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Predictors of urinary polycyclic aromatic hydrocarbon (PAH) metabolite concentrations in German children and adolescents using data from the German Environmental Survey 2014 – 2017 (GerES V)

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ABSTRACT

Children and adolescents are highly exposed to harmful polycyclic aromatic hydrocarbons (PAHs) that exist as mixtures of hundreds of compounds in air, soil, water, dust and food. This study investigated 21 personal, lifestyle and environmental predictors of urinary PAH metabolites from four PAHs (naphthalene, fluorene, phenanthrene and pyrene), using a dataset of 516 3–17-year-olds from 167 locations in Germany. Spearman correlation analysis showed weak to moderate associations ($\rho = 0.2\text{--}0.6$) between PAH metabolite concentrations. Regression analysis revealed high nicotine exposure, young age, and residence in large cities to be the most important predictors of multiple PAH burden, with estimated changes in metabolite concentrations of 124–1080%, 47–155%, and 22–57% respectively. Other notable predictors included: BMI, socioeconomic status, sex, consumption of chocolate, smoked and barbecued foods, chewing on plastic objects, road traffic, and heating type. Analysis of the ratios of 1-hydroxynaphthalene to 2-hydroxynaphthalene for subgroups with high cotinine level (3-fold increase) or moderate to high chocolate consumption (1.7–3.4-fold increase) was suggestive of exposure to carbaryl, a pesticide not permitted for use in the EU since 2007. Differences in the burden of PAH metabolites between East and West Germany was investigated using subgroup analysis. Consuming smoked food was both more prevalent ($p = 0.009$) and likely to contribute to a greater 1-hydroxynaphthalene burden in the East German subsample. Gaining a better understanding of exposure factors, especially using population data from a large number of locations, will guide policy makers and regulators in strengthening protective measures.

1. Introduction

Polycyclic aromatic hydrocarbons (PAHs) are universal environmental pollutants that negatively impact human health, targeting several endpoints and organs (Abdel-Shafy and Mansour, 2016; Armstrong et al., 2004; Borman et al., 2000; Legraverend et al., 1983; Mackenzie and Murray Angevine, 1981; Sombiri et al., 2024). They originally form as side products during the high temperature processing of organic materials, and contaminate the air (vapor and particulate phase), soil, water, dust, biota and food (EFSA, 2008; Keyte et al., 2013). There are over several hundred PAHs with differing physico-chemical and therefore toxicological properties, related to their structure and molecular weight. PAHs are transformed in vivo forming various activated metabolites, such as epoxides and diols, that mitigate the most harmful effects (Levin et al., 1982; Moorthy et al., 2015). PAHs are most

notorious for their known or suspected carcinogenic, genotoxic and reprotoxic properties (Bostrom et al., 2002; European Commission, 2002; Ohnishi et al., 2018). PAHs are also endocrine-disrupting chemicals (EDCs) and expected to mediate other negative health effects, such as respiratory, metabolic or reproductive issues (Haverinen et al., 2021; Perono et al., 2022; Vondráček and Machala, 2021; Ye et al., 2020; Zhang et al., 2016). It is even more challenging to comprehend the full extent of toxicity of PAHs as exposure involves mixtures of hundreds of PAHs and other xenobiotics, with multiple routes of uptake (ingestion, dermal absorption and inhalation) (Barbosa et al., 2023; Lao et al., 2018; Li et al., 2015).

A special focus on the environmental health of children and adolescents is important because they are more susceptible to pollutants and at risk of health effects that may last throughout their lifetime (WHO, 2018). The organs and metabolic processes of younger children are

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not fully developed and they have the highest intake of air, water and food per unit of body weight (Hauptman and Woolf, 2017; Schwartz, 2004; Sly and Carpenter, 2012). Furthermore, pre- and pubescent children are also more vulnerable to the effects of EDCs (Bali et al., 2023; Lughetti et al., 2015; Lucaccioni et al., 2020). Positive correlations between local airborne PAH concentrations and the rate of infants developing cancers and respiratory health issues have been reported (Heck et al., 2014; Jedrychowski et al., 2015; Jung et al., 2012; Karimi et al., 2015; Symanski et al., 2016; von Ehrenstein et al., 2016). However, it is very difficult to unravel the complex relationships between an individual's PAH exposure and negative health outcomes, particularly as it is hard to account for the many potential routes and sources of PAH exposure (WHO, 2021). A human biomonitoring (HBM) approach can overcome this by calculating the internal burden of a substance in an individual, thereby including all sources and routes of exposure. HBM refers to determining the concentrations of chemical pollutants and their metabolites, exposure biomarkers, in body fluids or target tissues (Angerer et al., 2007; Choi, 2015). The resulting HBM data can be analyzed alongside questionnaire data to identify risk factors for exposure in a population (Arnold et al., 2021; Clewell et al., 2008). These findings have important implications on an international level, where they inform and strengthen policy and chemical regulations towards a "toxic-free environment" (Ganzleben et al., 2017; Zare Jeddi et al., 2022).

PAH exposure in a population can be assessed successfully using HBM. Determination of urinary metabolites from lower molecular weight PAHs is the most suitable for HBM studies, because it is non-invasive, has excellent detection rates in the general population, and appears to correlate sufficiently with total PAH exposure (Göen et al., 1995; Jongeneelen, 1994; Kang et al., 2002; Karakoltzidis et al., 2025; Li et al., 2008; Nübler et al., 2023; Poursafa et al., 2017; Vyskocil et al., 2000; Wilhelm et al., 2007; Zhu et al., 2011). The metabolites 1-hydroxypyrene and hydroxynaphthalenes have been the most widely investigated in population studies (Jongeneelen, 1997; Jongeneelen et al., 1987; Santos et al., 2019; Styszko et al., 2023). In Germany, as part of the fifth German Environmental Survey 2014-17 (GerES V), urinary metabolites of naphthalene, fluorene, phenanthrene and pyrene were analyzed in children and adolescents (Kolossa-Gehring et al., 2012; Murawski et al., 2020b). The diverse size and structure of these four PAHs, which importantly affects their phase and environmental distribution, allows coverage of a reasonable portion of the spectrum of PAHs and their derivatives (Keyte et al., 2013; Peng et al., 2023; Ravindra et al., 2008). From an EU regulatory perspective, naphthalene is classified as suspected of causing cancer while naphthalene and phenanthrene are recognized as orally acutely toxic (ECHA, 2016, 2026; European Commission, 2024b). Though not formally under assessment as EDCs, monohydroxylated metabolites of all four PAHs have been shown in assays to be estrogenic (Kamiya et al., 2005; Sievers et al., 2013; Van de Wiele et al., 2005; Wenger et al., 2009).

Bivariate analysis of the GerES V HBM data highlighted several possible predictors of PAH burden (Murawski et al., 2020b). Age and smoking status were found to be highly significant across all metabolites, while population size of the community, consuming chocolate and chewing on plastic objects were reported for the first time as potential exposure factors. The participants' region of residence, East or West Germany, was also found to be significant (Fig. 1). The two regions refer to the two formerly distinct countries, the Federal Republic of Germany (FRG) and the German Democratic Republic (GDR), that reunified in 1990. East-West differences in PAH exposure in 2014-17 are somewhat surprising, more than 23 years after reunification (Fertmann et al., 2002; Schulz et al., 2007). East-West differences in PAH burden, first recorded in 1992 (GerES II), were then attributed to differences in coal heating and industrial activities. The combined exposure biomarker data from GerES V was also investigated in a mixture assessment, which found that the internal burden of PAH is linked to the burden of benzene and 4-*tert*-butylbenzoic acid (TBBA) (Rodríguez Martín et al., 2023). TBBA,

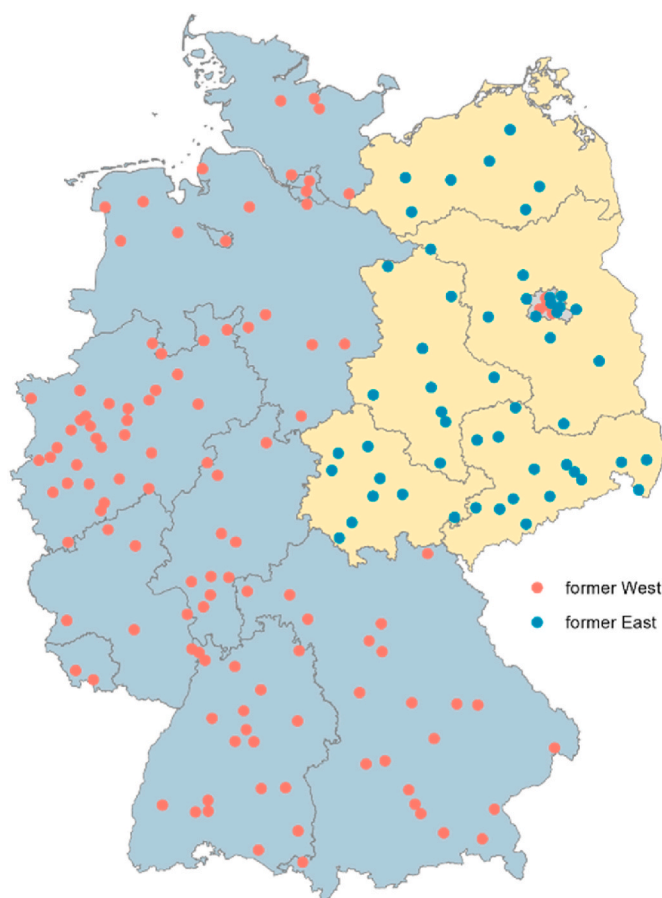


Fig. 1. Map of Germany showing the sampling locations in East and West Germany used in GerES V.

is a non-specific biomarker, mostly associated with exposure to the fragrance lysmeral (Scherer et al., 2017).

