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# A multi-day environmental study of polycyclic aromatic hydrocarbon exposure in a high-risk region for esophageal cancer in China

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## Abstract

Linzhou, China has one of the highest rates of esophageal squamous cell carcinoma in the world. Exposure to carcinogenic polycyclic aromatic hydrocarbons (PAHs), such as benzo[*a*]pyrene (BaP), may play a role in this increased risk. To better understand PAH sources, we measured PAHs in the air and food of 20 non-smokers over multiple days and compared the concentrations to a urinary PAH biomarker, 1-hydroxypyrene glucuronide (1-OHPG). Sampling occurred over four consecutive days. Kitchen air samples (days 2–3) and duplicate diet samples (days 1–4) were analyzed for 14 or more unique PAHs, including BaP. Daily urine samples (days 1–3) were analyzed for 1-OHPG. Mixed-effects models were used to evaluate the associations between air or food PAH concentrations and urine 1-OHPG concentrations. The median kitchen air BaP concentration was 10.2 ng/m$^3$ (inter-quartile range (IQR): 5.1–20.2 ng/m$^3$). The median daily food BaP concentration and intake were 0.08 ng/g (IQR=0.04–0.16 ng/g) and 86 ng/day (IQR=41–142 ng/day), respectively. The median 1-OHPG concentration was 3.36 pmol/mL (IQR=2.09–6.98 pmol/mL). In mixed-effects models, 1-OHPG concentration increased with same-day concentration of food BaP (p=0.07). Though PAH concentrations in air were not associated with 1-OHPG concentrations, the high concentrations of PAHs in both air and food suggest that they are both important routes of exposure to PAHs in this population. Further evaluation of the role of PAH exposure from air and food in the elevated rates of esophageal cancer in this region is warranted.

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**Conflict of Interest**
The authors declare no conflicts of interest.

**Keywords**

polycyclic aromatic hydrocarbons; cancer; China; dietary exposure; inhalation exposure; biomonitoring; multimedia exposure assessment

## Introduction

Esophageal cancer is the sixth most common cause of cancer death worldwide, and it occurs in a very uneven geographic pattern. In most parts of the world, the incidence rates of esophageal cancer are lower than 15 cases per 100,000 person-years. However, in certain areas, such as Linzhou (formerly Linxian), China and northeastern Iran, incidence rates approach 100 cases per 100,000 person-years, leading to a lifetime cumulative incidence of nearly 20 % (Ke 2002). In these high-risk populations, nearly all cases of esophageal cancer are esophageal squamous cell carcinoma (ESCC) (Blot et al. 2006). In low-risk countries, smoking tobacco products and heavy alcohol intake are the major causes of ESCC (Freedman et al. 2007), but in high-risk populations these exposures are only modestly associated with ESCC risk (Nasrollahzadeh et al. 2008; Tran et al. 2005). Also, fewer than 1 % of women in these high-risk populations smoke cigarettes or drink alcohol, but the rates of ESCC are similar in women and men (Nasrollahzadeh et al. 2008; Tran et al. 2005). High-risk populations may be exposed to some of the same carcinogenic compounds as those in tobacco smoke and alcohol (polycyclic aromatic hydrocarbons [PAHs], nitrosamines, and acetaldehyde); however the causative agents may come from other sources.

PAHs, such as benzo[*a*]pyrene (BaP), are ubiquitous environmental pollutants produced during incomplete combustion of organic material, and they include known and probable human carcinogens and mutagens (IARC 2010). Our previous studies suggest that high-level environmental exposure to PAHs may be a universal risk factor for ESCC (Abedi-Ardekani et al. 2010; Fagundes et al. 2006; Kamangar et al. 2005; Roth et al. 2001). While tobacco smoke is a major source of PAHs in low-risk ESSC regions, the major sources of PAHs in high-risk regions are less clear. This incomplete understanding has limited our ability to determine the etiologic significance of PAH exposure and to develop effective prevention strategies in these high-risk areas. In Linzhou, China, potential sources of PAH exposure include emissions from cooking and heating with unvented coal, gas, or wood stoves, burning crops, and gasoline-operated scooters and other vehicles. Previous studies in Linzhou have found high concentrations of PAHs in commonly eaten foods (Roth et al. 1998) and in soot deposits from indoor coal stoves (Wornat et al. 2001). In Iran, most PAH exposure is likely due to diet, though specific food sources have not been confirmed (Hakami et al. 2008). Inhabitants of southern Brazil, a moderate ESSC risk region, appear to be exposed to PAHs mainly from smoking, eating *churrasco* (grilled meat), and drinking maté (a tea made from roasted herbs) (Fagundes et al. 2006). Studies in Linzhou, northeastern Iran, and southern Brazil have found high concentrations of a urinary PAH metabolite, 1-hydroxypyrene glucuronide (1-OHPG) (Fagundes et al. 2006; Kamangar et al. 2005; Roth et al. 2001). While 1-OHPG provides a measure of recent PAH exposure, it does not directly provide information on PAH sources.

The current study reports a multi-day evaluation of the relative contributions of food and air to PAH exposure in Linzhou by comparing environmental and biomonitoring measurements. In addition, the relative concentrations of several different PAHs in food and air samples were examined to make inferences about potential sources of exposure.

