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Barbecue Fumes: An Overlooked Source of Health Hazards in Outdoor Settings?

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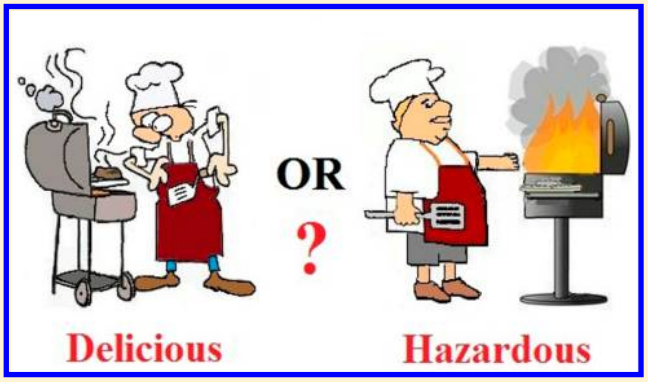
Supporting Information

ABSTRACT: Barbecuing or charcoal-grilling has become part of popular outdoor recreational activities nowadays; however, potential human health hazards through outdoor exposure to barbecue fumes have yet to be adequately quantified. To fill this knowledge gap, atmospheric size-fractioned particle and gaseous samples were collected near an outdoor barbecuing vendor stall (along with charcoal-grilled food items) in Xinjiang of Northwest China with a 10-stage micro-orifice uniform deposit impactor and a polyurethane foam (PUF) sampler and were analyzed for particulate matter and polycyclic aromatic hydrocarbons (PAHs). Exposure to PAHs through inhalation and dermal contact by adult consumers who spent 1 h per day near a charcoal-grilling vendor for a normal meal (lunch or dinner) amounted to a BaP equivalent (BaP_{eq}) dosage of 3.0−77 ng day^{-1} (inhalation: 2.8−27 ng day^{-1} of BaP_{eq}; dermal contact: 0.2−50 ng day^{-1} of BaP_{eq}), comparable to those (22−220 ng day^{-1} of BaP_{eq}) from consumer exposure through the consumption of charcoal-grilled meat, assumed to be at the upper limit of 50−150 g. In addition, the potential health risk was in the range of 3.1×10^{-10} to 1.4×10^{-4} for people of different age groups with inhalation and dermal contact exposure to PAHs once a day, with a 95% confidence interval (7.2×10^{-9} to 1.2×10^{-5}) comparable to the lower limit of the potential cancer risk range (1×10^{-6} to 1×10^{-4}). Sensitivity analyses indicated that the area of dermal contact with gaseous contaminants is a critical parameter for risk assessment. These results indicated that outdoor exposure to barbecue fumes (particularly dermal contact) may have become a significant but largely neglected source of health hazards to the general population and should be well-recognized.



INTRODUCTION

There are increased interests worldwide in recognizing the importance of cooking fumes as a source of negative impacts on air quality[1] and human health.[2,3] Cooking fumes are known to contain substances with mutagenic activity, such as fine particulate matter ($PM_{2.5}$),[4] black carbon, heterocyclic amines,[5] and polycyclic aromatic hydrocarbons (PAHs).[6] There is ample evidence that cooking fumes are associated with an increased risk of respiratory tract cancer in chefs, bakers, and other food-service workers.[7,8] As of now, many studies have been conducted to assess the significance of personal exposure to airborne particles from indoor sources such as indoor cooking,[5,9] which is understandable given that individuals spend the majority of their time in indoor environments. However, only limited information is available about human health risks arising from exposure in outdoor-cooking micro-environments.[10,11]

Eating outside the home increasingly makes up much of diets today for leisure and business. For example, American adults spent approximately 50% of their food expenditures on away-from-home foods.[12] Street foods, an attractive alternative to home-cooked foods[13] that originated in Asia, Latin America, and Africa, have become an integral component of the local food culture in various parts of Europe and North America.[14] Apart from street foods, outdoor barbecuing is also a popular event during summer and winter seasons in some parts of Asia, Europe, and North America. A study showed that annually, over 30 000 tons of charcoal are consumed with barbecues in England.[15] In North America, propane also has been used as an

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important type of fuel.[16] Barbecues and street foods are often prepared in residential, commercial, or recreational areas that have insufficient ventilation and poor air circulation.[14] Exposure of vendors, consumers, and residents to barbecue-linked pollutants may occur through incidental inhalation or dermal contact with cooking fumes, as well as via ingestion of chemicals absorbed by or deposited onto food. So far, there have been limited measurements of outdoor exposure to barbecue fumes. Particularly, dermal contact is often neglected in assessments of combustion-derived PAHs.

To address the above-mentioned knowledge gap, we selected an outdoor barbecuing vendor stall in Urumqi, Xinjiang Uygur Autonomous Region of China as a case study (Figure S1). Such a selection was made on the basis of the fact that charcoal grilling is a prevailing cooking method (Text S1) in this region, and beef and mutton dominate the daily diet for the local ethnic population under Islamic influence.[17] Gaseous and particle samples, as well as charcoal-grilled lamb meat samples, were collected and processed. Target pollutants in barbecue fumes and meat include particulate matter (PM) with aerodynamic diameters less than 10 μm (PM_{10}) and less than 2.5 μm ($PM_{2.5}$) and PAHs, which have been found to occur abundantly in cooking fumes.[18] The objectives of the present study were to (1) determine the occurrence of $PM_{2.5}$, PM_{10}, and PAHs in barbecue fumes; (2) examine the relative importance of inhalation, dermal contact, and food ingestion as the human exposure pathways; and (3) assess the potential human health risk associated with barbecue-fume exposure in outdoor barbecuing settings.

