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Risk of human exposure to polycyclic aromatic hydrocarbons: A case study in Beijing, China

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ABSTRACT

Polycyclic aromatic hydrocarbons (PAHs) can cause adverse effects on human health. The relative contributions of their two major intake routes (diet and inhalation) to population PAH exposure are still unclear. We modeled the contributions of diet and inhalation to the overall PAH exposure of the population of Beijing in China, and assessed their human incremental lifetime cancer risks (ILCR) using a Mont Carlo simulation approach. The results showed that diet accounted for about 85% of low-molecular-weight PAH (L-PAH) exposure, while inhalation accounted for approximately 57% of high-molecular-weight PAH (H-PAH) exposure of the Beijing population. Meat and cereals were the main contributors to dietary PAH exposure. Both gaseous- and particulate-phase PAHs contributed to L-PAH exposure through inhalation, whereas exposure to H-PAHs was mostly from the particulate-phase. To reduce the cancer incidence of the Beijing population, more attention should be given to inhaled particulate-phase PAHs with considerable carcinogenic potential.

1. Introduction

Polycyclic aromatic hydrocarbons (PAHs) emitted from the incomplete combustion of fossil fuels or biomass have attracted widespread public concern because of their adverse effects on human health, including carcinogenicity, teratogenicity, and mutagenicity (Boström et al., 2002). PAHs are ingested into the human body mainly through diet and inhalation (ACGIH, 2005). The relative contributions of the two routes to the total level of PAH exposure in the general population are crucial for PAH exposure assessment, especially in China with its high PAH emissions (Zhang et al., 2007, 2009). Unfortunately, the studies conducted to date are limited and have yielded somewhat inconsistent conclusions. Studies conducted in some East Asian regions have revealed that dietary exposure to PAHs contributes more to the overall exposure level of the local population; e.g., for the sum of the 16 EPA priority-controlled PAHs (ΣPAH_{16}) in Tianjin, China (Li et al., 2005), benzo[a]pyrene equivalent PAH (BAP_{eq}) in Taiyuan, China (Xia et al., 2010, 2013), and pyrene, benzo[b]fluoranthene, and benzo[a]pyrene in Tokyo, Japan (Suzuki and Yoshinaga, 2007). However, in the United States, the primary routes of exposure to low-molecular-weight PAHs (L-PAHs, generally PAHs with $\leq$4 benzene rings), including naphthalene, fluorene and pyrene, were inhalation, whereas BAP exposure was predominantly from food intake (Shin et al., 2013). This suggests that the major PAH exposure route varies among populations in different areas and according to PAH molecular weight. Previous studies have focused on total PAHs, BAP_{eq}, or a limited range of PAHs to investigate the relative contributions of various exposure routes to the overall exposure level of the general population. A critical knowledge gap remains with respect to the contribution of the 16 individual U.S. Environmental Protection Agency (EPA) priority-controlled PAHs.

The bio-accessibilities of different PAHs greatly depend on their physicochemical properties. For example, fine particulate matter with a relatively high content of high-molecular-weight PAHs (H-PAHs, generally PAHs with >4 benzene rings) can penetrate deep into the lungs when inhaled, resulting in greater bio-accessibility than L-PAHs (Ohura et al., 2005). Therefore, it was proposed that PAHs in the particulate phase might pose a greater adverse health effect on the human body than PAHs in the gaseous phase (Li et al., 2005; Zhang et al., 2009). Regarding dietary exposure, the difference in PAH bio-accessibility among food types is becoming a

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concern. It has been reported that the PAH concentrations vary among food types and according to PAH molecular weight (Xia et al., 2010; Yu et al., 2011). For these reasons, a systematic and detailed survey of the relative contributions of diet and inhalation is necessary.

Beijing is the capital of China, with the highest population (about 21 million in 2013) and vehicle volumes (about 5.4 million in 2013) in northern China. Both the annual energy consumption and PAH emission density in this area account for a great proportion of the national total (Zhang et al., 2007). The annual-average concentrations of the sum of the 15 EPA priority-controlled PAHs (ΣPAH_{15}), excluding naphthalene, in Beijing urban air is reported to be about 206 ng m^{-3}, with a BAP concentration of about 6 ng m^{-3}, which is considerably higher than the national standard (1 ng/m^3) (Li et al. 2014). Yu et al. (2011) reported that the median concentrations of ΣPAH_{15} in human milk, placenta, and umbilical cord blood for the Beijing population were 278, 819, and 1372 ng g^{-1} of fat, respectively, which were higher by almost an order of magnitude than corresponding levels in Japan and the United States, which may be caused by the higher PAH concentrations of various food types in the local area (Yu et al., 2011). An epidemiological investigation showed that there were 40,307 new cases of malignant tumors in Beijing in 2012 (Beijing Municipal Government, 2014), which was twice the level of 10 years ago. Among them, lung cancer was ranked first, followed by colorectal, liver, stomach, and prostate cancer for males. The ranking order for females was thyroid carcinoma, followed by breast, lung, colorectal, and uterine cancer. Animal experiments have shown that the position of a tumor is associated with the route of PAH exposure (Hecht, 1999; Latif et al., 2010). In the Beijing population, we found that L-PAHs could more readily penetrate the barrier between placenta and umbilical blood than H-PAHs (Yu et al., 2011). Our recent study showed that PAH concentrations in maternal serum had a strong association with the increased risks of fetal neural tube defects, but no relationships between human serum PAH concentrations and indoor air pollution were found (Wang et al., 2015). To evaluate the overall PAH exposure, the route from food consumption must be taken into consideration. Therefore, the relative contributions of diet and inhalation routes to the overall PAH exposure level of the Beijing population are important, and information is urgently required for environmental scientists, policy makers, and local residents of Beijing.

