Received: 15 July 2021 | Revised: 27 October 2021 | Accepted: 30 October 2021

DOI: 10.1111/ina.12956

ORIGINAL ARTICLE

WILEY

Characteristics and health risks of personal exposure to particle-bound PAHs for Hong Kong adult residents: From ambient pollution to indoor exposure

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Funding information
National Natural Science Foundation of China, Grant/Award Number: 41907181; Research Grant Council of the Hong Kong Special Administrative Region China, General Research Fund, Grant/Award Number: CUHK 14203719

Abstract

Research on individual level polycyclic aromatic hydrocarbons (PAHs) exposure is scarce. Moreover, the independent contribution of ambient- and indoor-origin PAHs to personal exposure remains poorly studied. We performed simultaneous ambient, residential indoor, and personal exposure measurements in a panel of healthy adults to investigate particle-bound PAHs, focusing on their carcinogenic congeners (cPAHs). Average PAH concentrations were much higher in ambient and residential indoor than personal exposure, with distinct seasonal variations. We employed chrysene as a tracer to investigate residential indoor and personal PAHs exposure by origin. Personal cPAH exposure was largely attributable to ambient-origin exposures (95.8%), whereas a considerable proportion of residential indoor PAHs was likely attributable to indoor emissions (33.8%). Benzo[a]pyrene equivalent (BaPeq) concentrations of cPAH accounted for 95.2%–95.6% of total carcinogenic potential. Uncertainties in estimated PAHs (and BaPeq) exposure and cancer risks for adults were calculated using the Monte Carlo simulation. Cancer risks attributable to ambient, residential indoor, and personal cPAH inhalation exposures ranged from 4.0×10^{-6} to 1.0×10^{-5}. A time-activity weighted model was employed for personal PAH exposure estimations. Estimated cPAH exposures demonstrate high cancer risks for adults in Hong Kong, suggesting that exposure to indoor-generated PAHs should be of great concern to the general population.

KEYWORDS
personal exposure, residential indoor, exposure factor, benzo[a]pyrene, inhalation cancer risks

1 | INTRODUCTION

The World Health Organization (WHO) has indicated that 7.0 million global premature deaths are annually attributed to ambient and indoor air pollution,[1] and about 92% of the world population lives in areas with fine particle ($PM_{2.5}$) concentrations exceeding the WHO air quality guideline (annual mean: 10 μg/m^3). In addition, the adverse impacts of $PM_{2.5}$ components on human health—including toxic organic species (e.g., polycyclic aromatic hydrocarbons, PAHs) and transition metals—have also been well documented.[2,3] Epidemiological studies regarding air pollution and health effects are generally based on ambient concentrations measured from fixed regulatory stations. However, people spend a significant proportion (80%–90%) of their daily time in indoor microenvironments and up

to 70% at home.[4] More recent research has indicated that indoor exposures result in equivalent or even higher adverse health effects compared to ambient air exposures.[5–7] Personal monitoring provides a more representative and accurate exposure measurement at the individual levels for subjects with similar economic, environmental, and behavioral variables.[8] Thus, many studies have indicated the necessity of personal and indoor monitoring for a more comprehensive exposure and health risk assessment.[9,10]

Increased lung cancer risks from occupational and environmental exposure to PAHs have been reported.[11] Concerning PAHs in indoor air and personal exposure, inhalation is one of the most important exposure pathways leading to lung cancer for adults in Asian cities.[12] Although assessing the harmful effects of individual PAH congeners is complicated, epidemiological studies have demonstrated associations between PAH mixture exposures and non-malignant respiratory diseases (e.g., asthma and chronic obstructive pulmonary disease).[13] Laboratory studies have indicated that variations in PAH toxicity were attributable to emission sources[14] and their synergistic (or) antagonistic effects compared to individual compounds (e.g., benzo[a]pyrene).[15] Studies in Hong Kong revealed that PAH compounds in the ambient atmosphere mainly originated from local vehicle emissions and regional pollution (e.g., industrial processes, coal combustion, and biomass combustion).[11,16] Guo et al.[17] (2003) suggested that high molecular weight PAHs contribute dominantly to the particulate-phase (78%–100%) in ambient $PM_{2.5}$ in Hong Kong. Moreover, PM-bound PAH congeners (including benzo[a]pyrene, dibenz[a,h]anthracene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, indeno[1,2,3-cd]pyrene) exhibited high carcinogenicity, mutagenicity and toxic potency.[2,3,18]

Many studies have investigated concentrations and sources of indoor and ambient PM-associated PAHs,[19–21] with growing evidence supporting the link between indoor PAHs exposure and adverse health risks.[22,23] Residential indoor PAHs due to outdoor infiltration, indoor emission sources (e.g., fuel and cooking, smoking, and incense burning), and personal activities are critical variables that need to be considered in quantifying indoor and personal exposure to PAHs.[24] Previous studies have employed chrysene that has limited indoor sources as a reference compound to estimate ambient-origin PAH exposures in indoor environments.[25–27] Nevertheless, characteristics of $PM_{2.5}$-bound PAH mixtures in simultaneous ambient-indoor-personal exposure are limited,[25] with less attention directed toward different exposure categories. Therefore, investigating the independent contribution of ambient- and indoor-origin PAH to total personal PAH exposures is of significant importance.

This work presents the results obtained from a panel of Hong Kong adult residents.[6] A subgroup of participants performed simultaneous ambient, residential indoor, and personal exposure sample collection for $PM_{2.5}$-bound PAH compounds. Subsequently, we estimated adults exposure to PAHs using a time-activity weighted model, which considers infiltration of ambient PAHs to indoor microenvironments, indoor-generated PAHs, and the influence of personal activities. The present study aims to: (1) characterize the variations of PAH mixture concentrations in ambient, residential indoor, and personal exposures; (2) explore the relative contribution of ambient- and non-ambient–origin (or indoor-origin) PAHs to personal and residential indoor exposures, respectively; and (3) assess cancer risks attributable to PAH inhalation exposure for Hong Kong adult residents using the Monte Carlo simulation.

Practical Implications

- Total personal PAH exposures originate from both ambient-origin and non-ambient generated exposures. Therefore, simultaneous ambient-residential indoor-personal exposure measurements are needed before carrying out epidemiological studies to distinguish the relative contributions of exposure metrics.
- The lower personal exposure to PAHs of ambient origin in adult participants than residential indoor PAH exposures of ambient-origin suggests a protective effect of PAH exposure from the office setting.
- The estimated carcinogenic risks attributable to PAH inhalation exposures exceed the acceptable level (1.0×10^{-6}) for Hong Kong adults. These results indicate priority should be given to controlling indoor and ambient air pollution emissions to reduce population exposure to PAHs.

2 | MATERIALS AND METHODS

2.1 | Participants and personal $PM_{2.5}$ monitoring

Details of study design (e.g., subject enrollment), personal $PM_{2.5}$ sample collection, and biomonitoring are described in other publications.[6,28] Briefly, we employed a random sampling strategy to recruit healthy non-smoking adult residents in Hong Kong, with no gender, occupation or spatial location restrictions. Seventy-nine healthy adults (>18 years of age, non-smokers with no pre-existing chronic respiratory or coronary diseases and related comorbidities) living in different districts of Hong Kong responded to the online advertisements.[6] Among the potential participants, 56 met the eligibility criteria and agreed to participate in personal exposure monitoring. All eligible participants were invited, and only 26 subjects agreed to participate in the indoor monitoring study. Figure S1 shows the residential locations of participants. One individual performed personal monitoring per household.

Twenty-four-hour (24 h) personal $PM_{2.5}$ exposure was measured directly using a personal environmental monitor (PEM) connected to a Leland Legacy pump (SKC Inc., Eighty-Four). Fifty-six participants performed repeated (two-day) personal $PM_{2.5}$ measurements between June 2014 and March 2016. Forty-five (45) and 43 participants were monitored in the summer and winter seasons, respectively, with 62.5% participating in both seasons. 2–6 personal $PM_{2.5}$ samples were collected from each participant; a total of 180 personal $PM_{2.5}$ samples were submitted for chemical analyses. In

addition, participants were required to fill out a 10-min questionnaire related to socio-demographics (e.g., age, gender, occupation, and social-economic status) and indoor physical conditions (e.g., living space, air conditioning, cooking fuel, ventilation). Participants were suggested to carry the sampler along with them (e.g., awake time outdoors) and maintain regular activities. They also completed an activity diary recording their daily activities at a 15–30 min resolution in each sampling session (24 h). These activity data have been classified into different microenvironments or activities, including residential indoor, office or school indoor, other indoors (shopping mall, restaurant), outdoor (e.g., walking), and commuting (bus, metro).

