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Co-exposure profiles of PAHs and their derivatives in coking plant workers' serum and associations with liver function

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ABSTRACT

Coking plant workers are at great exposure risk to polycyclic aromatic hydrocarbons (PAHs) and their derivatives, with mounting evidence indicating PAH exposure being associated with liver impairment. However, impacts of co-exposure to PAHs and their derivatives on liver function remain poorly understood. Herein, serum samples were collected from coking plant workers, nearby population, and control population to study 10 PAHs, 7 methylated-PAHs (MPAHs), 17 heterocyclic PAHs (HPAHs), and 1 oxygenated-PAH (9-fluorenone). Furthermore, association between them and liver function biomarkers was explored based on quantile g-computation, weighted quantile sum regression, and Bayesian kernel machine regression models. Median levels of PAHs, MPAHs, HPAHs, and 9-fluorenone in the serum of coking plant workers were 2230, 738, 1250, and 18.8 ng/g lipid, respectively, which were significantly higher than those in other two groups. Coking process workers had higher exposure levels to PAHs and their derivatives than those in other processes. Further, results revealed positive joint effect of PAHs and their derivatives with liver function biomarkers, including total bilirubin (TBIL), indirect bilirubin (IBIL), direct bilirubin, alanine aminotransferase, and aspartate aminotransferase (AST). Notably, PAHs and their derivatives were significantly positively correlated with IBIL, TBIL and AST, with 4-methyldibenzofuran, dibenzofuran, phenanthrene, 5,6-benzoquinoline, quinoline, and indole as primary contributor. PAH derivatives had more significant effect on liver function biomarkers from a co-exposure perspective, although their concentrations were much lower than those of parent PAHs. This study emphasizes significance of PAH derivatives and contributes to better understanding the underlying mechanisms of PAH and their derivative exposure on liver function impairment.

1. Introduction

As the world's largest producer and exporter of coke, China accounts for over 70 % of global coke output (Di et al., 2024). Numerous contaminants are formed and released into the environment during coal pyrolysis into coke, and the coking industry is usually regarded as a high energy consumption and high pollution emission industry (Cheng et al., 2024; Peng et al., 2025). Polycyclic aromatic hydrocarbons (PAHs) represent the most frequently detected class of contaminants in coking plants, and chronic high-level exposure poses adverse health risks to workers (Du et al., 2020; Kuang et al., 2013; Li et al., 2023a). More important, PAH derivatives, including methylated-PAHs (MPAHs), heterocyclic PAHs (HPAHs), and oxygenated-PAHs (OPAHs) are emerging pollutants that cannot be ignored in coking plants (Cao et al., 2023; Deng et al., 2023; Zhang et al., 2023; Zhou et al., 2024b). They have attracted much attention recently owing to their higher toxicity than unsubstituted PAHs (Krzyszczak and Czech, 2021; Peng et al., 2023; Titaley et al., 2016; Wincent et al., 2015). Some metabolites of PAH derivatives, including 3-hydroxycarbazole (3-OH-CBZ) and 5-hydroxyisoquinoline (5-OH-iQNL), in the urine of coking plant workers are linked

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to lipid peroxidation and DNA damage (Li et al., 2023a). In brief, the released PAHs and their derivatives are seriously threatening the health of the workers in coking plant.

Coking plants generally consist of different process workshops with distinct pollution emission characteristics and levels (Kuang et al., 2013; Li et al., 2023a). Clarifying the emission characteristics of different processes can provide a scientific basis for coking contamination control. Internal exposure measurements based on biological samples (blood, urine, and hair, etc.) provide a more accurate assessment of human exposure levels than external exposure measurements (Ekpe et al., 2021; Li et al., 2023a). Many studies have been conducted previously regarding coking plant workers' exposure to PAHs, but most of them primarily focus on urinary PAH metabolites, and few studies on PAH derivatives are available (Fu et al., 2022; Yang et al., 2017). Comparative analysis of serum and urine samples indicates that exclusive reliance on urinary metabolites could lead to an underestimation of the actual exposure level to PAHs (Yang et al., 2021). Furthermore, some PAH derivatives may exhibit potentially higher distribution rates in the blood than their parent PAHs (Zhao et al., 2022a). However, the investigation of PAHs and their derivatives in coking plant workers' Serum in different industrial processes has scarcely been conducted.

PAHs are primarily metabolized by hepatic cytochrome P450 enzymes, with the liver serving as their main target organ (Pálešová et al., 2023). As a crucial detoxification organ in the human body, the liver may become overwhelmed by excessive PAH exposure, leading to hepatocellular injury and impaired liver function (Wang et al., 2025). Abnormal liver function significantly increases the risk of liver diseases including cirrhosis, hepatocellular carcinoma, and fatty liver (Devarbhavi et al., 2023). Liver function tests serve as an important clinical tool for the diagnosis of liver diseases (Newsome et al., 2018). Total bilirubin (TBIL), indirect bilirubin (IBIL), direct bilirubin (DBIL), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) are often used as biomarkers for diagnosing liver injury in clinical medicine (Yang et al., 2021). As a vital antioxidant in the body, bilirubin (IBIL, DBIL, and TBIL) undergoes impaired hepatic metabolism and excretion upon hepatocellular injury, leading to elevated serum bilirubin levels (Oskay et al., 2022). ALT and AST are the most common biomarkers for hepatic injury, and their release into the bloodstream following hepatocellular damage leads to elevated serum enzyme activities (Hu et al., 2021). Epidemiological studies suggest that PAH exposure may impair human liver function (Xu et al., 2021; Yu et al., 2024). Although accumulating evidence indicates that exposure to high levels of PAHs may cause liver damage, research on the impact of co-exposure to PAHs and their derivatives on the coking plant worker's liver function is limited.

In light of the current study's limitations, serum samples were collected from workers of coking plant, nearby population, and the control population. The main aims were to (1) clear the distribution characteristics of PAHs and their derivatives (MPAHs, HPAHs, and OPAHs) in serum; (2) explore variations in PAHs and their derivatives among coking plant workers at different coking industrial processes; (3) clarify the associations of PAHs and their derivatives in serum with their metabolites in paired urine; (4) investigate the associations of PAHs and their derivatives with liver function biomarkers. These findings will provide critical insights into the underlying mechanisms linking co-exposure to PAHs and their derivatives to liver impairment.

2. Materials and methods

2.1. Chemicals and reagents

The chemicals and reagents employed in the present study are described in Text S1.

2.2. Study population and sample collection

In total, serum samples were collected from 239 subjects in the occupational exposure group (workers at a typical coking plant in Shanxi, China), 162 subjects in the nonoccupational exposure group (residents residing approximately 6.2 km from the plant), and 231 subjects in the control group (residents living over 50 km away from the plant) in China, in 2020. All participants were prohibited from consuming barbecued foods and alcoholic beverages for one week prior to sampling to prevent any dietary disturbances. Workers of the occupational exposure group had been employed at the coking plant for at least one year, and no other industrial pollution sources were present in the study area. Residents of the nonoccupational exposure and control groups had not worked in occupations associated with exposure to organics, such as coking, e-waste recycling, metal smelting, petrochemicals, and other related industries, and had resided in their respective areas (free of relevant pollution sources) for at least one year. The work-related information of occupationally exposed workers is described in Text S2. The demographic information (e.g. age, gender, drinking, smoking) of the study population were collected through questionnaires and are summarized in Table S1. The work was ethically vetted by the Ethics Committee of Guangdong University of Technology, and written informed consent was obtained from all study participants. All serum samples were centrifuged within 3 h of collection, aliquoted into lyophilized tubes, and immediately stored at −80°C prior to analysis. The information of 239 paired urine samples of exposed workers was provided in our previous publication (Li et al., 2023a).

