0.6	0.6 $\pm$ 0.3	0.4, 0.7	0.6 $\pm$ 0.3	0.4, 0.7	0.11	1.00 **
	Transition ^d	14	4.8 $\pm$ 5.3	0.3, 13.6	4.3 $\pm$ 3.3	0.8, 10.9	4.3 $\pm$ 3.3	0.8, 10.9	0.29	0.59
	Winter ^d	16	13.5 $\pm$ 12.1	3.8, 38.0	15.5 $\pm$ 9.6	5.4, 25.74	15.5 $\pm$ 9.6	5.4, 25.74	0.13	0.92 **
B[b]F	Summer ^d	4	0.8 $\pm$ 0.5	0.5, 1.1	1.1 $\pm$ 0.6	0.8, 1.5	1.1 $\pm$ 0.6	0.8, 1.5	0.16	1.00 **
	Transition ^d	14	7.3 $\pm$ 6.4	0.8, 16.8	7.3 $\pm$ 4.1	2.8, 14.2	7.3 $\pm$ 4.1	2.8, 14.2	0.28	0.45
	Winter ^d	16	17.1 $\pm$ 17.5	4.9, 55.8	17.7 $\pm$ 11.0	6.8, 34.6	17.7 $\pm$ 11.0	6.8, 34.6	0.12	0.95 **
B[k]F	Summer	4	0.2 $\pm$ 0.2	0.1, 0.3	0.4 $\pm$ 0.1	0.3, 0.5	0.4 $\pm$ 0.1	0.3, 0.5	0.26	1.00 **
	Transition	14	2.2 $\pm$ 2.0	0.1, 5.3	2.2 $\pm$ 1.1	1.0, 3.9	2.2 $\pm$ 1.1	1.0, 3.9	0.30	0.46
	Winter	16	5.2 $\pm$ 5.0	1.4, 16.2	5.7 $\pm$ 3.7	2.2, 11.5	5.7 $\pm$ 3.7	2.2, 11.5	0.13	0.91 **
B[ghi]P	Summer	4	0.6 $\pm$ 0.4	0.3, 0.9	0.8 $\pm$ 0.7	0.4, 1.3	0.8 $\pm$ 0.7	0.4, 1.3	0.09	1.00 **
	Transition	14	4.0 $\pm$ 3.8	0.4, 9.3	3.6 $\pm$ 3.0	1.3, 9.9	3.6 $\pm$ 3.0	1.3, 9.9	0.28	0.49
	Winter	16	8.3 $\pm$ 8.8	2.5, 29.1	7.8 $\pm$ 3.5	3.8, 13.8	7.8 $\pm$ 3.5	3.8, 13.8	0.12	0.92 **
B[a]P	Summer	4	0.4 $\pm$ 0.2	0.2, 0.6	0.5 $\pm$ 0.3	0.3, 0.7	0.5 $\pm$ 0.3	0.3, 0.7	0.11	1.00 **
	Transition	14	3.7 $\pm$ 3.6	0.3, 9.3	3.4 $\pm$ 2.4	0.8, 7.8	3.4 $\pm$ 2.4	0.8, 7.8	0.28	0.52
	Winter	16	9.9 $\pm$ 10.4	2.5, 33.1	10.4 $\pm$ 6.3	3.6, 19.3	10.4 $\pm$ 6.3	3.6, 19.3	0.13	0.94 **
Chrysene	Summer	4	0.6 $\pm$ 0.2	0.4, 0.7	0.7 $\pm$ 0.4	0.5, 0.9	0.7 $\pm$ 0.4	0.5, 0.9	0.09	1.00 **
	Transition	14	5.2 $\pm$ 5.2	0.4, 13.8	4.8 $\pm$ 3.2	1.3, 10.7	4.8 $\pm$ 3.2	1.3, 10.7	0.28	0.54
	Winter	16	12.0 $\pm$ 12.0	3.1, 37.5	13.1 $\pm$ 7.5	5.2, 24.3	13.1 $\pm$ 7.5	5.2, 24.3	0.14	0.94 **
D[ah]A	Summer	4	0.2 $\pm$ 0.1	0.1, 0.3	0.2 $\pm$ 0.2	0.1, 0.3	0.2 $\pm$ 0.2	0.1, 0.3	0.07	1.00 **
	Transition	14	0.9 $\pm$ 0.9	0.1, 2.3	0.9 $\pm$ 0.9	0.2, 2.7	0.9 $\pm$ 0.9	0.2, 2.7	0.26	0.58
	Winter	16	2.0 $\pm$ 2.1	0.7, 7.0	2.0 $\pm$ 0.8	0.8, 3.3	2.0 $\pm$ 0.8	0.8, 3.3	0.14	0.80 *
IP	Summer	4	0.7 $\pm$ 0.5	0.3, 1.0	0.9 $\pm$ 0.8	0.4, 1.5	0.9 $\pm$ 0.8	0.4, 1.5	0.11	1.00 **
	Transition	14	4.6 $\pm$ 4.4	0.4, 10.4	4.4 $\pm$ 3.7	1.4, 12.1	4.4 $\pm$ 3.7	1.4, 12.1	0.27	0.59
	Winter	16	10.4 $\pm$ 10.1	3.5, 34.0	10.0 $\pm$ 3.9	5.0, 15.8	10.0 $\pm$ 3.9	5.0, 15.8	0.12	0.89 **

^aRepresent any two monitored homes, irrespective of the locations;

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^b Number of available measurements the outdoor COD;
^c Pearson's r; ** significant to the $\alpha=0.01$ level; * significant to the $\alpha=0.05$ level;
^d Summer (June – August); Transition (April, May, Sept, & October); winter (November – March).

