INTRODUCTION

The World Health Organization (WHO) has indicated that 7.0 million global premature deaths are annually attributed to ambient and indoor air pollution, 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. 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. More recent research has indicated that indoor exposures result in equivalent or even higher adverse health effects compared to ambient air exposures. Personal monitoring provides a more representative and accurate exposure measurement at the individual levels for subjects with similar economic, environmental, and behavioral variables. Thus, many studies have indicated the necessity of personal and indoor monitoring for a more comprehensive exposure and health risk assessment.

Increased lung cancer risks from occupational and environmental exposure to PAHs have been reported. 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. 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). Laboratory studies have indicated that variations in PAH toxicity were attributable to emission sources and their synergistic (or) antagonistic effects compared to individual compounds (e.g., benzo[a]pyrene). 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). Guo et al. (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, benzo[a]anthracene, chrysene, benzo[k]fluoranthene, benzo[k]fluoranthene, indeno[1,2,3-cd]pyrene) exhibited high carcinogenicity, mutagenicity and toxic potency.

Many studies have investigated concentrations and sources of indoor and ambient PM-associated PAHs, with growing evidence supporting the link between indoor PAHs exposure and adverse health risks. 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. Previous studies have employed chrysene that has limited indoor importance. This work presents the results obtained from a panel of Hong Kong adult residents. A subgroup of participants performed simultaneous exposure monitoring per household.

### 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 ($1.0 \times 10^{-6}$) 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.

## 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. 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. 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 questionaire 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) (e.g., 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 diaries 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. The analyzed PAH compounds for this study—including acenaphthylene (Acy), acenaphthene (Ace), fluoranthene (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). 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 $\sum_{15}$PAHs 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.

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 $\sum_{15}$PAHs and cPAHs employing the following equations:

\[
f_{pex\_PAHs} = \frac{Chr_i}{Chr_Oj} \quad (1)
\]

\[
F_{inf\_PAHs} \propto \frac{Chr_i}{Chr_Oj} \quad (2)
\]

where $(Chr_i)_{Oj}$ represents the personal exposure to chrysene for subject $i$ on sampling day $j$, $(Chr_Oj)$ represents the ambient chrysene concentration on day $j$, and $(Chr_i)_{ij}$ 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:

\[
E_{pex\_PAH} = f_{pex\_PAH} \times O_{PAH} \quad (3)
\]

\[
E_{inf\_PAH} = F_{inf\_PAH} \times O_{PAH} \quad (4)
\]

where $E_{pex\_PAH}$ refers to personal exposure to PAHs of ambient origin and $E_{inf\_PAH}$ 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).
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/m³) multiplied by its corresponding toxic equivalency factor (TEF i), defined in Eq. 5.

\[
BaP_{eq} = \sum_{i=1}^{n} (PAH_i) \times TEF_i
\]  

(5)

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

\[
\text{Carcinogenic Potential (\%)} = \frac{PAH_i \times TEF_i}{BaP_{eq}} \times 100\%
\]  

(6)

Lifetime cancer risks attributable to PAH inhalation exposures were estimated by multiplying BaP eq exposure concentrations (ng/m³) with the inhalation cancer unit risks of exposure to BaP (UR BaP).

\[
\text{Cancer Risk} = BaP_{eq} \times UR_{BaP}
\]  

(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 \times 10^{-5} (ng/m³)^{-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/m³ over a 70-year lifetime. The California Environmental Protection Agency suggested a UR BaP value of 1.1 \times 10^{-6} (ng/m³)^{-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 BaP eq) for adults using the following equation:

\[
\text{Estimated exposure} = \sum_{k=1}^{n} C_{ik} t_{jk} / T_{ij}
\]  

(8)

where C ik represents the PAH concentrations in microenvironment k for subject i, and t ij refers to the time in microenvironment k for subject i on sampling day j, T j 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 ($r$) 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.$^{36}$

### 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 ($E_{ip-PAHs}$) | $f_{ipex} * O$                                 |
| Prediction-based, non-ambient PAHs exposure ($E_{ia-PAHs}$) | $P * f_{ipex} * O$                             |
| Prediction-based, indoor PAHs exposure of ambient origin ($E_{i-PAHs}$) | $F_{inf} * O$                                  |
| Prediction-based, indoor-generated generated PAHs exposure ($E_{ig-PAHs}$) | $I * F_{inf} * O$                              |