2.3. Sample preparation and instrumental analysis

Sample processing and instrumental analysis procedures of PAHs and their derivatives in serum have been described previously by us (Yue et al., 2026). That is, 1 mL of thawed serum was added to a Teflon centrifuge tube and 2.5 ng of surrogate standard (naphthalene-d8, acenaphthene-d10, phenanthrene-d10, chrysene-d12, perylene-d12, 9-fluorenone-d8, and anthraquinone-d8) was subsequently added. Then, 4 mL of ethyl acetate/acetonitrile (1:9, v/v) was added, vortexed and ultrasonicated for 10 min. The mixture was subjected to centrifugation at 4000 rpm at 4°C for 10 min and taken into the supernatant. The above procedure was carried out three times and combined the supernatant. The pooled extracts were then concentrated to 1 mL by rotary vaporization and further purified using Florisil solid phase extraction (1 g, 6 mL), and the targets were eluted with 10 mL of ethyl acetate/dichloromethane (1:1, v/v). The eluate was concentrated under a stream of pure nitrogen gas and transferred to amber glass vials, and finally reconstituted with 50 μL of isooctane and spiked with 5 ng internal standards (hexamethylbenzene, $^{13}C_6$-carbazole, and $^{13}C_{12}$-dibenzofuran) for instrumental analysis. Quantitative analysis of PAHs and PAH derivatives in serum were conducted using gas chromatography-tandem mass spectrometry (GC–MS/MS-TQ8040, Shimadzu, Japan) operated in electron ionization mode. Detailed information on instrumental analysis is introduced in Table S2 and Text S3. As PAHs and their derivatives are hydrophobic organics that predominantly accumulate in serum lipids, their measured concentrations can be influenced by total serum lipid levels. Accordingly, lipid normalization was employed in this study to minimize effects of inter-individual variations in lipid content on the evaluation of pollutant exposure levels. Total lipids (TL) concentrations in serum were quantified as follows: $TL = 1.12 \times CHOL + 1.33 \times TG + 1.48$ (g/L), where CHOL is total cholesterol and TG is triglycerides (Covaci et al., 2006; Ekpe et al., 2021).

2.4. Quality assurance and quality control

Each of 22 serum samples included a procedural blank and a spiked matrix to ensure the quality of data. The recoveries of standards in the spiked samples were 54.5 %−111 % for PAHs, 84.3 %−106 % for

MPAHs, 69.5 %−112 % for HPAHs, and 75.3 %−86.7 % for OPAHs, with relative standard deviations of 7.7 %−18.6 %, 7.9 %−15.4 %, 6.2 %−15.4 %, and 10.7 %, respectively. The limits of detection are defined as three times the signal-to-noise ratio, which was 0.011 − 0.026 ng/mL for PAHs, 0.016 − 0.028 ng/mL for MPAHs, 0.012 − 0.038 ng/mL for HPAHs, and 0.013 − 0.030 ng/mL for OPAHs. The method detection limits (MDLs) are defined as the average blank value measured in the whole procedure plus three times standard deviations (SD), or the signal level is five of the noise level, which was 0.026 − 0.320 ng/mL for PAHs, 0.085 − 0.280 ng/mL for MPAHs, 0.016 − 0.302 ng/mL for HPAHs, and 0.050 − 0.140 ng/mL for OPAHs. More details are provided in Table S3 and Text S4.

2.5. Determination of liver function biomarkers

The detection of liver function biomarkers was conducted using an automated clinical chemistry analyzer (Roche Cobas c702, Roche Diagnostics GmbH; Mannheim, Germany) (Yu et al., 2024). TBIL, IBIL, DBIL, AST, ALT and ALT/AST were chosen as liver function biomarkers. DBIL, IBIL and TBIL are routine liver function biomarkers primarily used to assess hepatic metabolic and reserve capacity, whereas ALT and AST, as liver enzymes, represent the most sensitive markers for hepatocellular injury (Liao et al., 2023). The AST/ALT ratio is usually recognized as a clinical indicator of alcoholic liver disease (Helena Nyblom et al., 2006).

2.6. Statistical analysis

Statistical analysis was performed with IBM SPSS Statistics version 26.0. Concentrations of contaminants below MDLs were taken as half of the MDLs during the statistical analysis. The Kolmogorov-Smirnov test was performed to assess the normality of PAH and their derivative concentrations. The significant differences of gender, age, smoking, and alcohol were assessed by Mann-Whitney *U* test. Further, multivariate linear regression models were applied to compare between-group differences in pollutant concentrations and liver function biomarkers, adjusted for age, gender, BMI, smoking, and alcohol consumption. Variables with a skewed distribution were log-transformed before regression analysis. Associations between PAHs and their derivatives in serum and their metabolites in urine were obtained by Spearman correlation analysis. Hierarchical cluster (HC) analysis was carried out using Origin 2021. Statistical significance was defined as *p*-value less than 0.05.

Weighted quantile sum (WQS) regression was performed to analyze associations between PAHs and their derivatives and liver function biomarkers using the R package "gWQS". WQS indices were constructed separately for each combination of the mixed exposure and liver function biomarkers, and the weights of individual PAHs and their derivatives were estimated to quantify their respective contributions to the overall mixture effect. In addition, quantile g-computation (QGC) was employed to evaluate the overall effects of PAHs and their derivatives on liver function biomarkers using the R package "qgcomp". The weights of individual components were estimated to quantify their positive and negative contributions to the mixed exposure. By combining efficiency of WQS regression with the flexibility of g-computation, QGC allows robust inference regarding unbiased mixture effects while maintaining appropriate confidence interval coverage (Keil et al., 2020). Bayesian kernel machine regression (BKMR) analysis was further conducted using the R package "bkmr". This model flexibly captures nonlinear relationships and non-additive effects (Bobb et al., 2015), and was used to estimate overall association between PAHs and their derivatives and liver function biomarkers, as well as to explore potential interactions and nonlinear dose-response relationships between individual pollutants and liver function marker levels. Key implementation details for WQS, QGC, and BKMR models are provided in Texts S5–S7. Gender, age, smoking status, and alcohol consumption were included as covariates in the WQS, QGC, and BKMR models. The effects of exposure to serum PAHs and their derivatives on liver function were examined only among coking plant workers, since if all three groups were included in the analysis, it would introduce population stratification and potentially leading to biased results (Lu et al., 2025; Wang et al., 2024). Only compounds with detection frequencies (DF) > 50 % were included in HC, WQS, QGC, and BKMR analyses. Concentrations of PAHs and PAH derivatives and liver function biomarkers were natural log-transformed for QGC, BKMR, and WQS analysis. Moreover, QGC, WQS, and BKMR analyses were conducted using R Studio version 4.4.1.

2.7. Sensitivity analysis

To further verify the robustness of our findings, sensitivity analyses were performed (Li et al., 2025). Given that lipid normalization and use of protective measures at work may confound the observed associations between exposures to PAHs and their derivatives and liver function biomarkers, TLs and workplace protective measures were additionally adjusted in the models for sensitivity analysis. The main associations in sensitivity analyses remained consistent with those from the primary analyses, further confirming the reliability of the associations between PAHs and their derivatives and liver function biomarkers.

3. Results and discussion

3.1. Characteristics of investigated participants

The demographic characteristics of the participants are provided in Table S1. The mean (±SD) ages of occupational exposure, nonoccupational exposure, and control groups were 44 ± 9.42, 57 ± 12.7, and 49 ± 12.0 years, and 77 %, 24 % and 28 % were males, respectively (unavailable data excluded). Among those exposed workers, 49 % and 38 % were smokers and alcohol drinkers, respectively. Correspondingly, more than half subjects were nonsmokers (90 % and 79 %, respectively) and nonalcohol drinkers (90 % and 90 %, respectively) in nonoccupational exposure group and control group. As the biomarkers of liver function, the mean (±SD) levels of DBIL, IBIL, TBIL, ALT, AST, and AST/ALT in the occupational exposure group were 3.44 ± 1.34, 11.4 ± 4.70, 14.8 ± 5.91 μmol/L, 31.1 ± 20.7, 26.2 ± 10.6, and 1.00 ± 0.42 U/L, respectively. There were significant differences between the occupational exposure group and the other two groups in all these biomarkers of liver function ($p < 0.05$).

