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Low Tar Level Does Not Reduce Human Exposure to Polycyclic Aromatic Hydrocarbons in Environmental Tobacco Smoke

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Supporting Information

ABSTRACT: Environmental tobacco smoke (ETS) is known to pose potential risk to human health, but the effects of tar level remain to be clarified. In the present study, ETS samples from two cigarette types with different tar levels in a 72.5 m^3 room were collected for measurement of 16 polycyclic aromatic hydrocarbons (PAHs). Urine samples of volunteers participating in smoking events were collected and analyzed for eight hydroxyl-PAHs. The concentrations, compositions, and particle size distribution patterns of PAHs from higher-tar and lower-tar cigarettes were similar, while the emission factors of PAHs from higher-tar cigarettes were lower than those from lower-tar cigarettes. Furthermore, the change in the concentrations of PAH metabolites in urine samples before and after smoking was not attributed to tar level. Assuming that a single cigarette was smoked in a 100 m^3 room, the estimated average inhalation cancer risks for different age groups from exposure to PAHs in ETS were below 1.0×10^{-6}, but potential risks should not be overlooked, especially considering that only inhaled particle-bound PAHs in ETS were included in this assessment. Apparently, reduced tar levels would not necessarily lead to lowered risk of exposure to PAHs. Kicking the habit is perhaps the best choice to minimize any potential health risk.

INTRODUCTION

The influences of smoking and passive smoking on human health have been a global concern, and it is widely accepted that smoking is harmful to human health. Under the appeal of the World Health Organization in 2003, 192 countries signed the Framework Convention on Tobacco Control in an attempt to curb the epidemic of tobacco use. Although some effective interventions have been put in place to reduce tobacco consumption,[1−4] the number of tobacco-related deaths worldwide continues to rise as a result of population growth and aging.[5] Earlier epidemiological studies suggested that lower-tar products were associated with lower lung cancer mortality.[6−9] To reduce the adverse impacts of smoking, tobacco companies started to market cigarettes with lower yields of carcinogenic tar and use these as the selling point.[10] Other studies,[11−13] however, found no difference in lung cancer risk among people who smoked light (with >6.5−14.5 mg tar) or ultralight cigarettes (with ≤6.5 mg tar). Unfortunately, ordinary smokers do not generally realize that low tar does not mean low hazard.[14,15]

Previous efforts in assessing the hazards of smoking to human health have mostly employed smoking machines to produce and collect mainstream smoke.[16,17] Few published studies have estimated tobacco emission factors (EFs) under real smoking scenarios. Smokers inhale large amounts of mainstream smoke drawn through filter tips and cigarette mouthpieces, while nonsmokers are exposed to secondhand smoke, including side-stream smoke and mainstream smoke exhaled by smokers. Compared with mainstream smoke, secondhand smoke, as the main source of environmental tobacco smoke (ETS), is characterized by small-sized particles and high toxicity.[18] It is considered as a risk factor for lung cancer and cardiovascular disease in nonsmokers.[19,20] Because of the difference in smoking patterns between smoking machines and human beings, tar reduction strategies based on smoking machines may not achieve expected benefits for smokers.[21] The potential health risk to nonsmokers from exposure to ETS is also disregarded in the process.

ETS is considered one of the most important sources of indoor air pollution and contains more than 60 carcinogens,[22] for example, abundant polycyclic aromatic hydrocarbon (PAHs).[23] Objective assessment of the health risk of inhalation exposure to PAHs in ETS requires information on the particle size distribution of PAHs because particle-bound PAHs of different sizes have different inhalation efficiencies and may predominantly deposit in different parts of the human respiratory system.[24] There have been some studies on $PM_{2.5}$ and PM_{10} from ETS,[25,26] but more detailed particle size distribution patterns have not been fully examined. Therefore,

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measuring EFs of size-fractionated PAHs in ETS is still significant for better understanding the risk of human respiratory exposure.

In view of the above-mentioned knowledge gap, the aim of this study was to evaluate the occurrence of particle-bound PAHs during indoor smoking and the potential health risk of inhaled particle-bound PAHs to human health. Instead of smoking machines used in most previous studies, we chose real-life smoking events to produce ETS. A micro-orifice uniform deposit impactor (MOUDI) was used to collect ETS samples. Concentrations of size-fractionated PAHs were obtained and used for assessment of potential health risk from inhalation of PAHs in ETS.

