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Lulu Huang†, Zhengyang Yuan†, Xue Cao, Lili Yao, Zulan Zhao, Li Li, Bairui Chen, Xinzhuo Hao, Chun Xie* and Qilong Liao*

†These authors contributed equally to this work.

***E-mails:** 1009207189@qq.com (C.X.); liaoqilong@scies.org (Q.L.)

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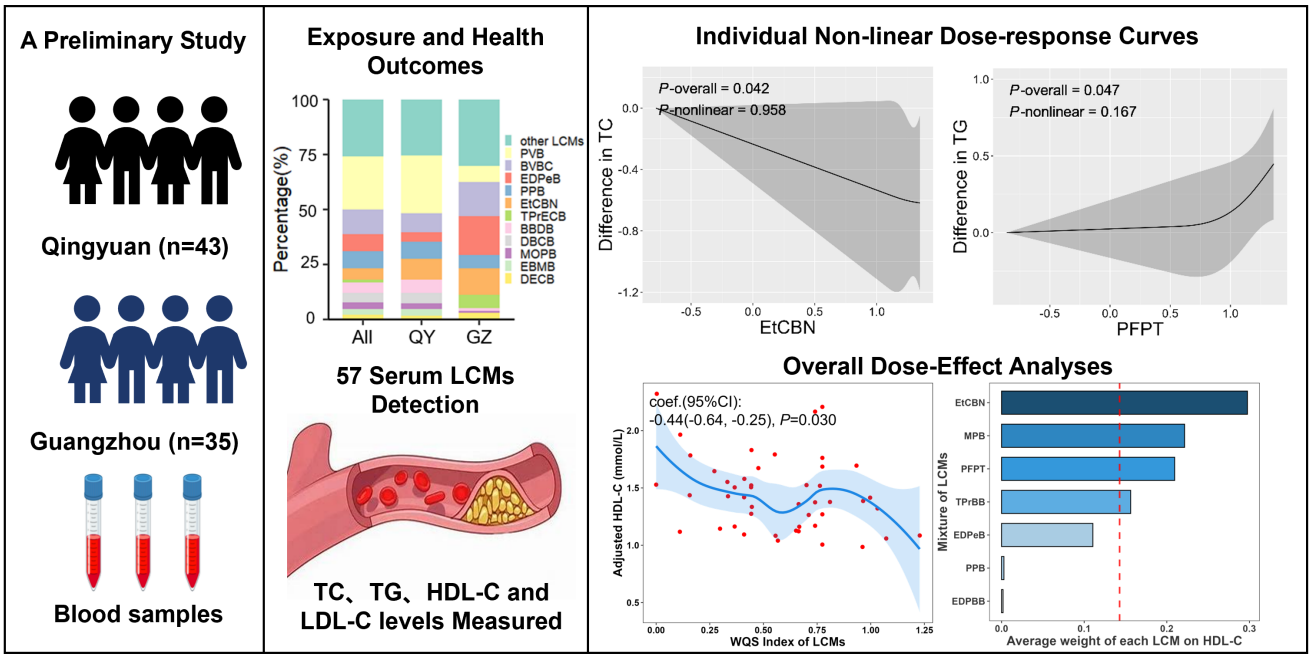
Graphical Abstract



Highlights:

- Positive association between PFPT and TG levels, and negative associations between EtCBN and TC as well as LDL-C levels.
- There is a non-monotonic dose-response relationship between LCM and lipid levels.
- High-density lipoprotein cholesterol levels were negatively correlated with LCM mixtures.
- EtCBN and PFPT contributed the most to dyslipidemia among the mixture.

Quantitative associations and combined effect on blood lipids of co-exposure to serum liquid crystal monomers: a preliminary study

LuLu Huang[1,2,†], Zhengyang Yuan[1,2,†], Xue Cao[2,3], Lili Yao[1,2], Zulan Zhao[1,2], Li Li[1,2], Bairui Chen[1,2], Xinzhuo Hao[2,3], Chun Xie[1,*] and Qilong Liao[2,*]

[1] The Key Laboratory of Environmental Pollution Monitoring and Disease Control, Ministry of Education, School of Public Health, Guizhou Medical University, Guiyang 561113, China

[2] State Environmental Protection Key Laboratory of Environmental Pollution Health Risk Assessment, Research Group of Emerging Contaminants, South China Institute of Environmental Sciences, Ministry of Ecology and Environment, Guangzhou 510655, China

[3] Department of Toxicology, School of Public Health, Guangxi Medical University, Nanning, Guangxi 530021, China

†These authors contributed equally to this work.