In this work, we build on the previous results of Murawski et al. (2020b) to gain a deeper understanding of multiple PAH exposure factors in the GerES V dataset. First relationships between the burden of PAHs as well as nicotine, benzene and TBBA was assessed using correlation analysis. Then multiple linear regression analysis was used to determine significant personal, lifestyle and environmental predictors of PAH internal burden for persons aged 3-17 in Germany and discussed in context with other studies. The predictors were then further analyzed to find potential differences in prevalence and exposure between East and West Germany. This study not only complements others that focus on diverse predictors of multiple PAH exposure for children or adolescents, but it is the first to use a large nationally representative HBM dataset outside the US (Alghamdi et al., 2015; Dobraca et al., 2018; Fernández et al., 2021; John et al., 2022; Liu et al., 2017; Smetanová et al., 2025; Tabatabaei et al., 2022).

2. Materials and methods

2.1. Study population

A total of 2294 participants aged 3-17 years from 167 sampling locations in Germany were recruited for GerES V from the German Health Interview and Examination Survey for Children and Adolescents (KiGGS Wave 2) (Fig. 1) (Mauz et al., 2017; Schulz et al., 2017, 2021). To increase statistical power, more participants were included at sampling points in East Germany, which refers to the German federal states that used to be part of the GDR, including East Berlin. This accounts for the relatively smaller proportion of the population living there. To assess

PAH internal burden, urine samples from 516 participants were randomly selected for HBM analysis, ca. 20% from each sampling location (Murawski et al., 2020b). A weighting factor provided by the Robert Koch Institute (RKI) was used to ensure results derived from the subsample were population-representative in terms of age structure, sex, migration background, region (East or West Germany), and parents' education level (Hoffmann et al., 2018; Lüttinger and König, 1988). The fundamental characteristics of the subsample, as well as the East and West German subsamples, are presented in Table S1 (Supplementary Data).

2.2. Human biomonitoring data

The complete details of the chemical analyses, including full QA/QC (Quality Assurance and Quality Control) measures, are described in Murawski et al. (2020b). HBM analysis was carried out on first void urine samples, which were collected from the GerES V participants at their home between January 2015 to June 2017 (with fieldwork preparation beginning in 2014). The hydroxy-PAH biomarkers of this work are listed with their descriptive statistics in Table 1. The PAH biomarker concentrations were determined using liquid chromatography-tandem mass spectrometry (LC-MS/MS) following enzymatic hydrolysis and solid-phase extraction; this method is validated in accordance with FDA (US Food and Drug Administration) guidelines (Ramsauer et al., 2011). To normalize for variations in dilution, creatinine-adjusted concentrations were also determined (Barr et al., 2005; UBA, 2005). Creatinine was measured using the Jaffé method (Błaszczewicz and Liesenhoff-Henze, 2010). These analyses were carried out at the Analytical Biological Research Laboratory, Munich (ABF GmbH). The sum parameter $\sum(\text{OH-phe})$ is used to represent the five metabolites of phenanthrene that are strongly correlated: 1-, 2-, 3-, 4- and 9-hydroxyphenanthrene (Murawski et al., 2020b). The concentrations of 2-OH-flu are overestimated 30-35% due to overlap with the signal of the other fluorene metabolites (3- and 9-hydroxyfluorene). 1-OH-nap is evaluated separately from 2-OH-nap since they are not strongly correlated and 2-OH-nap is dominant. 1-OH-naph is also the primary biomarker of carbaryl (1-naphthyl-N-methylcarbamate), a pesticide used internationally, but not permitted in the EU since 2007 (Knaak et al., 1965; Shealy et al., 1997). Other biomarkers of interest from GerES V – cotinine, S-phenylmercapturic acid (SPMA) and 4-tert-butylbenzoic acid (TBBA)), summarized in Table 1 – represent exposure to nicotine, benzene and 4-tertbutylphenyl derivatives respectively and have been previously published elsewhere.

2.3. Interview data

Data relating to personal, lifestyle and environmental factors was obtained from detailed in-person interviews of the participants (if 11 years and older) and their parent/guardian as part of KiGGS Wave 2 or GerES V. All sociodemographic data was obtained during KiGGS Wave 2. Sex refers to biological sex at birth. Socioeconomic status (SES) was calculated using a multidimensional index informed by the parent's education level, occupational status and equalized disposable income (Lampert et al., 2018). SES index >80th percentile is defined as high SES, SES index <20th percentile is defined as low SES. Migration background is categorized as none, one-sided or two-sided (Frank et al., 2018). One-sided is defined as having one parent not born in Germany or without German citizenship. Two-sided is defined as participants who migrated to Germany, have both parents not born in Germany or both parents without German citizenship. Body Mass Index (BMI) was determined using a physical examination and BMI percentile was calculated according to Kromeyer-Hauschild et al. (2001) as described in Schienkewitz et al. (2018). BMI >90th percentile is defined as overweight or obese, BMI <10th percentile is defined as underweight. The data concerning smoked food, barbecued food and fish consumption refer specifically to consumption within the 48 h prior to sample collection. Barbecued food here refers to any foods cooked over an open flame. The other dietary questions relate to the frequency of consumption of burgers and kebabs, red meat (meat excluding poultry and sausages) and chocolate considering a four week period not in connection with the time of sampling (Krug et al., 2018). Chocolate consumption in grams per day was estimated based on the frequency of consumption (ranging from never to five times per day) and the typical portion size relative to a 100 g chocolate bar. The variable 'wearing plastic shoes' was informed by a question asking whether rubber or plastic shoes are worn regularly without socks in summer. Road traffic rating was based on the participant or parent/guardian's categorization of the type of road (or busiest one if more than one) that their residence is situated on. 'Extremely busy' refers to a busy to extremely busy main road, 'busy' refers to a busy side street, 'moderate' refers to a moderately busy side street and 'low' refers to a quiet street with little to no traffic. Heating type and cooking device type refer to the type used predominantly in the household. 'Wood and fossil fuels' refer to oil, coal, wood and pellets. Renewable refers to solar and geothermal energy. 'Heating stove or fireplace' refers to the use of additional devices that burn for example coal, pellets, wood or ethanol at home.

Table 1
HBM analysis of biomarkers in GerES V.

Biomarker	Abbreviation	Parent substance	N	LOQ (µg/L)	% ≥ LOQ	GM (µg/L)	GM (µg/g _{crea})	P95 (µg/L)	Citation
PAH metabolites									
1-hydroxynaphthalene	1-OH-nap	naphthalene, carbaryl	516	0.05	96	0.785	0.688	7.14	[1]
2-hydroxynaphthalene	2-OH-nap	naphthalene	516	0.05	100	4.233	3.706	23.7	[1]
2-hydroxyfluorene ^a	2-OH-flu	fluorene	516	0.05	88	0.387	0.338	1.27	[1]
$\sum(\text{hydroxy-phenanthrenes})$	$\sum(\text{OH-phe})$	phenanthrene	516	^b	^b	0.511	0.448	1.80	[1]
1-hydroxypyrene	1-OH-pyr	pyrene	516	0.01	99	0.099	0.087	0.36	[1]
Other metabolites									
Cotinine	Cot	nicotine	2250	0.10	56	0.35	0.30	28.8	[2]
S-phenylmercapturic acid	SPMA	benzene	2260	0.02	98	0.097	0.083	0.41	[3]
4-tert-butylbenzoic acid	TBBA	4-tert-butylphenyl derivatives e.g. lysmeral	2133	0.20	100	10.21	8.658	53.40	[4]

N = total sample size, LOQ = (lower) limit of quantification, g_{crea} = grams of creatinine, GM = geometric mean, P95 = 95th percentile.

$\sum(\text{hydroxyphenanthrenes})$ = Sum of 1-, 2-, 3-, 4- and 9-OH-phenanthrene.

^a 2-OH-flu concentrations overestimated 30-35% due to overlap with other OH-Flu signals.

^b The LOQs for the individual OH-phe metabolites are ≤0.005 µg/L with 97-100% of values quantifiable (≥LOQ).

Citations: [1] Murawski et al., 2020b [2] Hahn et al., 2023 [3] Schwedler et al., 2021 [4] Murawski et al., 2020a

2.4. Statistical analysis

The HBM and interview data were analyzed using multiple linear regression. Where there were missing data points for any of the variables, these participants were excluded from the multivariable regression analysis, resulting in a reduced whole sample of $N = 469$. The logarithmized PAH biomarker concentration (volume-based) was set as the dependent variable for each model, which investigated all personal, lifestyle and environmental variables in the same model. Use of logarithmized concentrations sufficiently fulfills the requirements of a linear regression. Creatinine concentration (continuous) was included as a covariate to account for differences in dilution (Barr et al., 2005). All other variables were categorical. Urinary cotinine concentration, relative to a cut-off value of 50 $\mu\text{g/L}$, was used to reflect nicotine exposure from active smoking (Gruber and Schuurmans, 2018; Hahn et al., 2023; Haufroid and Lison, 1998). Sampling season was included as an interaction term for the heating-related variables. Sampling season was categorized as winter (October–March) and summer (April–September) to represent the heating and non-heating season (Jung et al., 2014). To be able to interpret the results of the regression analysis straightforwardly, the regression equation was exponentiated, turning the original additive model into a multiplicative model. The beta coefficients, when transformed as $[(\exp(\beta_i)-1)*100]$, can therefore be interpreted as % changes relative to the respective reference category (Wooldridge, 2013). Results are reported to 3 significant figures (or to 1 decimal place if the magnitude is less than 10%). Further analyses were carried out using the whole GerES V sample ($N = 516$). Correlations between the different PAH biomarkers were analyzed using Spearman-Rank Correlation (two-tailed). The prevalences of the predictors in East and West were analyzed for significant differences using chi-squared tests. Where differences in prevalence of a predictor were significant, t-tests were used to test the equality of means between subgroup of different exposure levels, using the logarithmized creatinine-based concentrations. The mean differences were also exponentiated using the above equation so they could be interpreted as % differences in real PAH concentrations. Levene's test for equality of variances was applied to calculate p-values and confidence intervals. All statistical analyses were carried out using IBM SPSS software, versions 20 and 26.