3.2. Level and distribution profiles of PAHs and their derivatives in serum

Total 35 PAHs and their derivatives, including 10 PAHs, 7 MPAHs, 17 HPAHs, and 1 OPAH were detected in the serum samples from the three groups. For parent PAHs, naphthalene (Nap), acenaphthene (Ace), acenaphthylene (Acy), phenanthrene (Phe), fluorene (Flu), pyrene (Pyr), and anthracene (Ant) were detected in > 70 % of serum samples from the three groups (Table 1). Total levels of 10 PAHs in exposed workers' serum were from 305 to 7590 ng/g lipid with a median concentration of 2230 ng/g lipid, which were significantly higher than those in serum samples of the nonoccupational exposure (range: 393 − 3700 ng/g lipid; median: 1290 ng/g lipid) and control (range: 89.6 − 2320 ng/g lipid; median 788 ng/g lipid) groups ($p < 0.001$). For PAH derivatives, the median concentrations of 7 MPAHs, 17 HPAHs, and 1 OPAH in exposed workers' serum were 738 (92.1 − 2564 ng/g lipid), 1250 (275 − 4586 ng/g lipid), and 18.8 ng/g lipid (not detected (n.d.) − 101 ng/g lipid), respectively. They were significantly higher than those in serum samples of the nonoccupational exposure (median: 597, 722, and 11.8 ng/g lipid, respectively) and control (median: 431, 348, and 8.86 ng/g lipid, respectively) ($p < 0.01$) groups. The results indicate that occupational exposure results in high accumulation of PAHs, MPAHs, HPAHs, and OPAHs in the serum of coking plant workers. To better assess the exposure levels of coking plant workers, the serum PAH levels were compared with those of other occupational workers (Table S4),

Table 1
Concentrations (ng/g lipid) of PAHs, MPAHs, HPAHs, and OPAHs in serum samples of the occupational exposure, nonoccupational exposure, and control groups.

Compounds	Occupational exposure Group (n = 239)			Nonoccupational exposure Group (n = 162)			Control Group (n = 231)		
	Median	range	DF (%) [a]	Median	Range	DF (%)	Median	Range	DF (%)
10 PAHs									
NaP	1090	n.d. [b] – 4750	98.3	414	n.d. – 1750	93.2	233	n.d. – 1400	91.8
Acy	30.6	n.d. – 959	96.2	26.3	n.d. – 756	90.7	24.4	n.d. – 223	87.9
Ace	67.2	n.d. – 811	95.8	76.4	n.d. – 359	96.9	33.2	n.d. – 213	87.9
Flu	370	n.d. – 1610	99.2	280	0.03 – 958	100	139	n.d. – 383	92.7
Phe	471	n.d. – 1490	99.6	320	n.d. – 992	99.4	296	n.d. – 807	93.1
Ant	68.0	n.d. – 458	95.4	66.2	n.d. – 320	97.5	19.9	n.d. – 105	96.1
FluA	22.1	n.d. – 225	87.0	19.9	n.d. – 104	77.2	7.00	n.d. – 50.2	51.5
Pyr	29.3	n.d. – 434	80.3	29.6	n.d. – 162	72.2	6.28	n.d. – 27.2	71.4
Chr	n.d.	n.d. – 93.0	35.6	n.d.	n.d. – 97.0	39.5	n.d.	n.d. – 38.1	22.1
BaP	n.d.	n.d. – 98.0	28.5	n.d.	n.d. – 126	24.1	n.d.	n.d. – 77.1	23.4
∑PAHs	2230	305 – 7590		1290	393 – 3700		788	89.6 – 2320	
7 MPAHs									
2,7-DMNap	207	n.d. – 1410	97.1	190	n.d. – 1320	95.1	127	n.d. – 685	89.7
1,3-DMNap	87.0	n.d. – 637	95.0	86.2	n.d. – 320	90.7	55.2	n.d. – 297	87.9
1,4-DMNap	30.3	n.d. – 555	90.8	20.8	n.d. – 357	87.7	16.1	n.d. – 186	88.3
1,6,7-TMNap	102	n.d. – 493	94.1	93.8	n.d. – 456	91.4	54.3	n.d. – 161	88.3
1,4,6,7-TMNap	34.3	n.d. – 269	91.6	27.6	n.d. – 335	88.3	20.7	n.d. – 127	81.0
1-Mflu	166	n.d. – 978	97.5	147	n.d. – 575	95.1	130	n.d. – 696	88.3
3-Mcholant	16.9	n.d. – 137	92.5	11.2	n.d. – 71.2	93.8	7.56	n.d. – 58.7	78.4
∑MPAHs	738	92.1 – 2560		597	92.6 – 1860		431	57.9 – 1700	
17 HPAHs									
QL	16.0	n.d. – 280	86.6	9.78	n.d. – 234	81.5	9.02	n.d. – 79.8	71.9
IQL	6.79	n.d. – 106	70.3	n.d.	n.d. – 32.8	25.9	n.d.	n.d. – 29.2	19.5
Indole	664	n.d. – 3090	99.6	335	n.d. – 1690	92.0	170	n.d. – 715	85.7
DBF	131	n.d. – 604	96.2	124	n.d. – 672	87.0	81.5	n.d. – 220	85.3
4-MDBF	116	n.d. – 667	97.1	105	n.d. – 412	87.7	40.4	n.d. – 115	83.5
DBT	23.6	n.d. – 172	94.1	21.6	n.d. – 98.2	85.8	12.3	n.d. – 103	70.6
Acridine	23.0	n.d. – 127	76.2	11.5	n.d. – 124	58.6	8.97	n.d. – 64.9	56.3
5,6-BQL	27.8	n.d. – 209	81.6	23.0	n.d. – 182	81.5	12.7	n.d. – 36.3	61.0
CBZ	n.d.	n.d. – 85.9	40.6	n.d.	n.d. – 20.4	36.4	n.d.	n.d. – 23.1	31.6
4-MDBT	47.8	n.d. – 275	97.1	9.76	n.d. – 56.2	63.0	n.d.	n.d. – 40.8	42.4
3-MDBT	n.d.	n.d. – 64.8	20.5	n.d.	n.d. – 32.5	13.0	n.d.	n.d. – 16.8	14.7
3 M−CBZ	40.6	n.d. – 267	91.2	n.d.	n.d. – 140	49.4	n.d.	n.d. – 59.4	38.5
4,6-DBT	n.d.	n.d. – 41.7	25.1	n.d.	n.d. – 45.9	19.8	n.d.	n.d. – 20.8	15.6
1,8-DMCBZ	3.19	n.d. – 56.0	61.5	n.d.	n.d. – 53.6	39.5	n.d.	n.d. – 21.4	28.6
2,8-DMDBT	n.d.	n.d. – 85.1	36.4	n.d.	n.d. – 74.9	19.1	n.d.	n.d. – 21.2	15.2
1,4-DMCBZ	n.d.	n.d. – 52.7	25.5	n.d.	n.d. – 44.2	20.4	n.d.	n.d. – 61.6	14.7
7-MB[b]nap[2,3-d] T	n.d.	n.d. – 75.8	21.3	n.d.	n.d. – 71.7	15.4	n.d.	n.d. – 26.8	13.0
∑HPAHs	1250	275 – 4590		722	161 – 2190		348	37.6 – 889	
1 OPAH									
9-Fluorenone	18.8	n.d. – 101	79.5	11.8	n.d. – 108	72.8	8.86	n.d. – 36.7	66.2

[a] DF: detection frequencies.
[b] n.d.: not detected.

since no study has reported PAH, MPAH, HPAH, and OPAH levels in coking plant workers' serum samples. The PAH levels in the serum of coking plant workers in this study were higher than that in the serum of aluminum smelter workers in eastern China (Zhao et al., 2024), male coal-fired power plant workers in Shandong, China (Zhao et al., 2022a), and firefighters in Korea (Ekpe et al., 2021), but lower than that in the serum of female coal-fired power plant workers in Shandong, China (Zhao et al., 2022a). This suggests that coking plant workers are generally exposed to higher levels of PAHs than other occupational workers.