2.2 | Simultaneous ambient, residential indoor, and personal $PM_{2.5}$ monitoring

In a subset of 26 (46.4%) subjects, we conducted simultaneous 24 h ambient, residential indoor, and personal $PM_{2.5}$ exposure measurements. Detailed information about the location and characteristics of the outdoor sampling sites is presented in Table S1. Briefly, we performed ambient $PM_{2.5}$ sample collection at three university campuses in Hong Kong, including one at Hong Kong Polytechnic University (HKPU) (eg, near the Cross Harbour Tunnel with dense traffic). The other two sites were at the Chinese University of Hong Kong (CUHK) and The University of Hong Kong (HKU), respectively (Table S1). These sampling sites represent air pollution exposures in typical urban areas (https://www.aqhi.gov.hk/en.html). Ambient $PM_{2.5}$ samples were collected using a Mini-Volume air sampler (Airmetrics) (Figure S2). Ambient sampling was performed on the same sampling days in summer and winter, respectively, as residential indoor and personal monitoring. Ambient samples were collected at the same region (Hong Kong Island, Kowloon, New Territories) where indoor monitoring was conducted. Moreover, residential indoor air sampling was consistent with the protocols followed for ambient $PM_{2.5}$ monitoring. The sampling device (i.e., Mini-Volume air sampler) was placed in the living room and set at the height of 1.2–1.5 meters above the floor to collect residential indoor $PM_{2.5}$ samples (Figure S2). For ambient measurements, the samplers were fixed on the roof of selected buildings, while personal $PM_{2.5}$ samples were collected in the breathing zone of participants (Section 2.1). A total of 126 $PM_{2.5}$ samples were obtained from residential indoors ($n = 63$) and ambient sites ($n = 63$). Research assistants would check the activity dairies after the daily sampling session. Paired samples (residential indoor-ambient-personal) with 24 h running time and 1440-min time-activity data were considered valid and included in the time-activity weighted model.

2.3 | Laboratory analysis of particle-bound PAHs

$PM_{2.5}$-bound PAHs were quantified using the thermal desorption-gas chromatography/mass spectrometer (TD-GC/MS) method, with details of the analytical protocol described in another publication.[29] The analyzed PAH compounds for this study—including acenaphthylene (Acy), acenaphthene (Ace), fluorine (Flu), phenanthrene (Phe), anthracene (Ant), fluoranthene (Flut), pyrene (Pyr), benz[a]anthracene (BaA), chrysene (Chr), benzo[b]fluoranthene (BbF), benzo[k]fluoranthene (BkF), benzo[a]pyrene (BaP), dibenz[a,h]anthracene (DBA), indeno[1,2,3-cd]pyrene (IcdP), benzo[ghi]perylene (BghiP)]—are on the U.S. Environmental Protection Agency (EPA) list of priority pollutants. The International Agency for Research on Cancer (IARC) and the U.S. EPA classify BaP as a human carcinogen (Group 1).[18] DBA was classified as a probable human carcinogen, and BaA, Chr, BbF, BkF, IcdP were classified as possible human carcinogens. Table S2 shows the method detection limits for individual PAH congeners. Field blanks were analyzed along with filter samples, and PAH concentrations were reported by subtracting blank results. The individual PAH congeners were detectable for >87.8% of the samples except DBA and IcdP (60.6%–72.8% detectable). Naphthalene was not detectable in any samples, thus was not reported in this study. The total of the 15 U.S. EPA priority PAHs and seven carcinogenic PAHs summed up as ∑15PAHs and cPAHs, respectively. $PM_{2.5}$, organic carbon, and elemental carbon mass concentrations in ambient air, residential indoor and personal exposure have been reported previously in another publication.[28]

2.4 | Estimation of ambient-origin PAHs exposure

Figure 1 shows the schematic framework of study design and the relations between personal and indoor PAH exposures with ambient concentrations. Both ambient-origin and non-ambient–origin (indoor-generated and (or) personal activity-related) sources contribute to residential indoor and personal PAH exposures. We employed chrysene as a proxy to calculate ambient exposure factor (f_{pex_PAHs}) and infiltration factor (F_{inf_PAHs}) of ∑15PAHs and cPAHs employing the following equations:

$$f_{pex\_PAHs} = Chr_{P_{ij}} / Chr_{O_j} \quad (1)$$

$$F_{\text{inf\_PAHs}} \propto Chr_{I_{ij}} / Chr_{O_j} \quad (2)$$

where $(Chr)_{Pij}$ represents the personal exposure to chrysene for subject i on sampling day j, $(Chr)_{Oj}$ represents the ambient chrysene concentration on day j, and $(Chr)_{Iij}$ refers to the residential indoor chrysene concentration measured on day *j* for subject *i*.

Subsequently, ambient-origin PAH exposures in personal exposure and residential indoors were calculated using the following equation:

$$E_{a\_PAHs} = f_{pex\_PAHs} \times O_{PAHs} \quad (3)$$

$$E_{i\_PAHs} = F_{\text{inf\_PAHs}} \times O_{PAHs} \quad (4)$$

where E_{a_PAHs} refers to personal exposure to PAHs of ambient origin and E_{i_PAHs} indicates residential indoor PAHs of ambient origin. Full details regarding ambient-origin PAH exposure estimation and the corresponding non-ambient exposures are described in the Supporting Information (Text 1).

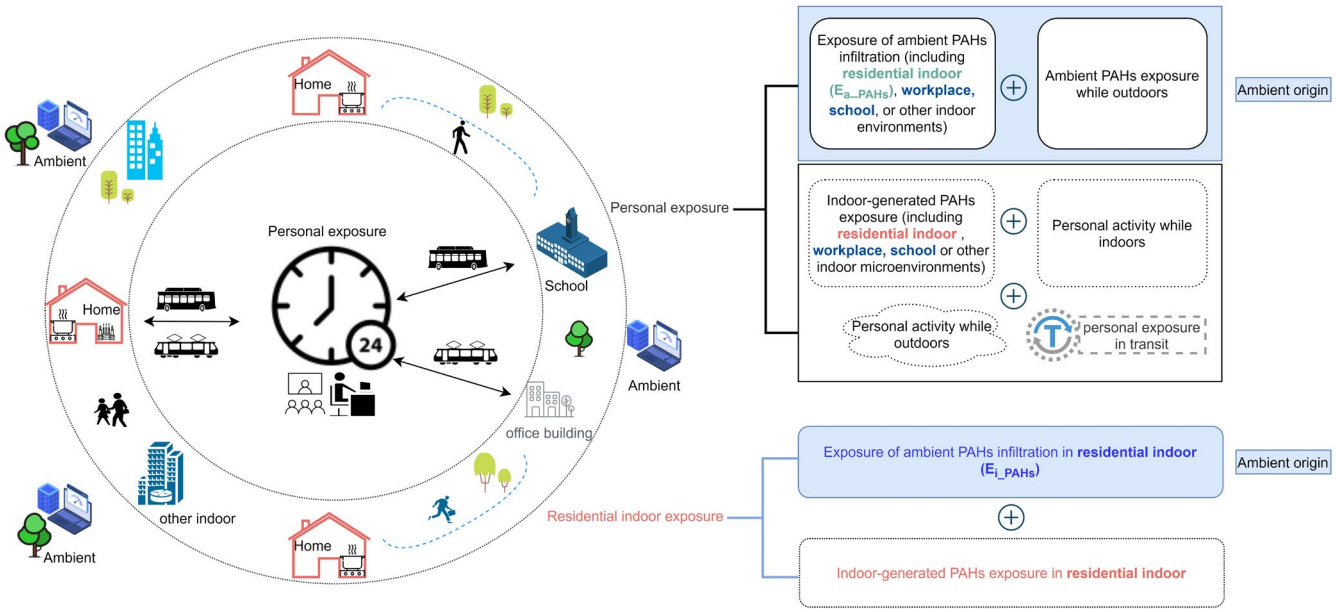

FIGURE 1 Schematic of the research framework relating measured ambient (O), residential indoor (I) and personal exposure (P) PAHs and diagram of factors influencing residential indoor and personal exposures. Notes: E_{a_PAHs} refers to personal exposure to PAHs of ambient origin; E_{i_PAHs} refers to residential indoor PAHs of ambient origin

2.5 | Health risks of inhalation exposure to PAHs

In the present study, we used BaP as an index to predict the carcinogenicity of PAH mixtures.[30] BaP equivalent concentrations (BaP_{eq}) were calculated as the summation of individual PAH concentrations (PAH_i) (ng/m^3) multiplied by its corresponding toxic equivalency factor (TEF_i), defined in Eq. 5.

$$BaP_{eq} = \sum_{i=1}^{n} (PAH_i) \times TEF_i \quad (5)$$

The carcinogenic proportion of individual PAH congeners to the total carcinogenic potency of BaPeq was calculated using Eq. (6):

$$\text{Carcinogenic Potential } (\%) = \frac{PAH_i \times TEF_i}{BaP_{eq}} \times 100\% \quad (6)$$

Lifetime cancer risks attributable to PAH inhalation exposures were estimated by multiplying BaP_{eq} exposure concentrations (ng/m^3) with the inhalation cancer unit risks of exposure to BaP (UR_{BaP}).

$$\text{Cancer Risk } = BaP_{eq} \times UR_{Bap} \quad (7)$$

The TEF_i values and included PAH congeners varied in different studies (Table S3). Some included the U.S. EPA priority PAHs, and other publications only included carcinogenic PAHs.[14] This study incorporates the TEF scheme for potency values of individual PAH compounds via the inhalation route developed by the U.S. EPA Integrated Risk Information System.[31] The WHO recommended a UR_{BaP} value of 8.7×10^{-5} (ng/m^3)$^{-1}$ based on an epidemiology study in coke-oven workers in Pennsylvania, USA,[30] and the UR_{BaP} value was defined as the theoretical upper limit for developing cancer when exposed to BaP at an average concentration of 1 ng/m^3 over a 70-year lifetime. The California Environmental Protection Agency suggested a UR_{BaP} value of 1.1×10^{-6} (ng/m^3)$^{-1}$ based on an animal study.[32] Thus, we used the WHO recommended UR_{BaP} value to calculate inhalation cancer risks in this study.