3. Results and discussion

3.1. Metabolite correlations

Since exposure to PAHs occurs in complex mixtures, it is valuable to assess the relationships between individual PAH metabolites to demonstrate how representative they are of the overall PAH burden. Spearman rank correlation analysis was used to quantify associations between PAH metabolites, as well as between the exposure biomarkers cotinine, SPMA and TBBA measured in GerES V (Table 1). The analysis revealed overall weak to moderate positive correlations between PAH metabolite concentrations in the range $\rho = 0.20$ – 0.60 (Fig. 2). The sum parameter $\Sigma(\text{OH-phe})$ was found to have the strongest associations with the other PAH metabolites giving moderate correlations with 1-OH-nap, 2-OH-nap and 1-OH-pyr ($\rho = 0.45$ – 0.60). 2-OH-nap was also moderately correlated with 1-OH-nap and 1-OH-pyr ($\rho = 0.40$ – 0.42). 2-OH-flu was most correlated with 1-OH-nap ($\rho = 0.37$). Consistent results were observed when using creatinine-normalized concentrations (Table S2) or with Pearson analysis of the logarithmized concentrations (not shown). In contrast, for adults from several European countries (aged 20–39) in a similar time period but in a non-population representative sample, PAH metabolites were much more strongly correlated. For 1-OH-nap, 2-OH-flu, $\Sigma(\text{OH-phe})$ and 1-OH-pyr, correlations between PAH metabolites were strong ($\rho > 0.68$), though associations with 2-OH-nap were mostly moderate ($\rho < 0.61$) (Karakoltzidis et al., 2025). More comparable studies of children in other regions, Valencia, Spain (5–12 years) or Northern California (girls 6–8 years), observed consistent

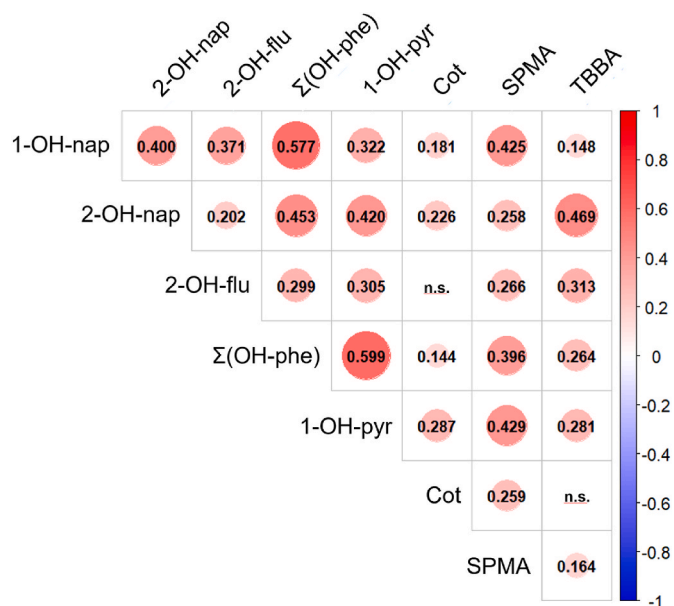


Fig. 2. Spearman correlation matrix for exposure biomarker volume-based concentrations in urine.

OH-nap = hydroxynaphthalene, OH-flu = hydroxyfluorene, $\Sigma(\text{OH-phe})$ = Sum of 1-, 2-, 3-, 4- and 9-OH-phenanthrene, OH-pyr = hydroxypyrene, Cot = cotinine, SPMA = S-Phenylmercapturic acid, TBBA = 4-tertbutylbenzoic acid, n.s. = not significant.

results in correlation analyses with the results of the European adults study (Dobraca et al., 2018; Fernández et al., 2021). A lack of strong association between 1-OH-nap and 2-OH-nap concentrations, is therefore documented and may be due to changes in metabolism with different types of exposure, as well as exposure sources (Smetanová et al., 2025). The weak correlations between some PAH metabolites in this study, particularly 2-OH-flu, are remarkable and perhaps suggest that correlations are weaker when analysing samples that feature a larger age range of both children and adolescents on a population-representative basis. This further underlines the importance of using national datasets and supports the use of different regression models for each biomarker.

Correlations between urinary cotinine, the primary biomarker of nicotine, and PAH concentrations indicate smoking-related exposures. Cotinine was found to have weak or no correlation with the PAH metabolites, but was most correlated with 1-OH-pyr ($\rho = 0.29$) (Fig. 2). Since the majority of the GerES V subsample are children under 14 (71%) and not all adolescents are active nicotine users, urinary cotinine concentration serves mostly as an indicator of ETS (Environmental Tobacco Smoke) exposure. The weak correlation between cotinine and PAH metabolites suggests that ETS exposure is not such an important contributor to PAH burden here. Weak or no correlations between cotinine and PAH metabolites were also reported for German adult students (Burkhardt et al., 2023). Changes in legislation are also believed to have led to decreased ETS exposure in Germany since 2007, such as the Federal Non-smokers Protection Act and Youth Protection Act (Burkhardt et al., 2023; Hahn et al., 2023; Kohler and Minkner, 2014). Poor correlations between PAH metabolites and cotinine in non-smoking populations has been reported in other studies (Lee et al., 2025; St.Helen et al., 2012).

The biomarkers SPMA and TBBA potentially share exposure sources with PAHs, as suggested by the network analysis of GerES V HBM data (Rodríguez Martín et al., 2023). SPMA is a biomarker of benzene while TBBA is a metabolite of synthetic compounds based on a 4-tert-butylphenyl moiety (Scherer et al., 2017; Stommel et al., 1989). Correlations with PAH metabolites were overall weak, except for moderate correlations between SPMA and 1-OH-nap or 1-OH-pyr ($\rho = 0.43$) and between

TBBA and 2-OH-nap ($\rho = 0.47$) (Fig. 2). Co-exposure to PAHs and volatile aromatic compounds like benzene is likely via airborne sources, with SPMA concentrations in GerES V being significantly associated with exposure to tobacco and vehicular pollution (Schwedler et al., 2021). Correlation of TBBA and 2-OH-nap cannot be straightforwardly explained but is suggestive of exposure to synthetic materials at home such as adhesives, coatings, plastic or rubber (Burkhardt et al., 2023; Hahladakis et al., 2018; Hoang and Park, 2024; Kang et al., 2012; Murawski et al., 2020a; Scherer et al., 2017). Furthermore, unpleasant odors emanating from children's plastic toys in Germany were found to contain naphthalene, other PAHs and VOCs (volatile organic compounds) (Denk et al., 2017; Even et al., 2019; Wiedmer et al., 2017). Overall, the correlation results demonstrate how mixture analysis can support the differentiation of PAH exposure sources and maybe even identify new ones.

3.2. Predictors of PAH internal burden

Multiple linear regression analysis was used to evaluate the various potential predictors of PAH burden, related to personal characteristics, lifestyle and environmental factors. Each of the five PAH biomarker or sum concentrations, listed in Table 1, were modelled with respect to all variables. The variables were chosen in accordance with the previous bivariate analysis (Murawski et al., 2020b). In the current work, the variable urinary cotinine level (above or below 50 $\mu\text{g/L}$) was used to represent active smokers and non-smokers, instead of self-reported smoking status (Gruber and Schuurmans, 2018). The dietary variables investigated were also expanded in this work to include red meat (excluding sausages), smoked food, fish, hamburgers and kebabs. The resulting regression models explain 15.7–38.5 % of the variance in metabolite or sum concentrations (Table 2). The covariate ‘creatinine concentration’ corresponds to significant increases of 0.06–0.08% in PAH metabolite or sum concentration per 1 mg/L increase in creatinine. The results of the regression analysis are presented in Tables 3a, 3b, and 3c and discussed in the following sections.

3.2.1. Personal characteristics

Investigation of demographic variables can help to identify at-risk groups. It is also important to control for age, sex and BMI when analysing metabolite concentrations, while SES and migration background may represent more complex exposure factors specific to Germany.

Urinary PAH metabolite concentrations strongly decrease with age. The most heavily burdened group is those aged 3-5 years with increases in the range of 47.0% (95% CI: 3.0%, 110%) – 155% (95% CI: 64.7%, 296%) for all five PAH biomarkers compared to 14–17-year-olds (Table 3a). The age trend is most clear for $\sum(\text{OH-phe})$ with significant increases for the age groups 6-10 and 11-13 years of 63.2% (95% CI: 33.2%, 100%) and 25.6% (95% CI: 6.2%, 48.6%) respectively, although similar trends are evident for the other metabolites even if not significant. Negative associations between age and PAH metabolites are well-

Table 2
Regression Models: R^2 values, intercepts and covariate urinary creatinine.

Model	R^2	R^2_{adj}	Intercept	Urinary creatinine (g/L)
log [1-OH-nap]	0.373	0.290	-3.194 (-4.376, -2.012)	0.599 (0.392, 0.805)
log [2-OH-nap]	0.357	0.271	-0.429 (-1.257, 0.399)	0.612 (0.427, 0.798)
log [2-OH-flu]	0.256	0.157	-2.812 (-4.169, -1.454)	0.752 (0.464, 1.04)
log [$\sum(\text{OH-phe})$]	0.407	0.328	-2.302 (-2.775, -1.830)	0.551 (0.439, 0.662)
log [1-OH-pyr]	0.457	0.385	-3.593 (-4.202, -2.983)	0.628 (0.462, 0.794)

OH-nap = hydroxynaphthalene, OH-flu = hydroxyfluorene, $\sum(\text{OH-phen})$ = Sum of 1-, 2-, 3-, 4- and 9-OH-phenanthrene, OH-pyr = hydroxypyrene.

Table 3a
Results of the regression analysis: Personal characteristics (N = 469).