# Methods

## Study population

Over 80 % of the Linzhou population is composed of rural farmers. Twenty adult participants, aged 35 to 76 years, were recruited from Gu Cheng Village, Rencun Commune, a rural, agricultural village in northern Linzhou. Participants included one male and one female from each of ten non-smoking, farming households. There were 9 husband-wife pairs and one father-daughter pair. In this village, women tend to spend the days indoors, while men spend most of the day working outside in the fields. In 9 of the 10 households, the main fuel for cooking and heating was coal; in one household it was gas. All subjects gave written informed consent. This study was approved by the institutional review boards of the Cancer Institute of the Chinese Academy of Medical Sciences and the U.S. National Cancer Institute. The study was conducted over four consecutive days in October 2006, to evaluate variability in exposures over a short time period and to maximize overlap in the windows of exposure captured by each sample type.

## Air samples

Active air samples were collected from the kitchen of each home on days 1 through 3 to assess the PAH concentrations attributable to the indoor stoves. To characterize personal exposure from all sources on these same days, personal active air samples were collected from one individual per home (8 males; 2 females) while they carried out their normal indoor and outdoor activities. Due to equipment failures resulting from participant misunderstandings of study instructions, all air samples collected from day 1 were excluded from the results and analysis. The instructions were clarified for improved compliance with the protocols for the subsequent days. The personal and kitchen air samples were collected for 8 h, from breakfast until shortly after dinner, because the daytime hours captured the time when the majority of fuel usage occurred. Kitchen and personal air samples were collected following National Institute for Occupational Safety and Health (NIOSH) Method 5506 (NIOSH 1998). The sample collection device consisted of a 37-mm Teflon filter to collect particulate-phase PAHs (SKC, Eighty Four, PA, USA), followed by an XAD-2 sorbent tube to collect gas-phase PAHs (SKC), and an AirChek pump (SKC) calibrated at a flow rate of 2 L/min. Stationary kitchen samplers were placed near the indoor stoves. Personal air samplers were worn by subjects at all times during the sample collection period with the inlet placed within 12 inches of the breathing zone. One field duplicate and one field blank were collected from one kitchen each day. Filters and sorbent tubes wrapped in foil and stored at 0°C or colder until analysis.

The filters and sorbent tubes were analyzed for 52 PAHs. Teflon filters and XAD-2 tubes were extracted using an Accelerated Solvent Extractor (Dionex ASE 200 at 100°C, 10.3 MPa) with dichloromethane (Richter B et al. 1996). The extract was concentrated into approximately 1 mL using a Turbo Vap II (Zymark, Hopkinton, MA, USA). During the concentration process, sample extracts were exchanged into hexane. PAHs were identified and quantified using a capillary gas chromatograph and a mass spectrometer (GC/MS) operated in selected ion monitoring (SIM) mode with helium as the carrier gas, using a 0.2 mm × 25 m column (DB-5MS, 0.33 μm film thickness, J & W Scientific, Inc., Folsom, CA, USA). PAHs were identified by the retention time relative to that of mixed standards (Supelco Separation Technologies, Bellefonte, PA, USA) and confirmed by the abundance of a secondary mass fragment relative to the molecular ion. The filter and sorbent-tube concentrations were summed to yield the total analyte concentration. Blanks were acceptable and revealed no contamination. The median coefficient of variation (CV) across all 52 PAHs was 20% (range: 1 to 42%). The NIOSH Method 5506 does not define an

acceptable CV. The three compounds that are the focus of this paper (naphthalene, pyrene, and benzo[a]pyrene) had CVs of 17%, 22%, and 12%, respectively.

## Food samples

On days 1 through 4, the participants in each home prepared one additional meal serving for all eating events on each day. Participants were instructed to eat identical meals on those days so that the duplicate meal servings collected would represent what was consumed by each participant in the home. Meals were placed in resealable plastic bags and stored in coolers on wet ice until pick-up each evening. Each day's meals were weighed to obtain the total mass of food ingested and then were combined and homogenized in a blender to create one daily food sample per home per day. Two 50-g aliquots were created from each daily food sample. One aliquot was stored at −80°C until analysis; the other was stored at 4°C until all food sample collections were complete. Subsamples of 12.5 g from each of the four 4°C daily aliquots were combined to create a 50-g, 4-day composite sample. The composite samples were then stored at −80°C until analysis.

Daily and composite food samples were analyzed for concentrations of 19 PAHs. Food samples were Soxhlet extracted in 250 mL of methylene chlorine for 16–22 h. Sodium sulfate was added to absorb any water. The extracts were concentrated to 0.5 mL using an automated evaporation system and eluted through an aminopropyl Sep-Pak column (Waters Corporation, Milford, MA, USA). The column was preconditioned with 15 mL of 20% (volume fraction) methylene chloride in hexane, and the samples were eluted using the same mixture. The eluents were concentrated to 0.3 mL using an automated evaporation system.

PAHs were quantified using GC/MS in SIM mode using a 0.25 mm × 30 m fused silica capillary column containing a 5 % (mole fraction) phenyl methyl-substituted polysiloxane phase (HP-5, 0.25 μm film thickness, Agilent Technologies, Wilmington, DE, USA). Helium was used as the carrier gas at a constant flow rate of 1.2 mL/min.