2.6 | Modeled PAH exposures and Monte Carlo simulation

We employed a time-activity weighted model to estimate personal exposure to PAH mixtures (and BaPeq) for adults using the following equation:

$$\text{Estimated exposure} = \sum_{k=1}^{n} C_{ik} t_{ijk} / T_{ij} \quad (8)$$

where *Cik* represents the PAH concentrations in microenvironment k for subject i, and t_{ijk} refers to the time in microenvironment k for subject i on sampling day j. T_{ij} is the total sampling time (24 h).

The time-activity weighted model has been employed in other publications for personal PAHs exposure modelling.[33,34] In the current study, residential indoor PAHs were directly measured; workplace, school, or other indoor PAH concentrations were estimated based on ambient PAH concentrations and infiltration factors (Table 1). In addition, a Monte Carlo simulation was employed to estimate the distribution of PAH exposures (Table S4) and cancer risks attributable to PAH inhalation exposure for adults.

2.7 | Statistical analysis

PAH concentrations are reported in ng/m^3. A Kolmogorov-Smirnov (K-S) test was used to investigate the normality of ambient, residential indoor, and personal exposure to PAHs. Individual PAH congeners and PAH mixtures were right-skewed (p-value for K-S test < 0.01). Spearman correlation (r_s) was employed to evaluate the correlations of PAH mixtures among ambient, indoor, and personal exposures. Statistical analyses were performed using R 3.5.1 (R Development Core Team, 2018: http://www.r-project.org). A p-value < 0.05 was considered statistically significant. The Monte Carlo simulation was performed 10,000 times to address the uncertainties of probabilistic risk assessment in R.

3 | RESULTS AND DISCUSSION

3.1 | Characteristics of study participants

Table 2 shows the characteristics of study participants and their activity patterns throughout the sampling campaign. These study subjects were classified as students and office workers with ages ranging from 18 to 42 years, of which 39.3% (N = 22) were females, and 60.7% (N = 34) were males. All participants reported that they were not exposed to environmental tobacco smoke in residential indoors or other indoor microenvironments (e.g., workplace, school) (Table 2). In general, participants spent more than 90% of their daily time indoors, and 72.3%–73.8% were at home. Results show on average the office workers and students spent approximately 7.2 h (29.9%) in the workplace and 4.9 h (20.3%) at school, respectively, 1-hour (2.6%–3.6%) outdoors, and more than 1-hour in the commute system (e.g., bus and MTR). Many previous studies in Hong Kong have confirmed these findings and revealed similar diurnal time-activity patterns for office workers and students.[4,35] Moreover, the time-activity data for all participants were consistent with those who participated in concurrent measurements. Consistent daily activities suggesting the subgroup characteristics were representative of all study subjects.

Characteristics of household environmental factors related to indoor air pollution are shown in Table S5. Air conditioning was widely used in indoor microenvironments during the summer season for Hong Kong people to cope with hot temperatures. The majority of study participants indicated no incense burning activity at home (Table S5). About 75% of the participants (or their family members in the same household) engaged in frequent cooking activities (e.g., 3–7 times/week). Town gas (i.e., natural gas), liquid petroleum gas (LPG) and electricity are the primary sources of household cooking energy. In addition, LPG is an important fuel for taxis and light buses and is commonly used for residential cooking in Hong Kong.[36]

3.2 | Occurrence of $PM_{2.5}$-bound PAHs at the individual level

The measured individual PAH congeners and PAH mixtures in personal exposure are listed in Table S6 and summarized in Table 3. The average concentrations of ∑15PAHs and cPAHs measured in personal exposures were 0.93 ± 0.43 ng/m^3 and 0.34 ± 0.17 ng/m^3, respectively. PAH exposure concentrations for all subjects were comparable with the exposure levels for those who performed simultaneous personal measurements (p > 0.05; Table S6). Higher PAH exposure concentrations were shown in winter compared to

TABLE 1 Summary of exposure metrics and input variables in time-activity weighted exposure model

Exposure metrics	Calculation
Ambient PAHs (O)	Measured
Residential indoor PAHs (I)	Measured
Personal exposure to PAHs (P)	Measured
Predicted personal exposure to PAHs (Estimated)	Modelled
Prediction-based, personal PAHs exposure of ambient origin (E_{a-PAHs})	f_{pex}*O
Prediction-based, non-ambient PAHs exposure ($E_{na-PAHs}$)	P - f_{pex}*O
Prediction-based, indoor PAHs exposure of ambient origin (E_{i-PAHs})	F_{inf}*O
Prediction-based, indoor-generated generated PAHs exposure ($E_{ig-PAHs}$)	I - F_{in}*O
Input variables for modelled $BaPeq_{-cPAHs}$ exposure concentration	
Ambient BaPeq concentration exposure (O_{-BaPeq})	O_{-BaPeq}*fraction of time outdoors[a]
Residential indoor BaPeq concentration (I_{-BaPeq})	I_{-BaPeq}*fraction of time in residential indoors
BaPeq concentration in the office building	f_{pex}(0.38)*O_{-BaPeq} *fraction of time in the workplace
BaPeq concentration at school	F_{inf}(0.66)*O_{-BaPeq} *fraction of time at school
BaPeq concentration in other indoors (eg, restaurant)	Enrichment factor(1.15)*O_{-BaPeq} *fraction of time in other indoors
BaPeq concentration in commute	Infiltration factor(0.92[b])*O_{-BaPeq} *fraction of commuting time

[a]Results about the time in different microenvironments were derived from the time-activity diary for participants.

[b]Data referenced from Gariazzo et al. (2015).

TABLE 2 Characteristics of participants and summary of activity data for adult participants during the study period

Item	Summer	Winter	Total[d]	Current measurement[e]
Sampling date	June – October 2014 and August– September 2015	November 2014–March 2015 and January–March 2016	June 2014–March 2016	June 2014–March 2016
Study subjects (*N*[a])	45 (32)	43 (32)	56	26
Never smokers[c] (Yes/No, %)	/	/	Yes, 100%	Yes, 100%
Female	19	15	22 (39.3%)	7 (26.9%)
Male	26	28	34 (60.7%)	19 (73.1%)
Age (years; median (range))	/	/	25 (18–42)	24 (18–42)
Weight (kg) (mean, SD[b])	/	/	60.3 (11.5)	60.4 (15.5)
College student (*n*, %)	29	24	36 (64.3%)	17 (63.0%)
Office worker (*n*, %)	16	19	20 (35.7%)	10 (37.0%)
Personal activity[f] (%), median (mean, SD)				
Indoors, total (%)	91.3 (83.5, 15.8)	95.8 (94.3, 5.2)	93.8 (92.5, 9.0)	93.8 (89.0, 19.1)
Indoors, at home	73.3 (72.3, 20.9)	79.2 (73.8, 22.8)	79.2 (74.6, 21.2)	73.3 (72.3, 24.6) *(57.1–59.4)*[g]
Indoors, cooking/dining	4.2 (4.5, 5.1)	5.7 (6.3, 5.5)	4.4 (6.1, 6.5)	4.2 (5.4, 5.3)
Indoors, cleaning activities	0 (1.5, 4.6)	0 (1.7, 4.8)	0 (2.1, 5.3)	0 (1.6, 4.6)
Indoors, workplace	0 (9.0, 16.5)	0 (11.2, 16.7)	0 (11.3, 16.9)	0 (10.1, 16.2) *(29.9, 13.4)*[g]
Indoors, school	0 (2.4, 7.9)	0 (3.8, 9.2)	0 (3.2, 8.6)	0 (3.1, 8.6) *(20.3, 11.8)*[g]
Time spent outdoor (%)	2.1 (2.9, 3.7)	2.6 (2.9, 3.2)	2.1 (3.7, 7.2)	2.4 (3.2, 4.0) *(2.6–3.6)*[g]
Time spent in transit (MTR, bus, minibus) (%)	1.2 (4.3, 5.6)	0 (2.2, 3.4)	0.7 (3.6, 5.1)	0 (3.2, 4.7) *(3.5–6.8)*[g]

[a]Number of participants.