MATERIALS AND METHODS

Materials. A standard solution including 16 priority PAHs designated by the United States Environmental Protection Agency (USEPA) was purchased from AccuStandard (New Haven, CT); these PAHs are naphthalene (Nap), acenaphthylene (Acy), acenaphthene (Ace), fluorene (Fle), phenanthrene (Phe), anthracene (Ant), fluoranthene (Flu), 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), the sum of which is defined as $\sum_{16}$PAH in the present study. Internal standards (2-fluorobiphenyl, *p*-terphenyl-d_{14}, and dibenzo[*a,h*]anthrancene-d_{14}) and surrogate standards (naphthalene-d_8, acenaphthene-d_{10}, phenanthrene-d_{10}, chrysene-d_{12}, and perylene-d_{12}) were purchased from Dr. Ehrenstorfer GmbH (Augsburg, Germany). SX-3 Bio-Beads used in gel permeation chromatography were purchased from Bio-Rad Laboratories (Hercules, CA).

Sample Collection. Sampling was conducted in August of 2013. Particle samples were collected with a micro-orifice uniform deposit impactor (MOUDI) (MSP Corporation, Shoreview, MN). Each sample was collected on 47 mm diameter glass microfiber filters (Whatman International, Maidstone, England) at a constant flow rate of 30 L min^{-1} and separated into 11 size fractions: >18, 10−18, 5.6−10, 3.2−5.6, 1.8−3.2, 1.0−1.8, 0.56−1.0, 0.32−0.56, 0.18−0.32, 0.10−0.18, and 0.056−0.10 μm. Gaseous samples were collected by a stainless steel unit, which contained polyurethane foam (PUF), connected with the MOUDI.[19] Both particle and air samples were collected at two downwind spots approximately 2 and 10 m away from an outdoor-barbecuing stove (≈1.2 m above the ground) at different time points, representative of heavily and moderately exposed scenarios, and a background site on the rooftop of a five-story building (≈15 m in height) at a horizontal distance of approximately 30 m from the barbecuing stove (Table S1). Samples were collected daily over a three-day period at each site during 12:30−14:30 and 19:30−21:30 (Beijing time) for the exposed sites and 09:30−21:00 (Beijing time) for the background site, which encompassed the lunch and dinner times in Urumqi. Charcoal-grilled lamb-meat samples were also collected from the stall on the last day of the sampling campaign and included kabobs of lamb meat, lamb rib, lamb liver, lamb kidney, lamb intestine, beef back strap, and *nan* (a specially made bread of local origin). Overall, 88 size-fractioned particle samples, eight gaseous samples, and samples of seven types of stall foods were obtained. Vapor samples (PUFs) were sealed consecutively with plastic zipper-closed bags and lightly vacuumized plastic bags, whereas particle samples were kept in membrane cell holders immediately after sampling. Food samples were first wrapped with aluminum foil and then placed in plastic zipper-closed bags. All samples were cooled with ice during transport to the laboratory and were stored at −20 °C until analysis.

Sample Extraction and Instrumental Analysis. Before extraction, the gaseous and particles samples were spiked with the surrogate standards and sonicated twice with 20 mL of hexane, dichloromethane, and acetone mixture (2:2:1 in volume). Each extract was concentrated, solvent-exchanged to hexane, and further concentrated with a Zymark TurboVap 500 (Hopkinton, MA). The concentrated extract was spiked with the internal standards before instrumental analysis. The food sample extracts were purified consecutively with Bio-Beads SX-3 and a Florisil column for lipid removal before final concentration.

All samples were analyzed with a Shimadzu gas chromatograph coupled to a mass spectrometer (GCMS-2010 Plus). An HP-5MS capillary column (30 m × 0.25 mm i.d. with a 0.25 μm film thickness) was used for chromatographic separation. The column temperature was programmed from 60 °C (initially held for 1 min) and elevated to 200 °C at 10 °C min^{-1}, ramped to 230 °C at 5 °C min^{-1} (held for 10 min), and further increased to 278 °C at 5 °C min^{-1} (held for 15 min). All samples were automatically injected (2 μL each) in a programmed temperature vaporizer with an initial temperature of 60 °C and then elevated to 280 °C at 400 °C min^{-1} (held for 20 min). The carrier gas was ultrahigh-purity helium at a flow rate of 1 mL min^{-1}. The ion source temperature was set at 250 °C. The mass selective detection was conducted in the electron impact mode. Mass spectra were acquired in the full scan mode with an electron impact energy of 70 eV.

Quality Assurance and Quality Control. A single procedural blank sample was analyzed for every batch of 20 samples. The recoveries of the surrogate standards, i.e., naphthalene-d_8, acenaphthene-d_{10}, phenanthrene-d_{10}, chrysene-d_{12}, and perylene-d_{12}, were 52 ± 8%, 54 ± 6%, 67 ± 12%, 82 ± 8%, and 96 ± 8% in blank samples and 38 ± 12%, 49 ± 11%, 60 ± 10%, 75 ± 13%, and 84 ± 20% in field samples. The average recovery of the target standards in matrix-spiked samples was 78 ± 21%. Concentrations of PAHs in all field samples, except for Nap, Ace, and Acy in particle and food samples that were excluded for further analyses, were corrected (by subtraction) for those detected in the corresponding procedural blanks within the same batch but were not corrected for the surrogate standard recoveries. The lowest calibration concentrations divided by the actual sample volumes were

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Table 1. Contents of Particulate Matter ($\mu g\ m^{-3}$), Benzo[a]pyrene (ng m^{-3}), and Airborne Polycyclic Aromatic Hydrocarbons (ng m^{-3}) around a Barbecuing Stove in Urumqi in Summer