## Urine samples

On days 1 through 3, all 20 participants provided a 10-mL urine specimen from the first morning void and a 10-mL urine specimen from the last evening void. Specimen cups containing collected urine samples were wrapped in aluminum foil and placed in a cooler on wet ice until pick-up by the field team. Each sample was divided into two 5-mL cryovials and stored at −70 °C.

Samples were analyzed for 1-OHPG using immunoaffinity chromatography and synchronous fluorescence spectroscopy in accordance with the method described byStrickland *et al.* (1994). Samples were assayed in batches of twenty. The limit of detection (LOD) was 0.03 pmol/mL. The inter-batch CV of the assay ranged from 18 to 37%. Samples were analyzed for creatinine spectrophotometrically (Perkin Elmer Lambda 5 model) with a commercial kit (Boehringer, Mannheim Germany) based on Jaffe's picrate method (Larsen 1972).

## Statistical Analysis

The analyses presented in this paper focus on the 14 PAHs that were measured in both the air and food samples. Three PAHs (BaP, pyrene, and naphthalene) were selected to represent the range of molecular masses (MWs). BaP is a known human carcinogen with a relatively high MW (252 g/mol) and exists predominantly in the particulate state. Pyrene is ubiquitous in environmental samples and represents a PAH with a mid-range MW (202 g/mol) that exists in both gaseous and particulate states. Pyrene is also the parent compound for the urinary metabolite 1-OHPG. Naphthalene has the lowest MW of the PAHs (128 g/

mol) and exists predominantly in the gaseous state. The concentrations of the 14 PAHs that were measured in both air and food samples were summed to yield a measure of the "total PAH" concentration in each medium. Samples below the LOD were assigned the LOD/2. The rate of detection of the three representative PAHs was 95% or greater in personal air, kitchen air, and food samples. PAH concentrations in personal air, kitchen air, food, and urine were natural log-transformed for all parametric analyses.

Differences in the means of the log-transformed PAH concentrations in personal and kitchen air samples were tested using random-effects ANOVA. PAH concentrations in the personal air samples were compared to the same day's corresponding PAH concentrations in the kitchen air samples for each measured PAH using the Spearman correlation statistic for measurements from days 2 and 3 combined. The concentration of each individual PAH was compared to the concentration of other individual PAHs within the same air sample (for days 2 and 3 combined) for both kitchen and personal air sample types and within each food sample (days 1–4) using the Spearman correlation statistic. The Spearman correlation statistic was also used to compare the mean concentration from the four daily food samples to the concentration in the four-day composite sample for each PAH.

The daily intake of PAHs via air and food was estimated by combining the concentrations of PAHs in air or food with intake rates (i.e., the volume of air breathed in each day or the mass of food consumed each day). For the ten participants with personal air monitoring, the personal air PAH concentrations ($ng/m^3$) were multiplied by a constant, default inhalation rate (15.8 $m^3$/day) to obtain the air PAH intake (in ng/day). This inhalation rate corresponds to that of an adult (male or female) 21 to 41 years of age doing light-intensity physical activity (U.S. EPA 2009). In all 20 participants, the daily food PAH concentrations (in ng/g) were multiplied by the measured mass of food consumed each day (in g/day) to obtain the food PAH intake (in ng/day). Because the meals collected were assumed to represent what each cohabitant consumed, the estimate of food PAH intake was equivalent for the two participants living in the same home. For the ten participants with personal air monitoring, the air and food PAH intakes were summed to obtain a total PAH intake for days 2 and 3. The percent of total intake attributable to either inhalation (calculated as the intake from air/[total intake from air + food] × 100%) or ingestion (calculated as the intake from food/[total intake from air + food] × 100%) was calculated in these ten participants.

Comparing the ratio of specific PAH concentrations to standard cut-points can help identify potential PAH sources, such as coal combustion (e.g., cooking and heating with coal in indoor stoves, power plant emissions); biomass combustion (wood burning, crop burning); petroleum combustion (e.g., vehicle emissions); or unburned petroleum products (e.g., gasoline or oil spills or leaks). Four widely used diagnostic ratios were computed in air and food samples for each home and sampling day. Ratios of anthracene/(anthracene + phenanthrene) greater than 0.1 are indicative of biomass, coal, or petroleum combustion as sources of PAHs, and ratios less than 0.1 are generally due to unburned petroleum products (Jiang et al. 2009; Lv et al. 2009; Yunker et al. 2002). Ratios of fluoranthene/(fluoranthene +pyrene) greater than 0.5 implicate coal or biomass combustion as a PAH source, while ratios from 0.2–0.5 suggest petroleum combustion (Jiang et al. 2009; Lv et al. 2009; Yunker et al. 2002). Ratios of benzo[*a*]pyrene/benzo[*ghi*]perylene in the range of 0.9–.6 implicate coal combustion (Lv et al. 2009). Ratios of benzo[*a*]anthracene/(benzo[*a*]anthracene +chrysene+triphenylene) of 0.2–0.35 are suggestive of petroleum or petroleum combustion, whereas ratios greater than 0.35 are indicative of general combustion (Jiang et al. 2009; Lv et al. 2009; Yunker et al. 2002).