MATERIALS AND METHODS

Materials. All standards of the target analytes are detailed in Text S1, along with internal and surrogate standards.[27] Two types of cigarettes with different tar yields (11 mg, higher-tar cigarette and 1 mg, lower-tar cigarette) selected are branded as Double Happiness and KENT, respectively, and were purchased in 2016 from a local supermarket in Guangzhou, China.

Sample Collection. Four indoor smoking events were arranged in a 72.5 m^3 room in Guangzhou on four consecutive Saturdays from August to September 2016. Each sampling event involved 12−14 males over the age of 21. There were 20 volunteers (some volunteers participated in more than one sampling event), and they all met the following inclusion and exclusion criteria for the present study: (1) female smokers were excluded because males are more likely to smoke and be exposed to secondhand smoke in China; (2) participants had to be healthy adults who are not with diagnosed heart disease or respiratory diseases; and (3) participants were not subject to occupational exposure to PAHs. The basic information of the participants is presented in Table S1. During each sampling, an air conditioner was set at 25 °C and the windows were slightly open. Participants started smoking simultaneously using the same smoking pattern for 1 h, during which each person consumed at least four cigarettes. A higher-tar cigarette was used in the first and second sampling events, while a lower-tar cigarette was used in the third and fourth events. The participants consumed a total of 50, 53, 60, and 57 cigarettes in the first, second, third, and fourth events, respectively. A fan was turned on to mix the air 6 min after smoking was initiated. Particles were collected using the MOUDI sampler (MSP; Shoreview, MN) placed on a table, with a constant flow rate of 30 L min^{-1}, during smoking, 12 h before smoking, and 12 h after smoking. Particles in each sample collected on 47 mm diameter glass microfiber filters (Whatman International, Maidstone, England) were segregated into 11 size fractions, that is, >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. After all participants completed smoking for 10 min, the samples collected by MOUDI were gathered and stored at −20 °C until further treatment.

Urine samples from the participants were collected with polypropylene centrifuge tubes, including pre-exposure samples (as background samples) collected in the morning prior to a smoking event and post-exposure samples. The urine samples were stored at −80 °C until analysis. No restrictions were placed on the diet and daily activities of the participants, but they were questioned to identify any alternative exposure. Overall, 132 particle samples and 98 urine samples were obtained.

Analytical Procedures. Each size-fractioned particle sample was spiked with the surrogate standards and sonicated with 20 mL of hexane, dichloromethane, and acetone mixture (2:2:1 in volume) for 30 min. After three extractions, the combined extract was concentrated and solvent-exchanged to hexane and further concentrated to ∼1 mL. A silica gel column for purifying the extract was eluted with 20 mL of dichloromethane and the eluate was collected and solvent-exchanged to hexane. Finally, the extract was concentrated to ∼50 μL under a mild stream of nitrogen. Internal standards were spiked to the extract before instrumental analysis with a Shimadzu model QP2010 Ultra gas chromatography/mass spectrometry. More detailed analytical procedures were reported previously.[28]

The detailed analytical method for OH-PAHs in urine was described elsewhere.[27] Briefly, 2 mL of each urine sample was spiked with the surrogate standards, followed by 1 mL of ammonium acetate buffer, 50 μL of β-glucuronidase, and 30 μL of mercaptoethanol (an antioxidant for minimizing instability of OH-PAHs). The mixture was then incubated overnight (16 h) at 37 °C. A C_{18} solid-phase extraction column was used to extract the analytes. The extract, filtered with a 0.22 μm nylon filter, was spiked with the internal standards. Instrumental analysis was performed with a Shimadzu LC-20A high-performance liquid chromatograph with a Zorbax Eclipse Plus-C18 column (2.1 × 100 mm and 1.8 μm film thickness) coupled to an AB SCIEX TRIPLE QUAD 5500 mass spectrometer. Urinary creatinine was measured by a 7600-020 automatic biochemical analyzer (Hitachi, Japan) at the Guangzhou Overseas Chinese Hospital.