Predictor	n (%)	% change in metabolite concentration				
		1-OH-nap	2-OH-nap	2-OH-flu	$\sum(\text{OH-phe})$	1-OH-pyr
Age group/years						
3 – 5	17.5	155 (64.7 , 296)***	47.0 (3.0 , 110)*	121 (48.1 , 231)***	97.7 (60.7 , 143)***	46.7 (18.3 , 82.0)***
6 – 10	32.6	112 (40.5 , 221)***	13.9 (-16.5, 55.4)	30.9 (-17.5, 107.6)	63.2 (33.2 , 100)***	17.3 (-5.9, 46.3)
11 – 13	20.7	29.9 (-14.4, 97.0)	-0.9 (-27.2, 34.9)	31.9 (-19.1, 115.1)	25.6 (6.2 , 48.6)**	10.2 (-11.3, 36.8)
14 – 17	29.2	0	0	0	0	0
Sex						
female	45.1	17.3 (-13.3, 58.7)	12.6 (-8.1, 37.6)	26.2 (-7.2 , 71.6)	15.7 (2.3 , 30.8)*	9.3 (-6.5, 27.9)
male	54.9	0	0	0	0	0
BMI percentile						
high (>P90)	18.5	0.2 (-29.9, 43.2)	36.9 (3.4 , 81.3)*	0.2 (-32.9, 49.5)	3.8 (-12.7, 23.5)	-5.6 (-22.9, 15.7)
normal low (<P10)	73.1 8.4	0 -17.2 (-43.7, 21.8)	0 -15.1 (-37.2, 14.7)	0 0.6 (-38.3, 63.9)	0 -6.3 (-21.9, 12.6)	0 1.7 (-19.0, 27.7)
SES index (percentile)						
low (<P20)	17.6	25.6 (-9.6, 74.5)	43.3 (5.5 , 94.7)*	7.2 (-32.8, 71.1)	10.2 (-6.8, 30.2)	23.4 (-1.2 , 54.3)
medium	65.1	0	0	0	0	0
high (>P80)	17.3	-19.5 (-43.1, 13.9)	-16.2 (-35.1, 8.2)	17.8 (-14.1, 61.5)	15.9 (-0.7 , 35.3)	7.9 (-11.9, 32.2)
Migration background						
none	73.7	0	0	0	0	0
one-sided	12.4	-6.5 (-33.1, 30.9)	-17.3 (-34.3 , 4.1)	35.3 (-3.4 , 89.5)	-11.8 (-25.5 , 4.5)	-1.4 (-20.5, 22.2)
two-sided	13.9	-2.0 (-31.4, 40.1)	5.1 (-23.5, 44.2)	36.0 (-24.8, 146.0)	-4.4 (-22.5, 18.0)	2.2 (-19.6, 30.1)

Significant and borderline significant results ($p \leq 0.1$) are shown in bold. The p-values for the significant results are denoted by * ($p < 0.050$), ** ($p < 0.010$) and *** ($p < 0.005$). The 95% confidence intervals are shown in brackets. N = total sample size, n (%) = subgroup size as a % of the total sample, OH-nap = hydroxynaphthalene, OH-flu = hydroxyfluorene, $\sum(\text{OH-phen})$ = Sum of 1-, 2-, 3-, 4- and 9-OH-phenanthrene, OH-pyr = hydroxypyrene, ref = reference group, BMI = Body Mass Index, SES = Socioeconomic Status.

established (Fernández et al., 2021; Hoseini et al., 2018; Huang et al., 2006; Jain, 2020; John et al., 2022; Lee et al., 2007, 2009b; Liu et al., 2017; Smetanová et al., 2025). Not only are young children more susceptible to PAHs physiologically, but they also engage in frequent hand-to-mouth behavior, crawling and playing on or closer to the ground, enabling a greater intake of PAHs from house-dust, soil and carpets (Stamatelopoulos et al., 2021). Younger children also spend more time outside, which has been shown to be a predictor of urinary 1-OH-pyr levels, and could act here as a confounding factor (Hansen et al., 2006; Peters et al., 2017). Results from the KiGGS module on physical activity (MoMo Study, 2014-17) found that 91% of German children aged 4-5 years play outside at least three times a week compared to just 15% of adolescents aged 14-17 years (Schmidt et al., 2020).

Sex has a small effect on urinary PAH metabolite concentration. This was significant for $\sum(\text{OH-phe})$ with an increase in 15.7% (95% CI: 2.3%, 30.8%) for girls (Table 3a). 2-OH-flu was also raised in girls with an increase of 26.2% (95% CI: -6.5%, 71.6%), as were the other metabolites but to a lesser extent. Sex differences in PAH internal burden in

children or adolescents are documented in some studies, mostly as a predictor of pyrene and/or phenanthrene metabolites (Fernández et al., 2021; Jain, 2020; Lee et al., 2007; Liu et al., 2017; Miri et al., 2018; Smetanová et al., 2025). This does seem to agree with the current knowledge of sex-related differences in susceptibility and metabolism of PAHs in adults (Uppstad et al., 2011; Zhang et al., 2014). Differences in behavior and diet as a result of gender may also contribute to differences in exposure and should not be excluded (Brettschneider et al., 2021; Krug et al., 2018; LoMauro and Aliverti, 2021).

BMI percentile was associated with urinary naphthalene metabolites levels. Being overweight or obese was a predictor of 2-OH-nap concentration with a significant increase of 36.9% (95% CI: 3.4%, 81.3%) compared to normal weight (Table 3a). Though not significant, small decreases in 1-OH-nap and 2-OH-nap levels were found for the underweight participants. This suggests a trend where excretion of OH-nap metabolites increases with BMI. For all other metabolites, changes in concentration with BMI percentile were negligible. BMI or BMI percentile is sometimes a predictor of PAH metabolites in children or adolescents (Alghamdi et al., 2015; Fernández et al., 2021; Jain, 2020; John et al., 2022; Liu et al., 2017). Other studies focused on childhood obesity risk found positive associations between naphthalene metabolites in particular and weight status, BMI z-score, cardiometabolic indicators or waist circumference (Bushnik et al., 2020; Kim et al., 2023; Poursafa et al., 2018; Scinicariello and Buser, 2014; Zhang et al., 2025). PAH exposure is in fact a risk factor for obesity due to underlying molecular mechanisms, such as proposed PAH-promoted changes in lipid metabolism (Wang et al., 2015; Ye et al., 2024). However, differences in BMI may also reflect differences in diet and eating habits. For example, in KiGGS, consumption of ultra-processed foods (UPFs) is also associated with obesity risk (Figueiredo Barata et al., 2025; Hoebel et al., 2022).

Socioeconomic status (SES) was found to affect all urinary PAH metabolites. Low SES is associated with increased 2-OH-nap by 43.3% (95% CI: 5.5%, 94.7%) and increased 1-OH-pyr by 23.4% (95% CI: -1.2%, 54.3%) (Table 3a). Both 1-OH-nap and 2-OH-nap concentrations appear to decrease with increasing SES. In contrast, high SES is associated with increased $\sum(\text{OH-phe})$ by 15.9% (95% CI: -0.7%, 35.3%). Other studies have also shown that higher poverty income ratio, parents' or mother's education level, or household income are associated with decreased urinary metabolites, mainly 2-OH-nap and 1-OH-pyr (Dobraca et al., 2018; Fernández et al., 2021; Jain, 2020; John et al., 2022; Liu et al., 2017). Only one study from Korea, which investigated the influence of residential distance from a steel mill, reported increased 1-OH-pyr levels with family income (Lee et al., 2007). The SES index used in this study was specifically developed to investigate health inequalities in Germany (Kuntz et al., 2018; Lampert et al., 2018). In other GerES studies, low SES index was found to be associated with higher indoor benzene and toluene air concentrations, higher urinary cotinine concentration or ETS exposure at home, and greater walking times to green spaces in urban areas (Hahn et al., 2023; Rehling et al., 2021; Wiehn et al., 2025). The latter may be relevant as distance from green spaces and vegetation index around the home were found to be significant predictors of 1-OH-pyr in one study (Miri et al., 2018). SES may also affect dietary factors, with low SES index linked to lower quality diets in preschoolers or adolescents and a higher proportion of caloric intake from fast-foods for adolescents in German studies (Brettschneider et al., 2021; Burgard et al., 2025; Moosburger et al., 2020).

Migration background also appears to affect PAH burden. Participants with one-sided migration background were associated with changes in the concentrations of 2-OH-flu by 35.3% (95% CI: -3.4%, 89.5%), 2-OH-nap by -17.3% (95% CI: -34.3%, 4.1%) and $\sum(\text{OH-phe})$ by -11.8% (95% CI: -25.5%, 4.5%), compared to no migration background (Table 3a). Results for two-sided migration background had large confidence intervals. One study from Valencia, Spain also found significantly higher OH-nap levels for children with no migration background (Fernández et al., 2021). Within GerES studies, two-sided migration background was a predictor of urinary cotinine level, even

when accounting for sources of ETS (Hahn et al., 2023), and associated with lower benzene concentration in indoor air (Wiehn et al., 2025). Migration background may also affect dietary habits and within KiGGS, two-sided migration background was also associated with 'disadvantageous food patterns' that are not explained by differences in SES (Koschollek et al., 2019; Schenk et al., 2016).

Overall, the most important predictors of urinary PAH metabolite concentrations among personal characteristics are age groups 3-5 years, 6-10 years and low SES, which increase concentrations of all or most metabolites. Being overweight or obese is a predictor of 2-OH-nap concentration specifically, though whether BMI should be regarded as a predictor of burden or as an exposure outcome needs to be considered. The effect of sex is small but interesting and further investigation of SES and migration background as variables is required to more clearly understand how they affect PAH burden.

3.2.2. Lifestyle factors

Studies show that PAH metabolites in urine spike most dramatically hours after smoking tobacco or consuming barbecued food (Li et al., 2010, 2012). More than 500 PAHs have been detected in tobacco smoke (Rodgman and Perfetti, 2006) while exposure of foods to smoke, flames, charcoal, oil-frying or direct heat, raises their PAH content substantially (Alomirah et al., 2011; Ekhtator et al., 2018; Rodgman and Perfetti, 2006; Sampaio et al., 2021; Yao et al., 2015). All types of foods may contain PAHs, though lipid-rich foods, such as meat or fish, tend to contain the highest PAH concentrations, (Wu et al., 2025). Use of plastic or synthetic products and surface materials is also a potential source of PAH exposure, particularly affecting low-cost or imported goods. PAHs can be used as plastic additives, e.g. softeners and pigments, or are present as contaminants (BfR, 2009; Preuss et al., 2003; UBA, 2016).