For the calculation of modelled $BaPeq_{cPAHs}$ exposure concentration:

- Ambient $BaPeq_{cPAHs}$ concentration exposure ($O_{BaPeq}$)
- Residential indoor $BaPeq_{cPAHs}$ concentration ($I_{BaPeq}$)
- $BaPeq_{cPAHs}$ concentration in the office building
- $BaPeq_{cPAHs}$ concentration at school
- $BaPeq_{cPAHs}$ concentration in other indoors (eg, restaurant)
- $BaPeq_{cPAHs}$ concentration in commute

- $O_{BaPeq}$ *fraction of time outdoors$^{a}$
- $I_{BaPeq}$ *fraction of time in residential indoors
- $f_{ipex}(0.38)*O_{BaPeq}$ *fraction of time in the workplace
- $F_{inf}(0.66)*O_{BaPeq}$ *fraction of time at school
- Enrichment factor(1.15)$*O_{BaPeq}$ *fraction of time in other indoors
- Infiltration factor(0.92)$*O_{BaPeq}$ *fraction of commuting time

$^{a}$Results about the time in different microenvironments were derived from the time-activity diary for participants.

$^{1}$Data referenced from Gariazzo et al. (2015).
| Item                          | Summer                                                                 | Winter                                                                 | Totald | Current measuremente |
|-------------------------------|------------------------------------------------------------------------|------------------------------------------------------------------------|--------|----------------------|
| Sampling date                 | June – October 2014 and August– September 2015                        | November 2014–March 2015 and January– March 2016                      |        |                      |
| Study subjects (N°)           | 45 (32)                                                                | 43 (32)                                                                | 56     | 26                   |
| Never smokersc (Yes/No, %)    | /                                                                      | /                                                                      | /      | /                    |
| Female                        | 19                                                                     | 15                                                                     | 22     | 7                    |
| Male                          | 26                                                                     | 28                                                                     | 34     | 19                   |
| Age (years; median (range))   | /                                                                      | /                                                                      | 25 (18–42) | 24 (18–42) |
| Weight (kg) (mean, SDb)       | /                                                                      | /                                                                      | 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 activityf (%), 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) |
| 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) |
| Indoors, school               | 0 (2.4, 7.9)                                                          | 0 (3.8, 9.2)                                                          | 0 (3.2, 8.6) | 0 (3.1, 8.6) |
| 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) |
| 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) |

aNumber of participants.
bSD denotes standard deviation.
cAll participants were non-smokers and not exposed to environmental tobacco smoke (ETS).
dSubjects who participated in personal PM$_{2.5}$ monitoring.
eSubjects conducted simultaneous personal/indoor/ambient measurement.
fA total of 169 personal activity diaries were collected.
gWorkday 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\(_{15\text{PAHs}}\)) 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\(_{c\text{PAHs}}\)). 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\(_{15\text{PAHs}}\) and BaPeq\(_{c\text{PAHs}}\) 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 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., \(\Sigma 15\text{PAHs}\) 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 \(\Sigma 15\text{PAHs}\) concentrations (2.03 ± 0.79 ng/m\(^3\)) were two times higher than those measured in personal exposures (p < 0.001). Much higher ambient \(\Sigma 15\text{PAHs}\) 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 \(\Sigma 15\text{PAHs}\) 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 \(\Sigma 15\text{PAHs}\) 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 \(\Sigma 15\text{PAHs}\) concentrations at the HKPU was 3.64 ng/m\(^3\) during 2014–2016, a dramatic decline of 90% from 2000 (average \(\Sigma 15\text{PAHs}: 33.96\) 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.\(^{46}\) (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 \(\Sigma 15\text{PAHs}\) 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 PAH 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, \(\Sigma 15\text{PAHs}\)) 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 (\(\Sigma 15\text{PAHs}\), 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