[b]SD denotes standard deviation.

[c]All participants were non-smokers and not exposed to environmental tobacco smoke (ETS).

[d]Subjects who participated in personal $PM_{2.5}$ monitoring.

[e]Subjects conducted simultaneous personal/indoor/ambient measurement.

[f]A total of 169 personal activity diaries were collected.

[g]Workday or school day, include only those that reported office/school time.

summer for participants ($p < 0.001$; Table S7). A similar seasonal trend of increased PAH exposure concentrations in winter was observed in other personal exposure studies.[37,38] This study showed no significant gender or occupational (e.g., university student vs office worker) differences in personal exposure to PAH congeners or PAH mixtures across participants (data not shown).

The average BaPeq exposure concentrations derived from the 15 U.S. EPA priority PAHs (i.e., BaPeq-$_{15PAHs}$) was 0.05 ± 0.03 ng/m^3 (Table 3). Similarly, the average concentration of cPAHs decreased from 0.34 ng/m^3 to 0.05 ng/m^3 after conversion to their BaPeq exposure concentrations (BaPeq-$_{cPAHs}$). The variation in BaPeq concentrations was attributable to the inclusion of different PAH congeners and their TEF values. In this study, although a substantial fraction of measured PAH concentrations comprised of low molecular weight compounds, comparable BaPeq-$_{15PAHs}$ and BaPeq-$_{cPAHs}$ concentrations were shown ($p > 0.05$) and exhibited a striking similarity with time-series distribution (Figure S3). Daily BaPeq concentrations in personal exposure (<0.2 ng/m^3) remained lower than the European Union annual average BaPeq standard level (1 ng/m^3)[39] throughout the study period. It should be stressed that the cited standard is only ambient pollution based, and the general adult populations would indeed have had non-ambient–origin exposures. For example, Liu et al.[40] (2007) investigated personal PAH exposures in traffic police officers in Beijing, China. They found that average personal BaPeq exposure (winter: 82.1 ng/m^3) was significantly higher than the ambient air standard. Data from another study showed considerably higher PAH exposures in industrial workers compared to the general population (e.g., non-smokers).[41]

3.3 | Variation of PAHs in ambient, residential indoor, and personal exposure

This study presents a unique comparison regarding PAH composition profiles and PAH mixture concentrations (e.g., ∑15PAHs and cPAHs) among ambient (O), residential indoor (I), and personal exposure (P) (Table 3). Flut (0.14–0.33 ng/m^3), Ant (0.14–0.30 ng/m^3), and Chr (0.11–0.26 ng/m^3) were found to be the most dominant PAH compounds in different exposure categories. The average ambient ∑15PAHs concentrations (2.03 ± 0.79 ng/m^3) were two times higher than those measured in personal exposures ($p < 0.001$). Much higher ambient ∑15PAHs concentrations were shown in other Chinese cities (e.g., Guangzhou: 6.77 ng/m^3; Xiamen: 4.35 ng/m^3) than the current findings.[16] Similar seasonal variability of PAH concentrations (winter >summer) was observed in ambient and residential indoors and consistent with other observations.[25,42] For example, Lv, Zhu (2013)[43] measured PAHs in different public places (e.g., supermarket and shopping center) in Hangzhou, China, and found that air conditioning reduces indoor particulate-bound PAH concentrations in summer. Ma et al.[44] (2016) suggested that the substantially higher PAH concentrations during the winter in Hong Kong can be attributed to the dominant contribution of regional pollution.

The average ∑15PAHs concentrations in ambient air ranged from 1.80 ng/m^3 to 2.30 ng/m^3 (Table S8). Slightly higher ambient PAH congeners, cPAHs, and ∑15PAHs concentrations ($p = 0.06$) were observed at the HKPU site. Spatial variations in ambient PAH concentrations were consistent with previous findings,[17] indicating that these PAH compounds at the road site (HKPU) were mainly attributable to traffic emissions in Hong Kong. The 95th percentile of ∑15PAHs concentrations at the HKPU was 3.64 ng/m^3 during 2014–2016, a dramatic decline of 90% from 2000 (average ∑15PAHs: 33.96 ng/m^3).[17] A recent study in Hong Kong corroborated these findings and revealed a remarkable decrease in PAH concentrations in ambient air.[44] In another study, Leung et al.[16] (2014) measured PAHs in ambient $PM_{2.5}$ in Hong Kong, in which the most abundant PAH species were IcdP, BghiP, BbF, and BkF in the winter season. Residential indoor ∑15PAHs concentrations varied from 0.64 ng/m^3 to 2.42 ng/m^3 with an average of 1.65 ± 0.94 ng/m^3. PAHs in residential indoors exhibited substantially higher variability (characterized by coefficients of variance, CV = 57.0%) than those in ambient air. Furthermore, previous studies indicated residential floor level and building types were factors affecting variability in residential PAHs concentrations.[45]

Table 3 also shows the average I/O, P/O, and P/I ratios for PAH compounds. The P/O PAH (including individual PAH congeners, cPAHs, ∑15PAHs) ratios were less than 1, suggesting personal PAH exposures were mainly attributable to ambient PAHs infiltration. Three subjects (out of 25) were exposed to higher PAHs than in ambient air or indoors (Figure S4). Individual PAH congener ($p < 0.05$) and PAH mixture concentrations (∑15PAHs, cPAHs) ($p < 0.001$) were the highest in ambient air on 89.8% of the sampling days than in personal exposure or residential indoor (Figure S5). In general, other than residential indoors, the workplace is likely to provide greater protection among office workers.[46] The results show 96% of the households have kitchen ventilation (Table S5). Gonzalez et al.[47] (2019) indicated that mechanical and natural ventilation could effectively decrease indoor $PM_{2.5}$ concentration during cooking, and particle concentration would return to the no event level (or baseline levels) in about 40 min (ranging from 20 min to 12.5 h).[48] These results indicate that cumulative measurement may not always capture the short-term spikes; however, the adverse effects of indoor cooking fuel and cooking process on long-term personal PAHs exposure cannot be ignored.

As for I/O PAHs ratios, low molecular weight PAHs (e.g., Acy and Ant) were characterized by higher I/O ratios (>1.0), implying the contribution of indoor emission. Diagnostic PAH ratios were employed to investigate the PAH emission sources in ambient, residential indoor and personal exposure (Table S9), with additional details shown in Supporting Material (Text 2). Ambient sources (vehicle emission, coal combustion) was the most dominant factor influencing personal PAHs exposure. These results agreed with a prior observation that revealed vehicle exhaust and regional pollution were significant contributors to ambient PAHs in Hong Kong.[44] In the current study, Flut was the most abundant PAH compound, suggesting coal and petroleum combustion were the dominant sources of origin. These findings could be attributable to the residential energy transition in Hong Kong over the past decades (e.g., increased

TABLE 3 Average concentrations of $PM_{2.5}$-bound PAHs monitored in ambient air (O), residential indoor (I), and personal exposure (P) along with the corresponding concentration ratios