For each PAH sample type, the between-subject ($\sigma^2_{BS}$) and within-subject ($\sigma^2_{WS}$) variance components were obtained using mixed-effects models that did not incorporate any fixed

effects and were used to calculate the intra-class correlation coefficient (ICC) [ICC=$\sigma^2_{BS}$/($\sigma^2_{BS}$ +$\sigma^2_{WS}$)]. The relationships between air PAH concentration and 1-OHPG concentrations were examined using mixed-effects models, where subject was incorporated in the model as a random effect using a uniform correlation structure to account for the repeated measurements collected on the same subject (Eq. 1)

$$\mathrm{Ln}(Y_{ij})=\beta_0+\beta_1 X1_{ij}+b_i+\varepsilon_{ij} \quad (1)$$

where $Y_{ij}$ was the PAH concentration in a given medium (air, food, or urine) for the *i*-th subject on the *j*-th day; $\beta_0$ was the overall mean concentration in the study population; $\beta_1$ was the regression coefficient for the variable $X1_{ij}$; $X1_{ij}$ was the PAH concentration for a given medium (air or food) for the *i*-th subject on the *j*-th day; $b_i$ was the random effect for *i*-th subject; and $\varepsilon_{ij}$ was the residual error associated with *i*-th subject on the *j*-th day. $b_i$ and $\varepsilon_{ij}$ were assumed normally distributed, with a mean of 0, and with between-subject ($\sigma^2_B$) and within-subject ($\sigma^2_W$) variances, respectively.

Creatinine was included as an independent covariate in all models where 1-OHPG was the dependent variable (Barr et al. 2005). Gender and time of day were also evaluated in the models as fixed effects. The relationships between food PAH concentrations and 1-OHPG concentration was examined using the same structure. In total, six PAH metrics were examined in separate models: previous-day PAH concentration in air or food, same-day PAH concentration in air or food, and average PAH concentration in air (arithmetic mean of days 2–3) or food (arithmetic mean of days 1–4). For the analyses using the previous-day air or previous-day food exposure metrics, we restricted the analyses to include only the morning 1-OHPG measurements. Otherwise, both morning and evening 1-OHPG measurements were included. Data were analyzed using Stata 11.0 (Statacorp; College Station, TX ©2009).

## Results

### PAHs in Air

Summary statistics for the air PAH concentrations and intakes for the 14 PAHs are shown in Table 1. In both personal and kitchen air samples, the lower MW PAHs, such as naphthalene, were more abundant by mass than the higher MW PAHs. In random effects ANOVA models, the concentrations of six PAHs were statistically significantly higher in the kitchen air samples than in the personal air samples. The kitchen air PAH concentrations in the home with the gas stove (median BaP concentration=6.2 ng/$m^3$) were consistently lower than those in the houses burning coal (median BaP concentration=11.3 ng/$m^3$). In general, the concentrations of the 14 individual PAHs in kitchen air samples were correlated with the concentrations of the corresponding PAHs in the personal air samples (median Spearman correlation coefficient=0.72; range=0.08–0.85) (data not shown).

The concentrations of the individual PAHs in the kitchen samples were generally highly correlated with other PAHs in the same sample, which supported our used of three indicator PAHs (online supplementary material, Table S.1). Spearman correlations comparing the individual PAHs to each other ranged from 0.67 to 0.99 with a median of 0.90. The magnitude of the correlations between PAHs in the same sample was lower in the personal samples (Spearman correlation range=0.25–1.0; median=0.76) (data not shown).

The between- and within-subject variance components for the three selected PAHs in the kitchen and personal air samples are shown in Table 2. The low ICCs in both sample types (all < 0.50) indicate larger within-subject variability than between-subject variability for all

three PAHs. The highest ICC was observed in the personal air samples for pyrene (ICC=0.49). The lowest ICC was observed in the kitchen air samples for BaP and pyrene (ICC=0.0).

### PAHs in Food

Table 3 shows the distributions of food PAH concentrations and intakes for the four days of sample collection. Similar to the air samples, the highest concentrations were observed in the lower MW PAHs. Spearman correlations comparing the individual PAHs with each other ranged from 0.23 to 0.92 (median=0.59) (data not shown). The food PAH concentrations in the 4-day composite sample and the mean of the 4 daily measurements were correlated, with Spearman correlation coefficients ranging from 0.59 to 0.99 (median=0.94) (data not shown). The ICCs for daily food concentrations of naphthalene, pyrene, and benzo[*a*]pyrene were 0.36, 0.03, and 0.15, respectively (Table 2), indicating that the within-subject variability exceeded the between-subject variability.

### 1-Hydroxypyrene Glucuronide

The medians of the creatinine-adjusted and unadjusted 1-OHPG concentrations for all urine samples were 0.58 (IQR=0.36–0.85) μmol/mol creatinine and 3.36 (IQR=2.09–6.98) pmol/mL, respectively. 1-OHPG concentration did not differ by time of collection (morning or evening) in mixed-effects models (p-value=0.9). 1-OHPG concentrations were higher in males compared to females, but the difference was not statistically significant and disappeared after adjustment for creatinine. The ICCs for creatinine-adjusted and unadjusted 1-OHPG concentrations were 0.18 and 0.36, respectively (Table 2).

### Evaluation of PAH Sources

The median diagnostic PAH ratios suggest a combination of coal and petroleum related sources of PAHs in both the air and food samples (Table 4).