| ng/m$^3$ | Ambient (n = 63) | Indoor (n = 63) | Personal (n = 63) | p-value$^b$ | I/O ratio$^c$ | P/O ratio$^d$ | P/I ratio$^e$ |
|----------|------------------|-----------------|------------------|------------|-------------|-------------|-------------|
|          | Mean ± SD$^a$    | 95th            | Mean ± SD$^a$    | 95th       |             |             |             |
| Acenaphthylene (Acy) | 0.05 ± 0.03      | 0.13            | 0.05 ± 0.03      | 0.11       | 0.03 ± 0.02 | 0.08        | <0.001      |
| Acenaphthene (Ace)    | 0.03 ± 0.03      | 0.09            | 0.03 ± 0.04      | 0.14       | 0.02 ± 0.02 | 0.06        | 0.03        |
| Fluorine (Flu)        | 0.07 ± 0.05      | 0.17            | 0.05 ± 0.04      | 0.12       | 0.03 ± 0.03 | 0.08        | <0.001      |
| Phenanthrene (Phe)    | 0.19 ± 0.11      | 0.42            | 0.13 ± 0.10      | 0.34       | 0.09 ± 0.08 | 0.24        | <0.001      |
| Anthracene (Ant)      | 0.30 ± 0.27      | 1.03            | 0.25 ± 0.17      | 0.60       | 0.14 ± 0.12 | 0.43        | <0.001      |
| Fluoronanthene (Flut) | 0.33 ± 0.15      | 0.69            | 0.27 ± 0.17      | 0.63       | 0.14 ± 0.08 | 0.27        | <0.001      |
| Pyrene (Pyr)          | 0.15 ± 0.06      | 0.28            | 0.11 ± 0.08      | 0.27       | 0.07 ± 0.05 | 0.14        | <0.001      |
| 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      |
| Chrysene$^f$ (Chr)    | 0.26 ± 0.14      | 0.58            | 0.21 ± 0.14      | 0.50       | 0.11 ± 0.06 | 0.21        | <0.001      |
| 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      |
| 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      |
| 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      |
| Dibenzo[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       |
| 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      |
| Benz[ghi]perylene (BghiP) | 0.15 ± 0.12 | 0.41            | 0.11 ± 0.09      | 0.34       | 0.06 ± 0.04 | 0.14        | <0.001      |
| Σ15PAHs             | 2.03 ± 0.79      | 3.64            | 1.65 ± 0.94      | 3.98       | 0.93 ± 0.43 | 1.69        | <0.001      |
| cPAHs               | 0.76 ± 0.39      | 1.67            | 0.65 ± 0.44      | 1.78       | 0.34 ± 0.17 | 0.64        | <0.001      |
| BaPeq-15PAHs         | 0.12 ± 0.07      | 0.25            | 0.10 ± 0.07      | 0.25       | 0.05 ± 0.03 | 0.11        | <0.001      |
| BaPeq-cPAHs          | 0.11 ± 0.07      | 0.25            | 0.09 ± 0.07      | 0.24       | 0.05 ± 0.03 | 0.10        | <0.001      |

$^a$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. Sources of residential indoor PAHs have been reported in another publication in Tong et al. (2019); the results indicated that vehicle emission, cooking activities, and indoor incense burning were the dominant sources. Other studies reported similar findings; for example, Zhu, Wang (2003) suggested that the abundance of 3-4 ring PAHs in residential indoors could be apportioned to cooking activities. Shi (2018) and Chen (China: 0.61). Higher I/O and P/O Chr ratios were observed in previous findings (Kraków, Poland: 0.54; Rome, Italy: 0.66; Guangzhou, China: 0.61).25, 26, 33 Filtration factors (I/O) were comparable with those reported in 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) demonstrated higher I/O cPAHs ratios in Hangzhou, China, suggesting substantial contributions of cooking practice and ambient infiltration. Minguillon et al. (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_i_PAHs) and residential indoor PAHs of ambient-origin (E_i_cPAHs). 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). 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_i_15PAHs; p < 0.001) and cPAHs of ambient-origin (E_i_cPAHs; p < 0.001) were shown for the study participants compared to residential indoor PAHs of ambient-origin (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. (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 PM2.5 exposure (33.2%) to total personal exposure in adult residents of Hong Kong. Moreover, E_i_cPAHs was relatively lower than E_i_cPAHs 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 Zhu, Zhao (2012) and Romagnoli et al. (2014). A previous study revealed that approximately 55% of total indoor PM2.5 was attributable to residential emissions in urban areas in China. Further, a study in Beijing demonstrated that 26.9%-32.6% of indoor BaPeq exposures resulted from indoor-origin pollution.