	PM_{10}	$PM_{2.5}$	BaP	PAH_g[a]	PAH_p[b]	$PAH_{g\text{-}BaPeq}$[c]	$PAH_{p\text{-}BaPeq}$[d]	$PAH_{g/t\text{-}BaPeq}$[e]
background	80 ± 30[f]	50 ± 20	0.15 ± 0.02	250 ± 120	6 ± 2	0.4 ± 0.3	0.2 ± 0.1	67%
10 m	530 ± 180	450 ± 170	2.2 ± 0.3	1800 ± 830	50 ± 14	2.5 ± 1.4	2.5 ± 0.5	50%
2 m	2600 ± 1700	2400 ± 1700	8.0 ± 0.8	2900 ± 480	320 ± 265	3.7 ± 1.7	10 ± 8.7	30%

[a]PAH_g: gaseous PAHs. [b]PAH_p: particle-bound PAHs. [c]$PAH_{g\text{-}BaPeq}$: BaP equivalent (BaP_{eq}) concentration for gaseous PAHs. [d]$PAH_{p\text{-}BaPeq}$: BaP_{eq} concentration for particle-bound PAHs. [e]$PAH_{g/t\text{-}BaPeq}$: fraction of BaP_{eq} for gaseous PAHs to total of BaP_{eq} for gaseous and particle-bound PAHs. [f](A ± B) represents the mean (A) and standard deviation (B).

defined as the reporting limits for the target compounds, i.e., 50 pg m^{-3} for MOUDI samples of 4 m^3 and 0.2 ng g^{-1} wet weight for a 10 g food sample. Analyte concentrations below the reporting limits were set to two-thirds of the reporting limits in the assessment of deposition fluxes and health risk.

Data Analysis. To estimate the deposition efficiency and flux of inhaled PAHs in the human respiratory tract (HRT), we adopted the International Commission on Radiological Protection (ICRP) model.[20] The model calculates the deposition fractions of inhaled particles in four anatomical regions: (1) the extrathoracic region (ET) that includes the nasal passage (ET_1), composed of the anterior nose and the posterior nasal passage, and the pharynx (ET_2), composed of the larynx and mouth; (2) the bronchial region (BB), combined by the airway from the trachea, main bronchi, and intrapulmonary bronchi; (3) the bronchiolar region (bb), joined by the bronchioles and terminal bronchioles; and (4) the alveolar-interstitial region (AR), the airway from the respiratory bronchioli through the alveolar sacs. The adult breathing rate under light exercise conditions was chosen as 1.5 $m^3\ h^{-1}$.[20] More details for ICRP-HRT model development and construction are given elsewhere.[20,21]

Daily intake of PAHs via inhalation (DI_{ig} for gaseous PAHs and DI_{ip} for particle-bound PAHs) or dietary intake (DI_{food}) and dermal contact (DI_{dg} for gaseous PAHs and DI_{dp} for particle-bound PAHs) and cancer risk (Risk) were estimated by[22−25]

$$DI_{ig} = \sum (C_g \times TEF_{PAH}) \times IR_{in} \times t_{event} \quad (1)$$

$$DI_{ip} = \sum (C_{ip} \times TEF_{PAH}) \times IR_{in} \times t_{event} \quad (2)$$

$$DI_{food} = \sum (C_{food} \times TEF_{PAH}) \times IR_{food} \quad (3)$$

$$DI_{dg} = \sum (C_g \times TEF_{PAH}) \times k_{p\_g} \times SA \times f_{sa} \times t_{event} \quad (4)$$

$$DI_{dp} = \sum (C_{dp} \times TEF_{PAH}) \times k_{p\_d} \times SA \times f_{sa} \times t_{event} \quad (5)$$

$$Risk = \sum (DI_i \times CSF_i) \times ED \times EF \times t_{event}/(BW \times AT) \quad (6)$$

where TEF_{PAH} is the toxicity equivalency factor of PAH based on BaP; the BaP equivalent (BaP_{eq}) concentration of an individual PAH compound equals the PAH concentration multiplying with its corresponding TEF; C_{ip} is the sum of BaP_{eq} concentrations of size-fractioned particle-bound PAHs distributed in five regions of the human respiratory tract (ng m^{-3}), calculated by the size-fractioned BaP_{eq} concentration of PAHs multiplying the deposition efficiency derived from the ICRP-HRT model; C_g and C_{dp} are the BaP_{eq} concentrations of gaseous and particle-bound PAHs, respectively (ng m^{-3}); C_{food} is the BaP_{eq} concentration of PAHs in food item (ng g^{-1}); IR is the rate of inhalation (IR_{in}, $m^3\ h^{-1}$) or daily dietary intake (IR_{food}, g day^{-1}); t_{event} is the event time, i.e., the time duration for a day with exposure (h day^{-1}); k_{p_g} is the transdermal permeability coefficient of gaseous compounds (m h^{-1}); SA is the skin surface area (m^2); f_{sa} is the exposed dermal fraction; k_{p_d} is the transdermal permeability coefficient of particle-bound compounds (m h^{-1}); CSF_i is the cancer slope factor of BaP for exposure route i (mg kg^{-1} $day^{-1})^{-1}$ and is age-dependent as specified by average body weight; ED is the lifetime exposure duration (year); EF is the exposure frequency, i.e., the number of days with exposure per year (days per year); BW is the average body weight; and AT is the average time for carcinogenic effects (day). Detailed values for these parameters are provided in Tables S2 and S3. Daily exposure was assumed to be 1 h at the charcoal-grilled-barbecue vendor for a normal meal (lunch or dinner), and cancer risk was thus estimated based on an eating-out frequency of once a day.