The aims of this study were to investigate: 1) the exposure levels of the Beijing population to 15 individual U.S. EPA priority-controlled PAHs through the diet and inhalation routes; 2) the main contributors to dietary and inhaled exposure to PAHs in the population; and 3) the potential cancer risk for the Beijing population caused by PAH exposure.

2. Materials and methods

2.1. Target population and PAHs of concern

The target population was residents of Beijing, with ages ranging from 1 to 72 years. The population was divided into two gender groups (male and female). Each group was further divided into four subgroups by age: children (1–6 years old), adolescents (7–18 years old), adults (19–65 years old), and seniors (66–72 years old). The following 15 U. S. EPA priority-controlled PAHs were selected: acenaphthylene (ACY), acenaphthene (ACE), fluorene (FLO), phenanthrene (PHE), anthracene (ANT), fluoranthene (FLA), pyrene (PYR), benzo[a]anthracene (BAA), chrysene (CHR), benzo[b]fluoranthene (BBF), benzo[k]fluoranthene (BKF), benzo[a]pyrene (BAP), indeno[1,2,3-cd]pyrene (IcdP), dibenzo[a,h]anthrancene (DahA), and benzo[g,h,i]perylene (BghiP). Naphthalene was excluded because of its higher volatility and poor quantification. Seven of the PAHs were classed as L-PAHs (ACY, ACE, FLO, PHE, ANT, FLA, and PYR) and eight as H-PAHs (BAA, CHR, BBF, BKF, BAP, IcdP, DahA, and BghiP).

2.2. Dietary exposure estimates

The residue levels in seven food categories—fruits, vegetables, cereals, fish, meat, eggs, and milk—of all selected PAHs were reported by us previously (Yu et al., 2011). To the best of our knowledge, the reported PAH concentrations of the seven food categories in Beijing are the only comprehensively published data. The amounts of the various food categories consumed and body weight of all subgroups were obtained from the Chinese national health and nutrition survey report (Zhai and Yang, 2006), and the details are listed in Table S1 in the Supplementary Information. The data for the concentration of PAHs in foodstuffs were tested to determine if they followed a logarithmic normal distribution, while food consumption and body weight were found to approximately follow a normal distribution. The dietary exposure level (*ED*, ng person^{-1} day^{-1}) of PAHs is the sum of exposures from intake of the seven food categories as follows:

$$ED = \sum C_i \times FI_i \quad (1)$$

where C_i and FI_i are the PAH concentration (ng g^{-1}) and intake rate (g day $^{-1}$) of food category (i) according to age group in Beijing. The body-weight (*BW*) adjusted dietary exposure to PAHs (ng kg^{-1} day^{-1}) was calculated by dividing *ED* by body weight (*ED*/*BW*). It should be noted that the differences in the bioaccessibility of PAHs in various food categories were neglected in our study because of the limited data available.

2.3. Inhaled exposure estimates

The inhaled exposure level (*EI*, ng person^{-1} day^{-1}) was calculated as:

$$EI = (C_{gPAH} + C_{pPAH}) \times BR \quad (2)$$

where C_{gPAH} (ng m^{-3}) and C_{pPAH} (ng m^{-3}) are the concentrations of gaseous-phase (gPAHs) and particulate-phase (pPAHs) PAHs in Beijing, respectively, as reported in our previous study (Liu et al., 2007a, b) and their detailed description was provided in the Supplementary Information. *BR* (m^3 day^{-1}) is the breathing rate of various age groups in Beijing; i.e., 9.3, 15, 16.5, 13 (m^3 day^{-1}) for children, adolescents, adults, and senior males, respectively, and 8.6, 12, 11, 9.9 (m^3 day^{-1}) for the corresponding subgroups in the female group, respectively (Wang et al., 2010, 2009). The *BR* variation was assumed to be 10%. The body weight-adjusted inhaled exposure dose of PAHs (ng kg^{-1} day^{-1}) was calculated by dividing *EI* by body weight (*EI*/*BW*). It was assumed that there was no difference in the bioaccessibility of the gaseous- and particulate-phase PAHs and the same equation was used to assess their PAH exposure levels, therefore, these results should be interpolated with care.

2.4. Carcinogenic risk assessment

The incremental lifetime cancer risk (ILCR) was used to express the carcinogenic risk caused by PAH exposure, as follows:

$$ILCR = (E \times SF \times E_D \times CF \times EF)/(BW \times AT) \quad (3)$$

where E (ng person^{-1} day^{-1}) is the exposure level of BAP_{eq}; *SF* is the