Limited studies have demonstrated variation of PAH concentrations in ambient-indoor-personal exposures. 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 = 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_15PAHs and BaPeq_cPAHs concentrations in ambient (0.11-0.12 ng/m³) and residential indoor (0.09-0.10 ng/m³) were about two times those measured in personal exposure assessments (0.05 ng/m³).

We performed a Monte Carlo simulation for adults’ exposure to BaPeq_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 modelled ∑15PAHs (r = 0.98; p < 0.001), cPAHs (r = 0.94; p < 0.001), and BaPeq_cPAHs concentration (0.09 ± 0.05 ng/m³) 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/m³), Houston, Texas (0.03 ng/m³); Grenoble, France (0.07 ng/m³), but significantly lower than those in Chinese cities (e.g., BaPeq_cPAHs: 1.47 ng/m³, BaPeq_15PAHs: 2.3 ng/m³ in Beijing). Carcinogenic PAHs and BaPeq_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. (2020) reported higher BaPeq concentrations indoors (e.g., dormitory, office, and laboratory) than the ambient air in Wuhan, China.
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 \( \Sigma 15 \text{PAHs} \) concentrations, while BaPeq\(_{cPAHs} \) accounted for 95.2%–95.6% of the total carcinogenic potential.
Strong correlations \( r = 0.99; \ p < 0.001 \) were shown between \( \text{BaPeq} \cdot \text{cPAHs} \) and \( \text{BaPeq} \cdot 15\text{PAHs} \) across different exposure metrics (Figure S7). These results were consistent with previous findings, providing evidence that \( \text{BaPeq} \cdot \text{cPAHs} \) is a suitable proxy for exposure to individual carcinogenic PAHs. Looking toward reducing exposures, Elzein et al. (2020) suggested focusing on mitigating carcinogenic PAHs emissions (e.g., \( \text{BaP}, \text{DBA}, \text{BbF}, \text{BkF}, \) and \( \text{IcdP} \)) 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 \( 1 \times 10^{-6} \) 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 \( (1.0 \times 10^{-5}) \), and residential indoor cPAHs \( (8.0 \times 10^{-6}) \) were higher than personal exposures \( (4.0 \times 10^{-6}) \). The high carcinogenic risk of residential indoor air could be...
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 $\Sigma_{15}$PAHs was 6.8 $\times$ 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 $\times$ 10$^{-6}$ and 2.45 $\times$ 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 $\times$ 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 $\times$ 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
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, 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 vaporize 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.
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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.

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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.

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REFERENCES

1. WHO Burden of disease from ambient and household air pollution. World Health Organization. 2016. http://www.who.int/phe/health_topics/outdoorair/databases/en/. Accessed May 2018.

2. Gerlofs-Nijland ME, Rummelhard M, Boere AJ, et al. Particle induced toxicity in relation to transition metal and polycyclic aromatic hydrocarbon contents. Environ Sci Technol. 2009;43(13): 4729-4736.

3. Kim KH, Jahan SA, Kabir E, Brown RJ. A review of airborne polycyclic aromatic hydrocarbons (PAHs) and their health effects. Environ Int. 2013;60:71-80.

4. Chau CK, Tu EY, Chan DW, Burnett J. Estimating the total exposure to air pollutants for different population age groups in Hong Kong. Environ Int. 2002;27(8):617-630.

5. Chi R, Chen C, Li H, et al. Different health effects of indoor- and outdoor-originated PM2.5 on cardiopulmonary function in COPD patients and healthy elderly adults. Indoor Air. 2019;29(2):192-201.

6. Fan ZL, Pun VC, Chen XC, et al. Personal exposure to fine particles (PM 2.5) and respiratory inflammation of common residents in Hong Kong. Environ Res. 2018;164:24-31.