We assessed three exposure scenarios for the age groups of 1−11 years (childhood), 12−17 years (adolescent), and 18−70 years (adulthood). The first scenario was for bystanders, food servers, or local residents who do not consume any charcoal-grilled foods (inhalation and dermal contact only). The second scenario was for people who consume the stalls' charcoal-grilled foods elsewhere (dietary intake only). The last scenario was for people who consume charcoal-grilled foods at a vendor (inhalation, dermal contact, and dietary intake). Crystal Ball software (version 2000.2, Decisioneering, Denver, CO) was employed to conduct Monte Carlo simulations, each with 10 000 iterations, to generate 2.5 and 97.5 percentiles at a 95% confidence interval for all calculations presented. The procedures of sensitivity analysis are described in Text S2.

RESULTS AND DISCUSSION

Concentrations of Airborne Particulate Matter and Polycyclic Aromatic Hydrocarbons. Airborne PM and PAHs are the major pollutants generated from cooking processes, with BaP as the individual PAH of greatest health concern.[1] As expected, the highest levels of PM_{10}, $PM_{2.5}$, BaP, and total PAHs were found 2 m from the stove, and the lowest levels at the background site (Table 1 and Tables S3−S5). The concentrations of PM_{10}, $PM_{2.5}$, and BaP (900−5300 and 700−4100 $\mu g\ m^{-3}$ and 7.2−8.8 ng m^{-3}, respectively) near the stove exceeded the national ambient air quality standards of China (24 h averages of 150 $\mu g\ m^{-3}$, 75 $\mu g\ m^{-3}$, and 2.5 ng m^{-3}, respectively).[26] Furthermore, the concentrations of PM_{10}, $PM_{2.5}$, and BaP (37−93 $\mu g\ m^{-3}$, 20−60 $\mu g\ m^{-3}$ and 0.13−0.17 ng m^{-3}, respectively) at the background site were below China's air quality standards. The concentrations of gaseous and particle-bound PAHs were 2400−3400 and 55−590 ng m^{-3}, 1000−2600 and 40−60 ng m^{-3}, and 130−370 and 4−8 ng m^{-3}, respectively, at distances of 2 and 10 m from the stove and

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at background sites. Furthermore, the concentrations of particle-bound PAHs in cooking fumes around the 2 m site were within the same order of magnitude with those inside coal-using houses and in the middle of the range for cooking fuels in indoor environments (Table S6).

In general, the data acquired in the present study were consistent with a previous finding that PM emissions from cooking processes were dominated by $PM_{2.5}$.[18] Genotoxicity studies also found that $PM_{1.0-0.56}$, containing the largest amount of carcinogenic PAHs such as BaP, induced the highest DNA-adduct levels.[27] Therefore, the relatively high levels of PM and BaP near the charcoal-grill stall stove should be of health concern for vendors and consumers who have regularly cooked or consumed foods near charcoal-grill stoves for extended time durations.

Deposition of Particle-Bound Polycyclic Aromatic Hydrocarbons in the Respiratory System. Deposition efficiency (Table S7) and fluxes of particle-bound PAHs in the adult male's respiratory tract were calculated using measured size-fractioned concentrations of inhaled PAHs and the ICRP model. The deposition fluxes of $\sum_{16}$PAH were 30−350, 21−44, and 2.5−6.0 ng h^{-1}, respectively, at the 2 m, 10 m, and background sites, with the mean values following the sequence of 2 m (165 ng h^{-1}) > 10 m (41 ng h^{-1}) > background (6.2 ng h^{-1}) (Figure 1). In addition, these deposition fluxes of $\sum_{16}$PAH were significantly different (*t*-test, $p < 0.05$) among the sampling sites.

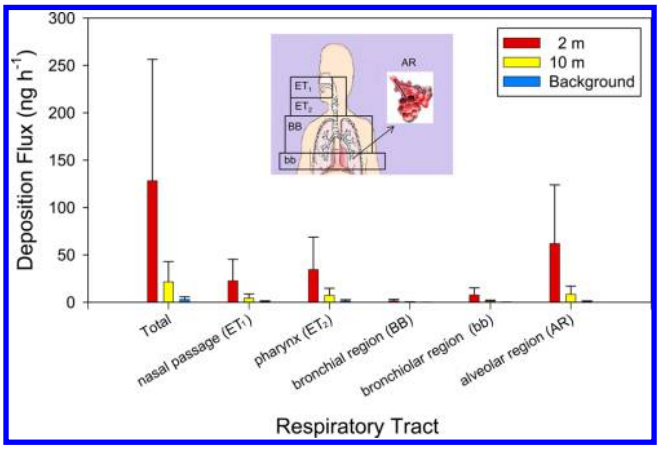

Figure 1. Deposition fluxes of particle-bound PAHs in the extrathoracic region (ET), including nasal passage (ET_1) and pharynx (ET_2), bronchial region (BB), bronchiolar region (bb), and alveolar-interstitial region (AR) of the human respiratory tract at the exposure distances of 2 and 10 m and a background site. Total exposure is the sum of ET, BB, bb, and AR.