	Ambient ($n = 63$)		Indoor ($n = 63$)		Personal ($n = 63$)					
ng/m^3	Mean ± SD[a]	95th	Mean ± SD[a]	95th	Mean ± SD[a]	95th	*p*-value[b]	I/O ratio[c]	P/O ratio[d]	P/I ratio[e]
Acenaphthylene (Acy)	0.05 ± 0.03	0.13	0.05 ± 0.03	0.11	0.03 ± 0.02	0.08	<0.001	1.13 ± 0.84	0.72 ± 0.65	0.83 ± 0.68
Acenaphthene (Ace)	0.03 ± 0.03	0.09	0.03 ± 0.04	0.14	0.02 ± 0.02	0.06	0.03	0.94 ± 0.96	0.78 ± 0.79	0.66 ± 0.71
Fluorine (Flu)	0.07 ± 0.05	0.17	0.05 ± 0.04	0.12	0.03 ± 0.03	0.08	<0.001	0.90 ± 0.77	0.80 ± 1.06	0.99 ± 1.03
Phenanthrene (Phe)	0.19 ± 0.11	0.42	0.13 ± 0.10	0.34	0.09 ± 0.08	0.24	<0.001	0.98 ± 1.19	0.67 ± 0.93	0.99 ± 0.80
Anthracene (Ant)	0.30 ± 0.27	1.03	0.25 ± 0.17	0.60	0.14 ± 0.12	0.43	<0.001	1.11 ± 0.88	0.68 ± 0.84	0.79 ± 0.70
Fluoranthene (Flut)	0.33 ± 0.15	0.69	0.27 ± 0.17	0.63	0.14 ± 0.08	0.27	<0.001	0.95 ± 0.80	0.50 ± 0.43	0.74 ± 0.53
Pyrene (Pyr)	0.15 ± 0.06	0.28	0.11 ± 0.08	0.27	0.07 ± 0.05	0.14	<0.001	0.86 ± 0.89	0.58 ± 0.88	0.94 ± 0.70
Benz[a]anthracene[f] (BaA)	0.05 ± 0.03	0.13	0.05 ± 0.04	0.13	0.02 ± 0.01	0.05	<0.001	0.76 ± 0.68	0.61 ± 0.86	0.97 ± 1.16
Chrysene[f] (Chr)	0.26 ± 0.14	0.58	0.21 ± 0.14	0.50	0.11 ± 0.06	0.21	<0.001	0.93 ± 0.76	0.49 ± 0.39	0.71 ± 0.45
Benzo[b]fluoranthene[f] (BbF)	0.17 ± 0.10	0.39	0.17 ± 0.13	0.46	0.09 ± 0.06	0.20	<0.001	0.93 ± 0.81	0.55 ± 0.37	0.92 ± 0.92
Benzo[k]fluoranthene[f] (BkF)	0.14 ± 0.08	0.31	0.12 ± 0.09	0.31	0.06 ± 0.04	0.14	<0.001	1.06 ± 1.00	0.52 ± 0.33	0.85 ± 0.80
Benzo[a]pyrene[f] (BaP)	0.06 ± 0.04	0.17	0.05 ± 0.04	0.13	0.02 ± 0.02	0.06	<0.001	1.01 ± 0.99	0.59 ± 0.83	0.81 ± 1.07
Dibenz[a,h]anthracene[f] (DBA)	0.01 ± 0.01	0.03	0.01 ± 0.01	0.03	0.01 ± 0.004	0.02	0.003	0.89 ± 0.70	0.94 ± 1.72	1.25 ± 0.95
Indeno[1,2,3-cd]pyrene[f] (IcdP)	0.09 ± 0.06	0.22	0.06 ± 0.05	0.18	0.04 ± 0.03	0.09	<0.001	0.99 ± 1.01	0.64 ± 0.65	1.11 ± 1.19
Benzo[ghi]perylene (BghiP)	0.15 ± 0.12	0.41	0.11 ± 0.09	0.34	0.06 ± 0.04	0.14	<0.001	1.05 ± 1.12	0.58 ± 0.71	0.94 ± 0.93
∑15PAHs	2.03 ± 0.79	3.64	1.65 ± 0.94	3.98	0.93 ± 0.43	1.69	<0.001	0.92 ± 0.74	0.48 ± 0.38	0.73 ± 0.46
cPAHs	0.76 ± 0.39	1.67	0.65 ± 0.44	1.78	0.34 ± 0.17	0.64	<0.001	1.15 ± 1.47	0.50 ± 0.30	0.79 ± 0.60
$BaPeq_{-15PAHs}$	0.12 ± 0.07	0.25	0.10 ± 0.07	0.25	0.05 ± 0.03	0.11	<0.001	1.09 ± 1.42	0.52 ± 0.35	0.81 ± 0.71
$BaPeq_{-cPAHs}$	0.11 ± 0.07	0.25	0.09 ± 0.07	0.24	0.05 ± 0.03	0.10	<0.001	1.00 ± 1.06	0.55 ± 0.43	0.85 ± 0.80

[a]SD denotes standard deviation.

[b]A p-value < 0.05 indicates the between-groups difference (personal-ambient-indoor) is statistically significant.

[c]I/O ratio: indoor-to-ambient concentration ratio; outliers (ratios greater than 10.0) was excluded.

[d]P/O ratio: personal-to-ambient concentration ratio.

[e]P/I ratio: personal-to-indoor concentration ratio; outliers (ratios greater than 10.0) was excluded.

[f]Carcinogenic PAHs.

natural gas consumption).[36] Sources of residential indoor PAHs have been reported in another publication in Tong et al.[49] (2019); the results indicated that vehicle emission, cooking activities, and indoor incense burning were the dominant sources.[49] Other studies reported similar findings; for example, Zhu, Wang (2003)[50] suggested that the abundance of 3–4 ring PAHs in residential indoors could be apportioned to cooking activities. Shi (2018)[20] and Chen et al.[51] (2017) indicated that indoor air pollution was a significant source of low molecular weight PAHs. In addition, the 95th percentile value of residential indoor cPAHs concentration was higher than in ambient with average I/O ratio > 1.0 (i.e., 1.15 ± 1.47), suggesting a substantial contribution of indoor-generated emissions (e.g., using LPG as cooking fuel) to residential PAH exposures.[20,22,24] We further calculated enrichment factors (EFs) for supporting this hypothesis (Figure S6). Previous studies focusing on indoor PAHs in the urban and rural areas reported similar findings, with evident residential indoor sources (e.g., kerosene, wood, and LPG as cooking fuel).[21,50,52,53] Zhu, Wang (2003)[50] demonstrated higher I/O cPAHs ratios in Hangzhou, China, suggesting substantial contributions of cooking practice and ambient infiltration. Minguillon et al.[54] (2012) indicated that indoor PM could induce accumulation of high molecular weight PAHs that were more carcinogenic than 3- or 4-ring PAHs.

3.4 | Estimation of PAH exposures of ambient origin

We employed chrysene as a tracer to estimate personal exposure to PAHs of ambient-origin (E_{a_PAHs}) and residential indoor PAHs of ambient-origin (E_{i_PAHs}). The median I/O and P/O Chr ratios were 0.66 and 0.38, respectively (Table S10). The particle-bound PAHs infiltration factors (I/O) were comparable with those reported in previous findings (Kraków, Poland: 0.54; Rome, Italy: 0.66; Guangzhou, China: 0.61).[25,26,33] Higher I/O and P/O Chr ratios were observed in winter compared to summer. These results agreed well with the previous finding; relatively lower infiltration efficiencies (0.40–0.45) were found in mechanically ventilated office buildings in Hong Kong than home indoors,[55] with higher infiltration efficiencies in the cold season than in the warm season.

Figure 2–3 show the distribution and contribution of ambient-origin ∑15PAHs and cPAHs to personal exposure and residential indoors. Lower levels of exposure to ∑15PAHs (E_{a_15PAHs}; $p < 0.001$) and cPAHs of ambient-origin (E_{a_cPAHs}; $p < 0.001$) were shown for the study participants compared to residential indoor PAHs of ambient-origin (E_{i_15PAHs}, E_{i_cPAHs}). Personal ∑15PAHs ($p = 0.80$) and cPAHs concentrations ($p = 0.70$) were comparable with their ambient-origin exposures (Figure 2). Moreover, as shown in Figure 3, ambient-origin ∑15PAHs and cPAHs contributed the most (95.8%–98.3%) to personal exposures. These results can be explained by the fact that participants spent their daytime hours in a school/office setting with limited indoor sources. Therefore, limited non-ambient generated ∑15PAHs and cPAHs exposure was shown. In a study conducted in an urban community in Camden, New Jersey, Zhu et al.[52] (2011) found that ambient-origin PAH exposure explained 44%–96% of the variability in personal exposures.

Indoor-generated PAH exposures (E_{i_PAHs}) accounted for 29.7%–33.8% of residential indoor PAHs concentrations (Figure 3). Such results were consistent with our previous findings regarding the contribution of non-ambient $PM_{2.5}$ exposure (33.2%) to total personal exposure in adult residents of Hong Kong.[56] Moreover, E_{a_PAHs} was relatively lower than E_{i_PAHs}, suggesting that the office setting was less prone to the influence of ambient-origin PAHs than the residence. These findings were consistent with the results reported in Zhou, Zhao (2012)[57] and Romagnoli et al.[58] (2014). A previous study revealed that approximately 55% of total indoor $PM_{2.5}$ was attributable to residential emissions in urban areas in China.[59] Further, a study in Beijing demonstrated that 26.9%–32.6% of indoor BaPeq exposures resulted from indoor-origin pollution.[60]

Limited studies have demonstrated variation of PAH concentrations in ambient-indoor-personal exposures.[25,37,53] Figure 4 shows the correlation matrix for PAH mixtures among ambient, residential indoor, and personal exposure. There was a moderate correlation between ambient and personal exposure to cPAHs ($r_s = 0.50$; $p < 0.001$) (Figure 4). No consistent associations were established for individual PAHs in residential indoor with those in ambient or personal samples, suggesting the substantial contribution of indoor-generated emissions. Correlation coefficients for individual PAH congeners were listed in Table S11.

3.5 | Estimation of inhalation cancer risks

Consistent with the results regarding PAH mixture concentrations, the average $BaPeq_{\text{-}15PAHs}$ and $BaPeq_{\text{-}cPAHs}$ concentrations in ambient (0.11–0.12 ng/m^3) and residential indoor (0.09–0.10 ng/m^3) were about two times those measured in personal exposure assessments (0.05 ng/m^3).

