

Article

Indoor Polycyclic Aromatic Hydrocarbons—Relationship to Ambient Air, Risk Estimation, and Source Apportionment Based on Household Measurements

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Abstract: Polycyclic aromatic hydrocarbons (PAH) are key components of particulate matter (PM) in terms of the toxicological risk of polluted air. Although commonly monitored in ambient air, PAHs are also present in indoor air, making the measurement of indoor PAH content essential for understanding the health risks associated with indoor environments. This study presents findings from measurements conducted across 37 households where children resided, using 7-day sampling campaigns to collect PM₁. The health risk assessment methods are detailed herein, along with a source apportionment analysis to explore the associations with potential sources and differences from ambient air concentrations. Additionally, the incremental lifetime cancer risk (ILCR) was calculated to assess long-term health risks associated with exposure to indoor PAHs. The results showed consistently higher PAH concentrations in outdoor environments (from 0.079 ng m^{−3} for dibenzo(a,h)anthracene to 1.638 ng m^{−3} for benzo(b)fluoranthene) compared to indoor environments (from 0.029 ng m^{−3} for dibenzo(a,h)anthracene to 0.772 ng m^{−3} for indeno(1,2,3-cd)pyrene), suggesting significant transfer of PAHs from outdoor to indoor air. The source apportionment analysis indicated that traffic emissions, fossil fuel combustion, and residential heating were the predominant sources of PAHs in both environments, with the concentration of indoor PAHs largely influenced by gasoline and liquid fossil fuel combustion. The diagnostic ratios supported these findings, with coal and biomass as additional sources impacting outdoor PAH levels. The ILCR analysis revealed that the exposure levels for both children (indoors at 1.78×10^{-5} , outdoors at 1.92×10^{-6}) and adults (indoors at 1.15×10^{-5} , outdoors at 1.24×10^{-6}) remained below the U.S. EPA's risk threshold, suggesting limited carcinogenic risk under typical household conditions in this study. These findings emphasize the complexity of PAH distribution between indoor and outdoor environments, illustrating how urban outdoor pollution sources contribute to indoor air quality and highlighting the relevance of effective air quality management strategies.

Keywords: indoor air quality; PM₁; particulate matter; outdoor air; ILCR; cancer risk



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1. Introduction

Humans spend most of their time indoors, as high as 90% of their time according to some studies [1], which implies a variety of microenvironments, with estimations of 70% for time spent at home. A 2017 systematic review revealed that household air pollution was associated with 1.8 million deaths and more than 60 million disability-adjusted life years globally [2]. Estimations show that in Europe, exposure to particulate matter (PM) reduces life expectancy by nearly one year, on average, for each person, primarily due to an increased risk of cardiovascular and respiratory diseases, neurological disorders, and lung

cancer [3]. Special care has to be directed toward vulnerable populations, such as children, older people, and individuals with respiratory conditions who appear to be more susceptible to these environmental risks than the general population [4]. Research shows that children living near highways with heavy-duty traffic have double the risk of developing respiratory issues compared to those living on less congested streets [5]. Indoor air quality (IAQ) is influenced by a range of factors and processes, including ambient air pollution levels, the transport of pollutants between indoor and outdoor environments, internal emissions, chemical reactions involving gases and particles, and dynamic processes such as resuspension, deposition, evaporation, growth, and coagulation, as well as ventilation and indoor air filtration [6–8]. Outdoor airborne pollutants can infiltrate indoor spaces through cracks, building shells, doors, and windows [7]. Indoor air chemistry also plays a significant role in determining IAQ [9]. Additionally, ventilation rates and deposition velocities can vary significantly—by as much as 30% on average—across different residential buildings and between seasons. Differences in ventilation habits, surface materials, and meteorological conditions contribute to the limitations of stationary and static IAQ investigations [10]. Despite more intensified research on IAQ in recent years, gaps remain regarding pollution sources and indoor–outdoor (I–O) interactions, and only a limited number of studies have focused on smaller particle fractions and chemical pollutants. One of the reasons is that small particle fractions (PM₁ particles—particles with an aerodynamic diameter less than 1 µm) can reach down to the alveoli in the lungs [11,12]. Once they have entered, their composition plays a role. The concentration of PM₁ can adsorb compounds that are suspected to be genotoxic, mutagenic, and carcinogenic, such as polycyclic aromatic hydrocarbons (PAHs) and their derivatives [13]. One of the methods to measure their health risk is using empirical models like the incremental lifetime cancer risk (ILCR). The ILCR is an empirical model based on exposure rates, toxicant concentrations and their carcinogenicity, and other estimations. The method relies on toxic equivalency factors (TEFs) [14] where PAHs are compared in terms of their toxicity to a reference compound. Within PAH studies, the reference is benzo(a)pyrene (BaP). The ILCR for PAHs provides a scale for assessing cancer risk: levels below ILCR = 10^{−6} are considered negligible to human health, levels between 10^{−6} and 10^{−4} indicate potential carcinogenic effect of PAHs to humans, and if levels exceed 10^{−4}, there may be indications of a high cancer risk due to human exposure to PAHs. A previous study conducted by this group [15] presented a case study on indoor dust in Croatia. Eleven PAHs were analyzed: fluoranthene (Flu), pyrene (Pyr), chrysene (Chry), benzo(a)anthracene (BaA), benzo(j)fluoranthene (BjF), benzo(b)fluoranthene (BbF), benzo(a)pyrene (BaP), benzo(k)fluoranthene (BkF), benzo(ghi)perylene (BgHiP), dibenzo(a,h)anthracene (DahA), and indeno(1,2,3-c,d)pyrene (IP). The results showed that all were present in each sample and that Flu and Pyr were the most abundant. A study from Serbia showed higher concentrations in dust compared to Croatia [16]. Both studies calculated health risks by means of the ILCR. The ILCR indicated that 85% of the studied locations were under high risk in Serbia, while Croatian households were found to be at a lower risk. Other studies from Greece [17,18] showed a comparable risk in Greek households to that in Croatia. These regional differences appear to be attributable to differences in pyrogenic sources and heating. Higher ILCR values, however, were calculated in a setting of kitchen workers [19], who showed deteriorated lung functions, presumably due to high levels of VOC and PAH and, consequently, higher concentrations of PAH-related metabolites in urine. The study reported alarmingly high ILCR values. Based on the listed works, one can observe that there is a certain sensitivity of the ILCR method to geographical distributions and pollutant sources, further presenting the validity of this methodology over a wide spectrum of use cases.