*E-mail: 1009207189@qq.com (C.X.); liaoqilong@scies.org (Q.L.).

Abstract: Humans are constantly exposed to liquid crystal monomers (LCMs) due to the widespread use of various electronic products. Previous studies have shown that LCMs can cause dysregulation in gene expression related to lipid homeostasis. However, the quantitative associations and effects of exposure to individual and mixed LCMs on blood lipid levels remain poorly understood. This study measured the serum concentrations of 57 LCMs, along with total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), in 78 adults in South China. Among the participants, 51 LCMs were detected in serum samples, with detection frequencies ranging from 1.30% to 75.6% and concentrations from 2.58 to 86.29 ng/mL. Results from generalized linear and restricted cubic splines models demonstrated distinct effects of individual LCMs on lipid levels, with non-monotonic dose-response relationship curves. The application of generalized weighted quantile sum (gWQS) analysis revealed that the constructed weighted quantile sum (WQS) index was negatively correlated with HDL-C levels (Coefficient: −0.44, 95% CI: −0.64 to −0.25, P = 0.030), with 4-(4-Ethylcyclohexyl) benzonitrile (EtCBN), 4-Methyl-4'-pentylbiphenyl (MPB) and 2'-Fluoro-4-Pentyl-4''-Propyl-1,1':4',1''-Terphenyl (PFPT) contributing most significantly to this association. Our findings highlight the overall dose-dependent effects of LCM exposure and provide further evidence of dyslipidemia associated with LCM mixtures.

Keywords: liquid crystal monomers; serum lipids; human exposure; dyslipidemias; quantitative associations; joint-effect

1. Introduction

Liquid crystal monomers (LCMs) are a class of synthetic organic chemicals that serve as key materials for liquid crystal displays (LCDs) [1,2], which are extensively applied in varied electronic devices [3,4]. The rapid growth of the electronic industry has led to a significant surge in the global demand for LCMs [2]. Typical LCM compounds consist of a hydrocarbon backbone and aromatic (or cyclohexyl) molecules decorated with side chains and/or terminal functional groups (e.g., fluorine, cyano substituents), and are usually classified into three categories according to their functional groups, *i.e.* biphenyls and analogs (BAs), cyanobiphenyls and analogs (CBAs), and fluorinated biphenyls and analogs (FBAs) [5]. During the production of LCD devices, multiple LCMs are co-added to LCDs to meet the specific requirements of various brands of LCD devices [6,7]. The lack of covalent bonding between LCMs and the base material of LCDs resulting in their inevitable release into the environment during production, use, or disposal. A preliminary estimation suggests that approximately 1.07−107 kg/year of LCMs are directly released globally from waste TV/computer LCD panels into various environmental compartments [2].

Recent studies have revealed the presence of numerous LCMs in various environmental matrices, including dust (67,400 ng/g) [8], air (68,800–385,000 pg/m^3) [9], and sediments from the Yangtze River (26.1 ng/g dry weight (dw)), Taihu Lake (0.076 ng/g dw), and Taizhou (1.15 ng/g dw) [10]. Additionally, LCMs have been detected in breast milk (11.97 × 10^4–2.82 × 10^4 ng/g lipid weight) [11], as well as in the serum of e-waste dismantlers (7.78–276 ng/mL) and the general population (3.16–28.5 ng/mL) [12]. These findings collectively demonstrate the widespread contamination of LCMs in the environment, posing potential risks to human health.

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The relatively high log Kow values (> 5) of LCMs further indicate their elevated potential for bioaccumulation in humans [13]. Consequently, most LCMs are considered to possess characteristics of persistence, bioaccumulation, and toxicity. It has been found that 476 out of 1,173 LCMs exhibit a tendency for environmental persistence and bioaccumulation, with halogenated substances accounting for approximately 99% of these [14]. Toxicological studies have also demonstrated that certain LCMs disrupt thyroid hormone levels in zebrafish [15], induce oxidative stress in fish species [16], and significantly modulate the expression of genes related to energy metabolic pathways [17]. Notably, liver-type fatty acid-binding protein (LBFABP), a gene involved in lipid homeostasis, is significantly upregulated by LCM exposure. Furthermore, Zhao *et al.* [18] found that LCMs induced dysregulation of the overall metabolome and transcriptome in human kidney epithelial cells, with significant disruption of fatty acid β-oxidation.