Quality Assurance/Quality Control. For each batch of 20 particle samples, there were one spiked blank, one field blank, and two procedural blanks. The recoveries of naphthalene-d_8, acenaphthene-d_{10}, phenanthrene-d_{10}, chrysene-d_{12}, perylene-d_{12}, and benzo[*ghi*]perylene-d_{10} were 54 ± 12, 56 ± 12, 53 ± 13, 66 ± 20, 65 ± 23, and 61 ± 18%, respectively, in particle samples. In addition, two procedural blanks, one spiked blank, one matrix sample, and a matrix-spiked sample were analyzed for each batch of 20 urine samples. The recoveries of 1-hydroxynaphthalene-d_7 and 1-hydroxyphenanthrene-d_9 were 98 ± 27 and 93 ± 17%, respectively, in urine samples. Some target compounds were found in blank samples, while only low levels of 1-hydroxynaphthalene and 2-hydroxynaphthalene were detected in urine samples. Therefore, the concentrations of PAHs in particle samples were corrected by corresponding procedural blank samples (by subtraction). All samples were not corrected for the surrogate standard recoveries. The reporting limit was defined as the lowest calibration concentration divided by the actual sample volume, that is, 3.6 pg m^{-3} for MOUDI samples with an air volume of 1.8 m^3 and 0.02 ng mL^{-1} for urine samples of 2 mL.

Data Analysis. Based on the criteria provided by the International Standards Organization and American Conference of Governmental Industrial Hygienists, particles can be divided into inhalable, thoracic, and respirable fractions, with different efficiencies of entering different regions of the human respiratory system.[29] Inhalable particles of different sizes have different deposition efficiencies in the head airway (HA; including nose, mouth, pharynx, and larynx), tracheobronchial (TB) area, and alveolar region (AR), which can be evaluated

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by a simplified model by the International Commission on Radiological Protection (Text S2).[30]

The International Agency for Research on Cancer considers benzo[*a*]pyrene (BaP) as the most potent carcinogen among PAHs.[31] The concentration of BaP is often positively correlated with those of other PAHs and total PAHs, allowing BaP to serve as a marker of PAHs in ETS.[16] The BaP equivalent (BaP_{eq}), which is calculated by multiplying the concentration of individual PAH with its toxicity equivalency factor (TEF), was used in the present study to facilitate the estimation of overall carcinogenicity.[32]

The incremental lifetime cancer risk (ILCR) was used to evaluate the potential risk for human health, in the present study, which was estimated per cigarette by[33]

$$\mathrm{ILCR} = \frac{\sum (\mathrm{TEF_{PAH}} \times \mathrm{EF_{PAH}}) \times \mathrm{IR} \times \mathrm{ET} \times \mathrm{ED} \times \mathrm{CSF} \times \mathrm{EFR}}{\mathrm{BW} \times \mathrm{AT} \times V_{\mathrm{room}}} \times 10^{-6}$$

where TEF_{PAH} is the TEF of PAH based on BaP (Table S2); EF_{PAH} is the EF of a particle-bound PAH released by each cigarette (ng cig^{-1}), which equals the concentration of PAHs multiplied by the room volume (72.5 m^3) and divided by the number of cigarettes consumed on the same day; IR is the inhalation rate (m^3 h^{-1}); ET is the event time, that is, the exposure duration in a day (h d^{-1}); ED is the lifetime exposure duration (year); CSF is the cancer slope factor of BaP [(mg kg^{-1} bw $d^{-1})^{-1}$]; EFR is the exposure frequency (d $year^{-1}$); BW is the average body weight (kg); AT is the average time for carcinogenic effects (d); and V_{room} is the room volume (m^3). Detailed values of these parameters vary with age groups (Table S3). The uncertainty in ILCR of respiratory exposure to particle-bound PAHs was evaluated by Monte Carlo simulation. The concentrations of PAHs were assumed to follow a logarithmic normal distribution, each with 10 000 iterations of simulation.

The creatinine-corrected concentrations of OH-PAHs in some participants reached a maximum before smoking and decreased throughout the entire study period, indicating significant accidental contributions from nonsmoking sources. Thus, 9 out of 49 pairs of urine samples before and after smoking were excluded for discussions, resulting in 40 pairs of urine samples for further consideration.

