Science of the Total Environment 443 (2013) 857–863

Contents lists available at SciVerse ScienceDirect

Science of the Total Environment

journal homepage: www.elsevier.com/locate/scitotenv

Human exposure to parent and halogenated polycyclic aromatic hydrocarbons via food consumption in Shenzhen, China

Chao Ding [a], Hong-Gang Ni [a,*], Hui Zeng [a,b]

[a] Shenzhen Key Laboratory of Circular Economy, Shenzhen Graduate School, Peking, University, Shenzhen 518055, China
[b] Department of Ecology, College of Urban and Environmental Sciences, Peking University, Beijing 100871, China

HIGHLIGHTS

- Dietary exposure to parent and halogenated PAHs for Shenzhen population was higher than that in other countries.
- The cancer risks induced by parent and halogenated PAHs were between 10^{-6} and 10^{-4}.
- Children faced the highest cancer risk, followed by adolescents and adults.
- Given all human exposure routes were considered, the cancer risk would be greater.

ARTICLE INFO

Article history:
Received 15 August 2012
Received in revised form 4 November 2012
Accepted 5 November 2012
Available online 12 December 2012

Keywords:
Parent and halogenated PAHs
Food
Human exposure
Cancer risk
Shenzhen

ABSTRACT

Human exposure to polycyclic aromatic hydrocarbons (PAHs) and halogenated PAHs (HPAHs) via food consumption is still not clear in south China so far. The goals of this work are to assess human exposure to parent and halogenated PAHs via food ingestion and the cancer risk for population in Shenzhen, a new urban center in south China. Sixteen PAHs and nine HPAHs were determined in vegetable, pork and rice samples collected from Shenzhen. In general, the population in Shenzhen was exposed to higher levels of PAHs via food ingestion in comparison to that reported for other countries in recent years, but lower than that estimated for two northern cities in China. The cancer risk values induced by exposure to PAHs and HPAHs for male and female on each subgroup were between the serious risk level (10^{-4}) and the acceptable risk level (10^{-6}). Children faced the highest cancer risk, followed by adolescents, seniors and adults.

1. Introduction

Since polycyclic aromatic hydrocarbons (PAHs) were regarded as carcinogenic and toxic to animals and humans, their distributions in the environment and exposure to human have generated great interest (Boström et al., 2002; Menzie et al., 1992). But people seldom notice that halogenated PAHs (HPAHs) have a larger toxicological affect on life body than their corresponding parent PAHs (Ohura et al., 2009). Therefore, different human exposure routes to PAHs have been assessed in previous studies (Fiala et al., 2001; Tang et al., 2009; Vyskocil et al., 2000; Zeng et al., 2010; Zhang et al., 2009). Although HPAHs have been detected in various environmental samples (Ohura et al., 2005; Sun et al., 2011), very limited information concerning their profiles in food was available so far (Ding et al., 2012). Of human exposure routes to PAHs, dietary ingestion has been identified as the principal route (>70%) for nonsmokers and non-occupationally exposed populations (Alomirah et al., 2011; Falco et al., 2003a; Fontcuberta et al., 2006; Menzie et al., 1992; Phillips, 1999; Vyskocil et al., 2000). But for HPAHs, we need to verify whether this is the case because of lacking of related information. We noticed that dietary exposures to PAHs have been assessed in three large cities (Beijing, Tianjin, and Taiyuan) in north China (Li et al., 2005, 2009; Xia et al., 2010). However, dietary exposure to both parent and halogenated PAHs for population in China has not been examined adequately so far.

Considering that vegetable, meat, and rice were the three most ingested food categories (~60% of the total diet for Shenzhen population) (Lei et al., 2011), these food samples were collected from wholesale markets for agricultural products in Shenzhen to quantify the profile of PAHs and HPAHs in foods and to evaluate toxic potencies and dietary intake of these pollutants on different ages. Based on that, the excess cancer risk induced by dietary exposure to these compounds was estimated as well.

2. Materials and methods

2.1. Sample collection and preparation

A total of 40 rice samples, 52 pork samples and 52 vegetable samples were collected from the largest four wholesale markets for agricultural

* Corresponding author. Tel.: +86 755 26033017; fax: +86 755 85293254.
E-mail address: nihg@pkusz.edu.cn (H.-G. Ni).