This study was conducted as part of the project “Evidence driven indoor air quality improvement” (EDIAQI) [20], which aims to improve indoor air quality (IAQ) guidelines and raise awareness across Europe and beyond by providing accessible information on exposures, pollution sources, and associated risk factors. Within the framework of EDIAQI, the Zagreb pilot focused on assessing the impact of indoor and outdoor air pollutants on

children's health, employing advanced monitoring technologies and biological sampling to explore the relationship between IAQ, health outcomes, and environmental influences in residential settings. Humans spend most of their time indoors, which can lead to health issues caused by indoor air pollution. To avoid or minimize these problems, efforts must be made to mitigate them. However, effective mitigation requires accurate indoor air quality data. This study focused on (i) expanding knowledge on PAH concentrations in households based on a measurement campaign conducted in 37 households located in Zagreb, Croatia; (ii) identifying the sources and driving factors by simultaneously measuring ambient and indoor air PAH concentrations; (iii) estimating the incremental lifetime cancer risks (ILCRs); and referring to a) our previous studies and b) studies across Europe.

2. Materials and Methods

2.1. Sampling and Chemical Analysis

This study was part of the Zagreb pilot within the EDIAQI project [20]. The thirty-seven (37) households spread over the city are shown on the map in Figure 1. Zagreb, the capital of Croatia, has an approximate population of 800,000 residents. With around 440,000 registered vehicles, the city faces ongoing challenges in managing air quality and urban traffic pollution (available online: https://www.zagreb.hr/UserDocsImages/001/SLJZG23_web.pdf (accessed on 19 December 2024)).

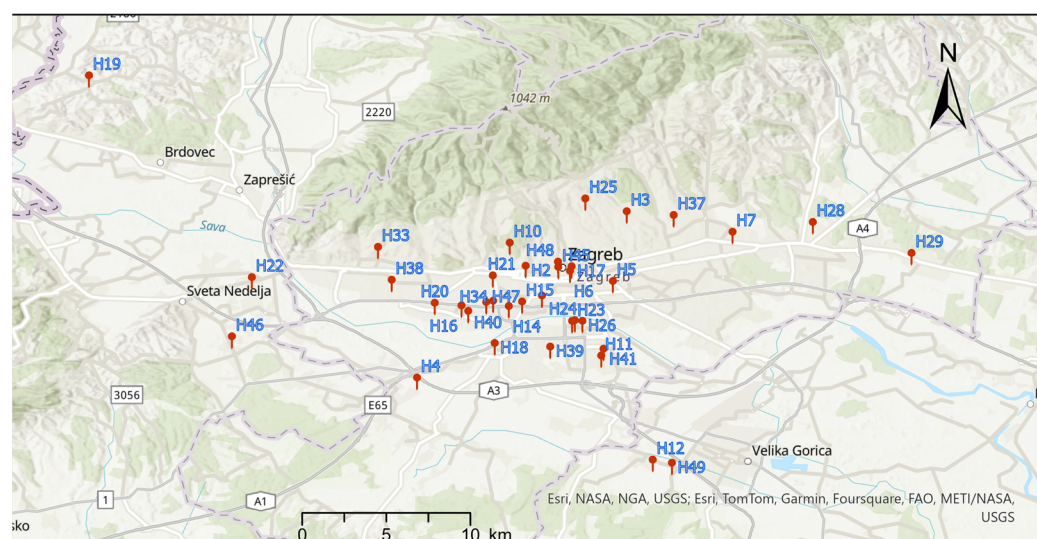


Figure 1. Sampling locations of the households in this study.

The primary contributors to pollution in Zagreb are traffic emissions and residential heating [20–22]. Within the EDIAQI project, PAHs in PM_{10} in the households (particulate matter with an aerodynamic diameter below $1 \mu m$) were collected on filters by active sampling using pumps over a 7-day period, both indoors and outdoors. Traditional reference samplers commonly used for outdoor/ambient air collection (with an airflow of $55 m^3 day^{-1}$) are unsuitable due to their size and noise level in indoor environments. Instead, smaller, quieter samplers, namely Sven-Leckel MiniVS-C (Berlin, Germany) and Mini Vol Portable Air Sampler, AirMetrics (Eugene, OR, USA), with a flow rate of $5 L min^{-1}$, are preferred. These samplers were equipped with a size-selective impactor inlet for the PM_{10} fraction of particulate matter, and a filter holder loaded with quartz filters (Whatman, Tisch Scientific, Cleves, OH, USA). The samplers were positioned in household bedrooms or living rooms. The preparation methods and analysis of PAHs were described in a study by Jakovljević et al. [23]. Briefly, the filters were extracted using a solvent mixture of toluene and cyclohexane (7:3) in an ultrasonic bath, centrifugated, and evaporated in a stream of nitrogen to dryness. Then, acetonitrile was used to re-dissolve the samples [23]. An Agilent Infinity 1260 coupled with a fluorescence detector and programmed changes in excitation and

emission wavelengths (Agilent Technologies, Santa Clara, CA, USA) was used to determine the following PAHs: benzo(a)pyrene (BaP), benzo(a)anthracene (BaA), benzo(ghi)perylene (BghiP), dibenzo(ah)anthracene (DahA), chrysene (Chry), benzo(k)fluoranthene (BkF), benzo(b)fluoranthene (BbF), benzo(j)fluoranthene (BjF), fluoranthene (Flu), pyrene (Pyr), and indeno(1,2,3-cd)pyrene (IP).

2.2. Statistical Analysis

All data processing and statistical analyses were conducted in the programming language Python 3.10 (www.python.org (accessed on 19 December 2024)) using the libraries pandas [24], scikit-learn [25], statsmodels [26], and pingouin [27]. The visualizations were created using seaborn [28] and matplotlib [29]. Households that did not have corresponding outdoor PAH measurements were removed from the dataset, which explains the non-sequential numbering of the households. This step was necessary to focus the analysis on households where both indoor and outdoor measurements were available, as these paired observations were crucial for comparing I-O PAH concentrations and evaluating transfer dynamics. To assess differences in I-O PAH concentrations, a non-parametric Wilcoxon signed-rank test was applied due to the left-skewed distribution of the data [30]. The Benjamini–Hochberg method was used to adjust for multiple comparisons, ensuring control of the false discovery rate at a significance level of 0.05 [31]. To complement these analyses, a probabilistic Monte Carlo (MC) simulation was conducted to model the variability in ILCR values for different age groups [32]. This approach enabled the random and independent sampling of variables to estimate cancer risk distributions more robustly, incorporating seasonal and population-specific factors. Principal component analysis (PCA) [33] was used to assess relationships in the high-dimensional data and interpret the sources. Canonical Correlation Analysis (CCA) [34] was used to understand differences in I-O PAH concentrations. CCA enabled the identification of the linear combinations of PAHs that were most strongly correlated between the indoor and outdoor environments, helping to understand PAH transfer dynamics. To assess and visualize the Euclidean distances between indoor and outdoor PAH concentrations, distances between households were calculated in the PCA space to represent the transfer or difference between environments. The relationships were further explored using diagnostic ratios (e.g., Flu/(Flu + Pyr), BaA/(BaA + Chry)) to assess emission sources and compare observed ratios with known values from the literature.