In sum, the concentrations of PAHs, MPAHs, HPAHs, and OPAHs were highest in the occupational exposure group, followed by the nonoccupational exposure group, and lowest in the control group. PAHs and their derivatives can be emitted from coking industries, spread to surrounding areas through ambient air diffusion (Deng et al., 2023), and subsequently enter the human body via the respiratory tract, resulting in higher exposure levels among the nonoccupational exposure group than the control group. Thus, PAHs and their derivatives detected in the serum of the three groups reflected the levels of coking-related exposure, the effect of coking industry on the surrounding environment and the background levels, respectively. Furthermore, the mean concentrations and standard deviations of PAHs (2476 ± 1291 ng/g lipid), MPAHs (864 ± 494 ng/g lipid), HPAHs (1376 ± 669 ng/g lipid), and OPAHs (20.5 ± 17.2 ng/g lipid) in the serum of coking plant workers were also significantly higher than those in nonoccupational exposure group and control group. This may be related to the large differences in exposure levels among different process workshops (Kuang et al., 2013; Li et al., 2023a).

The distribution characteristics of PAHs and their derivatives were further analyzed in the serum of three groups. Since only 9-fluorenone was detected in OPAHs, thus it was excluded from the discussion on OPAH distribution profiles. The distribution characteristics of PAHs in serum of the occupational exposure group were similar to those of the nonoccupational exposure group, but significantly differed from those of the control group (Fig. S1). Among PAH isomers detected, Nap (51 %) was the dominant PAHs in exposed workers' serum, followed by Phe (22 %) and Flu (17 %). This could be due to low-molecular-weight PAHs exhibiting higher solubility and volatility compared with high-molecular-weight PAHs, thereby facilitating their greater

accumulation in serum (Ekpe et al., 2021; Singh et al., 2007). Similar to occupational exposure group, NaP, Phe, and Flu were the dominant PAHs in the serum of nonoccupational exposure group, with a consistent order of proportions: NaP (34 %) > Phe (26 %) > Flu (23 %). The results further confirmed the effect of the coking industry on the surrounding environment. In contrast, Phe (39 %) was the most abundant PAH in the serum of the control group, followed by Nap (31 %) and Flu (18 %), which may be influenced by anthropogenic activities, such as transportation emissions, wood combustion, and food cooking (Lao et al., 2018; Peng et al., 2023; Shen et al., 2012).

For PAH derivatives, there was no significant differences in the distributions of MPAHs among three groups of serum (Fig. S2). The contribution ratios of the primary substances showed consistent ordering between the occupational and non-occupational exposure groups. Specifically, 2,7-dimethylnaphthalene (2,7-DMNap) (32 % and 31 %, respectively) exhibited the highest contribution in serum of occupational and nonoccupational exposure groups, followed by 1-methylfluorene (1-Mflu) (26 % and 26 %, respectively), 1,6,7-trimethylnaphthalene (1,6,7-TMNap) (16 % and 16 %, respectively), and 1,3-dimethylnaphthalene (1,3-DMNap) (15 % and 14 %, respectively). Differently, 1-Mflu (32 %) was the most abundant MPAHs in serum of control group, followed by 2,7-DMNap (31 %). This may be related to the significant abundance of methylfluorene in residential coal combustion (Cui et al., 2022a; Cui et al., 2022b). For HPAHs, the dominant substances in serum of three groups and their contribution proportions showed the same order: indole > dibenzofuran (DBF) > 4-methyldibenzofuran (4-MDBF) (Fig. S3). However, proportion of indole in occupational exposure group (60 %) was higher than that in nonoccupational exposure group (52 %) and control group (51 %), while proportion of DBF in control group (24 %) was higher than that in occupational (12 %) and nonoccupational (19 %) exposure groups. The difference may relate to distinct exposure sources: occupational exposure group is primarily linked to coking activities, while control group is primarily exposed to nearby anthropogenic activities (Li et al., 2023a).

3.3. Variations in PAHs and their derivatives among workers worked at different coking processes

Based on the above results, the levels of PAHs and their derivatives in the occupational exposure group exhibited higher variability than the other two groups, which may be attributed to different job categories among coking plant workers. Therefore, the variations of PAHs and their derivatives in exposed workers' serum from different processes were investigated. Exposed workers were stratified into two indirect exposure groups (maintenance as well as control and microcomputer rooms) and three direct exposure groups (coking, coal preparation, and chemical production) based on the type of operation. Workers in direct exposure groups have direct contact with various pollutants, whereas those in indirect exposure groups only experiences brief direct contact during equipment maintenance or repair.

Firstly, HC analysis was employed to assess the similarity of PAHs and their derivatives with DF > 50 % in coking plant workers' serum (Zhao et al., 2022a). Five clusters were divided based on correlation and distance (Fig. S4), i.e., the contaminants were categorized into five groups (Group 1 − 5). Specifically, Nap, 2,7-DMNap, indole, 9-fluorenone, Flu, Phe, 1-Mflu, DBF, fluoranthene (FluA), Pyr, 1,3-DMNap, 1,6,7-TMNap, 1,4,6,7-tetramethylnaphthalene (1,4,6,7-TMNap), and Ant were divided into Group 1; Acy was divided into Group 2; Ace, quinoline (QL), isoquinoline (IQL), acridine, 3-methylcarbazole (3-MCBZ), 4-MDBF, 4-methyldibenzothiophene (4-MDBT), and 1,8-dimethylcarbazole (1,8-DMCBZ) were divided into Group 3; 1,4-dimethylnaphthalene (1,4-DMNap) and 3-methylcholanthrene (3-Mcholant) were divided into Group 4, and 5,6-benzoquinoline (5,6-BQL) was divided into Group 5. As Fig. S5 shows, contaminant concentrations in direct exposure groups (median: 4400, 59.8, and 393 ng/g lipid, respectively) were significantly higher than those in indirect exposure groups (median: 2803, 21.8, and 324 ng/g lipid, respectively) for Groups 1 − 3 (Group 1: $p < 0.001$, Group 2: $p < 0.05$, and Group 3: $p < 0.01$). For Groups 4 and 5, contaminant concentrations were 1.2- and 1.5-time higher in direct exposure groups (median: 43.4 and 27.2 ng/g lipid) than those in indirect exposure groups (median: 35.2 and 18.3 ng/g lipid), but no statistically significant differences were detected ($p > 0.05$). The result demonstrates that contaminants in Groups 1 − 3 primarily originated from direct exposure groups and contaminants in Groups 4 and 5 simultaneously originated from direct and indirect exposure groups. Overall, the contaminant concentrations were higher in direct exposure groups than indirect exposure groups, which can be attributed to direct exposure workers being directly exposed to contaminants, whereas workers in indirect exposure groups are exposed to contaminants only when they are maintaining equipment in the site (Li et al., 2023a).