RESULTS AND DISCUSSION

Occurrence of Size-Fractionated PAHs. All PAHs discussed are particle-bound in the remaining part of this paper, except where specified. Concentrations of naphthalene, acenaphthylene, and anthracene in the particle samples were below the reporting limits; hence, these PAHs will be excluded for further discussions. The concentrations of PAHs increased considerably during smoking, and the average concentrations were 162 ± 84.0 and 242 ± 48.7 ng m^{-3}, respectively, in the smokes of higher-tar and lower-tar cigarettes (Table S4). The concentrations dropped to the environmental background levels by thorough cleaning and ventilation after each smoking event (Table S5). The composition profiles of individual PAHs released from higher-tar and lower-tar cigarettes were similar (Figure 1), with no significant difference (*t*-test; $p > 0.05$) between PAH concentrations in the two types of cigarettes.

The particle size distribution patterns of PAHs were generally similar between both the cigarette types, that is, PAHs were mainly concentrated on particles sized 0.10−1.8 μm. There was a unimodal peak for high-molecular-weight (HMW; 4−6 ring) PAHs in higher-tar tobacco smoke, while

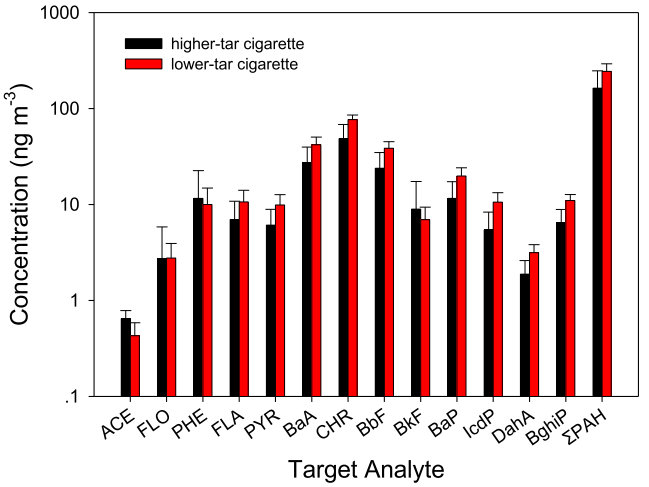

Figure 1. Concentrations of PAHs in ETS. All acronyms are defined in Table S2. Numerical concentration data are presented in Table S4.

HMW PAHs in lower-tar tobacco smoke showed a slightly bimodal distribution pattern (Figure S1). In addition, HMW PAHs in ETS of both higher-tar and lower-tar cigarettes were abundantly distributed in fine particles ($D_p < 1.8$ μm), whereas low-molecular-weight (LMW; 2−3 ring) PAHs were relatively more widely distributed in coarse particles ($D_p > 1.8$ μm) (Figure S2). These findings are consistent with previously reported results that the mass fraction of PAHs in coarse particles in indoor air of urban Guangzhou monotonically decreased with incremental molecular weight.[34] A plausible explanation is that LMW PAHs produced on fine particles during combustion easily migrate to coarse particles through volatilization and condensation,[35] while HMW PAHs are less volatile and therefore dominant in fine fractions.[36]

Emission Factors. The total EFs of PAHs were 230 ± 130 and 302 ± 71.3 ng cig^{-1} for higher-tar and lower-tar cigarettes, respectively, and highly particle size-dependent. In particular, particles in the range of 0.1−1.8 μm contained the most abundant PAHs (Figure 2). It should be noted that cigarettes with 10 times lower tar levels (marked on the packing boxes) actually produced higher total concentrations of particle-bound PAHs in ETS than cigarettes with higher tar level (Figure S3), suggesting no correlation between tar delivery levels in