2.3. Carcinogenic Activity and Population Exposure

To assess the toxicological risk of the compounds in the I-O air, the incremental lifetime cancer risk (ILCR) assessment presented in the Introduction was used. BaP was used as an indicator of the health impact of the PAH mixture on human health. The carcinogenic activity of individual PAHs was estimated based on toxic equivalency factors (TEFs) from the literature [14,35–37]. In this study, the TEFs reported by Nisbet and LaGoy [14] were used. To calculate the risk of the PAH mixture in ambient and indoor air, the carcinogenic potencies of individual PAHs were expressed relative to the potency of BaP. BaP equivalents (BaP_{eq}) were calculated by multiplying the mass concentration of each individual PAH with its respective toxic equivalency factor. Total carcinogenic potency (TCP) was calculated by summing the BaP_{eq} of each measured PAH. The equation used to calculate the TCP is presented below (1):

$$TCP = \sum_{PAH}^N BaP_{eq(PAH)} = \sum_{PAH}^N \gamma_{PAH} \times TEF_i \quad (1)$$

where TCP is the total carcinogenic potency [ng m^{-3}], BaP_{eq} is the BaP equivalent concentration, TEF_i the toxic equivalency factor of individual PAH, and γ_i the mass concentration of individual PAH [ng m^{-3}]. To determine the daily population exposures, it was assumed that the daily dose of inhaled PAHs was equal to their airborne concentrations. In this

study, we tried to estimate the most probable scenario. Based on the literature, we assumed that people spend 60% of their time indoors at home and 10% outdoors. The *ILCR* posed by exposure to PM-bounded *PAHs* was computed following Equation (2) [38].

$$ILCR_{inh} = \frac{SF_{inh} \times IEL \times EF \times ED \times y}{BW \times AT \times cf} \quad (2)$$

where SF_{inh} is the inhalation cancer slope factor of *BaP* [kg day mg^{-1}], *IEL* is the BaP_{eq} daily dose [ng day^{-1}], *EF* is the exposure frequency [day year^{-1}], *ED* the daily exposure level [$\mu\text{g g}^{-1}$], *BW* is the body weight [kg], *AT* is the average time [day], and *cf* is the conversion factor [10^{-6}]. In this study, SF_{inh} , *EF*, and *ED* were used as derived by Chen and Liao [39]. All of the parameters used in the calculation for adults (20–70 age) and children (5–19 age) are shown in Table A1 of the Appendix A. In this study, SF_{inh} of $3.14 \text{ kg day mg}^{-1}$ was used, and the proper description of how this value was derived had been explained by Chen and Liao [39]. The BaP_{eq} daily dose was calculated by multiplying the *TCP* concentrations [ng m^{-3}], inhalation rate, *IR*, [$\text{m}^3 \text{ day}^{-1}$], and daily exposure time span (% of the day). The equation used to calculate the *IEL* is presented below (3):

$$IEL = TCP \times t\%_{(indoor,outdoor)} \times IR \quad (3)$$

As the variables in the *ILCR* calculation display certain distributions, taking the average value into account to obtain the risk estimation can be misleading. Therefore, a probabilistic Monte Carlo (MC) simulation was employed to feed the distributions to the *ILCR* model for children and adults. All of the tested variables were sampled randomly and independently. The information on variable distributions was taken from Chen and Liao [39] and Liu et al. [40]. MC simulations for the *ILCR* models were generated using previously generated scripts [41,42]. The MC simulations were conducted to address variability and uncertainty in the *ILCR* estimates for children (5–19 years) and adults (20–70 years) under different exposure scenarios. By incorporating the distributions of the input variables, the simulations modeled real-world variability and avoided reliance on average values. Through 10,000 iterations of random and independent sampling, the approach accounted for differences in age-specific behaviors, time-activity patterns (e.g., 90% indoors and 10% outdoors), and seasonal factors, providing probabilistic risk estimates. This method effectively captured exposure variations, ensuring robust and realistic *ILCR* evaluations for the targeted populations.

3. Results

3.1. Chemical Analysis Results

The descriptive statistics of *PAH* concentrations, including the minimum, the maximum, and the mean and standard deviation, are presented in Table 1 for both environments.

Table 1. Descriptive statistics for indoor (Id) and outdoor (Od) *PAH* concentrations for all households (ng m^{-3}).

<i>PAHs</i>	Min _{Id}	Min _{Od}	Max _{Id}	Max _{Od}	Mean _{Id}	Mean _{Od}	Std _{Id}	Std _{Od}
Flu	0.007	0.012	1.117	2.188	0.261	0.512	0.284	0.566
Pyr	0.010	0.012	0.611	1.384	0.181	0.318	0.168	0.331
BaA	0.000	0.000	0.740	2.520	0.120	0.453	0.163	0.622
Chry	0.002	0.002	1.445	4.249	0.246	0.852	0.322	1.144
BjF	0.002	0.009	1.564	5.867	0.315	1.222	0.384	1.586
BbF	0.007	0.009	3.218	6.504	0.666	1.638	0.824	1.816
BkF	0.001	0.001	1.131	2.426	0.227	0.587	0.290	0.668
BaP	0.002	0.003	3.063	4.210	0.497	0.916	0.681	1.099
DahA	0.000	0.000	0.096	0.325	0.029	0.079	0.029	0.086
BghiP	0.002	0.003	3.308	4.348	0.637	1.099	0.792	1.189
IP	0.004	0.007	3.758	5.656	0.772	1.414	0.931	1.512

The results show considerable variability in PAH concentrations across households, with some PAHs, such as BbF and BghiP, showing higher average indoor and outdoor concentrations. Conversely, DahA and BaP were detected at lower concentrations across most households. Indoor (Id) concentrations were generally lower than outdoor (Od) concentrations, suggesting the potential influence of outdoor sources on indoor PAH levels.

Differences in indoor and outdoor PAH concentrations may be influenced by sampling season and geographic location. Also, some indoor PAH sources such as smoking, cooking, cleaning, human activity, and use of appliances (e.g., gas or wood stoves) can affect the PAH levels in indoor air.