7. Wu W, Jin Y, Carlsten C. Inflammatory health effects of indoor and outdoor particulate matter. J Allergy Clin Immunol. 2018;141(3):833-844.

8. Caplin A, Ghandehari M, Lim C, Glimcher P, Thurston G. Advancing environmental exposure assessment science to benefit society. Nat Commun. 2019;10(1):1236.

9. Morawska L, Afshari A, Bae GN, et al. Indoor aerosols: from personal exposure to risk assessment. Indoor Air. 2013;23(6):462-487.

10. Lei X, Chen R, Wang C, et al. Necessity of personal sampling for exposure assessment on specific constituents of PM2.5: Results of a panel study in Shanghai, China. Environ Int. 2020;141:105786.

11. Zhang Y, Tao S, Shen H, Ma J. Inhalation exposure to ambient polycyclic aromatic hydrocarbons and lung cancer risk of Chinese population. Proc Natl Acad Sci USA. 2009;106(50):21063-21067.

12. Ma Y, Harrad S. Spatiotemporal analysis and human exposure assessment on polycyclic aromatic hydrocarbons in indoor air, settled house dust, and diet: a review. Environ Res. 2015;84:7-16.

13. Lag M, Ovrevik J, Refsnes M, Holme JA. Potential role of polycyclic aromatic hydrocarbons in air pollution-induced non-malignant respiratory diseases. Respir. Res. 2020;21(1):299.

14. Dat ND, Chang MB. Review on characteristics of PAHs in atmosphere, anthropogenic sources and control technologies. Sci Total Environ. 2017;609:662-693.

15. Tarantini A, Maître A, Lefebvre E, Marques M, Rajahi A, Douki T. Polycyclic aromatic hydrocarbons in binary mixtures modulate the efficiency of benzo[a]pyrene to form DNA adducts in human cells. Toxicology. 2011;279(1-3):36-44.

16. Leung F, Wan H, Billah M, Cao J, Ho K, Wong CK. Chemical and biological characterization of air particulate matter 2.5, collected from five cities in China. Environ Pollut. 2014;194:188-195.

17. Guo H, Lee SC, Ho KF, Wang XM, Zou SC. Particle-associated polycyclic aromatic hydrocarbons in urban air of Hong Kong. Atmospheric Environ. 2003;37(38):5307-5317.

18. Boström CE, Gerde P, Hanberg A, et al. Cancer risk assessment, indicators, and guidelines for polycyclic aromatic hydrocarbons in the ambient air. Environ Health Perspect. 2002;110(suppl 3):451-488. 10.1289/ehp.110-1241197

19. Delgado-Saborit JM, Stark C, Harrison RM. Carcinogenic potential, levels and sources of polycyclic aromatic hydrocarbon mixtures in indoor and outdoor environments and their implications for air quality standards. Environ Int. 2011;37(2):383-392.

20. Shi SS. Contributions of indoor and outdoor sources to airborne polycyclic aromatic hydrocarbons indoors. Build Environ. 2018;131:154-162.

21. Naumova YY, Eisenreich SJ, Turpin BJ, et al. Polycyclic aromatic hydrocarbons in the indoor and outdoor air of three cities in the U.S. Environ Sci Technol. 2002;36(12):2552-2559.

22. Oliveira M, Slezkova K, Delerue-Matos C, Pereira MC, Morais S. Children environmental exposure to particulate matter and polycyclic aromatic hydrocarbons and biomonitoring in school environments: a review on indoor and outdoor exposure levels, major sources and health impacts. Environ Int. 2019;124:180-204.

23. Wang M, Jia S, Lee SH, Chow A, Fang M. Polycyclic aromatic hydrocarbons (PAHs) in indoor environments are still imposing carcinogenic risk. J Hazard Mater. 2021;409:124531.

24. Choi H, Harrison R, Komulainen H, Saborit JMD. Polycyclic aromatic hydrocarbons. In: WHO Guidelines for Indoor Air Quality: Selected Pollutants. Copenhagen, Denmark: World Health Organization; 2010:289-345.

25. Choi H, Spengler J. Source attribution of personal exposure to airborne polycyclic aromatic hydrocarbon mixture using concurrent personal, indoor, and outdoor measurements. Environ Int. 2014;63:173-181.