The studies mentioned above suggest a potential link between abnormal lipid metabolism caused by LCMs and the development of dyslipidemia in humans. Specifically, Su *et al.* [17] reported that *in vitro* exposure to LCM mixtures from six LCD devices significantly upregulated LBFABP, a key regulator of intracellular lipid transport and metabolism, while simultaneously downregulating fibroblast growth factor 19 (FGF19), a critical enterohepatic signaling molecule involved in bile acid and lipid homeostasis; these findings demonstrate that LCM exposure directly modulates the expression of core genes associated with lipid homeostasis and bile acid/cholesterol regulation, thereby establishing a critical mechanistic link between LCM exposure and the dysregulation of lipid metabolic processes that underlies potential LCM-induced dyslipidemia. And, it is widely recognized that dyslipidemia is a risk factor for a range of diseases [19,20], including cardiovascular disease (CVD), diabetes, and metabolic syndrome [21,22]. Therefore, understanding the impact of LCMs on lipid levels is essential for providing evidence for the primary prevention of dyslipidemia. Furthermore, our previous study indicated that LCM exposure levels were similar between the general population and occupationally exposed populations [23]. However, no studies have yet explored the associations between LCMs and lipid levels in general populations, leaving gaps in understanding the quantitative relationships between LCMs and lipids and the overall effects of LCM mixtures on lipid levels.

Hence, this study aims to elucidate the quantitative associations and dose-response relationship curves between individual LCMs and their mixtures with changes in lipid levels in the general population by employing generalized linear models (GLM), restricted cubic splines (RCS), and generalized weighted quantile sum (gWQS) regressions. Our findings Our findings provide preliminary human evidence for the dyslipidemic effects of LCM co-exposure, informing future risk assessment and toxicological studies.

2. Materials and methods

2.1. Study design and population

A cross-sectional epidemiological study was conducted in Guangdong Province, China. Given the emerging nature of LCM biomonitoring, this investigation was designed as an exploratory study. Volunteer recruitment was carried out in the cities of Qingyuan (QY) and Guangzhou (GZ) in 2023. The sampling frame comprised residents aged ≥ 18 years who had resided in the target cities for more than two years. Recruitment was conducted through multiple channels, including community posters and word-of-mouth referrals. After screening for eligibility, a total of 78 adults (QY: n = 43; GZ: n = 35) were enrolled. The inclusion criteria for study participants were as follows: (1) completion of the questionnaire survey and physical examination; (2) provision of sufficient serum samples for the detection of LCMs and blood lipid indices; (3) absence of self-reported or diagnosed diseases such as hypertension, diabetes, or tumors; (4) no recent surgical procedures or traumas within the past three months; and (5) no use of any blood lipid-related medications within the past three months. Blood samples from participants were collected during fasting in the early morning and were immediately used to analyze blood lipid indices in the clinic. The remaining blood samples were centrifuged to extract serum, which was stored at −80 °C for chemical analysis. All participants provided informed consent prior to participation in this study. The study was approved by the Medical Ethics Committee of Tongji Medical College, Huazhong University of Science and Technology (Ethics Approval No. [2020] S284).

2.2. Determination of blood lipids indices

Blood concentrations of triglycerides (TG, mmol/L), total cholesterol (TC, mmol/L), high-density lipoprotein cholesterol (HDL-C, mmol/L), and low-density lipoprotein cholesterol (LDL-C, mmol/L) were measured using an automatic biochemical analyzer (BS-2000M, Mindray Bio-Medical Electronics Co., Ltd., Shenzhen, China) following standard protocols. The measurements were performed by medical professionals at Guangzhou City Hospital for Occupational Disease Prevention and Treatment.