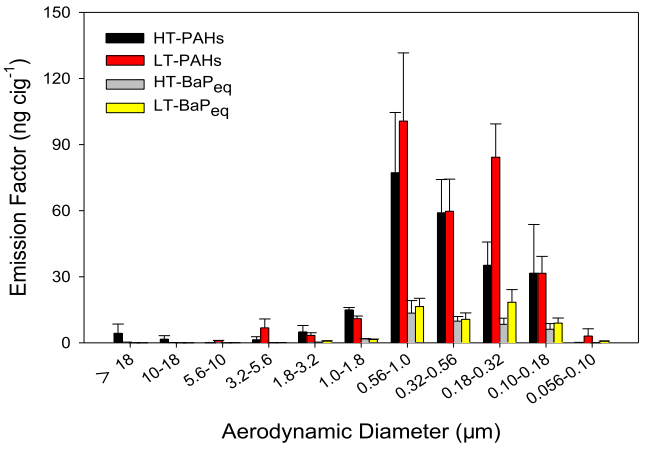

Figure 2. EFs of size-fractionated PAHs in ETS from higher-tar (HT) and lower-tar (LT) cigarettes.

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Table 1. Concentrations of PAH Metabolites (C; $\mu g\ g^{-1}$ Creatinine) in the Urine of Participants before and after Smoking

	higher-tar			lower-tar			
	C_{pre} [a]	C_{post} [b]	ΔC [c]	C_{pre}	C_{post}	ΔC	p value [d]
2-OH-Nap	1.7 (0.88, 8.8) [e]	3.4 (1.3, 16.1)	1.3 (0.10, 7.3)	2.3 (0.56, 38.9)	3.8 (1.3, 51.2)	1.4 (0.16, 12.3)	0.72
1-OH-Nap	1.0 (0.28, 6.7)	1.9 (0.42, 10.1)	0.96 (-, 5.5)	0.91 (0.09, 22.8)	2.2 (1.5, 40.9)	1.2 (0.05, 18.1)	0.31
2-OH-Flu	0.55 (0.15, 2.1)	0.92 (0.34, 6.2)	0.13 (-, 4.9)	0.66 (0.01, 8.1)	0.88 (0.60, 8.1)	0.28 (-, 4.3)	0.57
2-OH-Phe	0.11 (0.04, 0.26)	0.13 (0.08, 0.45)	0.01 (-, 0.22)	0.12 (0.01, 0.78)	0.15 (0.12, 1.1)	0.03 (-, 0.40)	0.34
3-OH-Phe	0.23 (0.11, 0.52)	0.29 (0.16, 0.74)	0.06 (-, 0.35)	0.27 (0.01, 1.49)	0.29 (0.21, 3.0)	0.08 (-, 1.5)	0.34
4-OH-Phe	0.06 (0.01,0.18)	0.11 (0.01, 0.21)	0.02 (-, 0.17)	0.06 (0.004, 0.10)	0.11 (0.03, 1.2)	0.03 (-, 0.34)	0.46
1 + 9-OH-Phe	0.22 (0.07, 0.63)	0.31 (0.10, 0.65)	0.09 (-, 0.25)	0.35 (0.01, 1.44)	0.30 (0.19, 2.5)	0.02 (-, 1.0)	0.30
∑OH-PAH	4.0 (2.1, 14.7)	8.2 (3.3, 26.6)	2.9 (1.3, 12.65)	4.7 (0.7, 72.4)	7.7 (5.4, 107.9)	2.9 (0.31, 35.8)	0.74

[a]Concentrations of PAH metabolites in urine before smoking, including 40 cases. [b]Concentrations of PAH metabolites in urine after smoking, including 40 cases. [c]Concentration change before and after smoking, that is, C_{pre} minus C_{post} for the same person. [d]The statistical significance of differences between changes of concentrations in urine (Mann−Whitney U test). [e]A (B, C) represents median (min, max).