0048-9697/$ – see front matter © 2012 Elsevier B.V. All rights reserved.
http://dx.doi.org/10.1016/j.scitotenv.2012.11.018

products in Shenzhen from March to April, 2011. The sampling details were summarized in Table S1 ("S" indicates tables presented in the supplementary data thereafter).

Detailed extraction procedures of PAHs and HPAHs for rice sample were described previously (Ding et al., 2012). As for the cleanup for all sample extracts, the same procedure as given in our previous study (Ding et al., 2012) was employed in the current study. Here only the extraction procedures for vegetable and pork samples were provided.

All of the vegetable samples were rinsed with distilled water carefully to remove the dust on the surface. After dried in the fume cupboard, each vegetable sample was crushed with a juice extractor and homogenized. About 50 g of pulverized sample was liquid–liquid extracted with 80 mL acetonitrile for 30 min in a separatory funnel. A known amount of mixture of surrogate standards (naphthalene-d_8, acenaphthene-d_{10}, phenanthrene-d_{10}, chrysene-d_{12}, and perylene-d_{12}) was added prior to the extraction. After filtered through a vacuum filter, the acetonitrile-extract was shaken for 1 min with 320 mL 2% sodium sulfate solution, and extracted three times with 30 mL hexane for 5 min each time. The extracts were then concentrated to 2 mL using a rotary evaporator.

About 20 g pork sample was freeze-dried and ground, and then was Soxhlet extracted using a mixture of 160 mL dichloromethane and hexane (v:v, 1:1) for 24 h. The surrogate standards (the same as that for vegetable samples) were spiked prior to extraction. The Soxhlet extract was concentrated to 3 mL and transferred to a separatory funnel. This extract in the separatory funnel was extracted with 30 mL acetonitrile saturated with hexane three times for 5 min each time. We obtained the acetonitrile-extract, which was then shaken for 1 min with 300 mL 5% sodium sulfate solution, and extracted three times with 30 mL of hexane for 5 min each time. The extract was concentrated to 2 mL.

2.2. Quality assurance/quality control

Instrumental analysis was detailed in our previous study (Ding et al., 2012). Here only quality assurance and quality control were provided. Procedural blanks and spiked blanks samples were processed with each batch of samples (12 samples). The recoveries of the surrogate standards spiked into food samples were 92% ± 30%, 92% ± 26%, 68% ± 15%, 66% ± 19%, and 58% ± 18% for naphthalene-d_8, acenaphthene-d_{10}, phenanthrene-d_{10}, chrysene-d_{12}, and perylene-d_{12}, respectively. The concentrations of the analytes were determined from an internal calibration method. The range of the calibration curve was from 5 ng/mL to 2000 ng/mL, and the linear coefficients were larger than 0.999 for all of calibration curves of PAHs and HPAHs. The lowest concentration level of the calibration curve was defined as the reporting limit (0.05 ng/g for vegetable samples, and 0.25 ng/g for pork and rice samples) for the target analytes. The concentrations of PAHs and HPAHs in the present study were given on a fresh weight basis (fw), and the reported concentrations were not corrected with the recoveries of the surrogate standards. In the ten procedural blanks samples, only naphthalene (Nap) and phenanthrene (Phe) were detected. Thus, the concentrations of Nap and Phe given in the present study were corrected with the content of Nap and Phe in the blank samples. Mean concentrations were used to dietary intake estimation and cancer risk calculation, for concentration data are normal distribution.