2.3. Serum LCMs concentrations detection

The analysis of LCMs in serum was performed as follows: Methanol was added to the serum samples for protein precipitation, and the samples were then sonicated with mixed solvents (hexane: acetone = 3:1, v/v). The extract was purified using a Si solid-phase extraction (SPE) column, and the eluate was concentrated to near dryness under a gentle stream of ultra-pure N2, then reconstituted with isooctane. LCMs were analyzed by gas chromatography-triple quadrupole tandem mass spectrometry (GC-MS/MS). The detailed sample pretreatment processes and the

parameter information for GC-MS/MS are provided in Supplementary Text S1 and Supplementary Table S1 in the Supporting Information (SI). The spiked recoveries of LCMs treated with the aforementioned pretreatment process ranged from 65.3% to 117%. For the internal standards (ISs), the recoveries ranged from 61.6% to 82.0%, with relative standard deviations (RSDs) less than 20%. Three polychlorinated biphenyl (PCB) congeners—PCB82, PCB141, and PCB198—were used as surrogate internal standards. The specific matching of each internal standard to target LCMs is provided in Supplementary Table S2. Quantification was performed using an internal standard calibration method, with a calibration curve consisting of no fewer than six points for each LCM ($R^2 > 0.99$ for all target analytes). All reported LCM concentrations were corrected for the recovery of their respective internal standards. The limit of quantitation (LOQ) was defined as the mean value in procedural blanks multiplied by three times the standard deviation (SD) for each analyte detected in the procedural blank samples. For analytes that were below the limit of detection (< LOD) in the procedural blank samples, the LOQ was defined as a signal-to-noise ratio of 10 (S/N = 10) [24]. The LOQs for LCMs ranged from 2.73E-05 to 2.67 ng/mL. The specific recoveries, RSDs, and LOQs for individual LCMs are listed in Supplementary Table S2. Further analyses included only specific LCMs with a detection frequency (DF) higher than 30%. This threshold was selected based on recent human biomonitoring studies of LCMs, which have employed DF criteria ranging from 20% to 40% depending on the matrix and analytical goals [25,26], to improve model stability, reduce the risk of spurious associations due to low detection rates, and focus on commonly detected compounds.

2.4. Statistical analysis

Demographic characteristics, serum lipids, and LCM concentrations were summarized. Concentrations below the limit of detection were substituted with 1/2 LOD. The normality of continuous data was assessed using the Kolmogorov-Smirnov test. Normally distributed data are presented as mean ± SD, while non-normally distributed data are presented as median with interquartile range (IQR). Categorical variables are expressed as numbers and percentages. For comparisons between the two groups, a t-test or Mann-Whitney U test was used for continuous variables, and the Chi-square test was used for nominal variables. To reduce the influence of outliers due to the skewed distribution of LCM concentrations, natural log-transformed (Ln-transformed) and normalized (Z-scored) values were employed. Correlations among individual LCMs were tested using Spearman correlation tests. Covariates included in the analysis were age (continuous), sex (male/female), body mass index (continuous), smoking status (yes/no), drinking status (yes/no), and physical exercise (yes/no), based on prior knowledge of dyslipidemia risk factors. Additionally, subgroup analyses were conducted for each city to assess the robustness of associations across different demographic profiles.

GLMs were employed to investigate the associations between individual LCMs and TC, TG, HDL-C, and LDL-C, with adjustments for the mentioned covariates. The results were expressed as regression coefficients (Coef.) with 95% confidence intervals (95% CIs), indicating the association between a one standard deviation increase in serum LCM levels (Ln-transformed) and blood lipid indices. To further explore potential non-linear patterns, each LCM concentration was categorized into tertiles (T1 to T3), with the T1 group serving as the reference group to estimate the Coef. (95% CIs) for the T2 and T3 groups. RCS with three knots at the 10th, 50th, and 90th percentiles were employed to flexibly model the association between individual LCMs and blood lipid indices. The *P*-overall value indicated the overall associations between LCMs and blood lipid indices, while the *P*-nonlinear value was used to assess the linear or nonlinear nature of these associations [27]. This complementary approach, which combines linear models to estimate overall trends with RCS to capture nonlinear patterns, enables a comprehensive assessment of the relationship between exposure and outcome.