Participants with cotinine levels above 50 µg/L were found to have the greatest changes in internal burden across all metabolites. The change in urinary 1-OH-nap concentration was exceptionally large with an increase of 1080% (95% CI: 624%, 1830%) (Table 3b). The average percentage increase for the other metabolites ranged from 124% (95% CI: 24.0%, 305%) for 2-OH-nap to 230% (95% CI: 78.8%, 510%) for 2-OH-flu. High cotinine level in German adolescents can be attributed to recent or frequent use of tobacco, waterpipe (hookah) and e-cigarettes, though the latter do not increase urinary PAH metabolites (Etemadi et al., 2019; Gali et al., 2022; Hahn et al., 2023; Maziak et al., 2015; Scherer et al., 2022). Urinary cotinine cut-offs, typically between 30 and 100 µg/L, are used to signify active nicotine use, though one recent study recommends a much lower cut-off of 7 µg/L for adolescents to reflect different behaviors and metabolism (Benowitz et al., 2016; Haufroid and Lison, 1998; Paci et al., 2018; Yang et al., 2025). Accordingly, the cut-off level in this study likely indicates the highest level of nicotine exposure, excluding ETS exposure and occasional or light nicotine use. Jain (2020) also found high serum cotinine concentration to be a predictor of the same urinary PAH metabolites in US adolescents. Research on adult smokers has shown that fluorene metabolites, especially 1-OH-Flu, and 2-OH-nap show the strongest relationships with nicotine biomarkers in toxicokinetic studies, while phenanthrene and pyrene metabolites are largely unaffected by smoking (Liu and Jia, 2016; St.Helen et al., 2012). This suggests that adolescent smokers may have higher burdens of pyrene and phenanthrene metabolites compared to adult smokers. The much greater increase in 1-OH-nap relative to 2-OH-nap also suggests that smokers are also exposed to carbaryl as a result of carbaryl use in tobacco cultivation (see Section 3.3).

Consuming foods processed by smoking, barbecuing or direct heat raised urinary naphthalene, or fluorene and pyrene metabolite concentrations. Recent consumption of smoked food corresponded with increased OH-nap levels of 23.2% (95% CI: 1.2%, 50.1%) to 37.7% (95% CI: 4.4%, 81.6%) (Table 3b). Meanwhile, recent consumption of barbecued food was associated with increases in most metabolite concentrations, particularly 2-OH-flu by 87.2% (95% CI: -13.8%, 307%)

Table 3b

Results of the Regression Analysis: Lifestyle factors (N = 469).

Predictor	n (%)	% changes in metabolite concentration				
		1-OH-nap	2-OH-nap	2-OH-flu	∑(OH-phe)	1-OH-pyr
Urinary cotinine/μg/L						
≥50	4.9	1080 (624, 1830)***	124 (24.0, 305)**	230 (78.8, 510)***	135 (79.5, 208)***	152 (78.8, 256)***
<50	95.1	0	0	0	0	0
Smoked food consumption						
yes, within 48 h	52.6	37.7 (4.4, 81.6)*	23.2 (1.2, 50.1)*	6.4 (−21.4, 44.1)	−1.8 (−13.6, 11.5)	12.4 (−3.9, 31.5)
no	47.4	0	0	0	0	0
Barbecued food consumption						
yes, within 48 h	8.0	24.1 (−16.5, 84.5)	−4.0 (−30.8, 33.2)	87.2 (−13.8, 307)	17.7 (−8.5, 51.4)	46.7 (9.6, 96.4)**
no	92.0	0	0	0	0	0
Fish consumption						
yes, within 48 h	17.2	−0.4 (−24.6, 31.6)	−16.6 (−36.1, 8.9)	−11.2 (−39.5, 30.4)	12.2 (−4.4, 31.7)	−12.4 (−26.7, 4.8)
no	82.8	0	0	0	0	0
Burger, kebab consumption/times/month						
≥4	6.7	20.5 (−15.9, 72.6)	−12.0 (−33.5, 16.4)	37.2 (−12.6, 115.3)	13.6 (−11.0, 45.0)	38.6 (−0.1, 92.3)
<4	93.3	0	0	0	0	0
Red meat consumption/times/month						
≥4	70.9	37.8 (−26.3, 158)	6.5 (−34.2, 72.5)	−29.5 (−59.1, 21.6)	−18.8 (−40.3, 10.4)	−45.8 (−59.2, −28.2)***
≥1	22.1	41.6 (−23.8, 163)	3.7 (−35.9, 67.6)	−30.8 (−60.2, 20.2)	−22.7 (−42.2, 3.3)	−42.0 (−57.4, −21.1)***
<1	7.0	0	0	0	0	0
Chocolate consumption/g/d						
≥50	5.3	168 (27.1, 465)**	32.0 (−34.2, 165)	32.6 (−60.5, 346)	44.4 (4.6, 99.4)*	1.0 (−37.9, 64.5)
25 – 50	10.4	113 (8.2, 320)*	27.5 (−26.3, 121)	48.7 (−46.5, 313)	31.4 (−3.9, 79.6)	0.9 (−37.5, 63.1)
0 – 25	81.7	111 (10.9, 303)*	33.9 (−20.4, 125)	72.5 (−33.2, 345)	37.9 (5.6, 80.1)*	8.4 (−28.2, 63.7)
0	2.6	0	0	0	0	0
Chewing on plastic objects						
yes	24.3	−8.7 (−30.8, 20.3)	2.6 (−17.2, 27.2)	2.8 (−27.5, 45.7)	0.3 (−12.6, 15.2)	20.5 (3.1, 40.9)*
no	75.7	0	0	0	0	0
Wearing plastic shoes						
yes, in summer	22.5	19.7 (−18.4, 75.6)	18.0 (−12.4, 59.0)	13.6 (−18.9, 59.2)	4.0 (−14.2, 26.2)	8.3 (−13.0, 34.9)
no	77.5	0	0	0	0	0

Significant and borderline significant results ($p \leq 0.1$) are shown in bold. The p-values for the significant results are denoted by * ($p < 0.050$), ** ($p < 0.010$) and *** ($p < 0.005$). The 95% confidence intervals are shown in brackets.

N = total sample size, n (%) = subgroup size as a % of the total sample, OH-nap = hydroxynaphthalene, OH-flu = hydroxyfluorene, ∑(OH-phen) = Sum of 1-, 2-, 3-, 4- and 9-OH-phenanthrene, OH-pyr = hydroxypyrene.

and 1-OH-pyr by 46.7% (95% CI: 9.6%, 96.4%). For participants who frequently consumed burgers and kebabs, increases were also found for 2-OH-flu by 37.2% (95% CI: −12.6%, 115.3%) and 1-OH-pyr by 38.6% (95% CI: −0.1%, 92.3%). Consumption of barbecued, grilled or smoked food types or items has also been reported as predictors of naphthalene, fluorene or pyrene metabolites for children in other publications (Alghamdi et al., 2015; Dobraca et al., 2018; John et al., 2022; Lee et al., 2007, 2009b; Liu et al., 2017; Shahsavani et al., 2017; Tabatabaei et al., 2022).

The effects of consuming red meat or fish on PAH burden were largely inconclusive. Consuming red meat (excluding sausages) at least once a month corresponded with significant decreases in 1-OH-pyr concentration of −42.0% (95% CI: −57.4%, −21.1%) – −45.8% (95% CI: −59.2%, −28.2%) (Table 3b). Though not significant, similar trends are evident for 2-OH-flu and ∑(OH-phe) concentrations, such as −22.7% (95% CI: −42.2%, 3.3%) for ∑(OH-phe), meanwhile 1-OH-nap appeared to increase e.g. by 41.6% (95% CI: −23.8%, 163%). This would suggest that the small number of participants who reported consuming red meat never or infrequently have an increased PAH burden overall, though it is not clear why. Recent consumption of fish had no notable effect on PAH burden. Few studies have identified meat or fish as predictors of PAH burden. The study of US national data reported ‘low temperature processed red meat’ consumed as a predictor of urinary pyrene, phenanthrene and fluorene metabolites for children (Jain, 2020). A study of Iranian children reported both increasing consumption of grilled food and meat separately as predictors of 2-OH-flu and 1-OH-pyr (Tabatabaei et al., 2022). Other studies did find fish or shellfish consumption to be a predictor of urinary naphthalene or pyrene metabolites (Lee et al., 2009a; Smetanová et al., 2025). In this dataset, the variables relating to grilled or smoked food may already account for a portion of the PAH exposure related to fish consumption, while the

effects of red meat consumption is likely influenced by unknown confounders and requires further investigation.

Regular consumption of chocolate corresponded with increases in naphthalene, fluorene and phenanthrene metabolite concentrations. Consuming any amount of chocolate regularly raised ∑(OH-phe) by 31.4% (95% CI: −3.9%, 79.6%) – 44.4% (95% CI: 4.6%, 99.4%) and 1-OH-nap by 111% (95% CI: 10.9%, 303%) – 168% (95% CI: 27.1%, 465%) (Table 3b). The average chocolate consumption data was also reported by the participants or their parents, making recall bias likely and differences between groups less distinct. The PAH content of numerous chocolate varieties in Germany was confirmed by Ziegenhals et al. (2009). Phenanthrene content is typically the highest for PAHs in chocolate, which could explain the increased ∑(OH-phe) concentrations in the present study (Sampaio et al., 2021). The strong effect of chocolate consumption on 1-OH nap concentrations relative to 2-OH-nap suggests carbaryl contamination (see Section 3.3). Other studies have not yet investigated links between chocolate consumption and urinary PAH metabolites to our knowledge (Murawski et al., 2020b).

Use of plastic products had a small effect on PAH burden. A habit of chewing on plastic objects (e.g. pens, temples of glasses, toys) increased urinary 1-OH-pyr concentration by 20.5% (95% CI: 3.1%, 40.9%) (Table 3b). Changes to the other metabolite levels were negligible. By comparison, wearing plastic or rubber shoes in summer resulted in non-significant increases of naphthalene and fluorene metabolites, such as 1-OH-nap by 19.7% (95% CI: −18.4%, 75.6%). No seasonal interaction term was included however, which could have ‘diluted’ the results by including winter samples. Bathing shoes available in Germany were recognized as a source of PAHs, with the detection of >100 mg/kg of naphthalene, fluorene and phenanthrene respectively in poly (vinyl chloride) (PVC) shoe samples (Kalberlah et al., 2011; UBA, 2016). The

use of plastic consumer goods has not yet been considered in other HBM studies. One study of children from Valencia (Spain) did identify consumption of plastic-packaged food in grams as a significant predictor of urinary 1-OH-pyr, $\sum(\text{OH-phe})$ and $\sum(\text{OH-flu})$ levels (Fernández et al., 2021).