Deposition fluxes of $\sum_{16}$PAH in the regions of ET_1, ET_2, BB, bb, and AR were 1.1−33, 2.0−50, 0.0−2.2, 0.2−11, and 1.1−92 ng h^{-1}, respectively (Figure 1). Furthermore, the mean deposition flux of $\sum_{16}$PAH in the AR region at the 2 m site (93 ng h^{-1}) was approximately 4−8 and 66−88 times those at the 10 m (12 ng h^{-1}) and background sites (1.1 ng h^{-1}) (*t*-test, $p < 0.05$). Figure 2 and Figure S2 show that more than 90% of the amounts of $\sum_{16}$PAH deposited in the BB, bb, and AR regions were contributed by fine particles (aerodynamic diameter <1.8 μm). Because fine particles can carry abundant PAHs into the deep regions of human lungs, such as the blood vessels and circulatory system,[3] they may be more likely to result in lung disease, such as lung cancer, than coarse

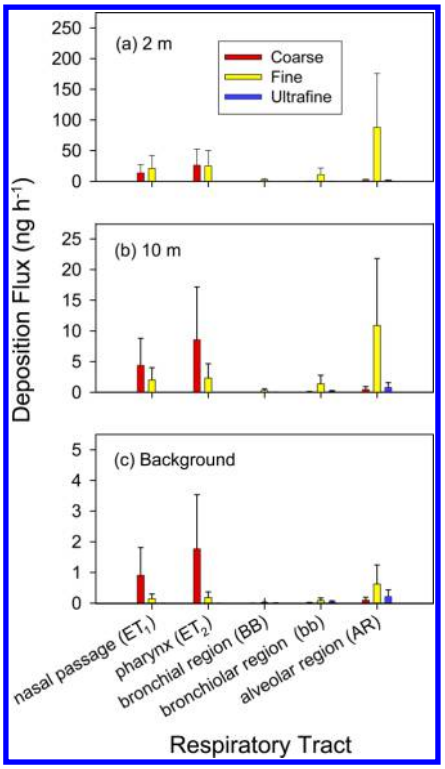

Figure 2. Deposition flux of size-fractioned particle-bound PAHs in five regions of the human respiratory tract at exposure distances of 2 and 10 m and a background site. The red, yellow, and blue bars represent coarse (aerodynamic diameter (Dp) > 1.8 μm), accumulation mode (1.8 μm > Dp > 0.1 μm), and ultrafine (Dp < 0.1 μm) particles, respectively.

particles.[28] Accordingly, the probability of respiratory tract infections related to atmospheric particle-bound PAHs is high for consumers with long-term exposure to barbecue fumes. This result further corroborated the previous views that particle size distribution is a critical factor dictating human inhalation exposure to particle-bound PAHs and related health risk.[29,30]

Inhalation of Gaseous Polycyclic Aromatic Hydrocarbons. The total BaP_{eq} concentrations in the gaseous and particle phases examined in the present study are summarized in Table 1. The 2 m site had the lowest fraction of gaseous PAHs, i.e., approximately 30−45% of the total BaP_{eq} concentrations (Table 1), probably because fume particles contain abundant heavy PAHs with high toxic equivalency factors.[31] However, the gaseous and particle fractions contributed equally to the total BaP_{eq} concentrations at the 10 m site. At the background site, the gaseous PAHs contributed more than 70% of the total BaP_{eq} concentrations. A previous study suggested that approximately 60% of BaP_{eq} concentrations and exposure was attributed to gaseous-phase PAHs for workers of a carbon black manufacturing plant and traffic officers in urban Beijing.[32,33] Hence, the importance of exposure to gaseous-phase pollutants through inhalation should be adequately addressed, or human health risk would be underestimated.

Uptake of Polycyclic Aromatic Hydrocarbons via Dermal Contact. The total fluxes of $\sum_{16}$PAH due to dermal contact (0.2−50 ng day^{-1} of BaP_{eq}, out of which ~99% was attributed to the uptake of gaseous PAHs) were comparable to or larger than those via inhalation (2.8−26 ng day^{-1} of BaP_{eq}) (Table 2), similar to what was reported previously.[24]

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Table 2. Daily Exposure (BaP equivalent (BaP_{eq}) dosage, ng day^{-1}) via Dermal Contact, Inhalation, and Stall Food Consumption by Adult Consumers[a]

	DI_{dg}[b] f_{sa}[g]		DI_{dp}[c] f_{sa}[g]		DI_{ip}[d]	DI_{ig}[e]	DI_{food}[f]
	whole	25%	whole	25%			
background	0.8 (0.1−4.7)[h]	0.2 (0.03−1.3)	< 0.01	< 0.01	0.1 (0.04−0.4)	0.5 (0.4−0.7)	71 (22−220)
10 m	5 (0.7−34)	1.4 (0.2−9)	0.03 (0.02−0.04)	0.01 (<0.01−0.01)	1.9 (1.4−2.5)	3.2 (1.4−7.8)	71 (22−220)
2 m	7.7 (1.1−50)	2.1 (0.3−13)	0.09 (0.03−0.33)	0.03 (<0.01−0.09)	5.4 (1.8−16)	5 (2.5−10)	71 (22−220)

[a]Daily exposure duration was assumed to be 1 h. [b]DI_{dg}: daily dermal exposure to atmospheric PAHs. [c]DI_{dp}: daily dermal exposure to particle-bound PAHs. [d]DI_{ip}: daily inhalation exposure to particle-bound PAHs. [e]DI_{ig}: daily inhalation exposure to atmospheric PAHs. [f]DI_{food}: daily dietary intake of charcoal-grilled food, which was assumed to be the upper limit for the consumption amount of 50−150 g. [g]f_{sa}: fraction of dermal adsorption. [h]A (B−C) represents the median A and a 95% confidence interval.