2.3. Data analysis

2.3.1. Human exposure model

Relative daily intake (RDI; ng/kg bw/day) and absolute daily intake of PAHs and HPAHs (ADI; ng/day) via food consumption were calculated with the following equations:

$$\mathrm{RDI}_j = \sum_{i=1}^{n} \left[\left(\mathrm{C}_i \times \mathrm{IR}_{i,j} \right) / \mathrm{bw}_j \right] \quad (1)$$

$$\mathrm{ADI}_j = \sum_{i=1}^{n} \left(\mathrm{C}_i \times \mathrm{IR}_{i,j} \right) \quad (2)$$

$$\text{Lifetime ADI} = \frac{\sum_{j=1}^{m} \left(\sum_{i=1}^{n} \mathrm{C}_i \times \mathrm{IR}_{i,j} \times \mathrm{ED}_j \right)}{\mathrm{ED}} \quad (3)$$

where C is the concentration (ng/g fw) of PAHs or HPAHs in food items; IR is the intake rate (g/day); bw is the average body weight (kg); *i* and *j* indicate food the *i*th item and *j*th subgroup, respectively. ED_j is the exposure duration (day) for the *j*th subgroup, Lifetime ADI is the average ADI in one's lifetime; and ED is the total exposure time (day) in one's lifetime.

2.3.2. Toxicity evaluation model

The toxicity levels of PAH and HPAH congeners were evaluated with toxic equivalency quotients (TEQ, ng-TEQ/g) in the Eqs. (4) and (5):

$$\mathrm{TEQ} = \sum_{i=1}^{n} \left(\mathrm{C}_i \times \mathrm{TEF}_{BaP,i} \right) \quad (4)$$

$$\mathrm{TEQ} = \sum_{j=1}^{n} \left(\mathrm{C}_j \times \mathrm{REP}_{BaP,j} \right) \quad (5)$$

where C_i and C_j is the concentration (ng/g fw) of PAH congener *i* and HPAH congener *j*, respectively. The toxic equivalency factor ($\mathrm{TEF}_{\mathrm{BaP}}$) of individual PAHs and relative potency ($\mathrm{REP}_{\mathrm{BaP}}$) of individual HPAHs indicate the degree of toxicity compared to BaP. $\mathrm{REP}_{\mathrm{BaP}}$ were established after the investigation of AhR-mediated activities of ClPAHs and BrPAHs in a yeast assay (YCM3 cell) system (Ohura et al., 2007, 2009).

2.4. Cancer risk assessment model

The excess cancer risk posed by dietary exposure to PAHs and HPAHs was assessed using the following equations (Ding et al., 2012; Xia et al., 2010; Yoon et al., 2007):

$$\mathrm{LECR} = \sum_{j=1}^{n} \left[\mathrm{Q}^* \times \mathrm{TEQ} \times \mathrm{IR}_j \times \mathrm{ED}_j / \left(\mathrm{bw}_j \times \mathrm{ED} \right) \right] \quad (6)$$

$$\mathrm{ECR}_j = \mathrm{Q}^* \times \mathrm{TEQ} \times \mathrm{IR}_j \times \mathrm{ED}_j / \left(\mathrm{bw}_j \times \mathrm{ED} \right) \quad (7)$$

where LECR is the lifetime excess cancer risk; ECR_j is the excess cancer risk for the *j*th subgroup; the cancer potency (Q^*) of BaP was assessed as $7.3\,(\mathrm{mg/kg/day})^{-1}$ by the integrated risk information system of the U.S.EPA. (2001); TEQ is the total toxic equivalency quotients (ng-TEQ/g) of all PAHs or HPAHs in food items; IR_j is the intake rate (g/day) of food items for the *j*th subgroup; bw_j is the average body weight (kg) for the *j*th subgroup; and ED is the total exposure time (day) in one's lifetime.

3. Results

3.1. PAHs and HPAHs in food items

Detection rates, relative abundance, and concentrations of PAHs and HPAHs were displayed in Fig 1 and Table S2. The percentage of samples above the reporting limit was defined as the detection rate here. As shown in Fig 1a, the eight non-carcinogenic PAHs (Nap, Acy, Ace, Fle, Phe, Ant, Flu, and Pyr) were detected at high proportions (>96%) in vegetable, pork, and rice. Noticeably, they altogether accounted for >92% of the Σ_{16}PAHs concentrations in all of the three food items (Fig. 1c). Of the eight carcinogenic PAHs (Σ_8CarPAHs: BaA, Chr, BbF, BkF, BaP, IcdP, DahA, and BghiP), BaA, Chr, BbF, and BaP in vegetable, Chr and BaP in pork, and Chr in rice were frequently detected (detection