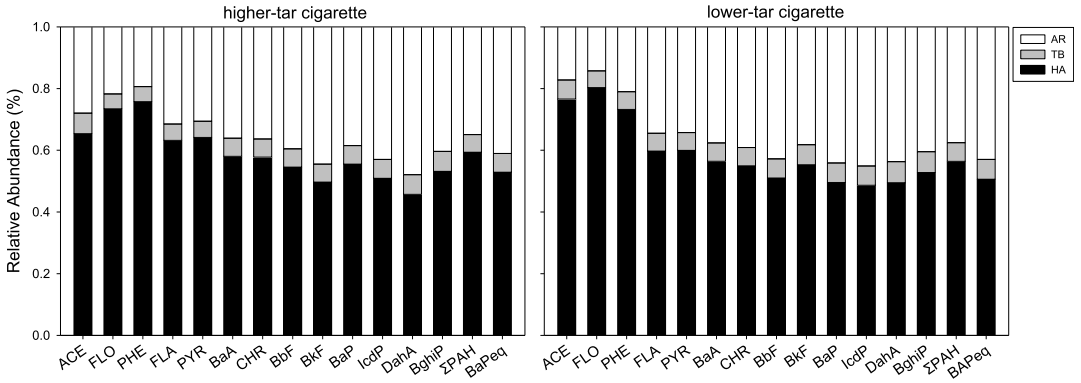

Figure 3. Relative abundances of PAHs deposited in the HA, TB region, and AR of the human respiratory tract. BaP_{eq} is the BaP equivalent of total PAHs.

mainstream cigarette smoke and ETS emissions of PAHs. Cigarette filter ventilation is expected to significantly impact tar delivery levels measured by machines and affect the efficiency of PAH emissions from cigarettes. A previous study on mainstream smoke found that strong ventilation reduced total smoke and thus the yields of PAHs.[37] However, different smoking patterns may alter ventilation effects. The Canadian Intense smoking regimen simulated by smoking machines obtained higher concentrations of individual PAHs in mainstream smoke than the International Organization of Standardization smoking regimen.[16] Smoking patterns for individual smokers can vary greatly, which can result in different yields of chemical compositions in tobacco smoke.[21]

Tobacco companies have used smoking machines to collect and analyze smoke samples for the yields of tar, nicotine, carbon monoxide, and other substances in order to produce the so-called low-tar cigarettes. However, machine-generated smoke is vastly different from that inhaled by humans. It is difficult to estimate the yields of carcinogens delivered per cigarette in mainstream smoke under realistic scenarios. Compared with mainstream smoke from Kentucky 3R4F reference cigarettes obtained by the International Organization of Standardization smoking regimen, concentrations of most HMW PAHs in ETS measured in the present study were higher, while LMW PAHs were relatively low.[16,17]

Urinary Excretion of PAH Metabolites. During sampling, the smokers were exposed to high concentrations of ETS and actively smoked at least four cigarettes. Thus, the creatinine-corrected concentrations of OH-PAHs in urine significantly increased after exposure to ETS (Mann−Whitney U test; $p < 0.05$) (Table 1). However, no significant difference was found between the cigarette types regarding the increased concentrations of PAH metabolites in urine upon exposure (Mann−Whitney U test; $p > 0.05$). This corroborates the findings that low-tar cigarettes do not deliver lower levels of nicotine and PAHs and are unlikely to reduce health risk than regular cigarettes.[12,13]

Inhalation Exposure to PAHs. Calculated based on the standard given by the International Standards Organization and American Conference of Governmental Industrial Hygienists,[29] the inhalable, thoracic, respirable, and deposition fractions of PAHs decreased in the sequence. Most particle-bound PAHs can be inhaled into the respiratory tract and transported deep into the respiratory system. Specifically, 83−99 and 67−99% of PAHs from higher-tar and lower-tar cigarettes, respectively, may reach the AR. However, only 17−41 and 18−53% of PAHs from higher-tar and lower-tar cigarettes were able to actually deposit in different regions of the human respiratory system (Table S6). Therefore, using bulk concentrations of PAHs may overestimate the risk of respiratory exposure to PAHs.

As determined by the simplified International Commission on Radiological Protection model, the relative abundances of PAHs (from both higher-tar or lower-tar cigarettes) deposited in different respiratory regions followed the sequence of HA, AR, and TB (Figure 3). Approximately 7.1−12 and 6.4−9.8% of inhaled PAHs from higher-tar and lower-tar cigarettes, respectively, are settled in AR. These values are comparable to those at selected indoor (8.6−10.2%) and outdoor (7.9−9.2%) environments of Guangzhou.[35] It is worth noting that inhaled