Additionally, the levels of PAHs and their derivatives in exposed workers' serum from different processes were also compared (Fig. S6). In Group 1, contaminant concentrations in coking process workers (median: 182 ng/g lipid) were significantly higher than those in other processes ($p < 0.001$), which may be high levels of PAHs and their derivatives produced by the cracking of coal briquettes at high temperatures during coking (Li et al., 2021). A significant difference of contaminants in Group 2 was found only between coking process (median: 72.2 ng/g lipid) and the control and microcomputer rooms (median: 16.9 ng/g lipid) ($p < 0.001$). Similar to Group 1, contaminant concentrations in coking process workers (median: 32.6 ng/g lipid) were significantly higher than those in other processes ($p < 0.01$), indicating contaminants in Group 3 primarily from coking process. Furthermore, comparable concentrations of contaminants in Group 4 were found in workers from coking, coal preparation, and maintenance processes (median: 21.4, 19.4, and 17.9 ng/g lipid, respectively), which were 1.40 − 1.76 times greater than those in chemical production process and the control and microcomputer rooms. Differently, 5,6-BQL in Group 5 showed similar levels in exposed workers from chemical production and coal preparation (median: 34.4 and 30.6 ng/g lipid), which were 1.21 − 2.02 times higher than those in other processes. In sum, coking process workers had the highest concentrations of PAHs and their derivatives in Groups 1 − 4 and chemical production workers showed the highest levels of 5,6-BQL.

The coking process is further divided into 11 different job categories, and concentrations of PAHs and their derivatives exposed workers' serum from these categories are shown in Fig. S7. Furnace door operators and coke oven workers generally had high levels of PAHs and their derivatives in each group, which may be due to pollutant emissions primarily occurring during the coking coal charging and hot coke pushing processes (He et al., 2015; Li et al., 2023a).

3.4. Associations between PAHs and their derivatives in serum and their metabolites in paired urine of coking plant workers

Apart from the unmetabolized PAHs and their derivatives in serum, the analysis of urinary metabolites is one of the assessment methods of PAH and their derivative exposure in humans. The concurrent investigation of unmetabolized PAHs in serum and their metabolites in urine facilitates accurate assessment of PAH exposure levels and health risks (Ekpe et al., 2021; Yang et al., 2021). Therefore, the exposure concentrations of PAHs and their derivatives were comparatively analyzed from both serum and urine perspectives, and the correlation between PAHs and their derivatives in the serum and their metabolites in paired urine was further explored.

The concentrations of PAHs and their derivative metabolites in coking plant workers' urine samples have been analyzed in our previous study (Li et al., 2023a; Li et al., 2023b). The detailed information was listed in Table S5. Among the hydroxy-PAHs (OH-PAHs) analyzed, 2-hydroxynaphthalene exhibited the highest median concentration (19.5 μg/g Cr) in urine, followed by 1-hydroxynaphthalene (16.2 μg/g Cr), 2-

hydroxyfluorene (8.12 μg/g Cr), and 1-hydroxypyrene (1-OH-Pyr) (4.20 μg/g Cr). Notably, 1-OH-Pyr exhibited a relatively high concentration in urine, while its parent compound, Pyr, remained at a relatively low level (29.3 ng/g lipid) in serum compared to other PAH isomers (Table 1). Furthermore, Phe exhibited a relatively high concentration (471 ng/g lipid) in serum, whereas its metabolite, hydroxyphenanthrene (OH-Phe, sum of 2/3-, 4-, and 1/9- OH-Phe), exhibited a comparatively low concentration (2.86 μg/g Cr) in urine. The difference in exposure profiles between OH-PAHs in urine and PAHs in serum may be primarily attributed to the various metabolic rates of PAH isomers (Yang et al., 2021). In terms of HPAH exposure levels, indole had the highest concentrations in serum (median: 664 ng/g lipid), 3- and 4-hydroxyindole, metabolites of indole, were detected in the urine of these workers, but at very low concentrations (median: 0.28 and 0.59 ng/mL, respectively) (Li et al., 2023b). It is worth noting that L-tryptophan, an essential amino acid, can be metabolized into indole after being absorbed by the body (Paats et al., 2020; Wu et al., 2023; Zhao et al., 2022b). This may also contribute to high concentrations of indole in the serum. Further study on the sources of indole and the metabolic pathway in humans is needed. Moreover, IQL concentrations were low (median: 6.79 ng/g lipid) in serum, whereas high concentration of 5-OH-iQNL (8.22 μg/g Cr), a metabolite of IQL, was detected in the urine of these workers (Li et al., 2023a). Studies on the metabolic transformation of IQL after entry into humans are still very limited and need to be further explored. The result indicates that the assessment based solely on serum or urine samples might underestimate PAH and HPAH exposure levels.

After absorption by human body, PAHs and their derivatives are transported via the systemic circulation to the liver, where they undergo biotransformation to form hydroxylated metabolites ultimately excreted in urine (Peng et al., 2023). PAHs and their derivatives in serum are the primary precursors of urinary hydroxylated metabolites, to clarify their correlation, Spearman correlation analysis was performed between PAHs and HPAHs in serum and their corresponding metabolites in paired urine samples. As Fig. S8 and Table S6 show, significant correlations were found between Nap and ∑OH-NaP ($r_s = 0.174$, $p < 0.05$), Phe and ∑OH-Phe ($r_s = 0.147$, $p < 0.05$), Pyr and 1-OH-Pyr ($r_s = 0.227$, $p < 0.05$), indicating that these hydroxyl metabolites of PAHs with low molecular weight are mainly excreted via urine. In contrast, there was no significant correlation between Flu and ∑OH-Flu (sum of 2-hydroxyfluorene and 3-hydroxyfluorene) ($p > 0.05$), which may be attributed to the unmeasured common urinary hydroxy metabolites of Flu, such as 9-hydroxyfluorene (Kuang et al., 2013; Yang et al., 2017). For HPAHs, no significant correlation was observed between serum levels of IQL, DBF and carbazole (CBZ) and urinary concentrations of their metabolites, 5-OH-iQNL, 2-OH-DBF and 3-OH-CBZ ($p > 0.05$). In terms of IQL and DBF, in addition to 5-OH-iQNL and 2-OH-DBF, they have other major metabolites such as 2-OH-iQNL and 1-OH-DBF (Li et al., 2023b). However, these metabolites were excluded in the quantitative analysis in the current work due to lacking standard reference materials, which may result in no significant correlation being observed. The low detection rate of CBZ (40.6 %) in serum may be the primary factor accounting for the lack of significant correlation between CBZ and its metabolite 3-OH-CBZ. Furthermore, substantial individual variations exist in the catalytic activities and expression levels of human xenobiotic-metabolizing enzymes, leading to divergent metabolic processes and transformation profiles of PAHs and their derivatives in vivo (Shimada, 2006). This may constitute another important explanation for the lack of a significant correlation between serum levels of parent PAHs and their derivatives and urinary levels of their corresponding metabolites. In sum, although the levels of serum PAHs and urinary OH-PAH exhibited a statistically significant yet weak correlation, and HPAH concentrations in serum showed no significant correlation with OH-HPAH concentrations in urine. These findings suggest that urinary OH-PAH and OH-HPAH levels in coking workers may not reliably reflect their corresponding serum PAH and HPAH levels. This emphasizes the necessity of simultaneous analysis of serum and urine samples.

3.5. Associations of PAHs and their derivatives in serum with liver function biomarkers

PAH exposure is known to impair liver function (Yu et al., 2024; Zhou et al., 2024a), but the specific effects of co-exposure to PAHs and their derivatives on liver function impairment remain unclear. To better understand effects of PAH and their derivative exposure on liver function, associations between PAHs and their derivatives and liver function biomarkers (IBIL, DBIL, TBIL, ALT, AST, and AST/ALT) were explored using WQS, GGC, and BKMR models in this study. Confounding factors including age, gender, smoking, and alcohol consumption were adjusted during analysis to eliminate their potential impacts on serum levels of PAHs and their derivatives (although not all of these factors exhibited statistically significant influence) (Text S8 and Figs. S9–S12), thus enabling a more precise evaluation of independent associations between these pollutants and liver function biomarkers. In WQS regression models, co-exposure to PAHs and their derivatives was significantly positively associated with DBIL ($\beta = 0.032$, 95 % confidence interval (CI): 0.026 − 0.038), IBIL ($\beta = 0.037$, 95 % CI: 0.028 − 0.045), TBIL ($\beta = 0.033$, 95 % CI: 0.026 − 0.040), ALT ($\beta = 0.033$, 95 % CI: 0.020 − 0.046), and AST ($\beta = 0.019$, 95 % CI: 0.011 − 0.027), but not significantly associated with AST/ALT (Fig. 1), indicating that mixed exposure plays a important role in liver function. For weights of individual PAHs and their derivatives contributing to the overall joint effect on liver function biomarkers, Ant showed the highest weight in relation to DBIL (weight: 34 %) and TBIL (20 %), 5,6-BQL (25 %) exhibited the highest weight for IBIL, while indole contributed the highest weight to ALT (26 %) and AST (23 %), underscoring their potential impact on liver function.