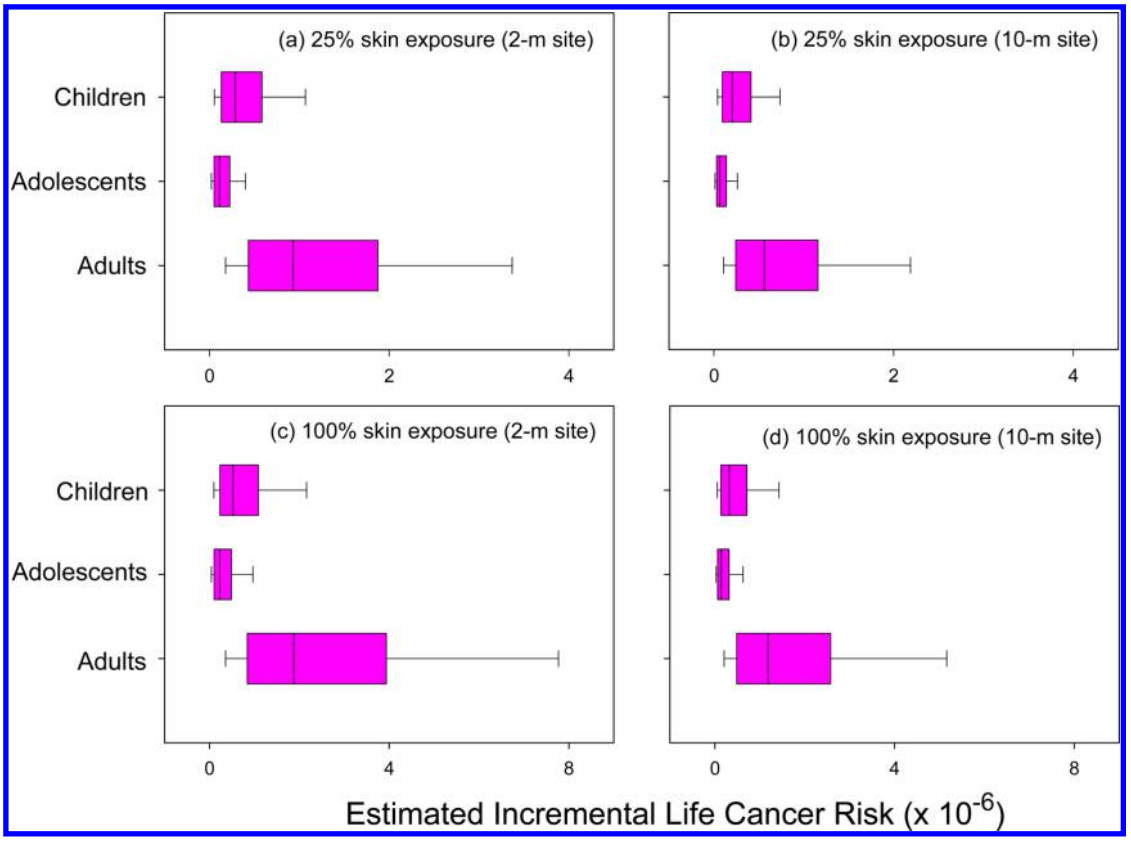

Figure 3. Estimated incremental life cancer risks induced by PAHs for consumers and bystanders of different age groups with exposure once a day at distances of 2 and 10 m from a charcoal-grill stall stove through inhalation and dermal contact: (a) and (b) 25% dermal contact surface and (c) and (d) 100% dermal contact surface. Detailed values of all parameters are provided in Table S2.

Furthermore, light and thin cloths are expected to provide negligible resistance to the transport of gaseous organic compounds from air to skin,[22] further emphasizing the possibility of dermal exposure in warmer months. Uncertainty analyses indicated that the transdermal permeability coefficient (k_{p_g}) had the greatest contribution (>83%) to the total variance of transdermal fluxes. Apparently, additional research is warranted because there are limited measured values of k_{p_g} available in the literature.[34]

In fact, dermal exposure from air is common in occupational exposure scenarios[35−38] yet is largely overlooked as a pathway for human exposure to semivolatile organic compounds, with only a few assessments done in indoor microenvironments.[24,39] Previous studies found that levels of aromatics and some PAHs and their metabolites were significantly elevated in urinary samples of firefighters post-firefighting.[40,41] Because firefighters' protective gear was effective in blocking external inhalation exposures during firefighting, higher levels of PAH metabolites post-firefighting suggested that dermal contact was the main input route of airborne pollutants in the firefighters. Consequently, human exposure to outdoor air pollutants, e.g., charcoal-grilled-barbeque fumes, for extended time durations may substantially increase the possibility of dermal contact as a major route of air-pollutant intake.

Comparison of Pollutant Exposure by Dietary Intake, Inhalation, and Dermal Contact. The concentrations of PAHs in meats were highly dependent on cooking methods, content of fat, additives, and cooking fuels (Table S8). The average concentration of PAHs in charcoal-grilled foods (0.9 ± 0.5 ng g^{-1} of BaP_{eq}; Table S9) was generally within the range of PAH concentrations in similar food items from other regions (Table S8)[42,43] but were lower than 5 ng g^{-1}, the European

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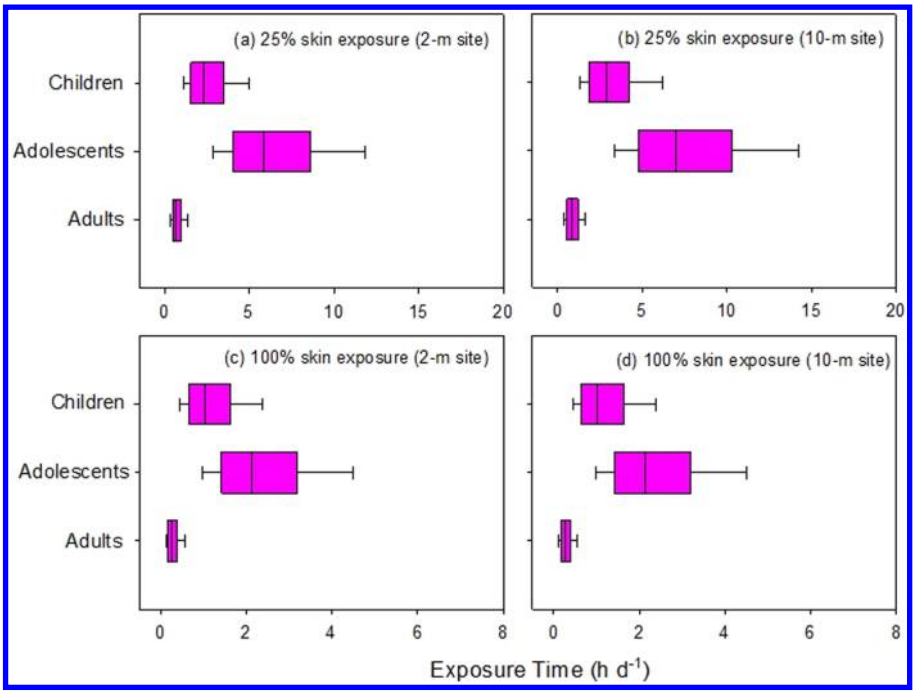