We performed a Monte Carlo simulation for adults' exposure to $BaPeq\text{-}_{cPAHs}$ concentrations. An I/O infiltration ratio of 0.66 and a P/O exposure factor of 0.38 were employed in exposure modelling for school and office settings, respectively (e.g., input variables are shown in Table 1). As shown in Figure 4, the distributions of modeled ∑15PAHs ($r_s = 0.98$; $p < 0.001$), cPAHs ($r_s = 0.94$; $p < 0.001$), and $BaPeq\text{-}_{cPAHs}$ concentration (0.09 ± 0.05 ng/m^3) agreed well with measured residential indoor exposures. From this study conducted in Hong Kong, average BaPeq concentrations in ambient and indoor settings were comparable or higher than those in Los Angeles, California (0.07 ng/m^3), Houston, Texas (0.03 ng/m^3)[21]; Grenoble, France (0.07 ng/m^3), but significantly lower than those in Chinese cities (e.g., $BaPeq_{\text{-}cPAHs}$: 1.47 ng/m^3, $BaPeq_{\text{-}15PAHs}$: 2.3 ng/m^3 in Beijing).[19,61] Carcinogenic PAHs and $BaPeq\text{-}_{cPAHs}$ exposure concentrations in some residential indoor Hong Kong locations were higher than those of ambient concentrations, demonstrating the potential risk of indoor pollution to human health. Other studies revealed similar findings; for instance, Wang et al.[62] (2020) reported higher BaPeq concentrations indoors (e.g., dormitory, office, and laboratory) than the ambient air in Wuhan, China.

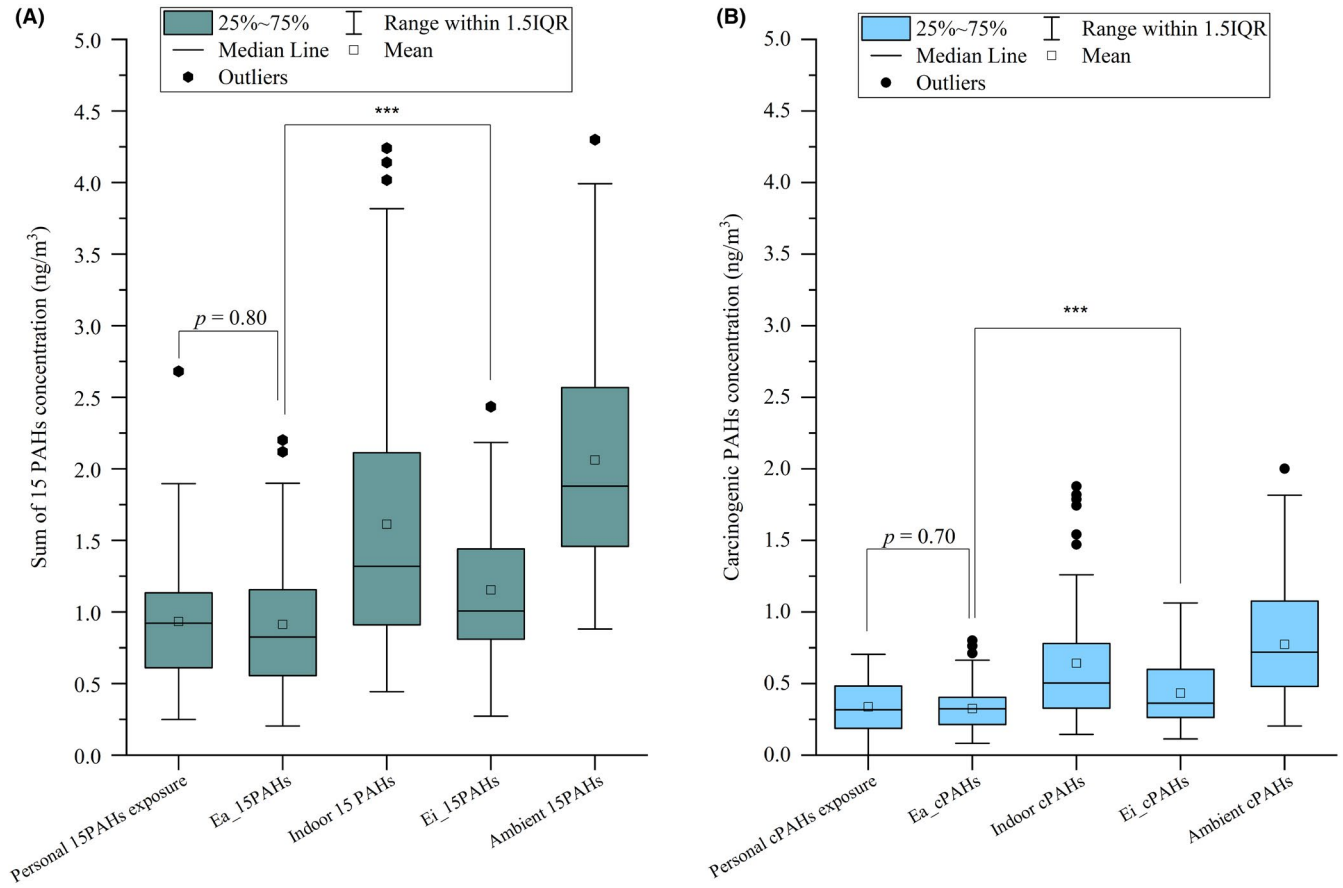

FIGURE 2 Boxplots of (A) ∑15PAHs and (B) cPAHs in ambient, residential indoor and personal exposure monitoring along with personal exposure to PAHs of ambient-origin (E_{a_PAHs}) and residential indoor PAHs of ambient origin (E_{i_PAHs}) using chrysene as a tracer. Notes: I/O chrysene ratios greater than unity were replaced with F_{inf} of 0.66 (median value of chrysene I/O ratio). $^{***}p < 0.001$

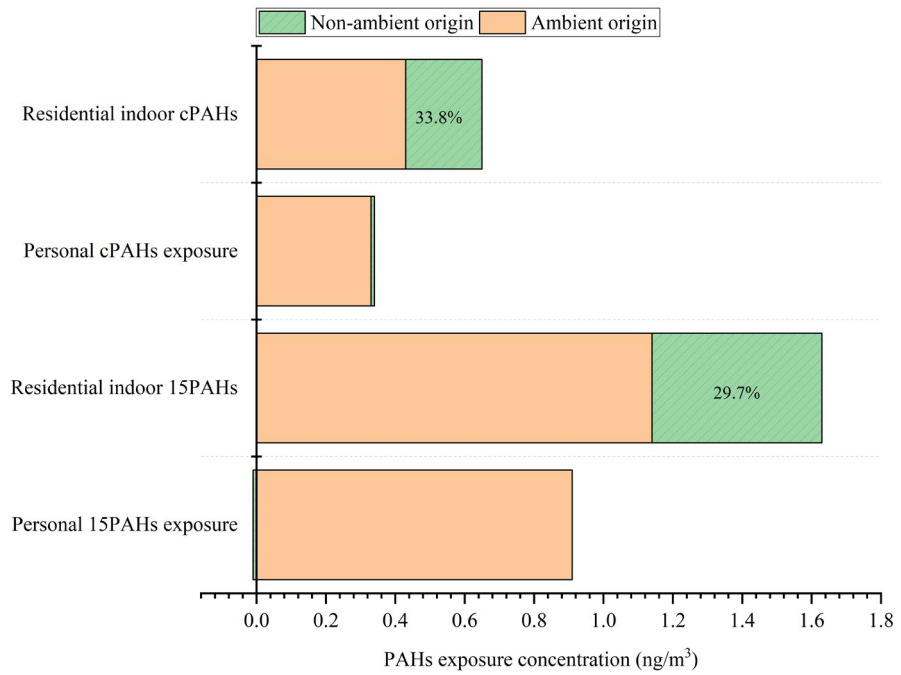

FIGURE 3 Estimated personal exposure to PAHs of ambient-origin (E_{a_15PAHs}, $E_{a\_cPAHs,}$) and residential indoor PAHs of ambient origin (E_{i_PAHs}, E_{i_cPAHs}) along with non-ambient PAH exposures

As shown in Figure 5, BaP (46.5%), BbF (18.4%), and BkF (14.1%) contribute most to the total BaPeq carcinogenicity. The relative contribution of BaP to the overall carcinogenicity of PAH mixtures was the highest in residential indoors (48.8%). Average cPAHs contributed 36.6%–39.4% to ∑15PAHs concentrations, while BaPeq-$_{cPAHs}$ accounted for 95.2%–95.6% of the total carcinogenic potential