However, WQS regression may be prone to amplified bias due to unmeasured confounding (Keil et al., 2020). Thus, our findings were further validated using QGC model. First, the joint effects of PAHs and their derivatives on liver function biomarkers were explored via QGC, the regression coefficients for three scenarios (Models 1 − 3, Model 1: unadjusted; Model 2: adjusted for age, gender; Model 3: adjusted for age, gender, smoking, and alcohol consumption) are presented in Table 2. The exposure concentrations of PAHs and their derivatives showed significant positive correlations with IBIL and TBIL ($p < 0.05$), but not with AST/ALT ($p > 0.05$) in all models. In contrast, no significant correlation was observed between ALT and AST with PAHs and their derivatives in Models 1 and 2 ($p > 0.05$). However, a significant positive correlation was observed after adjusting for gender, age, alcohol consumption, and smoking ($p < 0.05$), with the correlation coefficients increasing from 4.77 and 5.41 to 7.06, and 6.23, respectively. These suggest that smoking and alcohol consumption are key factors influencing PAHs and their derivatives on ALT and AST. Consistent with the WQS model, in fully adjusted model, exposure to PAHs and their derivatives was significantly associated with DBIL, IBIL, TBIL, ALT, and AST ($p < 0.05$), with a β 0.576 (95 % CI: 0.271, 0.881), 1.261 (0.196, 2.327), 1.837 (0.503, 3.171), 7.06 (1.49, 12.6), and 6.23 (0.351, 12.1), respectively. This indicates that quantile increases in exposure to the mixture of PAHs and their derivatives positively correlated with liver function indicators, and long-term co-exposure to these compounds may increase risk of liver function damage. Moreover, the weights of individual compounds in mixture on liver function biomarkers were explored (Table 2). No significant correlation was found between the mixture of PAHs and PAH derivatives and AST/ALT in all models, which will not discuss further. Among the examined compounds, Ant (19 %), FluA (14 %), Pyr (13 %), and DBF (10 %) were the main contributors to the positive effects of DBIL; Phe (13 % and 11 %), 4-MDBF (13 % and 13 %), DBF (11 % and 13 %), and 5,6-BQL (10 % and 9 %) were significant in IBIL and TBIL, while indole (25 % and 24 %) was the dominant contributor to ALT and AST (Fig. 2). The results clearly indicate that PAH derivatives exert more pronounced damaging effect on liver function than their parent PAHs.

Further, the associations of PAHs and their derivatives with liver

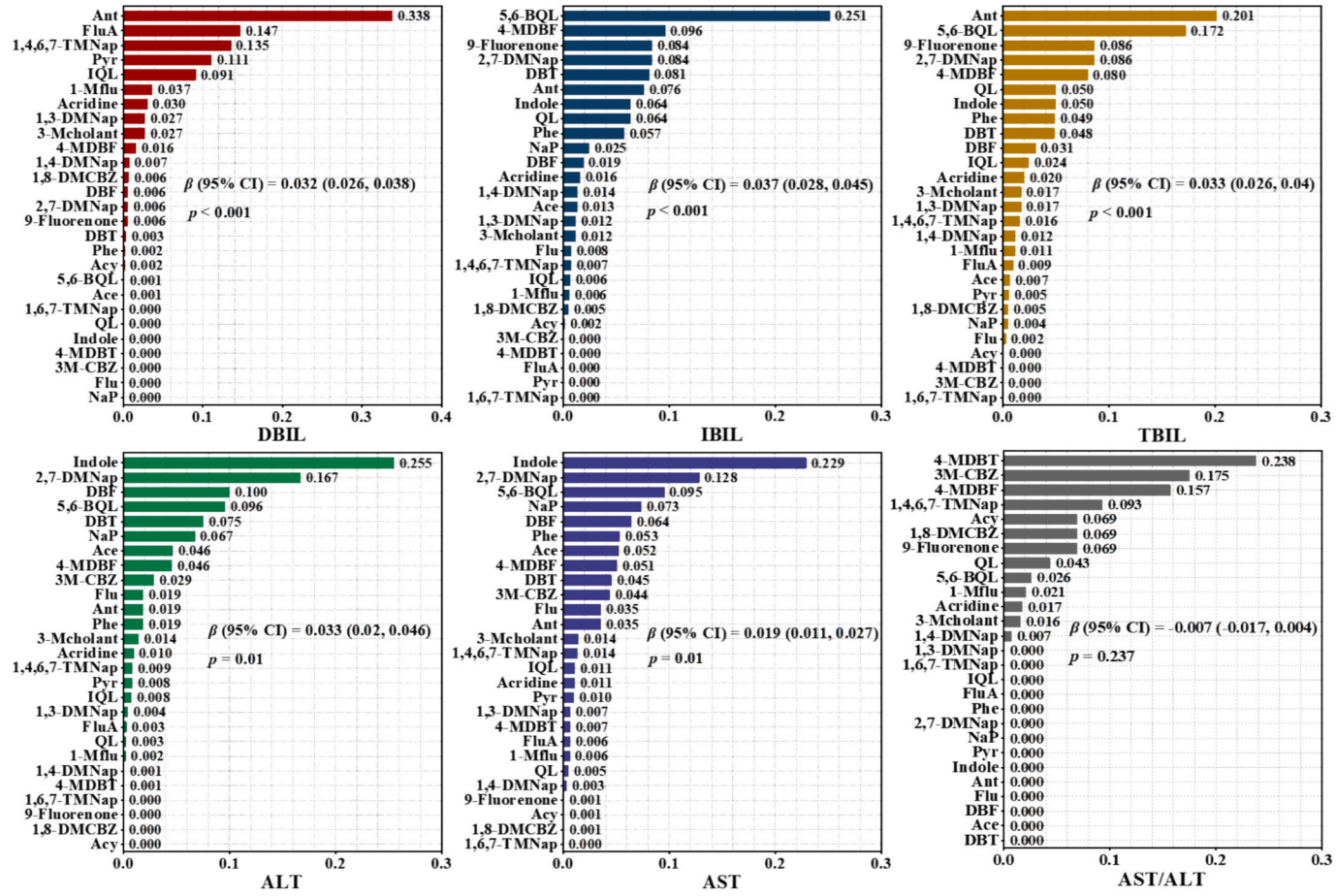

Fig. 1. Weights of individual PAHs and their derivatives contributing to the overall joint effect on liver function biomarkers (DBIL, IBIL, TBIL, ALT, AST, AST/ALT) in the WQS regression model (adjusted for age, gender, smoking, and drinking). The overall β coefficient (95 % credible intervals) and p-value for the WQS mixture index reflect the strength and statistical significance of the overall association between mixed PAH exposure and the corresponding liver function biomarker.

Table 2
Regression coefficients (β, 95 % CI) for the associations of PAHs and their derivatives with liver function biomarkers in exposed workers by using quantile g-computation model.