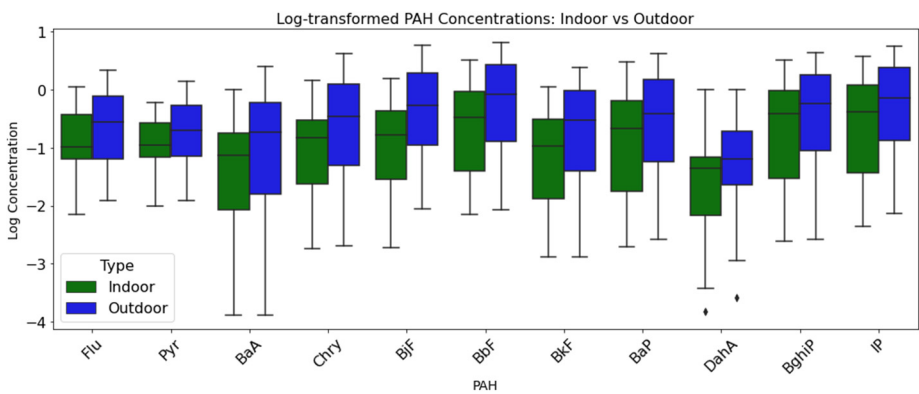
One of the research questions in this study was how indoor and outdoor PAH concentrations relate to one another. For this purpose, various statistical analyses were utilized. When comparing indoor and outdoor concentrations (Table 1, Figure 2), it could be observed that outdoor PAH concentrations were consistently higher than indoor concentrations across all of the measured compounds. The trend of higher outdoor levels was further supported by the standard deviation (Std) values, where outdoor measurements consistently showed greater variability than indoor values, particularly for compounds such as BbF and BaP.

3.2. Indoor and Outdoor PAH Relationships

To investigate whether there were significant differences in the concentrations of I-O PAHs, a non-parametric Wilcoxon signed-rank test was conducted for each PAH. Given that the data were not normally distributed and left-skewed, the Wilcoxon test was chosen to compare paired I-O measurements.

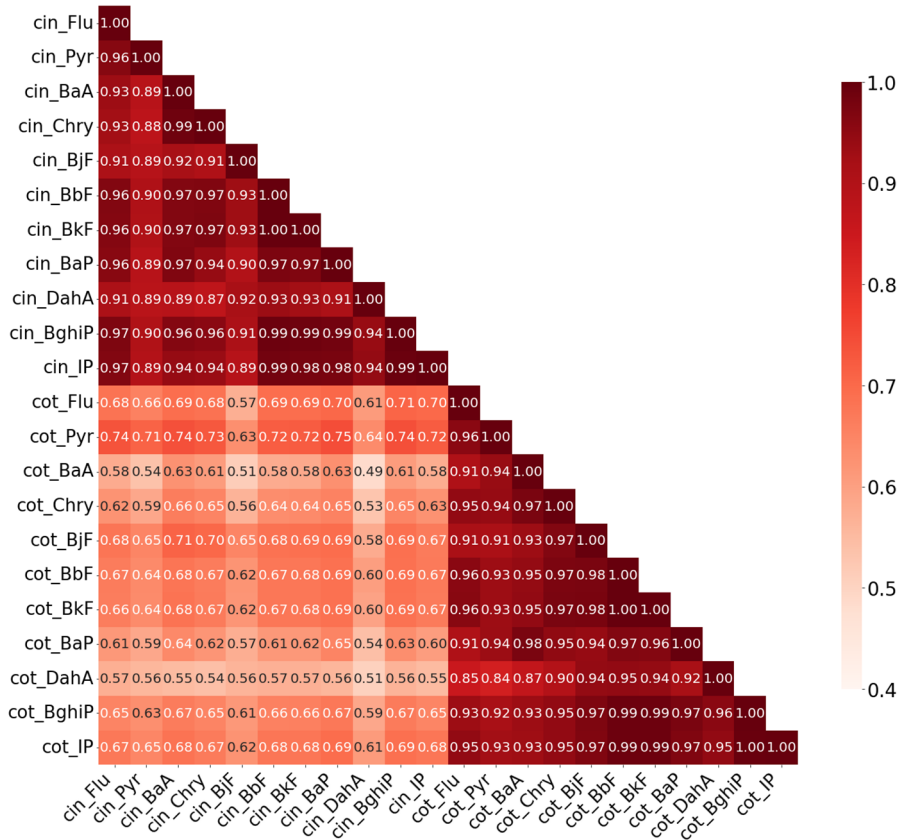
A correction for multiple comparisons was applied using the Benjamini–Hochberg method to control the false discovery rate at a significance level of 0.05. The results of the analysis, including the adjusted *p*-values, showed statistically significant differences between indoor and outdoor concentrations after correction, which included BaA, Chry, BbF, BkF, and DahA, with the adjusted *p*-values all below 0.05. For Flu, Pyr, BaP, BghiP, and IP, no statistically significant differences were observed for I-O concentrations. Figure 2a shows the log-transformed PAH concentrations, comparing I-O environments across households. Overall, outdoor concentrations tended to be higher than indoor concentrations for all PAHs, suggesting that outdoor sources might significantly contribute to indoor levels. For compounds like BaA, BbF, and BkF, indoor concentrations were notably lower than outdoor concentrations, indicating limited indoor sources or reduced transfer from outdoor environments. These results are in accordance with the studies by Delgado-Saborit et al. [43] and Naumova et al. [44], where the authors found that pooled PAH levels measured outdoors were generally higher than the levels indoors. The correlation heatmap (Pearson correlation) which illustrates the relationships between indoor (denoted as cin_) and outdoor (denoted as cot_) concentrations of PAHs is presented in Figure 2b. The correlations between different indoor PAH concentrations were consistently high, with most coefficients close to 1 (ranging between 0.88 to 0.99). This suggests that the indoor concentrations of PAHs were inclined to vary in a similar manner across households, potentially indicating common indoor sources or uniform behavior of PAHs in indoor environments. Similarly, strong correlations existed among the outdoor PAH concentrations, with values ranging from 0.85 to 1.00. This indicates that the outdoor PAH levels were also closely related to each other, potentially reflecting shared outdoor sources such as traffic emissions, industrial activities, or other combustion processes. The intercorrelations for I-O PAH concentrations were moderate to strong for certain PAHs, though generally lower compared to the intra-group (indoor–indoor and outdoor–outdoor) correlations. Notable I-O pairs included cin_Pyr and cot_Pyr (correlation coefficient of 0.71), followed by the pairs cin_Flu-cot_Flu, cin_BkF-cot_BkF, and cin_IP-cot_IP (correlation coefficient of 0.68), then the pairs cin_BbF-cot_BbF and cin_BghiP-cot_BghiP (correlation coefficient of 0.67), and the pairs cin_BaP-cot, cin_Chry-cot_Chry, and cin_BaA-cotBaA (correlation coefficient of 0.65). A moderate relationship between indoor and outdoor levels for these PAHs suggested their

possible common origin, especially considering that for most of them (Flu, Pyr, BaP, BghiP, and IP), no significant difference was found between indoor and outdoor concentrations. However, some PAHs, such as DahA, exhibited weaker correlations between outdoor and indoor concentrations, with a correlation coefficient of 0.51 for DahA, implying that, for this compound, the outdoor concentrations may not have strongly influenced the indoor levels.