26. Hu YJ, Bao LJ, Huang CL, Li SM, Liu P, Zeng EY. Assessment of airborne polycyclic aromatic hydrocarbons in a megacity of South China: spatiotemporal variability, indoor-outdoor interplay and potential human health risk. Environ Pollut. 2018;238:431-439.

27. Madruga DG, Ubeda RM, Terroba JM, Dos Santos SG, Garcia-Cambero JP. Particle-associated polycyclic aromatic hydrocarbons in a representative urban location (indoor-outdoor) from South Europe: assessment of potential sources and cancer risk to humans. Indoor Air. 2019;29(5):817-827.
CHEN E t al.

32. OEHHA. 2020;188:109780.

33. Gariazzo C, Lamberti M, Hanninen O, et al. Assessment of population exposure to Polycyclic Aromatic Hydrocarbons (PAHs) using integrated models and evaluation of uncertainties. Atmospheric Environ. 2015;101:235-245.

34. Aquilina NJ, Delgado-Saborit JM, Gauci AP, Baker S, Meddings C, Harrison RM. Comparative modeling approaches for personal exposure to particle-associated PAH. Environ Sci Technol. 2010;44(24):9370-9376.

35. Chen XC, Ward TJ, Cao JJ, et al. Determinants of personal exposure to fine particulate matter (PM2.5) in adult subjects in Hong Kong. Sci Total Environ. 2018;628–629:1165-1177.

36. Transport and Housing Bureau Energy saving plan for Hong Kong’s Built Environment 2015. 2015. Accessed May 2015.

37. Choi H, Perera F, Pac A, et al. Estimating individual-level exposure to airborne polycyclic aromatic hydrocarbons throughout the gestational period based on personal, indoor, and outdoor monitoring. Environ Health Perspect. 2008;116(11):1509-1518.

38. Svecova V, Topinka J, Solansky I, Rossner P Jr, Sram RJ. Personal exposure to carcinogenic polycyclic aromatic hydrocarbons in the Czech Republic. J Expo Sci Environ Epidemiol. 2013;23(4):350-355.

39. European Commission. Air quality standards. 2001. https://ec.europa.eu/environment/air/quality/standards.htm. Accessed 25 January, 2010.

40. Liu Y, Tao S, Yang Y, Dou H, Yang Y, Coveney RM. Inhalation exposure of traffic police officers to polycyclic aromatic hydrocarbons (PAHs) during the winter in Beijing. China. Sci Total Environ. 2007;383(1–3):98-105.

41. Petit P, Maitre A, Persoons B, Picout DJ. Lung cancer risk assessment for workers exposed to polycyclic aromatic hydrocarbons in various industries. Environ Int. 2019;124:109-120.

42. Zhu L, Lu H, Chen S, Amagai T. Pollution level, phase distribution and source analysis of polycyclic aromatic hydrocarbons in residential air in Hangzhou, China. J Hazard Mater. 2009;162(2–3):1165-1170.

43. Lv J, Zhu L. Effect of central ventilation and air conditioner system on the concentration and health risk from airborne polycyclic aromatic hydrocarbons. J Environ Sci (China). 2013;25(3):531-536.

44. Ma Y, Cheng Y, Qiu X, Lin Y, Cao J, Hu D. A quantitative assessment of source contributions to fine particulate matter (PM2.5)-bound polycyclic aromatic hydrocarbons (PAHs) and their nitrated and hydroxylated derivatives in Hong Kong. Environ Pollut. 2016;219:742-749.

45. Jung KH, Bernabé K, Moors K, et al. Effects of floor level and building type on residential levels of outdoor and indoor polycyclic aromatic hydrocarbons, black carbon, and particulate matter in New York City. Atmosphere. 2011;2(2):96-109.

46. Barratt B, Lee M, Wong P, et al. A dynamic three-dimensional air pollution exposure model for Hong Kong. Res Rep Health Eff Inst. 2018;194:1-65.

47. Gonzalez A, Boies A, Swanson J, Kittelson D. Measuring the effect of ventilation on cooking in indoor air quality by low-cost air sensors. Int J Environ Eng. 2019;13(9):568-576.