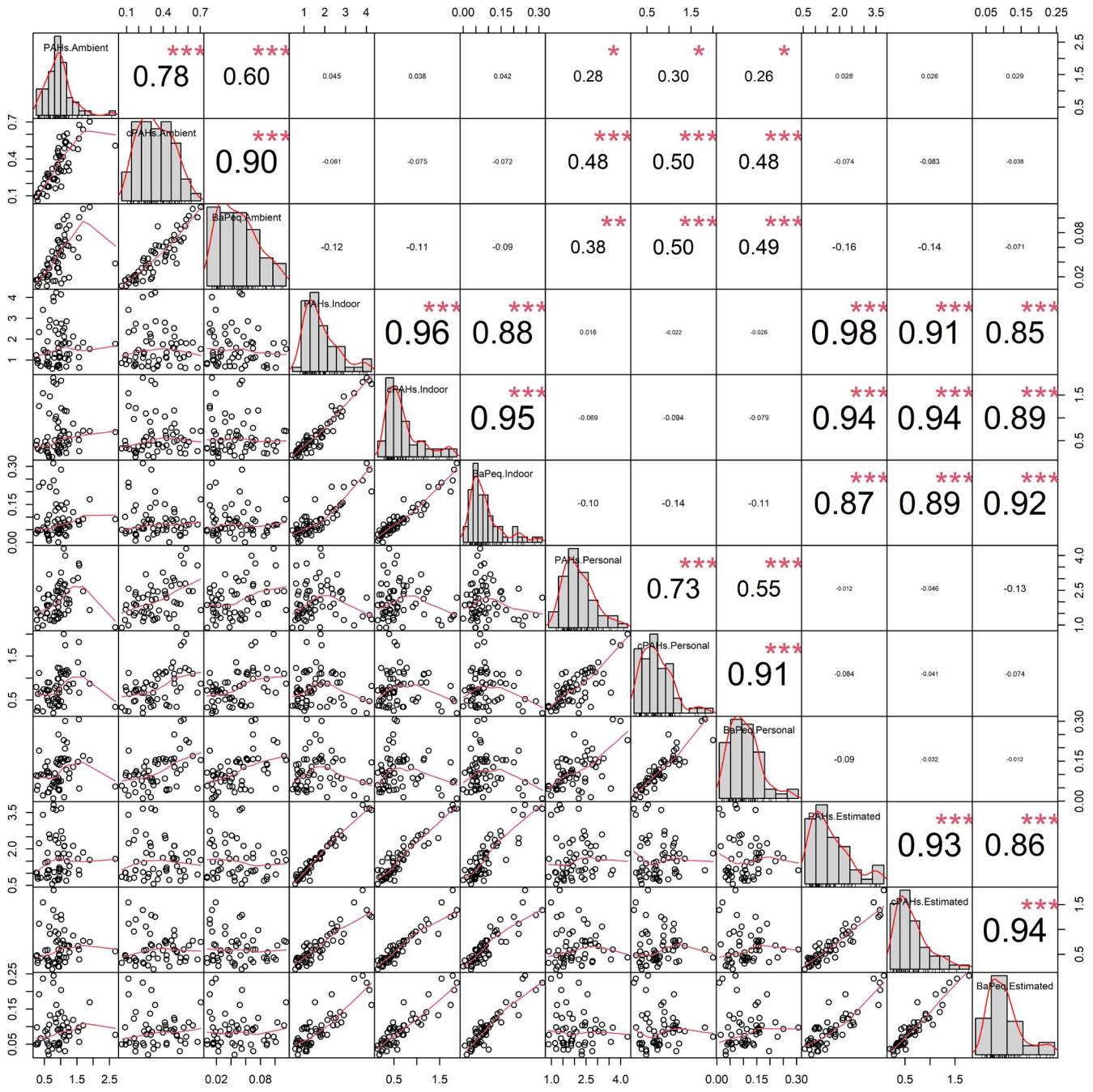

FIGURE 4 Spearman's correlation matrix between PAHs (∑15PAHs, cPAHs and BaPeq-$_{cPAHs}$) exposure categories. Notes: Ambient, indoor and personal PAHs were measured directly. The modelled value refers to adults' PAH exposures estimated using the time-activity weighted model. Notes: $^{*}p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$

(Figure 5). Strong correlations (r_s = 0.99; $p < 0.001$) were shown between BaPeq-$_{cPAHs}$ and BaPeq-$_{15PAHs}$ across different exposure metrics (Figure S7). These results were consistent with previous findings,[19] providing evidence that BaPeq-$_{cPAHs}$ is a suitable proxy for exposure to individual carcinogenic PAHs. Looking toward reducing exposures, Elzein et al.[61] (2020) suggested focusing on mitigating carcinogenic PAHs emissions (e.g., BaP, DBA, BbF, BkF, and IcdP) to reduce the adverse effects of exposure to ambient PAHs in Beijing, something that is also relevant to Hong Kong.

The cancer risks attributable to inhalation exposure of cPAHs exceeded 1×10^{-6} for all exposure scenarios (Figure S8), implying that the abundance of cPAHs mainly determines inhalation carcinogenic risks. Cumulative frequency distributions of cancer risks associated with cPAHs inhalation exposure are demonstrated in Figure 6. The differences are shown as cancer risks caused by ambient concentration (1.0×10^{-5}), and residential indoor cPAHs (8.0×10^{-6}) were higher than personal exposures (4.0×10^{-6}). The high carcinogenic risk of residential indoor air could be

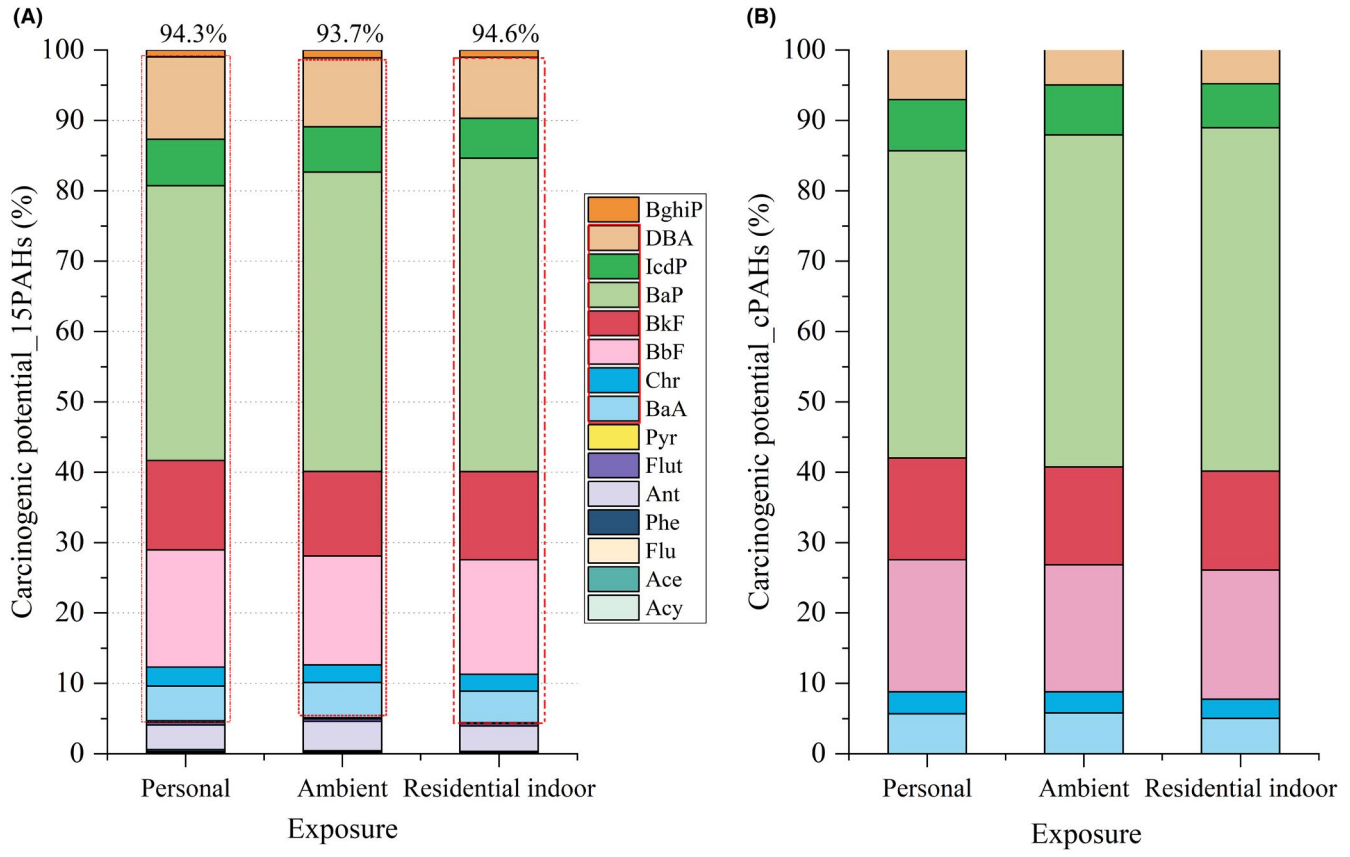

FIGURE 5 Percentage contribution of individual PAH congeners to PAH mixtures (A: ∑15PAHs; B: cPAHs) total carcinogenic potential in personal, ambient, and residential indoor, respectively

attributable to the simultaneous impacts of ambient-origin and indoor-generated PAHs. The 95th percentile value of cancer risks posed by PAH exposures was lower than the acceptable level when the CalEPA UR_{BaP} value was employed (data not shown). However, the adverse effects of PAH exposures should not be overlooked. These findings corroborate previous studies, where positive associations between cPAH exposure concentrations with $PM_{2.5}$ toxicity in residential indoor and airway inflammation in adult participants were observed.[6,49]