The magnitudes of PAH intake from air and food were similar (Tables 1, 4). The relative contribution to total daily PAH intake was greater from air sources for naphthalene and BaP, with mean percentage of total intake from inhalation of 63% (range=23–88%) and 60% (7–98%), respectively. In contrast, the relative contribution to total daily PAH intake was greater from food sources for pyrene and total PAH, with the percentage of total intake from inhalation of 21% (2–73%) and 46 % (11–80%), respectively (data not shown).

Table 5 presents the parameters and variance components from the linear mixed-effects models that evaluated the association between air and food PAHs with 1-OHPG concentrations. 1-OHPG concentrations increased with increasing same-day concentrations of food BaP, pyrene, and total PAH; the associations were statistically significant for pyrene and total PAH. The same day kitchen and personal air concentrations of BaP, pyrene, naphthalene, and total PAH were not positively associated with 1-OHPG concentrations (all p-values ≥0.68). The previous-day food and air PAH concentrations and the 4-day average food PAH concentrations were not associated with 1-OHPG concentrations (data not shown). The estimated same day total intake from air and food of the representative PAHs was not associated with 1-OHPG concentrations.

The mixed-effects model evaluating the relationship between kitchen air PAH and food PAH concentrations showed that food pyrene concentrations increased with increasing air pyrene concentrations (regression coefficient $\beta$=0.08, p<0.001). The kitchen air concentrations of BaP, naphthalene and total PAH were not associated with the food concentrations of these PAHs.

## Discussion

This study builds on previous studies in Linzhou by evaluating air, food, and urine PAH concentrations to determine if a specific source dominates PAH exposure in this region. High levels of PAHs were observed in multiple media in this non-smoking population. However, air, food, and urine PAH concentrations were relatively homogenous across participants, limiting our ability to more specifically characterize the relationships between these environmental exposures and urinary 1-OHPG.

Kitchen air BaP concentrations in Linzhou (median=10.2 ng/$m^3$, mean=39.6 ng/$m^3$) were 3 to 50 times higher than indoor air concentrations in most previously measured areas, including Chicago (median=0.2 ng/$m^3$) (Li et al. 2005); Los Angeles (geometric mean=0.08 ng/$m^3$) (Naumova et al. 2002); Berlin (median=0.3 ng/$m^3$) (Fromme et al. 2004); Hangzhou, China (mean=4.48 ng/$m^3$) (Zhu et al. 2009); and Agra, India (mean=13.1 ng/$m^3$) (Masih et al. 2010). Though all the homes sampled in Linzhou were non-smoking, the kitchen air BaP measurements were higher than in smokers' apartments in Berlin (median=0.7 ng/$m^3$) (Fromme et al. 2004). However, the BaP concentrations in Linzhou were lower than those observed by Lv *et al.* in 9 houses using bituminous coal as daily fuel in Xuanwei and Fuyuan counties, China (mean=95.7 ng/$m^3$) (Lv et al. 2009). This difference may be related to differences in season, geography, coal type, and/or home ventilation.

Daily dietary intake of BaP in our study (median=86 ng/day; mean=106 ng/day) was similar to that of cases (mean=99.0 ng/day) and controls (mean=91.4 ng/day) in northeastern Iran, another high-risk ESCC region (Hakami et al. 2008). The median dietary BaP intake in Linzhou was higher than the reported intake estimated for the U.S. population (median=40 to 60 ng/day) (Kazerouni et al. 2001) and that observed in the Lower Rio Grande Valley (n=9; median<LOD) (Berry et al. 1997), but lower than that observed in New Jersey (n=8 homes; median=176 ng/day) (Buckley et al. 1995). The BaP concentrations in the Linzhou meals (median=0.09 ng/g) were within the wide range of concentrations measured in cooked meats and grain products in the United States (0.01 to 4.86 ng/g) (Kazerouni et al. 2001), but were lower than the raw (median=13.8 ng/g) and cooked wheat (median=4.6 ng/g) and raw (median=3.1 ng/g) and cooked corn (median=4.9 ng/g) previously measured in Linzhou (Roth et al. 1998).

The urinary 1-OHPG concentrations (median=3.36 pmol/mL) in the current survey were higher than previous measurements in Linzhou (median=2.06 pmol/mL) (Roth et al. 2001), a smoking, maté-drinking population in a moderate-risk ESCC region in Brazil (median=2.09 pmol/mL) (Fagundes et al. 2006), occupationally exposed workers in Korea (mean=2.16 pmol/mL), and current smokers in Korea (mean=1.82 pmol/mL) (Kang et al. 1995a). The Linzhou 1-OHPG concentrations were not as high as those measured immediately following consumption of well-done charbroiled beef (10–83 pmol/mL) (Kang et al. 1995b) or in a high-risk ESCC population in Iran (median=4.2 pmol/mL) (Kamangar et al. 2005).

A previous study in Linzhou indicated that raw wheat had higher concentrations of PAHs than cooked wheat (Roth et al. 1998), raising the possibility that raw foods were exposed to airborne PAH during storage within the home, where foods were generally stored on open surfaces prior to cooking. We examined this issue by evaluating whether or not kitchens with relatively high PAH content in their air samples were more likely to have food samples with a relatively high PAH content. Our results were inconclusive, showing that kitchen air pyrene concentration, but not naphthalene, BaP, or total PAH concentration, was significantly associated with the corresponding food PAH concentration. Additional days of sampling, in more households, will be needed to more fully explore this issue.