(a)

Indoor vs Outdoor PAHs



(b)

Figure 2. (a) Boxplots of indoor and outdoor log-transformed PAH concentrations (the box represents the interquartile range (IQR) where 50% of the data points are concentrated, with the line inside the box indicating the median value, and the whiskers extend to 1.5 times the IQR, with outliers shown as individual points); (b) Pearson correlation heatmap for indoor (_cin) and outdoor (_cot) PAH concentrations.

3.3. Analysis of PAH Transfer

A PCA of indoor and outdoor PAH concentrations across different households was conducted to visualize and assess the degree of I-O variation. The concentrations of I-O PAHs were aligned vertically in a flat table. Hence, each household had two values in the “same feature space”: one for indoors and one for outdoors. For a strong I-O correlation to be justified, the households would need to have short Euclidean distances in the PCA 2-dimensional plots (Figure 3a). Hence, shorter distances imply a stronger association for I-O concentrations, which may reflect higher I-O transfer or similar pollution sources for both environments. Larger distances indicate more substantial differences for I-O PAH levels, suggesting limited I-O transfer or significant indoor sources. PCA was used to reduce the dimensionality of the data, with PC1 (87.17% of variance explained) and PC2 (7.97% of variance explained) capturing most of the variability in the dataset. The boxplot of the Euclidean distances (Figure 3a) between indoor and outdoor PAH concentrations revealed that most households had moderate differences. The variability observed in households with higher Euclidean distances (H5, H7, H14, H15, H16, and H18) may have been influenced by their specific spatial locations and environmental conditions. These households, sampled during the summer, primarily used gas or electric stoves. Their proximity to urban areas with heavy traffic or industrial zones, as indicated in Figure 3, could have contributed to the elevated outdoor PAH levels. Additionally, summer sampling might have recorded increased PAH volatilization and outdoor concentrations, while higher ventilation rates, such as frequent window openings, could have influenced the indoor–outdoor transfer. These factors highlight the importance of localized urban influences and ventilation behaviors in explaining the differences in I-O PAH relationships. Canonical Correlation Analysis (CCA) was used to explore the I-O relationships further. CCA allows for the simultaneous analysis of two sets of variable spaces—in this case, again, outdoor PAH concentrations (X_1) and indoor PAH concentrations (X_2) were aligned in a flat table. The goal here is to determine the linear combinations that capture the maximum shared variance between these sets (X_1 and X_2). This method was chosen to see which PAHs are most influential in explaining the I-O relationships. Figure 3c shows the canonical loadings for outdoor PAHs and indoor PAHs. The loadings represent the contribution of each PAH to the first canonical variate, which captures the strongest shared variance between the two environments. One of the key findings is that BbF had a strong positive loading (0.72) on the outdoor canonical variate, indicating its significant contribution to outdoor PAH variability.

Meanwhile, BkF behaved differently, showing a strong negative loading (−0.61) outdoors and an even stronger negative loading indoors (−0.83). This pointed to BbF and BkF being key variables in differing between these two environments. BbF’s strong positive loading outdoors indicates that it is a significant contributor to outdoor PAH variability, which may reflect sources like traffic emissions and industrial activities that are more prevalent or impactful outdoors. BbF is commonly associated with combustion sources, particularly vehicle exhaust, which could dominate the outdoor environment, making it a primary outdoor PAH marker. BkF’s strong negative loading in both environments, especially indoors, suggests that it might be less influenced by typical outdoor sources and could instead reflect indoor sources or reduced penetration indoors.

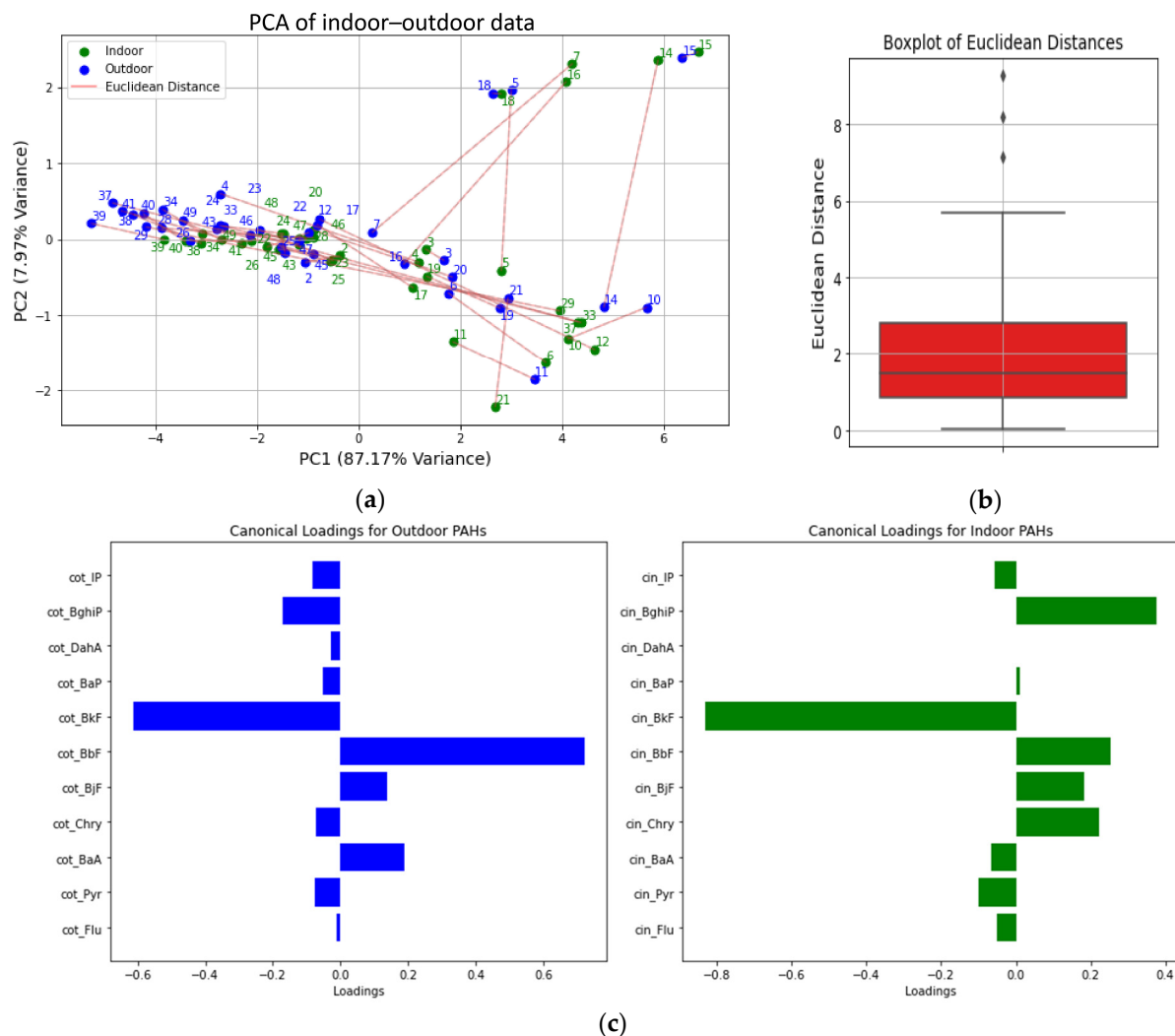


Figure 3. (a) Distance plot of the indoor and outdoor settings using PCA; (b) boxplot of the Euclidean distances; and (c) CCA loadings for indoor and outdoor PAHs.