Table 3

Descriptive statistics for directly monitored personal exposure concentrations (ng/m³) vs. modelled concentrations.

	<i>Observed</i>					<i>Predicted</i>									
						Model 1					Model 2				
	N	Mean ± SD	Min	Max		Mean ± SD	Min	Max			Mean ± SD	Min	Max		
B[a]A	266	2.64 ± 3.86	0.37	54.60		2.65 ± 3.18	0.66	12.55			2.38 ± 3.40	0.16	26.05		2.09 ± 3.07
B[b]F	266	4.44 ± 3.45	0.37	54.60		4.26 ± 3.04	1.00	18.17			5.11 ± 3.16	0.27	56.26		3.80 ± 2.79
B[k]F	266	1.45 ± 3.46	0.14	20.09		1.44 ± 3.05	0.34	6.11			1.67 ± 3.16	0.09	18.54		1.27 ± 2.76
B[ghi]P	266	2.48 ± 3.21	0.14	20.09		2.49 ± 2.88	0.53	12.81			3.07 ± 2.68	0.25	25.53		2.19 ± 2.68
B[a]P	266	2.82 ± 4.13	0.09	42.10		2.51 ± 3.51	0.47	11.94			3.08 ± 3.43	0.13	39.25		2.13 ± 3.26
Chrysene	266	2.46 ± 3.46	0.14	20.09		2.52 ± 2.87	0.70	10.38			2.86 ± 3.16	0.15	31.19		2.14 ± 2.74
D[ah]A	266	0.50 ± 3.72	0.14	7.39		0.53 ± 3.26	0.10	3.22			0.69 ± 3.16	0.04	7.77		0.44 ± 3.09
IP	266	2.93 ± 3.46	0.25	39.65		2.99 ± 3.09	0.58	16.12			3.88 ± 2.91	0.25	36.97		2.56 ± 2.89
pyrene	266	5.31 ± 2.77	1.00	54.60		1.60 ± 2.29	0.56	7.24			4.86 ± 2.20	0.85	22.65		4.40 ± 2.07
															1.55
															16.78

^a benz[a]anthracene (B[a]A), benzo[b]fluoranthene (B[b]F), benzo[k]fluoranthene (B[k]F), indeno[1,2,3-c,d]pyrene, dibenz[a,h]anthracene (D[a,h]A), and benzo[ghi]perylene (B[ghi]P)

Table 4

Fitness statistics of the Models I, II, and III.

	IOA ^a			RMSE ^b			percentile of difference (ng/m ³) ^c						<i>r</i> ^d		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
							50 th	90 th	50 th	90 th	50 th	90 th			
B[a]A	0.91	0.91	0.86	2.04	2.12	2.38	1.51	3.58	1.69	3.26	1.53	5.06	0.85	0.84	0.79
B[b]F	0.90	0.91	0.87	2.02	1.84	2.18	1.51	3.33	1.55	2.83	1.60	3.65	0.83	0.84	0.79
B[k]F	0.89	0.91	0.87	2.10	1.84	2.19	1.51	3.78	1.55	3.06	1.50	3.81	0.81	0.84	0.78
B[ghi]P	0.91	0.91	0.87	1.93	1.75	2.09	1.51	3.08	1.50	2.79	1.53	3.31	0.83	0.83	0.79
B[a]P	0.91	0.91	0.85	2.16	1.98	2.58	1.60	3.88	1.64	3.61	1.64	5.07	0.84	0.84	0.77
Chrysene	0.90	0.91	0.86	2.03	1.87	2.20	1.51	3.39	1.59	3.22	1.54	4.02	0.82	0.83	0.78
D[a]h]A	0.94	0.92	0.93	2.04	1.92	2.25	1.67	3.20	1.68	3.31	1.53	4.09	0.84	0.83	0.80
IP	0.91	0.91	0.87	1.97	1.82	2.19	1.53	3.08	1.55	2.82	1.54	3.66	0.84	0.84	0.79
pyrene	0.66	0.85	0.79	3.83	1.94	2.12	3.19	7.24	1.55	2.83	1.44	3.96	0.80	0.76	0.70

^a Index of Agreement between observed and modeled.^b Root mean squared error (RMSE) between observed and modeled in ng/m³.^c Percentiles of absolute concentration differences between observed and modeled (ng/m³).^d Pearson correlation between observed and modeled. **Indicates significant to the $\alpha = 0.01$ level.^e benz[a]anthracene (B[a]A), benzo[b]fluoranthene (B[b]F), benzo[k]fluoranthene (B[k]F), B[a]P, indeno[1,2,3-c,d]pyrene, dibenz[a,h]anthracene (D[a,h]A), and benzo[ghi]perylene (B[ghi]P)