The gWQS regression model was employed to estimate the combined effect of a mixture of LCMs on blood lipids. These weighted percentiles sum approach is particularly effective in analyzing high-dimensional datasets, as is commonly encountered in environmental epidemiology studies [28,29]. In this model, concentrations of individual LCMs were converted into ordinal quartile variables, scored as 0, 1, 2, or 3 (representing the 1st to 4th quartiles, respectively). Each chemical is assigned a weight ranging from 0 to 1, and the sum of all weights equals 1. The weighted quantile sum (WQS) index was calculated as the weighted sum of these quartile scores. The WQS index for each participant was used to characterize the level of co-exposure to the LCMs mixture. Subsequently, the WQS index was included in a regression model with relevant covariates to examine the combined effect on the outcome. The regression coefficient (β1) for the WQS index represents the change in blood lipid levels associated with a one-unit increase in the WQS index, equivalent to an average increase of one quartile across all LCMs in the mixture. The weight of each LCM indicates its relative contribution to the overall association. The dataset was randomly split into a training set (40%) and a validation set (60%) for 100 independent iterations. For each iteration, the gWQS model was run with 1000 bootstrap samples in the training set to estimate weights, and the mixture effect coefficient (β) was evaluated in the validation set. The final weights and effect estimates (β and 95% CI) were calculated as the mean across all 100 iterations. This approach is specifically recommended for finite sample sizes to obtain stable and reproducible estimates, as it reduces the influence of any single random partition [29] The results of the gWQS models were presented as coefficients (Coef.) with 95% confidence intervals (95% CI), and key LCMs related to the overall dose effect were identified based on the relative strength of their weights (averaged across iterations). The results of the gWQS models were presented as coefficients (Coef.) with 95% confidence intervals

(95% CI), and key LCMs related to the overall dose effect were identified based on the relative strength of their weights.

Specifically, considering potential heterogeneities within the two groups, GLM, RCS, and gWQS analyses were conducted for both groups. Statistical analyses were performed using SPSS (version 24.0) and R software (version 4.1.3), utilizing the R packages "glm", "rcs", and "gWQS". A significance level of $P < 0.05$ was set for a two-tailed test.

3. Results

The characteristics of the study participants are presented in Supplementary Table S3. The average age (mean ± SD) of all participants was 41 ± 13.9 years, with the QY population (49 ± 11.0 years) being older than the GZ population (32 ± 11.0 years). The average body mass index (BMI) for the QY group (22.7 kg/m²) was slightly higher than that of the GZ group (21.4 kg/m²). However, the GZ group had a higher proportion of smokers and a higher annual income compared to the QY group. No significant differences were observed between the two groups in terms of sex composition, alcohol consumption, and frequency of physical exercise. Given these demographic differences, all subsequent statistical models adjusted for age, location, and socioeconomic factors to minimize confounding.

The detection frequencies (DF) and concentrations of the analytes are presented in Table 1 and Supplementary Table S4. A total of 51 LCMs were detected in the serum samples of this study. The DFs of LCMs ranged from 1.28% to 75.6%, with the concentrations of $\sum_{51}$ LCMs ranging from 2.58 to 86.3 ng/mL. Among the 51 detected LCMs, seven had relatively high DFs in the serum samples, ranging from 30.8% to 75.6%, with individual concentrations ranging from 0.49 to 21.7 ng/mL. The levels of ΣLCMs were from 2.58 to 86.3 ng/mL in the QY group and from 2.86 to 36.4 ng/mL in the GZ group. The composition profiles of individual LCMs among all participants, the QY group, and the GZ group are shown in Figure 1. In the serum of all participants, PVB (19.6%) was found to be the most abundant chemical, followed by BVBC (10.8%), EtCBN (10.6%), EDPeB (9.13%), PPB (7.15%), BBDB (4.27%), and DBCB (3.02%). The major LCMs in the serum of the QY group were generally similar to those found among all participants. For the GZ group, EDPeB (17.8%) was observed as the most abundant LCM, followed by BVBC (15.4%), EtCBN (12.3%), PVB (7.13%), TPrECB (5.97%), and PPB (5.95%). The proportions of BAs, CBAs, and FBAs were 57.8%, 2.24%, and 39.9%, respectively, for the QY population, and 31.9%, 19.6%, and 48.5%, respectively, for the GZ population.