Overall, 1-OH-pyr and 1-OH-nap seem to be the most strongly impacted by lifestyle factors. Smoking tobacco is the most important predictor of urinary PAH levels overall, especially for 1-OH-nap. Consuming barbecued food recently was a predictor of 1-OH-pyr concentration, while consuming chocolate regularly was a predictor of 1-OH-nap and $\sum(\text{OH-phe})$ concentrations. Minor predictors were consumption of smoked food, burgers and kebabs and chewing on plastic objects. Small group sizes for some of the variables likely reduced statistical power. Although diet is viewed as the most dominant contributor to PAH burden for non-smokers, dietary variables are less reliable as predictors due to large variation in habits and types of foods consumed, as well as the short metabolite elimination half-lives (3–11 h in adults) (Choi et al., 2023).

3.2.3. Environmental factors

The general population is exposed to airborne PAH pollution from indoor and outdoor sources, which also depends on weather conditions, ventilation measures and time spent outdoors. Naphthalene exposure and lighter PAHs are typically associated with indoor sources, while total PAH concentrations outdoors are generally greater (Jia and Batterman, 2010; Kang et al., 2012; Lovrić et al., 2024; Oliveira et al., 2017; Račić et al., 2025; Vyskocil et al., 2000). PAHs from diesel and gasoline are major constituents of traffic-related air pollution (TRAP),

particularly phenanthrene, which also contaminates roadside soils (Fu et al., 2024; Marr et al., 1999; Vyskocil et al., 2000). Burning fuels inside the home also causes large increases in PAH concentrations in air both inside and outside the home, particularly in winter (Cazier et al., 2016; Chen et al., 2018; Cirillo et al., 2006; Gustafson et al., 2008; Jung et al., 2014; Lim et al., 2022). The effects of season on PAH concentration are overall complex, but average temperature, humidity, wind and solar radiation are important factors (Amarillo and Carreras, 2016; El-Saeid et al., 2015).

The population size of the community is a predictor of urinary naphthalene, phenanthrene and pyrene metabolites. Participants residing in large cities had significantly greater concentrations of 2-OH-nap, $\sum(\text{OH-phe})$ and 1-OH-pyr in the range 21.9% (95% CI: 0.7%, 47.4%) – 56.5% (95% CI: 11.8%, 119%) (Table 3c). Participants living in small towns (<50,000) or city outskirts had increases in 1-OH-pyr of 28.0% (95% CI: 0.2%, 63.6%) – 39.8% (95% CI: 6.3%, 83.9%), while 1-OH-nap increased 49.4% (95% CI: –6.4%, 139%) for suburban residents. These results could indicate an association between population size and PAH exposure; however, for residents living in smaller cities (50,000–500,000) the effect was mostly negligible. To our knowledge this is the first examination of population size of the community on urinary PAH metabolite concentration in children or adolescents on a national scale. An older study from Duisburg, Germany, found elevated 1-OH-pyr and $\sum(\text{OH-phe})$ levels in children living in areas closer to industrial activities, which could be still be relevant to this study (Wilhelm et al., 2007). In Czech studies, urban environment was a predictor of the same metabolites and $\sum(\text{OH-Flu})$ (Smetanová et al., 2025). Numerous other studies have identified increased PAH

Table 3c
Results of the Regression Analysis: Environmental factors.

Predictor	n (%)	% changes in metabolite concentration				
		1-OH-nap	2-OH-nap	2-OH-flu	$\sum(\text{OH-phe})$	1-OH-pyr
Population size of the community.						
≥500,000	22.3	30.7 (–14.0, 98.7)	56.5 (11.8, 119)**	–1.9 (–34.4, 46.7)	21.9 (0.7, 47.4)*	44.2 (11.5, 86.5)**
50,000 – 500,000	15.5	–4.9 (–42.0, 55.8)	2.5 (–29.4, 48.7)	–22.0 (–53.3, 30.9)	2.1 (–17.9, 27.0)	20.2 (–11.0, 62.5)
20,000 – 50,000	13.3	29.3 (–24.4, 121)	–0.5 (30.9, 43.3)	23.4 (–29.2, 115.2)	3.7 (–16.9, 29.4)	39.8 (6.3, 83.9)*
city outskirts	29.3	49.4 (–6.4, 139)	25.4 (–10.5, 75.7)	15.3 (–23.3, 73.2)	6.6 (–13.0, 30.6)	28.0 (0.2, 63.6)*
<20,000	19.6	0	0	0	0	0
Road traffic rating						
extremely busy	14.5	–8.0 (–35.9, 31.9)	28.3 (–1.7, 67.6)	–0.7 (–34.1, 49.7)	27.9 (8.0, 51.4)***	12.2 (–6.7, 35.0)
busy	15.7	–0.8, (–26.7, 34.2)	11.6 (–18.2, 52.2)	11.0 (–30.5, 77.2)	14.7 (–5.9, 39.7)	12.9 (–12.3, 45.5)
moderate	26.4	15.4 (–14.3, 55.4)	–0.8 (–21.9, 26.0)	–10.5 (–37.1, 27.2)	25.9 (8.4, 46.2)***	17.1 (0.7, 36.1)*
low	43.4	0	0	0	0	0
Sampling season						
winter	51.9	–22.8 (–50.1, 19.7)	18.8 (–12.2, 60.7)	4.6 (–33.5, 64.6)	–4.5 (–21.5, 16.3)	19.0 (–8.2, 54.3)
summer	48.1	0	0	0	0	0
Heating type						
wood or fossil fuels	32.9	1.8 (–30.5, 49.2)	24.6 (–10.3, 73.2)	–44.8 (–68.9, –2.1)*	15.1 (–8.0, 44.0)	14.7 (–12.8, 50.8)
gas	47.6	0	0	0	0	0
district or renewable	19.5	66.5 (4.6, 165)*	21.8 (–14.7, 74.1)	15.5 (–22.9, 72.9)	20.3 (–9.3, 59.4)	–10.7 (–32.0, 17.3)
Fuels heating * season						
winter	-	29.8 (–24.2, 122)	–20.3 (–49.3, 25.2)	123 (16.5, 327)*	14.3 (–14.3, 52.4)	14.4 (–18.6, 60.8)
summer	-	0	0	0	0	0
District or renewable heating * season						
winter	-	–37.1 (–66.2, 17.1)	–35.6 (–61.2, 7.1)	–33.4 (–65.8, 29.6)	–20.1 (–44.0, 14.1)	13.9 (–21.6, 65.5)
summer	-	0	0	0	0	0
Heating stoves or fireplace						
yes	38.9	4.0 (–29.0, 52.3)	–9.9 (–30.3, 16.4)	64.6 (–1.1, 174)	13.4 (–5.5, 36.2)	4.8 (–15.7, 30.2)
no	61.1	0	0	0	0	0
Heating stoves or fireplace * season						
winter	-	10.9 (–32.4, 81.9)	8.9 (–26.1, 60.3)	–49.7 (–71.8, –10.5)*	–0.2 (–22.1, 27.9)	12.2 (–15.3, 48.5)
summer	-	0	0	0	0	0
Cooking device type						
wood, fossil fuels or gas	6.1	57.1 (–13.8, 186)	33.0 (–14.5, 107)	6.4 (–31.3, 64.7)	–7.1 (–22.6, 11.4)	18.4 (–16.7, 68.4)
electric	93.9	0	0	0	0	0

Significant and borderline significant results ($p \leq 0.1$) are shown in bold. The p-values for the significant results are denoted by * ($p < 0.050$), ** ($p < 0.010$) and *** ($p < 0.005$). The 95% confidence intervals are shown in brackets.

N = total sample size, n (%) = subgroup size as a % of the total sample, OH-nap = hydroxynaphthalene, OH-flu = hydroxyfluorene, $\sum(\text{OH-phen})$ = Sum of 1-, 2-, 3-, 4- and 9-OH-phenanthrene, OH-pyr = hydroxypyrene.

metabolite excretion for children in urban or industrial areas, with the majority focusing on 1-OH-pyr concentrations and a small number of locations in one region (Alghamdi et al., 2015; Dobraca et al., 2018; Fernández et al., 2021; Freire et al., 2009; Hansen et al., 2006; John et al., 2022; Lee et al., 2007, 2009a, 2009b; Liu et al., 2017; Ruchirawat et al., 2007; Schrijen et al., 2008; Tuntawiroon et al., 2007; Wang et al., 2014, 2015; Wilhelm et al., 2007). Further variables relating to local land use could help to distinguish the effects of municipality size further, though results from other studies were often inconclusive (John et al., 2022; Lee et al., 2007, 2009a, 2009b; Miri et al., 2018; Smetanová et al., 2025; van Wijnen et al., 1996).

Road traffic rating is connected with increased urinary naphthalene, phenanthrene or pyrene metabolites. A road traffic rating of 'extremely busy' corresponded with increased $\sum(\text{OH-phe})$ by 27.9% (95% CI: 8.0%, 51.4%) and 2-OH-nap by 28.3% (95% CI: -1.7%, 67.6%) compared to a 'low' road traffic rating (Table 3c). A 'moderate' road traffic rating corresponded with increased $\sum(\text{OH-phe})$ and 1-OH-pyr concentration by 25.9% (95% CI: 8.4%, 46.2%) and 17.1% (95% CI: 0.7%, 36.1%) respectively, but no notable increases were found in connection with a 'busy' road traffic rating. The results do not indicate clear trends between increasing road traffic and PAH metabolite concentration. Road traffic rating, though subjective, serves as an easy-to-obtain indicator of exposure to TRAP in the near-road zone, distinguished from background air pollution (Whaley et al., 2020). In a study from Granada (Spain), TRAP exposure was a predictor of 1-OH-pyr represented by estimated NO₂ concentration or road traffic rating (Freire et al., 2009). However, associations between estimated traffic density or distance to highways and urinary PAH metabolites were not significant in US studies (Dobraca et al., 2018; Kim et al., 2021). Other less direct indicators, such as neighborhood walkability, walking to school and use of transport are also predictors of urinary PAH metabolites in children (John et al., 2022; Miri et al., 2018; Shahsavani et al., 2017).