Figure 4. Ranges of exposure time (h day^{-1}) to maintain a cancer risk value of less than 10^{-6} for people of different age groups subject to inhalation and dermal-contact exposure once a day at distances of 2 and 10 m from a charcoal-grill stove.

Commission's standard for smoked and grilled meat.[44] Based on the results from the present study, dietary intake of BaP_{eq} via consumption of popular charcoal-grilled foods in Xinjiang, such as grilled whole lamb and *yangrou chuar* (lamb kebabs), can be estimated. The daily meat consumption for local residents is approximately 50−150 g, with low consumption of vegetables. If all are charcoal-grilled foods, an upper limit of dietary exposure could be 22−220 ng day^{-1} of BaP_{eq} for adults. Uncertainty analyses indicated that daily meat consumption (DI_{food}) had the greatest contribution (>85%) to dietary intake of BaP_{eq}.

Generally, diet is the primary source of human exposure to PAHs, contributing to more than 70% of the total exposure to PAHs.[45] This is also true in the present study, such that the amounts (50−150 g) of charcoal-grilled meat consumed would lead to the upper limit of the estimated dietary exposure at 22−220 ng day^{-1} of BaP_{eq}, substantially greater than the inhalation of airborne PAHs (1.4−16 ng day^{-1} of BaP_{eq}) and of dermal contact with particle-bound PAHs (<0.01−0.33 ng day^{-1} of BaP_{eq}) (Table 2). However, dermal contact with gaseous PAHs (0.7−50 ng day^{-1} of BaP_{eq}) in the present study was comparable to the upper limit of estimated dietary exposure, i.e., consumers near a charcoal-grill barbecue setting similar to that in the present study would also be subject to combined exposure through inhalation and dermal contact of cooking fumes.

Health Risk Assessment. For the first exposure scenario, the cancer risk through inhalation and dermal contact of Σ_{16}PAH at distances from 2 to 10 m was 6.1×10^{-8} to 1.2×10^{-5}, 7.2×10^{-9} to 1.5×10^{-6}, and 2.4×10^{-8} to 3.2×10^{-6} for adults, adolescents, and children, respectively (Figure 3 and Table S10). The cancer risk for the second exposure scenario (dietary intake only) was 1.8×10^{-6} to 2.0×10^{-5}, 1.9×10^{-7} to 2.2×10^{-6}, and 5.5×10^{-7} to 6.9×10^{-6} for adults, adolescents, and children, respectively (Table S10). These risk levels are essentially the same as the total dietary exposure to $\sum_{16}$PAH (2.35×10^{-5}) reported for the general Korean population[46] and higher than the total dietary exposure to Σ_{16}PAH (5.8×10^{-6}) reported for adults in Shenzhen, China.[47] The cancer risk for the third scenario (inhalation, dermal contact, and dietary intake combined) was 2.5×10^{-6} to 2.8×10^{-5}, 2.8×10^{-7} to 3.2×10^{-6}, and 7.7×10^{-7} to 9.1×10^{-6} for adults, adolescents, and children, respectively (Table S10). It should be noted that cancer risk was estimated based on an eating-out frequency of once a day and an event-exposure duration (t_{event}) of 1 h. Cancer risk may vary with different exposure durations, food consumption patterns, and changes in other parameters. These changes can be easily accounted for by rescaling the cancer risk using the actual values of the parameters used.

The exposure time was measured at the stall, i.e., a customer spent approximately 10−15 min waiting for charcoal-grilled foods to be cooked. Conversely, if customers were not in rush, they spent approximately 40−60 min or more. The results of sensitivity analyses (Figures S3−S5) indicated that t_{event} had the greatest contribution (>45%) to the total variance of the health risk for all age groups, followed by gaseous PAH concentration at the skin surface under the first exposure scenario. The PAH concentrations in stall foods and food consumption are the influential variables, with more than 45% and 38% of sensitivity contributions, respectively, under the second exposure scenario, and the third scenario is similar to the second scenario. Besides t_{event} and other parameters linked to the stall's microenvironment, factors such as cancer slope factors and transdermal permeability coefficients could also contribute (≥10%) to the total variance, which indicated that improving the accuracy of these factors is critical for risk assessment.[34]

In most regulatory programs, an incremental lifetime cancer risk (ILCR) between 10^{-6} and 10^{-4} is defined as a potential risk, whereas an ILCR >10^{-4} indicates a high risk.[48] Thus, the cancer risks for all age groups and three exposure scenarios (<10^{-4} and >10^{-6}) were at least moderate. In particular, the

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cancer risks for the first exposure scenario (i.e., 6.1×10^{-8} to 1.2×10^{-5} and 2.4×10^{-8} to 3.2×10^{-6} for adults and children) suggested that outdoor charcoal-grill cooking fumes may be a source of potential health hazards even to bystanders who do not consume any cooked meat. Because emissions from high-temperature frying have been classified in Group 2A (probable carcinogens) as being probably carcinogenic to humans by the International Agency for Research on Cancer (IARC),[49] our results indicated that risks from exposure to outdoor charcoal-grill fumes should also be adequately recognized in general.