We expected the ratios of PAHs in kitchen air, personal air, and food samples to implicate coal and biomass combustion as dominant PAH sources in Linzhou, where these fuels are commonly used for cooking and heating. In addition to coal combustion, our comparison with diagnostic PAH ratios suggested that other sources, such as petroleum-based sources, may also contribute to exposure. Future studies in the region aimed at identifying PAH sources for intervention strategies may want to consider a broader array of combustion sources in addition to the indoor stoves as potential contributors to PAH exposure.

Fuel type has been identified as an important determinant of PAH emissions (Iwegbue 2011); Oanh et al. 2002). We observed lower concentrations of PAHs in the kitchen air of the home with the gas stove than in the homes burning coal, but this finding was based on only 2 measurements collected in one home with a gas stove. The effect of fuel type should be explored as a possible intervention for exposure mitigation.

The within-subject variability for air PAH, food PAH, and 1-OHPG concentrations was much greater than the between-subject variability in this population, which limited our ability to examine the relationship between food and air PAH exposures and 1-OHPG concentrations. Same-day food BaP, pyrene, and total PAH concentrations were associated with 1-OHPG concentrations, suggesting that the dietary PAH exposure is an important route of exposure. Neither air PAH concentrations nor total intake of PAHs from food and air were associated with 1-OHPG concentrations. However, we cannot rule out air as an important contributor to total PAH exposure because of the lack of between-subject variability and the small sample size. In fact, air and food contributed roughly equally to estimated total BaP intake. Although we explored several time relationships of PAH exposure (same day, previous day, and average exposure), we only observed a positive relationship between concurrently measured food PAH concentrations and 1-OHPG concentrations. Based on the half-life of 1-OHPG (6–35 hr) (Buckley and Lioy 1992)), we had speculated that the prior day's environmental measurements would be most strongly related to the subsequent day's biomonitoring measurements. However, without air samples on day 1 or urine samples on day 4, our small numbers limited our ability to fully investigate the temporal relationship between the environmental and biological measurements. Inter-individual differences in metabolism could explain some of the lack of association or lack of strong association between estimated intake from air or food and urine 1-OHPG. Two controlled feeding studies have demonstrated differences in toxicokinetics in participants eating identical meals (Kang et al. 1995b; Buckley et al. 1995).

The current study allowed the evaluation of a number of sampling considerations that may inform the more efficient design of future studies. The correlation analysis of an extensive list of PAHs can serve as a guide for a less comprehensive analytical panel for larger and longer environmental surveys. For example, our study lends additional support to using BaP as a surrogate of total PAHs in air and food samples, because of its relatively strong correlation with other probable carcinogenic PAHs and total PAHs. However, incorporating a select group of PAHs, rather than a single compound, would provide a more complete and reliable exposure profile in an environmental sample (Phillips 1999). In addition, because the PAH values in single composite of the four daily food samples were well correlated with the average of the PAH values of the same daily food samples measured individually, use of single composite sample to represent the average over the included individual days could reduce analytical costs. Because of the high day-to-day variability and low ICCs observed, however, PAH concentration from a single air, food, or urine sample would not reliably predict an individual's long-term exposure in this population.

Strengths of the study were its repeated measures design, the multi-media sampling, and the broad array of PAHs measured. Limitations of this study include the small sample size and

the homogeneity in exposures within the population. In addition, we were unable to account for differences in toxicokinetics between individuals and between the inhalation and ingestion routes. We also lacked detailed information on other potential sources of PAH exposure, such as agricultural burning and vehicle exhaust.

In summary, the PAH concentrations measured in this study in air, food, and urine demonstrate that this Linzhou population is highly exposed to PAHs. These findings provide strong justification for further evaluation of the potential etiologic role of PAH exposure in the elevated rates of ESSC in this area. Because both inhalation and ingestion seem to be important routes of exposure to PAHs in this population, future epidemiologic studies in Linzhou should examine the relationship between both airborne and foodborne PAH sources and ESCC risk.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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**Table 1**

Distributions of selected polycyclic aromatic hydrocarbons (PAHs) in kitchen and personal air samples for days 2 and 3.