	Model 1 [a]		Model 2 [b]		Model 3 [c]	
	β 95 % CI	p-value	β 95 % CI	p-value	β 95 % CI	p-value
DBIL	0.560 (0.291, 0.829)	0.0001*	0.502 (0.222, 0.782)	0.0635	0.576 (0.271, 0.881)	0.0002*
IBIL	1.24 (0.302, 2.189)	0.0101*	1.10 (0.116, 2.09)	0.0292*	1.261 (0.196, 2.327)	0.0209*
TBIL	1.81 (0.624, 2.99)	0.0029*	1.60 (0.371, 2.84)	0.0112*	1.837 (0.503, 3.171)	0.0073*
ALT	4.77 (−1.43, 11.0)	0.1323	5.71 (−0.047, 11.5)	0.0527	7.06 (1.49, 12.6)	0.0135*
AST	5.41 (−0.911, 11.7)	0.0943	5.12 (−0.551, 10.8)	0.0777	6.23 (0.351, 12.1)	0.0385*
AST/ALT	0.045 (−0.064, 0.112)	0.4159	0.030 (−0.060, 0.119)	0.5167	0.042 (−0.054, 0.138)	0.3957

[a] Model 1: unadjusted.
[b] Model 2: adjusted for age, gender.
[c] Model 3: adjusted for age, gender, smoking, and alcohol consumption

function biomarkers were estimated using the BKMR model (Fig. 3). The joint effects of PAHs and their derivatives exhibited a positive overall association with IBIL, TBIL, and AST, which were consistent with those from WQS and QGC models. Notably, the percentile concentrations of serum PAHs and their derivatives exhibited a statistically significant association with both IBIL and TBIL levels. This suggests that exposure to PAHs and their derivatives may induce accumulation of IBIL and TBIL in body, thereby potentially compromising liver function. Further, the individual effects of serum PAHs and their derivatives on liver function biomarkers were explored via BKMR. Distinct from results from QGC and WQS models, QL yielded the most notable contributions to IBIL and TBIL, yet exerted no impact on DBIL (Figs. S13–S15). This discrepancy can be ascribed to numerous factors associated with each respective model (Keil et al., 2020). Consistent with QGC and WQS models, indole also served as the most significant contributor to ALT and AST in BKMR analyses, presenting a positive correlation (Figs. S16 and S17). Furthermore, univariate dose–response curves were plotted to explore the associations between individual contaminants and liver function biomarkers. QL concentration correlated positively with IBIL and TBIL and indole concentration with ALT and AST (sS23). In addition, the posterior inclusion probability (PIP) illustrated impact of exposure variables on the results. The PIP values of QL for IBIL and TBIL were 0.741 and 0.770, respectively, and the PIP values of indole for ALT and AST were 0.874 and 0.798, respectively (Table S7). These further

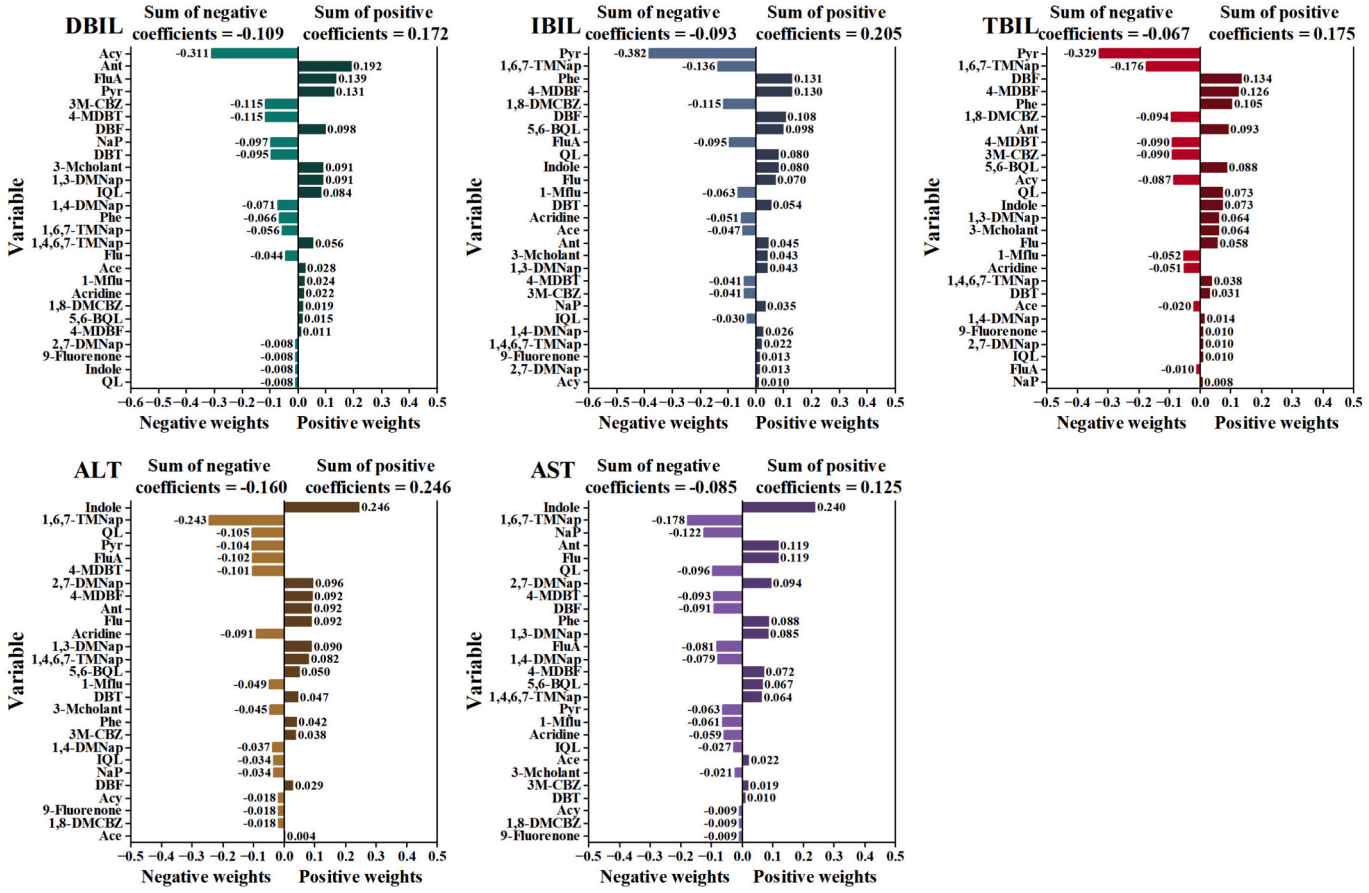

Fig. 2. Weights for the proportion of positive and negative effects of each PAH and their derivatives on liver function biomarkers (DBIL, IBIL, TBIL, ALT, AST) in the QGC model (adjusted for age, gender, smoking, and drinking). The values at the top of each subplot denote the sum of all negative coefficients and positive coefficients, respectively, reflecting the overall direction and magnitude of the mixture effect.

demonstrates their significant contributions to liver function biomarkers.