3.4. Indoor and Outdoor PAH Diagnostic Ratios

Source apportionment of I-O PAHs was conducted using a set of diagnostic ratios widely applied to differentiate between petrogenic, pyrogenic, and traffic-related sources of PAHs. The diagnostic ratios based on the mass fraction ratios of specific PAHs were used to further evaluate the potential sources of PAHs. Flu and Pyr were used to classify petroleum and combustion sources. According to Agudelo-Castañeda and Teixeira [45] and Hanedar et al. [46], exhaust gases have a Flu/(Flu + Pyr) ratio of 0.2 to 0.5, liquid fossil fuel combustion has a ratio of 0.4 to 0.5, while a ratio higher than 0.5 is characteristic for coal, grass, or wood combustion. Petroleum combustion is characterized by an IP/(IP + BghiP) ratio less than 0.5, whereas wood combustion is indicated by a ratio greater than 0.7. Car exhausts have BaP/BghiP values between 0.3 and 0.4, diesel combustion has values from 0.46 to 0.81, and coal combustion has values from 0.9 to 0.6 [45–47]. Vehicles with catalysts have a value of BaP/(BaP + Chry) ratio less than 0.33, while the values are 0.49 for gasoline and 0.73 for diesel [45,48]. Finally, a diagnostic ratio BaA/(BaA + Chry) below 0.38 is characteristic for gasoline, while for diesel, the values should be above 0.38 [49–51]. The ratios used in this study included Flu/(Flu + Pyr), IP/(IP + BghiP), BaA/(BaA + Chry), BaP/BghiP, and BaP/(BaP + Chry). These ratios were calculated separately for both indoor and outdoor PAH concentrations across households, allowing for the identification of potential sources and assessment of the transfer of PAHs from outdoor to indoor environments (see Table 2). The Flu/(Flu + Pyr) ratio suggested a dominance of combustion-related sources, with most

values falling within the range typically associated with fossil fuel combustion. This was consistent across I-O environments, indicating that combustion processes, potentially from traffic or residential heating, are key contributors to the PAH concentrations found in these households. The IP/(IPnd + BghiP) ratio pointed toward diesel engine emissions as a likely source, particularly in outdoor environments, where higher values of this ratio were observed. The BaA/(BaA + Chry) ratio provided further evidence of mixed combustion sources. Ratios above 0.35 are typically indicative of wood or biomass combustion, while values below this threshold suggest coal or oil combustion. Pan-frying and steaming, as two different types of gas cooking methods, had BaA/(BaA + Chry) ratios of 0.29 and 0.31, respectively [52]. Some households exhibited values for specific diagnostic ratios, such as Flu/(Flu + Pyr), that aligned with coal/biomass combustion, especially in indoor environments. This may suggest contributions from indoor sources like heating systems or cooking to the overall indoor PAH levels. According to See and Balasubramanian [52], the Flu/(Flu + Pyr) ratio for different gas cooking methods ranged from 0.51 to 0.56 for steaming (to cook over boiling water) and stir-frying (to fry quickly in oil). In addition, the BaP/(BaP + Chry) and BaP/BghiP ratios showed that both gasoline and diesel emissions were significant contributors to outdoor PAHs.

Table 2. Median values for indoor and outdoor diagnostic ratios and potential sources based on the literature.

Diagnostic Ratio	Indoor (Median Value)	Conclusion	Outdoor (Median Value)	Possible Source
Flu/(Flu + Pyr)	0.52	Liquid Fossil Fuel Combustion	0.55	Coal/Biomass Combustion
IP/(IP + BghiP)	0.53	Diesel Combustion	0.57	Diesel Combustion
BaA/(BaA + Chry)	0.28	Gasoline Combustion	0.31	Gasoline Combustion
BaP/BghiP	0.65	Diesel Combustion	0.71	Diesel Combustion
BaP/(BaP + Chry)	0.53	Gasoline and Diesel Mix	0.51	Gasoline Combustion

The relatively higher outdoor ratios of BaP/BghiP suggest traffic-related emissions are a major source, while the lower indoor ratios may indicate the influence of indoor activities or the transformation of PAHs as they migrate indoors. By comparing these diagnostic ratios between indoor and outdoor environments, it was possible to identify PAHs that may be transferred from outdoor sources, such as traffic emissions and residential heating, into indoor spaces. The differences in ratios between these environments suggest that, while outdoor sources have a dominant role, indoor sources such as cooking, smoking, or the infiltration of outdoor air also contribute to the overall PAH burden in households. Overall, our diagnostic ratios confirmed that traffic emissions, fossil fuel combustion, and residential heating were the primary sources of PAHs in both the indoor and outdoor environments. Indoor PAHs were largely influenced by liquid fossil fuel combustion and gasoline sources, as reflected in the Flu/(Flu + Pyr) and BaA/(BaA + Chry) ratios, which had median values of 0.52 and 0.28, respectively. These values suggested a significant contribution from traffic-related emissions indoors. On the other hand, outdoor PAHs appeared to be more strongly impacted by coal/biomass combustion (median Flu/(Flu + Pyr) ratio of 0.55), a common source in areas where residential heating or industrial activities rely on solid fuels. The IP/(IP + BghiP) ratio consistently pointed to diesel as a key contributor in both environments, with median values of 0.53 for indoors and 0.57 for outdoors, indicating limited differentiation between indoor and outdoor PAH sources for this specific ratio. However, the BaP/BghiP ratio suggested gasoline combustion as a significant contributor in both environments, with a slightly higher mean value for outdoors (0.722) compared to indoors (0.661). The BaP/(BaP + Chry) ratio revealed a more complex behavior: for indoors,

a mix of gasoline and diesel sources was suggested (mean ratio of 0.537), while for outdoors, diesel combustion was more dominant (mean ratio of 0.512). This difference may reflect the stronger influence of traffic emissions in outdoor environments, with diesel engines playing a more significant role. The diagnostic ratios obtained in this study were lower than the ratios in the study by Rogula-Kozłowska et al. [53] in Warszawa and Gliwice, and similar to the results found in a university building in an urban area of Belgrade, Serbia, for PAHs in the PM_{2.5} particle fraction [54].

The scatter plot of the diagnostic ratios (Figure 4) provides additional insights into the relationship between indoor and outdoor PAH concentrations, highlighting varying contributions from outdoor and indoor sources.