The concentrations of seven chemicals were detectable in more than 30% of all participants. Statistical analysis revealed significant correlations between the concentrations of these seven chemicals (r = −0.058 to 0.41, $P < 0.05$) (Figure 1c). Specifically, there were significant positive correlations between MPB and EDPeB (r = 0.27, $P < 0.05$), 2'-Fluoro-4-Pentyl-4''-Propyl-1,1':4',1''-Terphenyl (PFPT) and EDPBB (r = 0.25, $P < 0.05$), TPrBB and EDPBB (r = 0.41, $P < 0.05$), Conversely, there was a significant negative correlation between PPB and PFPT, EDPBB, and TPrBB concentrations and between EDPeB and EtCBN, EDPBB, and TPrBB, as well as between MPB and EtCBN, EDPBB, and TPrBB (r = −0.058 to −0.30, $P < 0.05$). The concentrations of TC, TG, HDL-C, and LDL-C in the serum of the QY group were 3.61 to 9.07 mmol/L, 0.32 to 3.46 mmol/L, 0.97 to 2.56 mmol/L, and 1.79 to 6.31 mmol/L, respectively (Table 1). Higher levels of TC and HDL-C were observed in the QY population compared to the GZ population (Mann-Whitney U test, $P < 0.05$).

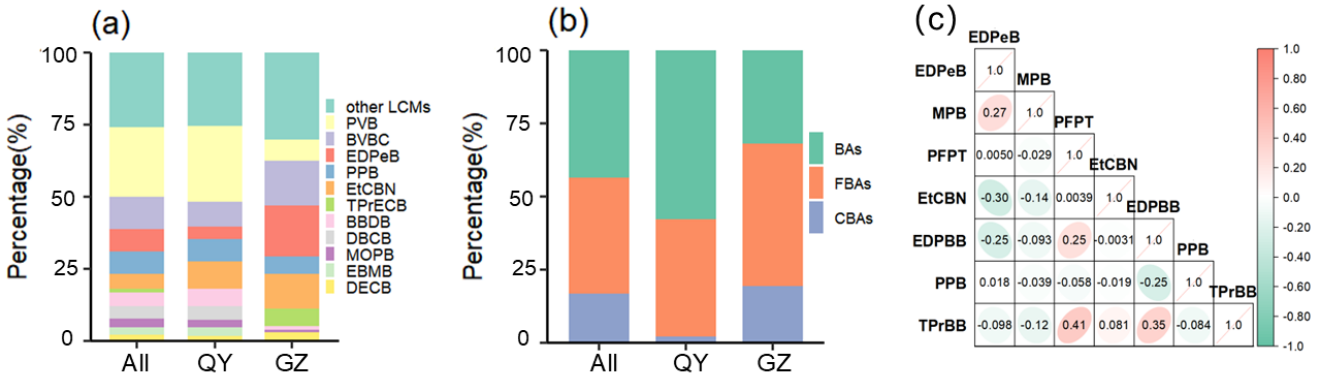

Figure 1. Compositional profiles of **(a)** major LCMs, **(b)** relative proportions of the three types of LCMs, and **(c)** correlation analysis. Note: All: Total population; QY: Qingyuan group; GZ: Guangzhou group; LCMs with DF > 30% were used for correlation analysis.

Table 1. Concentrations of serum LCMs and lipids indices for all participants.