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Table 2. ILCR (10^{-6}) upon Exposure to PAHs in ETS for Different Exposure Scenarios

	bulk concentration			deposition concentration		
	higher-tar	lower-tar	combined[a]	higher-tar	lower-tar	combined
			Residence			
children	0.07 (0.009−0.60)[b]	0.11 (0.01−0.85)	0.09 (0.01−0.71)	0.02 (0.002−0.13)	0.02 (0.003−0.17)	0.02 (0.002−0.15)
adolescents	0.10 (0.007−0.44)	0.09 (0.01−0.60)	0.07 (0.01−0.52)	0.01 (0.002−0.09)	0.02 (0.002−0.12)	0.02 (0.002−0.11)
adults	0.18 (0.02−1.8)	0.28 (0.04−1.9)	0.23 (0.03−1.7)	0.04 (0.005−0.30)	0.05 (0.008−0.37)	0.05 (0.007−0.33)
seniors	0.04 (0.006−0.34)	0.068 (0.01−0.43)	0.06 (0.008−0.38)	0.009 (0.001−0.07)	0.01 (0.002−0.08)	0.01 (0.002−0.08)
			Office			
adults	0.11 (0.01−0.81)	0.17 (0.03−1.1)	0.14 (0.02−0.96)	0.02 (0.003−0.17)	0.03 (0.005−0.22)	0.03 (0.004−0.20)

[a]Calculated on the basis of the average values of two cigarette types. [b]*A* (*B*−*C*) represents a median value and 95% confidence interval. Detailed values of all parameters are provided in Table S3.

particles deposited in the respiratory tract, especially in the innermost AR, can be quickly transferred into the systemic circulation.[38]

The size distribution patterns of PAHs deposited in different respiratory regions were similar between the two cigarette types (Figure S4); that is, PAHs in coarse particles deposit more abundantly in the HA, while those in fine particles tend to deposit deeper into the respiratory tract. LMW PAHs have higher deposition fraction in the human respiratory tract than HMW PAHs, but they are mostly intercepted by the HA because they have higher affiliations with coarse particles. The dominance of HMW PAHs in AR is particularly significant because HMW PAHs are generally more toxic than LMW PAHs.[39]

Health Risk Assessment. Based on the relative TEF values, the BaP_{eq} values in ETS from higher-tar and lower-tar cigarettes were 28.1 ± 13.0 and 46.3 ± 97.5 ng m^{-3}, respectively (Table S4). The yield of BaP_{eq} per cigarette was 40.0 ± 20.0 ng cig^{-1} in ETS from higher-tar cigarettes and 57.6 ± 14.2 ng cig^{-1} in ETS from lower-tar cigarettes. Assuming smoking in a 100 m^3 room free of air pollution, BaP_{eq} emission from approximately two cigarettes may exceed the European Union's annual average BaP_{eq} standard (1 ng m^{-3}).[40] In a typical school, office, and residency of Guangzhou, China, the average concentrations of total BaP_{eq} were 1.2 ± 0.6, 1.4 ± 0.6, and 1.5 ± 0.6 ng m^{-3}, respectively,[41] which were slightly higher than the European Union's standard. Therefore, the BaP_{eq} concentrations in such a 100 m^3 room may exceed the Chinese daily average BaP_{eq} standard (2.5 ng m^{-3})[42] from smoking of just two cigarettes. Castro et al.[26] found that the concentration of particle-bound and gaseous BaP_{eq} in a smoking room was 47.4 ng m^{-3}, 2.5 times that (19.1 ng m^{-3}) in a nonsmoking room. Clearly, ETS from smoking habits has a strong impact on indoor air quality and human health.

The respiratory risk assessment needs to be considered in conjunction with the bioavailability and absorption of particle-bound PAHs, which is largely determined by the deposition of particles in different regions of the human respiratory tract. Therefore, the bulk and deposition concentrations of particle-bound PAHs were both used to estimate the ILCR, which represent the most severe and conservative respiratory exposure risks, respectively.