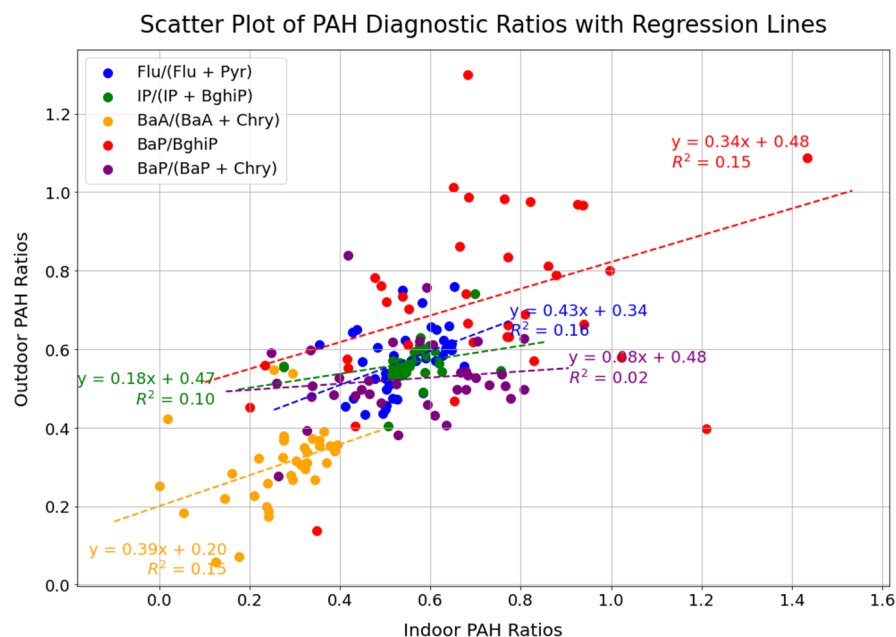


Figure 4. Scatter plot of PAH diagnostic ratios with regression lines. Each regression line represents a linear relationship between indoor and outdoor values for a specific diagnostic ratio, with the corresponding equation and R^2 value. The equation describes the slope, while R^2 indicates the strength of the relationship, with values closer to 1 reflecting a stronger correlation between the two environments.

The BaA/(BaA + Chry) ratio, with an increment of 0.20 and a slope of 0.39, suggests that outdoor values are generally higher, pointing to traffic-related emissions as a dominant outdoor source, while the lower indoor ratios could reflect cooking activities or limited infiltration. The Flu/(Flu + Pyr) ratio, with a slope of 0.43, indicates a relatively balanced but outdoor-dominated pattern, consistent with emissions from biomass in less-gasified areas, especially during colder months when windows remain closed. The scattered BaP/BghiP ratio exhibits a correlation slope of 0.34, reflecting diverse sources, including diesel and solid fuel combustion outdoors, but also suggesting varying household-specific indoor influences. The BaP/(BaP + Chry) ratio has the lowest slope (0.08) and the lowest R^2 , showing stable outdoor values linked to a gasoline–diesel mix and greater variation indoors, which is potentially driven by localized sources such as gas stoves or air conditioning systems. Lastly, the IP/(IP + BghiP) ratio demonstrates strong correlation and low variability, with a slope of 0.18, emphasizing diesel combustion as a consistent source for both environments. Seasonal factors, such as heating and ventilation practices, may also influence these patterns. Further investigations extending from this study will focus on analyzing a larger sample of households and detailed questionnaires to better understand the impact of household types, seasonal variations, and other parameters on the indoor–outdoor PAH dynamics.

3.5. Toxicological Risk Assessment

To determine the daily population exposures, total carcinogenic potency (TCP) was used to calculate the daily dose according to Equation (3). This study estimated the most probable exposure scenario for two age groups: children (5–19 years) and adults (20–70 years). It was inferred from the literature that people spend 90% of their time indoors and 10% outdoors. The ILCR from exposure to PM-bound PAHs was calculated using Equation (2). Equation (3) was applied to calculate the IEL for indoors and outdoors, and the results are shown in Figure 5. The variables in the ILCR calculation followed specific distributions, so relying solely on average values could lead to inaccurate risk estimations. To address this, a probabilistic Monte Carlo (MC) simulation was used, which allowed the input of these distributions into the ILCR model for different age groups—children and adults—for indoors and outdoors. Each variable was sampled randomly and independently. The MC simulation was run for 10,000 iterations per sampled variable, with the same number of iterations run for the IEL and ILCR models. The parameter values used for the MC simulation were detailed in our previous work [41].

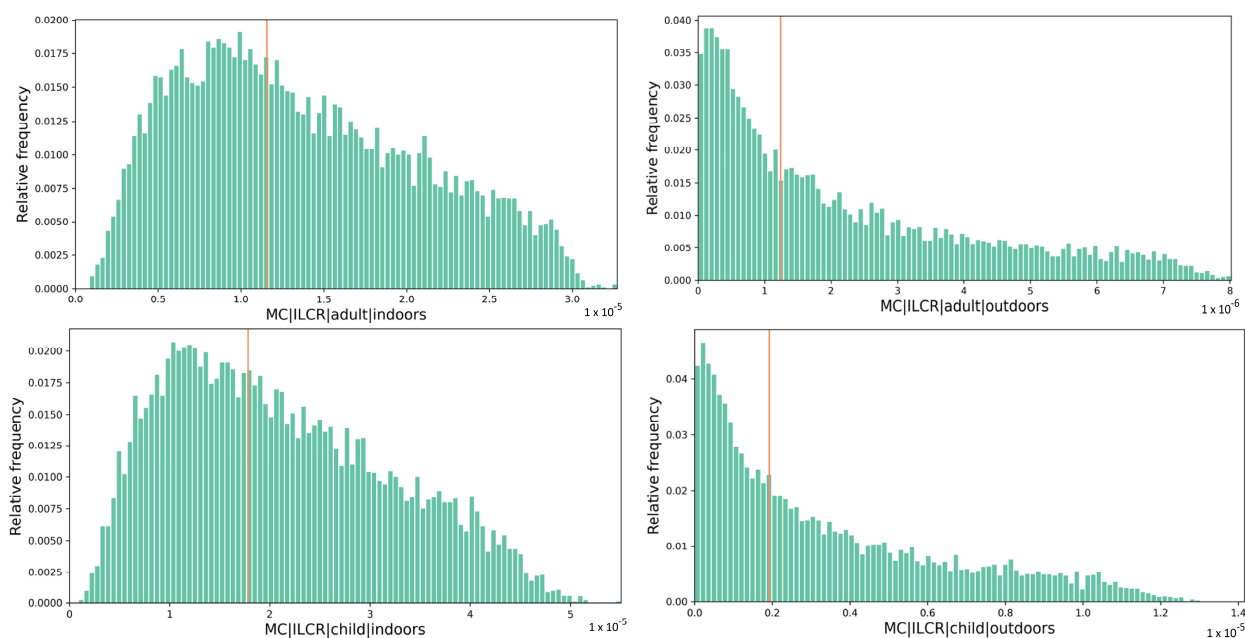


Figure 5. Distribution of incremental lifetime cancer risk for adults and children, derived using a Monte Carlo simulation. The orange line represents the median value.