Sampling season, when considered independently of heating variables, may have a small effect on naphthalene and pyrene metabolite concentrations. 2-OH-nap and 1-OH-pyr levels were ca. 19% higher in samples taken in winter e.g. for 1-OH-pyr 19.0% (95% CI: -8.2%, 54.3%) (Table 3c). 1-OH-nap appears to decrease in winter but the uncertainty is large. These results are reasonable as concentrations of lighter PAHs in ambient air, such as phenanthrene and pyrene, are relatively stable over the year, which is why they are expected to correspond more with consistent vehicle emissions rather than seasonal heating emissions (Schauer et al., 2003; Schladitz et al., 2015; Szramowiat-Sala et al., 2025; Whaley et al., 2020). Small changes in PAH burden in winter could correspond with seasonal changes in time spent outdoors and frequency of opening windows. In the only central European study, therefore the most relevant geographically (Smetanová et al., 2025), winter sampling was a predictor of increased urinary 2-OH-nap and $\sum(\text{OH-phe})$ in Czech children, where heating type was not significant. Other studies in the US found spring or autumn sampling to be predictors of OH-naps or 1-OH-pyr (Dobraca et al., 2018; John et al., 2022; Morgan et al., 2015).

Household wood and fossil fuel heating is associated with increased fluorene and naphthalene metabolite concentrations. For participants with 'wood or fossil fuel' heating, 2-OH-flu concentration increased 123% (95% CI: 16.5%, 327%) and 1-OH-nap increased 29.8% (95% CI: -24.2%, 122%) in winter compared to summer (Table 3c). However, when comparing 'wood or fossil fuel' heating with gas heating over both seasons, 2-OH-flu concentration was decreased by -44.8% (95% CI: -68.9%, -2.1%). Participants who reported using heating stoves or fireplaces, had increased 2-OH-flu levels of 64.6% (95% CI: -1.1%, 174%); other metabolites did not change notably. However, 2-OH-flu levels decreased -49.7% (95% CI: -71.8%, -10.5%) with the use of fireplaces or stoves and winter sampling compared to summer sampling, which would contradict the typical use of fireplaces and stoves during the cold season. It should be noted that this variable 'use of fireplaces or

heating stoves' did not account for the type of fuel or device used, or ventilation measures, while the type or age of the house/apartment was not included in the models. Only a couple of studies have investigated burning of fuels at home as predictor of urinary metabolites in children (Freire et al., 2009; Peters et al., 2017). John et al. (2022) found that the frequency of burning wood or synthetic logs in fireplaces was not significant while frequency of candle use was a predictor of 2-OH-nap.

Household renewable or district heating is connected with increased naphthalene metabolites. For participants with district and renewable heating, urinary 1-OH-nap increased 66.5% (95% CI: 4.6%, 165%) compared to gas heating (Table 3c). OH-nap concentrations were ca. 36% lower for those with district or renewable heating in winter compared to summer, e.g. for 2-OH-nap -35.6% (95% CI: -61.2%, 7.1%). It is not assumed that these heating types increase domestic emissions at home, which could indicate confounders. Other studies examining changes in urinary PAH metabolites with gas and electric heating gave differing results, but no studies have referenced district or renewable heating types specifically (Freire et al., 2009; John et al., 2022; Smetanová et al., 2025; Sochacka-Tatara et al., 2018).

Household cooking with wood, fossil fuels or gas is associated with increased naphthalene metabolites. Participants with gas, wood or fossil fuel cooking have increased 1-OH-nap by 57.1% (95% CI: -13.8%, 186%) and 2-OH-nap by 33.0% (95% CI: -14.5%, 107%) (Table 3c). A subtle increase in 1-OH-pyr concentration is also present. In HBM studies that specifically compared gas and electric cooking devices, changes in other metabolites or no significant differences were reported (John et al., 2022; Morgan et al., 2015; Peters et al., 2017; Smetanová et al., 2025; Sochacka-Tatara et al., 2018). This would be consistent with evidence that cooking-related naphthalene emissions do not differ between electric and gas stoves (Sjaastad et al., 2010). So, the increases in naphthalene metabolites observed here may derive from the burning wood or fossil fuels, rather than gas. Other research has shown that the fumes released from cooking food contain large amounts of PAHs, meaning that the use of ventilation or extraction systems in the kitchen may be more important than cooking fuel/device type, which should be investigated further (Singh and Agarwal, 2022; Yao et al., 2015).

Overall, residing in a community of over 500,000 inhabitants and on a road with elevated traffic are the most important environmental predictors of urinary PAH metabolite levels, leading to increases in 2-OH-nap, $\sum(\text{OH-phe})$ and 1-OH-pyr. Burning wood and fossil fuels at home is also an important predictor of 2-OH-Flu and OH-naps. However, the combined effects of sampling season and household heating type are largely unclear from the models.

3.3. 1-OH-nap/2-OH-nap ratio and carbaryl exposure

In the lifestyle factors discussion (Section 3.2.2), particularly large increases in 1-OH-nap relative to 2-OH-nap were observed for the predictors: cotinine level and consumption of chocolate. The pesticide carbaryl is also excreted as 1-OH-nap in urine and can therefore inflate the total 1-OH-nap concentration (Knaak et al., 1965; Shealy et al., 1997). This can be assessed by comparing metabolite ratios among subgroups. The ratio of the volume-based geometric means 1-OH-nap/2-OH-nap for the whole GerES V sample is 0.185 (Table 4). High exposure to nicotine increased the 1-OH-nap/2-OH-nap ratio 3-fold from 0.176 to 0.530. Consuming a small or medium amount of chocolate daily increased the 1-OH-nap/2-OH-nap ratio 1.7-fold from 0.101 to average 0.173, while consuming a large amount of chocolate daily increased the ratio 3.4-fold to 0.348. In young populations outside Europe, the 1-/2-OH-nap ratio can be greater than 1 due to significant carbaryl exposure (Smetanová et al., 2025; Souza et al., 2022; Thai et al., 2020).

Although carbaryl has been banned in the EU since 2007, its use in agricultural activities worldwide means that imported food and beverage products may contain carbaryl residues (UBA, 2007; WHO, 1994). Carbaryl residues have only been detected in food products

Table 4

1-OH-nap/2-OH-nap ratio according to cotinine level and chocolate consumption.

		n	GM 1-OH-nap (µg/L)	GM 2-OH-nap (µg/L)	1-OH-nap/2-OH-nap ratio
Urinary cotinine level (µg/L) for all participants (N = 516)	>50	23	6.650	12.545	0.530
	<50	492	0.709	4.019	0.176
Chocolate consumption (g/day) for participants with low cotinine (n = 492)	>50	25	2.424	6.957	0.348
	25 - 50	52	0.699	4.181	0.167
	0 - 25	403	0.732	4.079	0.179
	0	12	0.442	4.397	0.101
	-	516	0.785	4.233	0.185

OH-nap = hydroxynaphthalene, GM = geometric mean.

from inside the EU (e.g. tomatoes in 2016) to our knowledge (EFSA, 2018). In China, currently the world's largest cultivator of tobacco, carbaryl was detected in 600 tobacco samples (FAO, 2025; Yang et al., 2014). Carbaryl use in tobacco cultivation in the US is also documented, as well as in South America and Indonesia for use in cocoa growing (Bateman and Jayne, 2023; EPA, 2020). Carbaryl can be used, for example, against cocoa pod rot disease (Gassa and Junaid, 2015).

3.4. Differences in PAH burden and exposure factors between East and West Germany

In the bivariate analysis presented in Murawski et al. (2020b), differences in the PAH metabolite concentrations were established between GerES V participants living in East and West Germany. The differences in the GMs were found to be significant for concentrations of 1-OH-nap, 2-OH-flu and $\sum(\text{OH-phe})$. In this work, mean differences in the logarithmized concentrations between East and West were analyzed with t-tests (Table S3). For East German participants, metabolite concentrations were significantly increased by 67.0% (95% CI: 24.7%, 123%) for 1-OH-nap, 35.8% (95% CI: 15.5%, 60%) for $\sum(\text{OH-phe})$ and 33.6% (95% CI: 0.1%, 78.4%) for 2-OH-flu.

The prevalence of various predictors of PAH metabolites in East and West Germany were compared using chi-squared tests. Three predictors were found to have significant differences in prevalence between East and West: chocolate consumption, smoked food consumption and residential traffic intensity (Table 5, Fig. S1a–b). Cotinine level was also identified as marginally significant. Differences in the heating-related predictors were evident and revealed that prevalence of wood or fossil-fuel heating as well as the use of stoves or fireplaces are actually greater in the West German subsample and therefore previously relevant differences in wood or fossil fuel heating are unlikely to explain differences in urinary metabolites in 2014–17.

Subgroup analyses were then performed to test which of these predictors were likely to contribute to the observed differences between East and West German participants. The differences in 1-OH-nap, $\sum(\text{OH-phe})$ and 2-OH-flu levels were calculated for the selected subgroups (Table 6). For the subgroup with low cotinine level (<50 µg/L), East German participants demonstrated higher 1-OH-nap, $\sum(\text{OH-phe})$ and 2-OH-flu concentrations, with the % difference largely unchanged from that for whole GerES V sample. For the subgroup who reported moderate chocolate consumption (0–25 g/d), the East German participants exhibited significantly higher 1-OH-nap, 2-OH-flu and $\sum(\text{OH-phe})$ levels. However, the subgroups reporting no (0 g/d) or high chocolate consumption (>50 g/d) were too small to allow for comparison. With ≤5% of participants belonging to these subgroups, the effect on the whole sample is small, suggesting that any differences in prevalence of these exposure factors are unlikely to be responsible for differences in the whole sample.

Table 5

Differences in the prevalence of selected predictors between East (n = 82) and West Germany (n = 434).