This paper provides a unique case study to explore PAH concentrations across different exposure categories—from ambient pollution, indoor-origin exposure to total personal exposure—and the potential cancer risks apportioned to PAH inhalation exposures in very high-density environments of Hong Kong. Leung et al.[16] (2014) reported inhalation cancer risks attributable to ambient ∑15PAHs was 6.8×10^{-6} in Hong Kong.[16] Hong et al. (2016) Hong et al.[63] (2016) performed an air monitoring program to characterize PAH concentrations in five Asian countries, determining the lifetime excess carcinogenic risks caused by PAH exposure were 1.36×10^{-6} and 2.45×10^{-6} in Japan and South Korea. Another study demonstrated that excess annual lung cancer incidence attributable to inhalation PAHs in the Chinese population was 6.5×10^{-6}.[11]

Our modelling results provide additional information on personal exposures to carcinogenic PAHs in adult residents of Hong Kong. We extended our analysis to include both ambient and indoor-generated PAHs in light of these findings. Thus, a time-activity weighted model that incorporated ambient exposure concentration, indoor exposure, and subjects' activity patterns were established. The 95th percentile value of inhalation cancer risks posed by modelled cPAHs exposure was 1.68×10^{-5}, indicating moderate potential cancer risks for adults in Hong Kong (Figure 6). Zhang et al.[53] (2019) suggested using LPG as cooking fuel may not effectively alleviate the risks of inhalation exposure to PAHs. Our findings indicate that cancer risks are underestimated by 57% if non-ambient–origin PAHs inhalation is not considered in risk assessment. Such models could be applied to estimate personal exposure to PAHs for Hong Kong adult residents with no environmental tobacco smoke exposure. They assume greater importance as cities become more compact and populous, forcing individuals and households to live at higher net densities. Falling per capita indoor living space in the centers of many high-income cities and the low-income cities of the developing world is largely accepted by those who calculate that living and working in the city brings net benefits over time.

This work has its uncertainties and limitations. Although repeated personal measurements were performed, the research findings regarding personal PAH exposure concentrations should be interpreted cautiously. The fitted exposure model was derived from a small sample size of personal data (with similar activity patterns) for the given season/year and may not be fully transferable to the general populations (e.g., tobacco smokers) or other localities. A larger

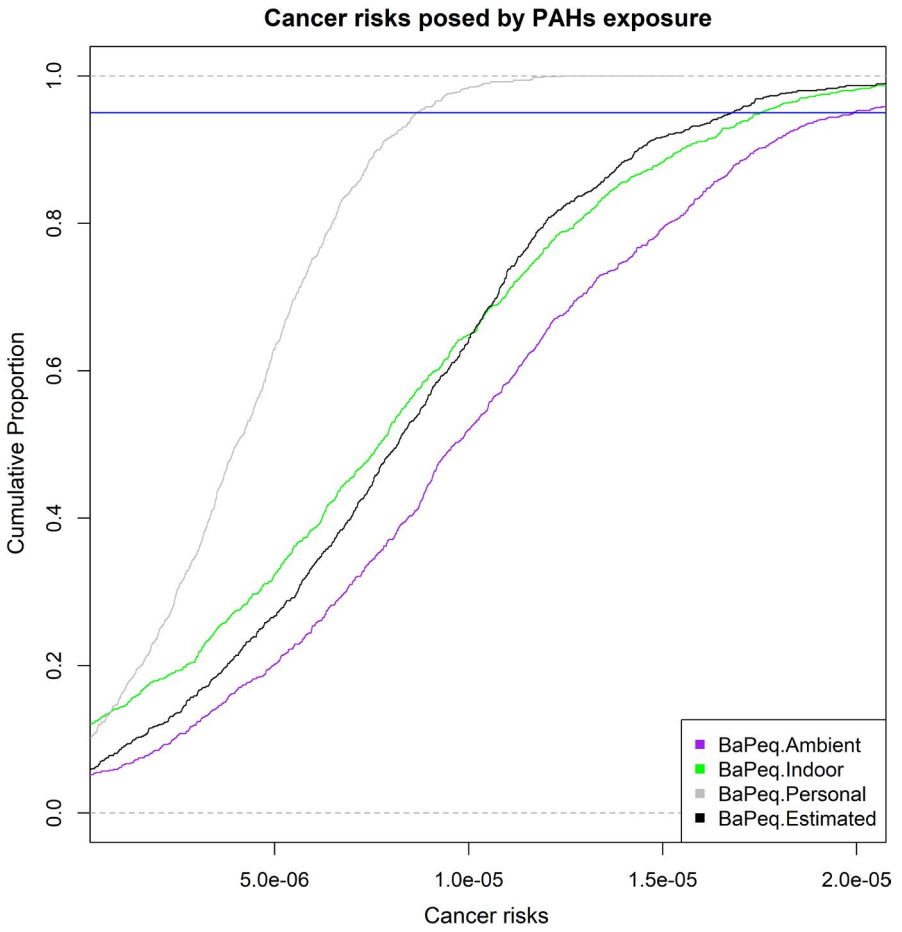

FIGURE 6 Comparison of the cumulative probability of cancer risks attributable to PAH inhalation exposures—ambient, residential indoor, personal exposure, estimated exposure—for adults in Hong Kong. Notes: We repeated the random event 10 000 times to perform the Monte Carlo simulation; Blue line: 95th percentile

dataset with a longer sampling time would improve the estimation accuracy. Other influencing factors of exposure to PAHs, for example, residential floor level, building types, and road proximity, warrant further investigation. Many of the health costs of high-density urban living, such as the carcinogenic indoor air studied in this paper and density-related stress and mental health problems,[64,65] are not immediately detected or understood by residents. Secondly, investigating cancer risks of PAH inhalation exposures presents some challenges, and the uncertainties are inherent in cancer risk assessments. The carcinogenicity of PAH relative to other ambient and indoor carcinogens is an area of research. Our findings add to a body of evidence that points to public health education (for example, indoor incense burning and cooking practices that vapourize oil) and perhaps density regulations. Despite the limitations, this study has the merit of measuring and modelling total personal PAH exposures from a panel of adults, including ambient- and indoor-origin exposure, which is essential for risk assessment and management in a major urban airshed (Hong Kong).

4 | CONCLUSIONS

Residential indoor environments are of fundamental importance where urban residents spend >70% of their daily time. As a result of COVID-19 lockdown, more people have shifted toward working from home and spending more time indoors—along with being exposed to emissions from intensive indoor activities (e.g., cooking and cleaning). As a result, understanding our residential indoor air quality is more important than pre-pandemic. This study investigated characteristics and variation in simultaneous ambient, residential indoor, and personal exposure to individual PAH and PAH mixtures. Notable seasonal variation was found for most PAH congeners in residential indoor and personal exposure, with higher concentrations measured in the winter compared to summer. Residential indoor and personal PAH exposures were more heterogeneous compared to ambient PAHs. Personal PAH exposures were strongly affected by PAHs from ambient air. Apart from ambient-origin exposures, indoor-generated PAHs were also an important factor affecting residential indoor PAHs. Notably, we report a 57% under-estimation of lung cancer risks if non-ambient–origin PAHs are disregarded in the risk assessment calculations exemplifying the importance of improving the quality of the residential indoor environment. In the current study, compared with ambient samples, the estimated PAH exposures were more reliable in determining cancer risks because they capture the unique and combined effects of both ambient-origin and non-ambient–origin exposures. These findings provide a deeper scientific understanding of the complex associations between different exposure categories and health effects and suggest that mitigation efforts are necessary to reduce PAH emissions within ambient and indoors to protect public health.

ACKNOWLEDGEMENTS

Xiao-Cui Chen acknowledges the National Natural Science Foundation of China [grant no. 41907181]. This study was supported by grant from the Research Grant Council of the Hong Kong Special Administrative Region China, General Research Fund (Project No. CUHK 14203719).

CONFLICT OF INTEREST

No conflict of interest declared.

AUTHOR CONTRIBUTIONS

XCC and **KFH** conceived and planned the experiments. **XCC** analyzed the data, performed the modelling, and prepared the original draft. **TJW, CS,** and **CW** aid in review and editing the manuscript. All authors provided critical feedback and approved the final version of the manuscript.

PEER REVIEW

The peer review history for this article is available at https://publons.com/publon/10.1111/ina.12956.

DATA AVAILABILITY STATEMENT

The data source is from Prof. Kin-Fai Ho, The Jockey Club School of Public Health and Primary Care, The Chinese University of Hong Kong, Hong Kong, China (kfho@cuhk.edu.hk).

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

How to cite this article: Chen X-C, Ward TJ, Ho K-F, Sarkar C, Webster C. Characteristics and health risks of personal exposure to particle-bound PAHs for Hong Kong adult residents: From ambient pollution to indoor exposure. *Indoor Air*. 2022;32:e12956. https://doi.org/10.1111/ina.12956