The incremental lifetime cancer risk (ILCR) values due to PAH exposure in both adults and children in this study were significantly higher than those reported in previous research, such as the study by Jakovljević et al. [41] on the PM₁ particle fraction from outdoors at a residential area. In our findings, the ILCR for adults was 1.24×10^{-6} for outdoors and 1.15×10^{-5} for indoors, while for children, the values were 1.92×10^{-6} for outdoors and 1.78×10^{-5} for indoors. These values indicated potential cancer risk levels that exceed the U.S. EPA's acceptable risk threshold of 10^{-6} , suggesting that more than one in a million individuals may develop into an additional cancer case over the lifetime at this level. The higher ILCR values observed in this study compared to those reported by Jakovljević et al. [41] may stem from differences in measuring sites; Jakovljević et al. [41] determined PAHs in the PM₁ particle fraction at a residential area in the north side of Zagreb, while in this study, outdoor sampling was performed in different parts of Zagreb that are close to traffic areas. The ILCR values obtained in this study were one magnitude lower than the values reported for rural households in northern China (1.69×10^{-4}) [55] and similar to the values reported by Chen et al. [56]. In Europe, the annual ILCR value was estimated in an urban setting in Madrid, Spain, for outdoor, indoor, and indoor-generated PM₁₀ particle

fractions, and the risk was 6.2×10^{-6} , 3.1×10^{-6} and 2.3×10^{-6} , respectively [57]. Also, ILCR was estimated for other environments such as Warsaw and Gliwice University for lecturers with 40 years of work experience, and the risk was 4.39×10^{-7} in Warsaw and 1.16×10^{-6} in Gliwice [38].

This study focused on PAHs bound to PM_{10} , reflecting real-time exposure, while Jakovljević et al. [15] assessed PAHs in indoor dust collected by vacuum cleaners. While PAHs in dust typically accumulate over longer periods and may theoretically result in higher total concentrations, the higher ILCR values observed in this study may be attributed to several factors. Firstly, this study assessed PAHs bound to PM_{10} , which are more bioavailable and capable of penetrating deeper into the respiratory tract compared to larger dust particles. Secondly, PM_{10} -bound PAHs reflect real-time pollution levels, potentially capturing higher concentrations during pollution peaks, particularly in winter. Furthermore, the inhalation pathway, emphasized in this study, is more efficient in delivering PAHs to the systemic circulation compared to ingestion or dermal absorption, which were the primary pathways assessed by Jakovljević et al. [15]. Differences in sampling methodology, exposure assumptions, and toxic equivalency factors (TEFs) also likely contributed to the observed discrepancies. The study by Jakovljević et al. (2020) [41] investigated PAHs in the PM_{10} particle fraction in an urban location in Zagreb, highlighting seasonal differences in concentrations and associated health risks. The annual BaP concentration was reported to be 0.765 ng m^{-3} , with the total carcinogenic potency (TCP) averaging 0.14 ng m^{-3} in the warm period and 2.14 ng m^{-3} in the cold period. Their study attributed the higher PAH levels in the cold period to emissions from domestic heating and stable atmospheric conditions, which hindered pollutant dispersion. The incremental lifetime cancer risk (ILCR) values for outdoor PAHs were significantly lower than the U.S. EPA's threshold of 10^{-6} , indicating an acceptable level of risk for all population groups. Compared to the findings in this study, the lower ILCR values in the cited work can be explained by the focus on outdoor air, which generally exhibits less concentrated PAHs than indoor environments due to the absence of confined spaces and localized indoor sources. Future studies should integrate simultaneous measurements of PAHs in dust and PM_{10} , along with a unified risk assessment framework, to provide a more comprehensive understanding of PAH exposure risks.

4. Conclusions

This study aimed to characterize indoor and outdoor PAH concentrations in 37 urban households in Zagreb, Croatia, providing insight into the sources and health risks associated with PAHs in urban residential environments. The results showed consistently higher outdoor PAH levels across all compounds, suggesting a transfer of PAHs from outdoors to indoors. Traffic emissions, fossil fuel combustion, and residential heating were identified as the primary PAH sources in both environments based on the diagnostic ratios of the tested PAHs. Indoor PAH concentrations appeared to be influenced by liquid fossil fuel combustion and gasoline sources, while outdoor PAH concentrations showed stronger impacts from biomass combustion. Significant differences in indoor–outdoor PAH relationships were observed across households, highlighting the importance of local factors such as ventilation and indoor sources. Interestingly, PAHs such as DahA were observed at extremely low levels in both environments, with negligible differences between indoor and outdoor concentrations, highlighting minimal environmental fluctuation for certain lower-concentration PAHs. The overall higher outdoor PAH concentrations can likely be attributed to external pollution sources, including vehicular emissions, industrial activities, and combustion processes, which are more prevalent in outdoor environments. The variability in correlation suggests that outdoor-to-indoor PAH transfer differs between compounds. Overall, while indoor and outdoor PAH concentrations show strong internal correlations, the strength of the relationship between indoor and outdoor levels varies across different PAHs. The Canonical Correlation Analysis (CCA) identified BbF as a dominant contributor to outdoor PAH variability, likely from traffic emissions, while BkF showed strong negative loadings indoors, indicating that it may be less affected by outdoor

sources. The ILCRs obtained in this study across households for children and adults were between 10^{-6} and 10^{-5} , indicating a potential health risk that is probably lower than the priority risk level value (10^{-4}).

This study provides valuable information into the dynamics of PAH distribution in urban households, highlighting the relationships between indoor and outdoor air quality. These findings highlight the necessity of comprehensive air quality management strategies that integrate both environmental and public health considerations. The study's conclusions lead to the following recommendations for further research: investigate additional potential indoor sources of PAHs such as cooking practices, cleaning products, smoking, household heating, and some other factors that facilitate PAH transfer from outdoor to indoor environments (e.g., building materials and ventilation systems).

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Appendix A

Table A1. Parameters used in ILCR calculation for children and adults.

Definition	Units	Children	Adults
Inhalation Cancer Slope Factor of BaP (SF _{inh})	kg day mg ⁻¹	3.14	3.14
Exposure Frequency (EF)	day year ⁻¹	252	252
Daily Exposure Level (ED)	µg g ⁻¹	8.8×10^{-4}	7.1×10^{-4}
Body Weight (BW)	kg	40	70
Average Time (AT)	day	25550	25550
Inhalation Rate (IR)	m ³ day ⁻¹	24.87	32.74

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