Predictor		n (%) East	n (%) West	t-test p-value
Urinary Cotinine Level	≥50 µg/L	9.3	3.6	0.076
	<50 µg/L	90.7	96.4	
Chocolate consumption*	>50 g/d	11.0	4.1	0.016
	25–50 g/d	5.1	11.6	
	0–25 g/d	79.1	82.4	
Smoked food consumption**	0 g/d	4.8	1.9	0.009
	within 48 h	65.8	49.9	
	no	34.2	50.1	
Road traffic rating*	extremely busy	24.2	12.5	0.021
	busy	16.8	15.5	
	moderate	28.8	25.9	
	low	30.1	46.1	
Heating stove or fireplace	yes	27.6	37.6	0.095
	no	72.4	62.4	
Heating type	fuels	23.2	36.7	0.118
	gas	53.6	44.4	
	district or renewable	23.2	18.9	

n = subsample size, n (%) = subgroup size as a % of the specified subsample, East = East German subgroup, West = West German subgroup.

Table 6

Subgroup Analysis - East-West differences in metabolites levels (N = 516).

Subgroup	n (%)	% increase in metabolite concentrations for East German participants		
		1-OH-nap	$\sum(\text{OH-phe})$	2-OH-flu
Cot <50 µg/L	95.4	54.7 (15.3, 107)***	38.8 (17.2, 64.4)***	29.6 (-4.6, 75.9)
		8.3 (-47.0, 121)	-16.2 (-51.0, 43.5)	25.6 (-28.8, 121)
Cot ≥50 µg/L	4.6			
0 g/d chocolate	2.4	194 (-16.4, 939)	98.5 (-5.3, 317)	-70.7 (-99.2, 1027)
0–25 g/d chocolate	81.9	82.9 (30.1, 158)*	37.0 (12.7, 66.5)**	55.1 (11.6, 116)**
25–50 g/d chocolate	10.6	49.6 (-81.3, 1,1094)	86.8 (4.8, 233)*	24.6 (-68.7, 396)
>50 g/d chocolate	5.2	-39 (-82.1, 103)	-3.2 (-35.9, 45.8)	-20.7 (-70.8, 115)
Low traffic	43.4	98.2 (12.1, 250.8)*	46.5 (12.3, 91.2)**	35.9 (-18.2, 126)
Moderate traffic	26.4	93.5 (14.7, 226)*	16.4 (-15.9, 61.1)	31.4 (-24.7, 129)
Busy traffic	15.7	104.2 (12.0, 272)*	51.6 (10.3, 108)*	-14.4 (-58.4, 75.6)
Extremely busy traffic	14.4	-1.7 (-53.0, 105)	27.9 (-9.5, 80.5)	37.4 (-19.4, 135)
No smoked food	47.6	14.6 (-32.0, 93.3)	33.2 (3.9, 71.1)*	29.9 (-23.5, 121)
Recent smoked food	52.4	93.7 (38.5, 170.4)***	37.3 (10.4, 70.7)**	37.0 (-1.6, 90.9)
GerES V sample	100	67.0 (24.7, 123)***	35.8 (15.5, 59.5)***	33.6 (0.1, 78.4)*

Significant and borderline significant results ($p \leq 0.1$) are shown in bold. The p-values for the significant results are denoted by * ($p < 0.050$), ** ($p < 0.010$) and *** ($p < 0.005$). The 95% confidence intervals are shown in brackets.

n (%) = subgroup size as a % of the total sample, OH-nap = hydroxynaphthalene, $\sum(\text{OH-phen})$ = Sum of 1-, 2-, 3-, 4- and 9-OH-phenanthrene, OH-flu = hydroxyfluorene, Cot = cotinine.

Across the subgroups with different levels of road traffic exposure, 1-OH-nap was significantly higher by 98–104% for East German participants who reported low, moderate or busy traffic (relative to 67% in the whole sample) but close to 0% for those who reported extremely busy traffic (Table 6). However, uncertainty around these estimates is high

and road traffic rating was not found to be a predictor of 1-OH-nap in the models, when controlled for the other variables. Meanwhile, moderate and extremely busy road traffic rating are highly significant predictors of $\sum(\text{OH-phe})$ ($p < 0.005$) and in both these subgroups, changes in the $\sum(\text{OH-phe})$ burden for East German participants were not significant, compared to 46-52% increase in burden for East Germans in the low or busy road traffic rating groups, a % difference fairly similar to the 36% increase in the whole sample. Since the prevalence of East Germans who reported residing on a road with moderate or extremely busy traffic was significantly greater, this could be partially responsible for differences in the phenanthrene burden observed in the whole sample.

Meanwhile smoked food consumption is in fact a significant predictor of 1-OH-nap. For the subgroup, who had not recently eaten smoked food, the difference in 1-OH-nap burden between East and West German participants was not significant while for the subgroup, who had recently consumed smoked food, East Germans participants had increased 1-OH-nap by 94% ($p < 0.0005$) relative to a 67% increase in the whole sample (Table 6). This larger increase suggests that the East German participants ate larger portions of smoked food or that the smoked food they consumed contained larger amounts of naphthalene. This result together with the greater prevalence of recently consuming smoked food for the East German participants indicates that different dietary habits concerning smoked food in East and West Germany is likely responsible for the observed difference in 1-OH-nap burden in the GerES V sample.

Regional differences between East and West Germany in terms of dietary and smoking behavior have been repeatedly discussed since German reunification. There is a consensus in the literature that due to different accessibility to consumer products during the German division, different dietary and smoking patterns emerged, which are still present today (Atzendorf et al., 2020; BMEL and Landwirtschaft, 2017; Brettschneider et al., 2021; Mensink and Beitz, 2004; RKI, 2008; Thiele and Weiss, 2003). In the Eating Study of KiGGS (EsKiMo II, 2015-17), small differences in diet between adolescent girls and boys in East and West Germany were also observed (Brettschneider et al., 2021).

3.5. Strengths and limitations

Analysis of a large nationally representative dataset of 3–17-year-olds delivers results that are not limited to a particular age group, sex or region. The inclusion of 21 diverse predictors, including demographic, lifestyle and environmental factors, across four chemically distinct PAHs, makes it possible to identify relevant exposure sources and at-risk groups. Due to the complexity of PAH exposure, it is of course not possible to consider and account for all possible confounders. Although the inclusion of a large number of variables can lead to overfitting, our primary goal was to explore and compare trends across all PAH biomarkers rather than optimize each model for predictive performance. Categorization of the predictors allows for simple interpretation and comparison of the results. Though in some cases, categorization may impact the results e.g. cotinine level with a single cut-off value and sampling season as winter or summer. Predictors were also largely based on interview data of participants or their parent/guardian, which does increase the potential for recall, social desirability and interviewer biases. The use of KiGGS Wave 2 interview data, pertaining to some dietary variables (red meat, burgers and kebabs, chocolate consumption), also did not relate directly to the weeks before sampling. The KiGGS home visit took place 2-6 months before the GerES home visit and the dietary habits of the participants could have changed in this time. Additionally, the average amount of chocolate consumed per day was estimated based on the questionnaire results, which introduces further uncertainty. In both the regression and subgroup analyses, small group numbers often gave large confidence intervals and may have weakened the significance of the results. As with all cross-sectional studies, the data cannot be used to draw any conclusions about causality. There are also no direct relationships between urinary metabolites and exposure

due to larger inter- and intra-individual variations in uptake, metabolism and excretion.

4. Conclusions and outlook

Regression analysis of the GerES V dataset identified 9 major predictors of urinary PAH concentrations, likely to be important for the German population under 18 years. These consist of systemic or chronic exposure factors: age; SES; community size; road traffic rating, as well as specific or acute exposure factors: nicotine use; wood or fossil fuel heating; and consuming chocolate, barbecued or smoked food. Some variables were investigated here for the first time as predictors of PAH burden in children and adolescents: community size, chocolate consumption, chewing on plastic objects, wearing plastic shoes in summer, and renewable or district heating. The subgroup analysis identified differences in consuming smoked food and road traffic rating as contributing to increased burdens of 1-OH-nap and $\sum(\text{OH-phe})$ for the East German participants respectively.

These results highlight the need for targeted prevention measures to protect children and adolescents from the health risks that exposure, and especially long-term exposure, to PAHs can pose. In 2024, the European Parliament and Council agreed on stricter limits for air pollutants such as particulate matter, which hopefully will reduce inhalation exposures (European Commission, 2024a). Nonetheless, extra measures should be taken to monitor air quality and reduce traffic in and around where children spend time, particularly those in large cities, lacking green spaces and with low SES. Though health guidance is available in Germany, the public should be more specifically informed of the risks of frequent chocolate consumption, barbecuing, and indoor wood or charcoal burning, particularly for young children and by extension pregnant people. This is important as current measures to encourage healthy diets are not so successful. For example, adolescents in Germany in 2015-17 consumed ca. 1.5 times the recommended daily amount of confectionary (Brettschneider et al., 2021). Research on the carbaryl and naphthalene content of chocolate is urgently needed to confirm and limit dietary exposure. Carbaryl was recently included in a call for toxicological data from the European Food Safety Authority (EFSA) to review Maximum Residue Limits (MRLs) in foods due to potential consumer risks (EFSA, 2026). Regarding plastic products, recent EU regulations to limit the carcinogenic PAH content of consumer products on the market may help to reduce PAH exposure in future (European Commission, 2021, 2025).

In future surveys or targeted studies, complementary indoor and outdoor air and house-dust measurements of the same PAHs included in this study could provide further information about inhalation and dermal exposures in the home. More details of the residence and residential area (proximity to industrial sites and green spaces); time spent inside and outside in the last 48 h; and meteorological conditions could serve as interesting variables. Evaluating exposure data for PAHs alongside metabolites of VOCs in future HBM studies could help to identify further unexpected exposure sources as well as contribute to understanding of mixtures and total exposure.

Overall, the results underline the need for permanent population-representative monitoring of the exposure of the population to PAHs. Only a follow up of exposure time trends and success of measures integrating all sources, pathways and living conditions allows for an effectful protection of the population against the impact of such harmful substances.

CRedit authorship contribution statement

Alexandra Roth: Conceptualization, Formal analysis, Investigation, Methodology, Visualization, Writing – original draft. **Artemis Saddington:** Investigation, Visualization, Writing – review & editing. **Marika Kolossa-Gehring:** Funding acquisition, Project administration, Supervision, Writing – review & editing. **Aline Murawski:**

Conceptualization, Supervision, Writing – review & editing.

Conflict of interest

The authors declare no conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ijheh.2026.114829>.

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