| | | Kitchen Air Concentration[a] (n=19) | | Personal Air Concentration[b] (n=20) | | Estimated Daily Air Intake[c] (n=20) | |
|---|---|---|---|---|---|---|---|
| **Polycyclic Aromatic Hydrocarbon** | **Relative Molecular Mass** | **Median (ng/m$^3$)** | **IQR (ng/m$^3$)** | **Median (ng/m$^3$)** | **IQR (ng/m$^3$)** | **Median (ng/day)** | **IQR (ng/day)** |
| Naphthalene | 128 | 584.6 | (354.1, 1114.6) | 508.8 | (275.6, 841.6) | 8040 | (4350, 13300) |
| Fluorene | 166 | 18.6 | (12.9, 27.5) | 19.3 | (30.5, 45.9) | 305 | (181, 482) |
| Phenanthrene * | 178 | 62.0 | (44.2, 117.6) | 35.7 | (24.8, 45.9) | 565 | (391, 725) |
| Anthracene * | 178 | 4.5 | (3.0, 9.1) | 3.3 | (1.9, 4.5) | 52 | (30, 71) |
| Fluoranthene * | 202 | 26.8 | (14.9, 42.3) | 15.7 | (11.2, 22.8) | 247 | (176, 361) |
| Pyrene * | 202 | 23.2 | (12.7, 36.9) | 12.5 | (9.2, 18.6) | 197 | (146, 293) |
| Benz[*a*]anthracene | 228 | 10.8 | (4.0, 21.3) | 6.2 | (3.6, 9.4) | 98 | (57, 148) |
| Chrysene+Triphenylene *[d] | 228 | 16.1 | (9.6, 52.4) | 11.5 | (7.4, 22.4) | 181 | (116, 355) |
| Benzo[*b*]fluoranthene * | 252 | 21.8 | (12.2, 58.8) | 16.2 | (9.9, 31.0) | 255 | (157, 489) |
| Benzo[*k*]fluoranthene | 252 | 4.8 | (2.6, 7.7) | 3.8 | (2.5, 5.4) | 60 | (40, 85) |
| Benzo[*a*]pyrene | 252 | 10.2 | (5.1, 20.2) | 9.0 | (5.1, 12.3) | 142 | (80, 194) |
| Benzo[*e*]pyrene | 252 | 13.2 | (7.6, 36.0) | 9.7 | (5.6, 18.2) | 154 | (88, 287) |
| Perylene | 276 | 2.3 | (1.2, 3.2) | 1.9 | (1.0, 2.3) | 29 | (15, 36) |
| Benzo[*ghi*]perylene | 276 | 11.4 | (6.0, 26.6) | 10.8 | (5.4, 14.2) | 170 | (86, 224) |
| Total PAHs | | 840.2 | (494.1, 1550.2) | 654.0 | (384.8, 1116.1) | 10300 | (6080, 17600) |

[a] All kitchen samples were above the LOD.

[b] All personal samples were above the LOD except fluorene, phenanthrene, anthracene, pyrene, and perylene, which each had one sample below the LOD.

[c] Intake calculated only for subjects with personal air monitoring, using the following formula: air PAH intake (ng/day) = personal air concentration (ng/m$^3$) × 15.8 m$^3$/day.

[d] Analysis unable to separate the two compounds

* Mean log-transformed concentrations were significantly different ($p<0.05$) between personal and kitchen measurements using random effects ANOVA.

## Table 2

Variance components and intraclass correlation coefficients (ICCs) in air, food, and urine 1-OHPG concentrations for selected PAHs.[a]

| | $\sigma^2_{BS}$ (SE) | $\sigma^2_{WS}$ (SE) | ICC[b] |
|---|---|---|---|
| ***Kitchen Air Concentrations (ng/m$^3$) (n=19)***[c] | | | |
| Naphthalene | 0.04 (0.32) | 0.91 (0.42) | 0.03 |
| Pyrene | 0.00 (0) | 1.57 (0.52) | 0.00 |
| Benzo[a]pyrene | 0.00 (0) | 1.64 (0.55) | 0.00 |
| ***Personal Air Concentrations (ng/m$^3$) (n=20)***[d] | | | |
| Naphthalene | 0.13 (0.24) | 0.60 (0.27) | 0.18 |
| Pyrene | 0.60 (0.46) | 0.64 (0.29) | 0.49 |
| Benzo[a]pyrene | 0.30 (0.36) | 0.75 (0.34) | 0.29 |
| ***Food Concentrations (ng/g) (n=40)***[e] | | | |
| Naphthalene | 0.04 (0.03) | 0.07 (0.02) | 0.36 |
| Pyrene | 0.008 (0.04) | 0.25 (0.06) | 0.03 |
| Benzo[a]pyrene | 0.15 (0.18) | 0.84 (0.22) | 0.15 |
| ***Urine 1-OHPG Concentrations (n=120)***[f] | | | |
| 1-OHPG (pmol/mL) | 0.53 (0.22) | 0.93 (0.13) | 0.36 |
| Creatinine-adjusted 1-OHPG (μmol/mol creatinine) | 0.05 (0.03) | 0.23 (0.03) | 0.18 |

PAH, polycyclic aromatic hydrocarbons; $\sigma^2_{BS}$, between-subject variance; $\sigma^2_{WS}$, within-subject variance; SE, standard error

[a] Variance components obtained from random intercept models of natural log-transformed PAH concentrations.

[b] $ICC=\sigma^2_{BS}/(\sigma^2_{BS}+\sigma^2_{WS})$

[c] 10 homes days 2 and 3, with 1 missing sample.

[d] 10 participants, days 2 and 3.

[e] 10 homes, days 1 through 4.

[f] 10 homes, 2 participants per home, days 1 through 3, morning and evening samples.