48. Morawska L, Ayoko GA, Bae GN, et al. Airborne particles in indoor environment of homes, schools, offices and aged care facilities: the main routes of exposure. Environ Int. 2017;108:75-83.

49. Tong X, Chen X-C, Chuang H-C, et al. Characteristics and cytotoxicity of indoor fine particulate matter (PM 2.5) and PM 2.5-bound polycyclic aromatic hydrocarbons (PAHs) in Hong Kong. Air Qual Atmos Health. 2019;12(12):1459-1468.

50. Zhu L, Wang J. Sources and patterns of polycyclic aromatic hydrocarbons pollution in kitchen air, China. Chemosphere. 2003;50(5):611-618.

51. Chen Y, Li X, Zhu T, Han Y, Lv D. PM2.5-bound PAHs in three indoor and one outdoor air in Beijing: concentration, source and health risk assessment. Sci Total Environ. 2017;586:255-264.

52. Zhu X, Fan ZT, Wu X, et al. Ambient concentrations and personal exposure to polycyclic aromatic hydrocarbons (PAH) in an urban community with mixed sources of air pollution. J Expo Sci Environ Epidemiol. 2011;21(5):437-449.

53. Zhang J, Liu W, Xu Y, et al. Distribution characteristics of personal exposure with polycyclic aromatic hydrocarbons and particulate matter in indoor and outdoor air of rural households in Northern China. Environ Pollut. 2019;255(Pt 1):113176.

54. Minguillon MC, Schembari A, Triguero-Mas M, et al. Source apportionment of indoor, outdoor and personal PM2.5 exposure of pregnant women in Barcelona, Spain. Atmos Environ. 2012;59:426-436.

55. Barratt B, Lee M, Wong P, et al. A dynamic three-dimensional air pollution exposure model for Hong Kong. Res Rep Health Eff Inst. 2018;194:1-65.

56. Chen XC, Chow JC, Ward TJ, et al. Estimation of personal exposure to fine particles (PM2.5) of ambient origin for healthy adults in Hong Kong. Sci Total Environ. 2019;654:514-524.

57. Zhou B, Zhao B. Population inhalation exposure to polycyclic aromatic hydrocarbons and associated lung cancer risk in Beijing region: contributions of indoor and outdoor sources and exposures. Atmospheric Environ. 2012;62:472-480.

58. Romagnoli P, Balduzzi C, Perilli M, et al. Indoor PAHs at schools, homes and offices in Rome, Italy. Atmospheric Environ. 2014;92:51-59.

59. Yun X, Shen GF, Shen HZ, et al. Residential solid fuel emissions contribute significantly to air pollution and associated health impacts in China. Science Advances. 2020;6(44):eaba7621.

60. Xie Y, Zhao B, Zhao Y, et al. Reduction in population exposure to PM2.5 and cancer risk due to PM2.5-bound PAHs exposure in Beijing, China during the APEC meeting. Environ Pollut. 2017;225:338-345.

61. Elzein A, Stewart GJ, Swift SJ, et al. A comparison of PM 2.5-bound polycyclic aromatic hydrocarbons in summer Beijing (China) and Delhi (India). Atmospheric Chem and Phys. 2020;20(22):14303-14319.

62. Wang G, Wang Y, Yin W, et al. Seasonal exposure to PM2.5-bound polycyclic aromatic hydrocarbons and estimated lifetime risk of cancer: a pilot study. Sci Total Environ. 2020;702:135056.

63. Hong WJ, Jia H, Ma WL, et al. Distribution, fate, inhalation exposure and lung cancer risk of atmospheric polycyclic aromatic hydrocarbons in some Asian countries. Environ Sci Technol. 2016;50(13):7163-7174.

64. Regoecci WC. Crowding in context: an examination of the differential responses of men and women to high-density living environments. J Health Soc Behav. 2008;49(3):254-268.
65. Chan SM, Wong H, Chung RYN, Au-Yeung TC. Association of living density with anxiety and stress: a cross-sectional population study in Hong Kong. *Health Soc Care Community*. 2021;29(4):1019-1029.

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