Figure 4a,b present the ranges of exposure time necessary to maintain a cancer risk of less than 10^{-6} for different age groups when staying near or at downwind areas (≤10 m exposure distance) of charcoal-grill stoves, based on an eating-out frequency of once a day. The simulated median exposure time for the first exposure scenario is 2.3−2.8, 5.9−7.0, and 0.7−0.8 h day^{-1} (at 2 and 10 m) for children, adolescents, and adults, respectively, with only a 25% skin surface exposure to gaseous PAHs. If 100% dermal surface exposure is assumed, the suggested exposure time would become less than 0.9−1.0, 2.0−2.1, and 0.2−0.3 h day^{-1} for children, adolescents, and adults, respectively (Figure 4c,d). For the third exposure scenario (Figure S6), the simulated median exposure time is 0.3, 0.9, and 0.1 h day^{-1} for children, adolescents, and adults, respectively, if the dietary intake amounts are assumed to be 70, 80, and 100 g of charcoal-grilled street foods for children, adolescents, and adults, respectively. The suggested exposure time to induce a cancer risk of less than 10^{-6} may increase or decrease proportionally with decreased or increased eating-out frequency and the amount of grilled food consumed.

Global Importance of Outdoor Barbecue Fumes as a Significant Source of Health Hazards. A survey conducted by the Hearth, Patio, & Barbecue Association in 2013 found that 41% of Americans were inclined to purchase grills for outdoor events.[16] Although the summer months see the most outdoor cooking festivals, increasing use of outdoor grills year-round has become a new trend. For example, Americans and Canadians perform outdoor barbecue approximately 23−38 times a year on average, and the use of dry rubs for meat in charcoal grilling increased from 17% in 2003 to 33% in 2013.[16] Another study by AXA PPP Healthcare found that U.K. adults consumed up to three times more grilled meat at a barbecue party than for a normal meal.[50] Moreover, a typical barbecue party may last 5.6 h for Australians and 3.9 h for the English.[51] Furthermore, the BaP equivalent concentrations in fumes with different grilling techniques (charcoal grilling, gas grilling, or electric oven roasting) may be within a factor of 2 (Table S6). Accordingly, barbecue fumes from backyard parties or other settings may have become a significant but largely neglected source of health threats to all participants.

Barbecuing or charcoal-grilling, perhaps one of the most popular ancient food-processing methods, has now become an important outdoor cooking style. Sidewalk snack booths or similar establishments are widely seen in many Asian and Latin American countries to serve a variety of street foods, including barbecued or charcoal-grilled ones. Barbecuing in residential backyards and recreational centers is part of increasingly popular outdoor activities worldwide despite the potential linkage between cooking-fume exposure and health risks by numerous epidemiological studies. One focal area of such studies is the linkage of lung cancer in rural women with exposure to indoor charcoal-cooking fumes.[7,52,53] Currently, most studies have focused on pollutant emissions from indoor commercial grills and kitchens,[4,5,7] while outdoor exposure to barbecue fumes and related health hazards have largely been overlooked. Although the present study was somewhat limited due to the lack of biomarkers for quantifying internal exposure dosage (such as urinary hydroxy-PAHs) and other mutagenic substances (such as heterocyclic amines), the results presented herein suggested that it would be prudent to further investigate barbecue fumes as an important source of human health concerns. Particularly, the risk of dermal exposure to barbecue fumes containing gaseous contaminants has often been underestimated and cannot be adequately examined without the development of proper methods to deal with nonsteady-state exposure conditions.[23]

Furthermore, recent studies have shown that third-hand smoke, i.e., noxious residues that cling to virtually all surfaces (such as walls, carpets, furniture, and dust particles) long after second-hand smoke has cleared out, may contain abundant toxic airborne pollutants[54] and cause significant genetic damage in human cells.[55] Barbecue fumes are expected to contain pollutants with similar gas-to-surface partitioning properties as those in cigarette smoke. Hence, under the same logic as third-hand smoke exposure, family members of eating-out consumers and barbecue participants may also be exposed to pollutants contained in outdoor cooking smoke and barbecue fumes through re-emission into indoor environments. Such exposure routes should be adequately recognized and included in health impact assessments.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge on the ACS Publications website at DOI: 10.1021/acs.est.5b01494.

Additional text pertaining to processes for preparing charcoal-grilled foods in Xinjiang and to sensitive analysis. Tables showing information about samples collected from outdoor barbecuing stoves; risk parameters for estimating human exposure to PAHs; toxicity equivalency factors and measured gaseous concentrations; particle-size-dependent concentrations of PAHs; geometric mean diameter and geometric standard deviations of particle-bound PAHs; concentrations of BaP, particulate matter, and airborne PAHs from previous and present studies; radios of deposition fractions to inhalable fractions of size-fractioned PAHs; measured concentrations of PAHs in stall foods and of BaP and PAHs in meat; and estimated incremental life cancer risks induced by PAHs. Figures showing the location of the sampling site and the surrounding city; relative contributions of particles to the deposition amount of PAH in the human respiratory tract,; sensitivity contributions of parameters to estimating incremental life cancer risks induced by PAHs related to exposure to charcoal-grill stall stove surfaces, the consumption of charcoal-grilled foods, or both; and the range of exposure time to maintain life cancer risks pertaining to inhalation, dermal contact, and dietary exposure related to charcoal-grill stoves. (PDF)

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The authors declare no competing financial interest.

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