**Table 3**

Distributions of food PAH concentrations and food PAH intakes, days 1–4 (n=40).

| | | | Concentration (ng/g) | | Intake (ng/day) | |
|---|---|---|---|---|---|---|
| **Polycyclic Aromatic Hydrocarbon (PAH)** | **Molecular Weight** | **% Samples < LOD[a]** | **Median** | **IQR** | **Median** | **IQR** |
| Naphthalene | 128 | 0 | 4.19 | (3.44, 5.54) | 3500 | (2820, 5360) |
| Fluorene | 166 | 0 | 0.85 | (0.54, 1.06) | 638 | (397, 1110) |
| Phenanthrene | 178 | 0 | 3.17 | (2.28, 4.40) | 2490 | (1680, 4730) |
| Anthracene | 178 | 0 | 0.22 | (0.15, 0.47) | 236 | (121, 410) |
| Fluoranthene | 202 | 0 | 1.28 | (0.90, 1.73) | 1081 | (702, 1850) |
| Pyrene | 202 | 0 | 0.99 | (0.78, 1.31) | 1020 | (556, 1310) |
| Benz[*a*]anthracene | 228 | 0 | 0.25 | (0.17, 0.37) | 280 | (179, 579) |
| Chrysene+Triphenylene[a] | 228 | 0 | 0.51 | (0.34, 0.84) | 473 | (323, 684) |
| Benzo[*b+j*]fluoranthene[a] | 252 | 0 | 0.33 | (0.23, 0.51) | 303 | (179, 498) |
| Benzo[*k*]fluoranthene | 252 | 40 | 0.03 | (0.03, 0.10) | 44 | (23, 85) |
| Benzo[*a*]pyrene | 252 | 10 | 0.08 | (0.04, 0.16) | 86 | (41, 142) |
| Benzo[*e*]pyrene | 252 | 0 | 0.17 | (0.09, 0.33) | 153 | (77, 260) |
| Perylene | 276 | 20 | 0.05 | (0.03, 0.12) | 46 | (33, 97) |
| Benzo[*ghi*]perylene | 276 | 50 | 0.03 | (0.03, 0.34) | 36 | (23, 250) |
| Total PAH | | | 13.5 | (9.59, 18.4) | 10800 | (7630, 1780) |

LOD, limit of detection; IQR, inter-quartile range

[a]Analysis unable to distinguish between the two compounds.

## Table 4

Distributions of diagnostic PAH ratios in the air and food samples.

| PAH Ratio | Characteristic Sources[a] | Median (Range) | | |
|---|---|---|---|---|
| | | Kitchen Air | Personal Air | Food |
| Ant/(Ant+Phen) | >0.1: coal, petroleum, or biomass combustion<br><0.1: unburned petroleum | 0.08 (0.03, 0.19) | 0.09 (0.05, 0.12) | 0.07 (0.03, 0.41) |
| Fla/(Fla+Pyr) | >0.5: biomass & coal combustion<br>0.2–0.5: petroleum combustion<br><0.2: unburned petroleum | 0.55 (0.48, 0.60) | 0.56 (0.45, 0.73) | 0.57 (0.38, 0.67) |
| BaP/BghiP | 0.9 to 6.6: coal combustion | 0.86 (0.46, 1.39) | 0.91 (0.59, 1.10) | 1.12 (0.04, 23.26) |
| BaA/(BaA+Chr+Tri) | 0.2 to 0.35: petroleum or petroleum combustion<br>>.35: general combustion | 0.30 (0.19, 0.43) | 0.32 (0.17, 0.39) | 0.32 (0.23, 0.47) |

[a] Lv et al. 2009, Jiang et al., 2009; Yunker et al. 2002

Ant, anthracene; Phen, phenanthrene; Fla, fluoranthene; Pyr, pyrene; BaP, benzo[*a*]pyrene; BghiP, benzo[*ghi*]perylene; BaA, benz[a]anthracene; Chr, chrysene+triphenylene

## Table 5

Relationship between same-day PAH exposures and urinary 1-hydroxypyrene (1-OHPG) concentration.[a,b,c]

| | Benzo[*a*]pyrene | | Pyrene | | Naphthalene | | Total PAH | |
|---|---|---|---|---|---|---|---|---|
| | **Fixed Effect Estimate (SE)** | **P-value** | **Fixed Effect Estimate (SE)** | **P-value** | **Fixed Effect Estimate (SE)** | **P-value** | **Fixed Effect Estimate (SE)** | **P-value** |
| Kitchen Air Concentration (ng/m$^3$)[d] (n=80) | 0.02 (0.08) | 0.76 | −0.003(0.08) | 0.98 | −0.01 (0.10) | 0.92 | −0.01 90.10) | 0.91 |
| Personal Air Concentration (ng/m$^3$)[e] (n=40) | −0.03 (0.10) | 0.79 | 0.04 (0.10) | 0.68 | −0.009 (0.11) | 0.94 | 0.02 (0.12) | 0.87 |
| Food Concentration (ng/g)[f] (n=120) | 0.15 (0.08) | 0.07 | 0.54 (0.21) | 0.01 | −0.23 (0.36) | 0.52 | 0.71 (0.29) | 0.02 |
| Food Intake (ng/day)[f] (n=120) | −0.23 (0.19) | 0.22 | 0.33 (0.18) | 0.07 | −0.16 (0.26) | 0.52 | 0.36 (0.23) | 0.12 |
| Total Intake from Personal Air and Food (ng/day)[e] (n=40) | −0.10 (0.14) | 0.47 | 0.17 (0.21) | 0.44 | −0.042 (0.18) | 0.82 | 0.06 (0.23) | 0.80 |

[a] All variables were natural log-transformed.

[b] Creatinine was included in all models as an independent covariate. Sex, day, and time of day were non-significant and were not included in the models.

[c] Each metric was examined in a separate regression model.

[d] 20 participants, days 2 and 3, morning and evening.

[e] 10 participants, days 2 and 3, morning and evening.

[f] 20 participants, days 1 though 3, morning and evening.