As people generally spend the majority of their time indoors, indoor air pollution is closely related to human health, especially for the elderly and children.[43] In the present study, the discussion of ILCR is based on the estimated concentration of PAHs in ETS released by a single cigarette in an ideal room of 100 m^3. To accommodate different exposure times and individuals' intake capacities, four age groups with various exposure scenarios (Table S3) were considered. The average inhalation ILCRs of total particle-bound PAHs under the residence scenario based on BaP_{eq} concentrations followed the sequence of adults (2.3×10^{-7} with 95% CI of 3.4×10^{-8} to 1.7×10^{-6}) > children (9.3×10^{-8} with 95% CI of 1.2×10^{-8} to 7.1×10^{-7}) > adolescents (7.3×10^{-8} with 95% CI of 1.0×10^{-8} to 5.2×10^{-7}) > seniors (5.6×10^{-8} with 95% CI of 8.3×10^{-9} to 3.8×10^{-7}) (Table 2). The ILCR values of the adults group may reach the risk threshold of 1.0×10^{-6} under this scenario,[33] and adults who are exposed to ETS in both offices and homes have an ILCR value of 3.7×10^{-7} (95% CI: 5.2×10^{-8} to 2.5×10^{-6}). When adults spend more than 10 h in rooms filled with ETS, potential cancer risk may become non-negligible. If a person has been exposed to secondhand smoke during the entire life span, the ILCR value would be 5.51×10^{-7} (95% CI: 1.2×10^{-7} to 2.7×10^{-6}) or 1.1×10^{-7} (95% CI: 2.5×10^{-8} to 5.3×10^{-7}) estimated by deposition concentrations. Although the estimated cancer risks are low, they are derived simply based on inhaled particle-bound PAHs. Exposure to gaseous PAHs and dermal contact with PAHs are also key contributors to cancer risk.[27,44] Hence, the actual health risks from exposure to PAH would be higher than those estimated herein. Cancer risk may vary with the number of cigarettes consumed in a room, room size, and other factors. For example, if there is a heavy smoker in the family, these values may have been underestimated. Statistical studies have shown that exposure to ETS from husbands, at work, and in childhood is associated with increased female mortalities, cancer hazards, and cardiovascular disease-induced deaths, respectively.[19,45]

Health Perspectives on Smoking. Epidemiological and environmental studies agree that chronic exposure to ETS will increase the incidence and mortality of lung and nasal cancer, as well as cardiovascular and respiratory diseases.[46,47] It is noteworthy that besides secondhand smoke, the residues left after smoking, the so-called thirdhand smoke, should not be overlooked.[48] Even if smoking stops for a long time, thirdhand smoke would continue to subject nonsmokers to inhalable nicotine and other tobacco smoke ingredients from dust.[49] Ventilation is often the main mechanism for achieving acceptable indoor air quality after smoking, but the effects are limited.[50] Clearly, stopping smoking is the only effective way to minimize potential adverse outcome from exposure to ETS.

To quell fear and reduce consumption, many smokers have tried to shift from one cigarette type to another, such as low-tar and electronic cigarettes. However, low-tar cigarettes do not consistently deliver fewer carcinogens than regular cigarettes, so the potential cancer risk is not necessarily reduced.

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Electronic cigarettes are not free of pollutant emissions, which may pose health hazards to smokers and secondhand smokers.[51] Reducing the consumption of cigarettes does reduce exposure to specific substances, but even low levels of smoking can lead to significant toxin exposure.[52] Therefore, kicking the habit is an irreplaceable choice to return to a healthy lifestyle and prevent/minimize any major health issue because of smoking in the later stage of life.

Although the perception of "low tar, less harmful" has proven to be wrong, China still has laws that require technological advances to reduce tar content, as well as explicitly tagging of tar delivery level in tobacco packaging boxes.[53] This undoubtedly makes tar level the reference standard for tobacco selection, so that the market and consumers are misguided. The government needs to take responsibility for building a nonsmoking environment and helping smokers quit smoking instead of promoting the use of the so-called low-hazard products. When smoking becomes unacceptable in a society, smokers with a sense of smoking-related stigma are more likely to quit smoking.[54]

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge at https://pubs.acs.org/doi/10.1021/acs.est.9b05802.

Basic characteristic of participants; acronyms, molecular weights, and toxicity equivalency factors; risk parameters; bulk and deposition concentrations; concentrations before and after smoking event; inhalable, thoracic, respirable, and deposition fractions; size distributions and distribution of PAHs; EFs of PAHs; and size-dependent deposition concentrations (PDF)

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Notes

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