Herein, positive associations between PAHs and their derivatives and IBIL, TBIL, and AST were identified using WQS, QGC, and BKMR models. Bilirubin, the terminal metabolite of heme degradation, serves as a crucial biomarker for assessing liver function (Liao et al., 2023). TBIL is composed of both conjugated (DBIL) and unconjugated (IBIL) bilirubin fractions. Conjugated hyperbilirubinemia is mostly related to hepatocellular injury or biliary tract obstruction, while unconjugated hyperbilirubinemia is associated with impaired hepatic conjugation or hemolysis (Gazzin et al., 2016). The elevation of serum bilirubin levels induced by PAH and PAH derivative exposure may be explained by the following mechanism: bilirubin acts as a potent antioxidant to counteract exposure-related oxidative stress, thereby further inducing heme oxygenase expression (Pálešová et al., 2023). As classic biomarkers of liver function, ALT and AST are present at extremely low levels in serum under physiological conditions; they are released into blood in large quantities when liver is damaged, resulting in significantly elevated serum concentrations (Hu et al., 2021; Xu et al., 2021). In this study, exposure to PAHs and their derivatives was significantly positively associated with AST levels in WQS and QGC models, and showed a similar positive association in BKMR model, although it did not reach statistical significance. These findings indicate that exposure to PAHs and their derivatives may link to liver function damage and pose potential adverse effects among coking-exposed populations. Previous epidemiological studies have also demonstrated that PAH exposure may impair human liver function (Xu et al., 2021; Yu et al., 2024; Zhou et al., 2024a). Collectively, comprehensive analyses using the WQS, QGC, and BKMR models suggest that co-exposure to PAHs and their derivatives was associated with elevated levels of IBIL, TBIL, and AST. Although parent PAHs exhibited high concentrations, they were not the primary contributors to liver function biomarkers from the perspective of co-exposure. For example, 4-MDBF, DBF, QL, and indole contributed significantly to the increased levels of IBIL, TBIL, and AST. This indicates that focusing solely on parent PAHs cannot fully reveal the internal relationship between pollutant exposure and liver function in coking plant workers.

3.6. *Results of sensitivity analysis*

After adjusting for TLs and workplace protective measures, the associations between PAHs and their derivatives and liver function biomarkers remained robust. Effect sizes and key contributor rankings changed minimally, and the direction and significance of joint effects were consistent with the primary analysis (Tables S8 and S9). These findings suggest that TLs and workplace protective measures exerted minimal confounding influence, further confirming the robustness of our results.

4. Strengths and limitations

This study is the first to systematically characterize the co-exposure profiles of PAHs and their derivatives (MPAHs, HPAHs, and 9-fluorenone) in the serum of coking plant workers, and their associations with liver function biomarkers. By employing multiple models (e.g., QGC, WQS, and BKMR), the joint effects and key constituent-specific contributions of these pollutants were evaluated and the dominant role of PAH derivatives in influencing liver function biomarkers was

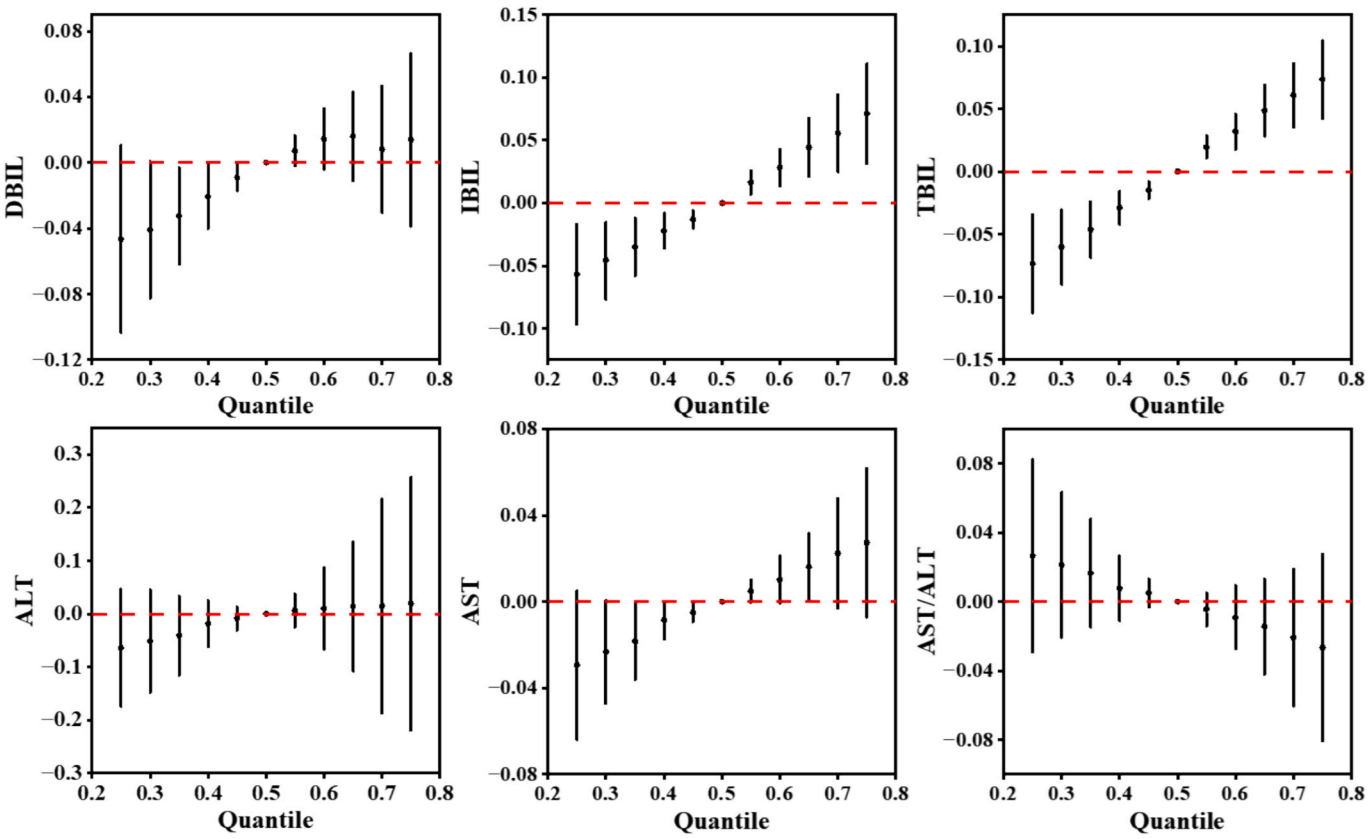

Fig. 3. Overall effect on liver function biomarkers (DBIL, IBIL, TBIL, ALT, AST, AST/ALT) of each 5-quantile increase in PAHs and their derivative mixture concentrations from the 25th to 75th quantile relative to the median by the BKMR model (adjusted for age, gender, smoking, and drinking). Black circle indicates effect estimates, red dotted lines represent the null, and black vertical lines represent 95 % credible intervals (CIs). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

validated. These findings address critical knowledge gaps regarding the occupational internal exposure of PAH derivatives, and provide important scientific evidence for occupational health risk management in the coking industry. Nevertheless, several limitations should be noted. First, this study can only demonstrate an association between exposure to PAHs and their derivatives and liver dysfunction, but cannot establish a definitive causal relationship. Second, the limited sample size may affect stability and reliability of some statistical models. Third, potential confounders including household cooking practices, medical history, and employment duration were not fully adjusted, which may result in residual confounding. Thus, large-sample prospective cohort studies combined with mechanistic toxicological research are warranted to validate our findings and enhance causal inference.

5. Conclusions

This study systematically characterized the exposure levels and compositional profiles of PAHs, MPAHs, HPAHs, and OPAHs in serum, explored exposure disparities across workers involved in different coking processes, and further elucidated the associations of these pollutants with their corresponding urinary metabolites and liver function biomarkers. From a combined co-exposure perspective, PAH derivatives exerted a more significant influence on liver function biomarkers than parent PAHs. The current work is study offers fresh light on the occupational exposure risks of coking plant workers and emphasizes the importance of comprehensively assessing the health impacts of exposure to PAHs and their derivatives.

6. Statement

This work has received approval for research ethics from Guangdong University of Technology and a proof/certificate of approval is available upon request.

CRediT authorship contribution statement

Congcong Yue: Writing – original draft, Methodology, Investigation, Formal analysis. **Hailing Li:** Visualization, Validation, Methodology. **Chang He:** Methodology, Data curation. **Yao Tang:** Visualization, Validation. **Guiying Li:** Writing – review & editing, Visualization. **Shengtao Ma:** Methodology. **Xin Zhang:** Resources. **Taicheng An:** Writing – review & editing, Supervision, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2026.110291.

Data availability

No data was used for the research described in the article.

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