Analytes	All participants (n = 78)			QY (n = 43)			GZ (n = 35)			*P*-value
	DF (%)	P_{50}	Range	DF (%)	P_{50}	Range	DF (%)	P_{50}	Range	
LCM concentration (ng/mL)										
EDPeB	75.6	2.25	0.31–7.84	55.8	1.73	0.31–3.86	100	2.62	0.89–7.84	< 0.001
MPB	69.2	0.26	0.06–2.73	69.8	0.15	0.06–2.73	68.6	0.49	0.16–2.08	0.005
PFPT	43.6	0.16	0.02–0.91	53.5	0.27	0.02–0.91	31.4	0.12	0.02–0.16	0.006
EtCBN	38.5	4.56	1.96–18.3	53.5	4.28	1.96–7.50	20.0	8.33	6.61–18.3	0.047
EDPBB	35.9	0.58	0.07–2.35	65.1	0.58	0.07–2.35	0	< LOD	< LOD	-
PPB	34.6	2.82	0.78–14.5	23.3	6.62	3.01–14.5	48.6	1.38	0.78–4.04	0.191
TPrBB	30.8	0.28	0.04-0.98	46.5	0.36	0.04–0.98	11.4	0.17	0.08–0.21	0.001
DPrB	28.2	0.67	0.27–1.63	37.2	0.70	0.27–1.63	17.1	0.41	0.29–0.86	0.029
8OCB	26.9	0.88	0.42–7.04	34.9	0.74	0.42–1.13	17.1	5.05	3.75–7.04	0.255
EFPT	25.6	0.12	0.04–0.61	37.2	0.17	0.04–0.61	11.4	0.07	0.04–0.18	0.006
EDPB	24.4	0.47	0.16–1.21	37.2	0.56	0.16–1.21	8.6	0.33	0.24–0.40	0.002
TePrB	23.1	1.06	0.04–5.70	11.6	0.18	0.04–0.62	37.1	1.56	0.51–5.70	0.002
PVB	21.8	11.1	2.46–58.1	20.9	24.5	11.1–58.1	22.9	4.46	2.46–8.13	0.765
DMPMB	21.8	0.71	0.20–1.94	25.6	0.55	0.20–1.16	17.1	1.05	0.65–1.94	0.564
BBDB	21.8	2.33	0.65–11.1	23.3	5.46	1.38–11.1	20.0	0.96	0.65–1.82	0.432
BVBC	20.5	5.98	2.66–46.9	4.7	42.2	37.5–46.9	40.0	5.77	2.66–11.4	< 0.001
EPB	20.5	0.12	0.03–0.71	27.9	0.18	0.06–0.71	11.4	0.04	0.03–0.06	0.036
BDPrB	20.5	0.88	0.25–2.55	7.0	0.45	0.43–0.68	37.1	1.20	0.25–2.55	0.001
Σ_7 LCMs[a]	-	4.47	0.49–21.7	-	4.63	0.49–21.2	-	4.18	1.12–21.7	0.517
Σ_{other} LCMs[b]	-	8.72	0.09–69.6	-	8.28	0.09–69.6	-	8.72	0.94–31.8	0.284
Σ_{51} LCMs	-	14.3	2.58–86.3	-	12.5	2.58–86.3	-	16.1	2.86–36.4	0.640
Lipids level (mmol/L)										
TC	-	5.08	3.02–9.07	-	5.28	3.61–9.07	-	4.80	3.02–6.42	0.031
TG	-	0.97	0.32–3.46	-	1.11	0.32–3.46	-	0.78	0.48–3.09	0.054
HDL-C	-	1.40	0.91–2.56	-	1.50	0.97–2.56	-	1.23	0.91–2.23	0.001
LDL-C	-	3.02	1.73–6.31	-	3.06	1.79–6.31	-	2.98	1.73–4.48	0.872

Note: DF, detection frequency. a: LCMs with DF ≥ 30%, the $\sum_7$LCMs include EDPeB, MPB, PFPT, EtCBN, EDPBB, PPB and TPrBB. b: 44 LCMs with DF < 30%. -: No data. *P*-values for LCM concentrations were estimated by the Mann–Whitney U test, and bolded values indicate statistical significance. For each LCM, the P_{50} and range are calculated based only on samples with detectable concentrations (*i.e.*, values > LOD).

The associations between ln-transformed LCM concentrations and changes in lipid levels in the total population are summarized in Table 2. A one-unit increase in ln-transformed PFPT concentrations was associated with a 0.14 mmol/L increase in TG concentration (95% CI: 0.01, 0.28, $P = 0.046$). Similarly, a one-unit increase in ln-transformed EtCBN concentrations was associated with decreases of 0.29 mmol/L in TC concentration (95% CI: −0.52, −0.07, $P = 0.013$) and 0.21 mmol/L in LDL-C concentration (95% CI: −0.40, −0.03, $P = 0.028$). Additionally, EtCBN concentration showed a negative trend with HDL-C levels (coefficient: −0.07, 95% CI: −0.15, 0.00, $P = 0.066$). Furthermore, at the T2 tertile of MPB, LDL-C levels were positively associated with MPB concentration (coefficient: 0.47, 95% CI: 0.01, 0.94, $P = 0.048$).

In the QY subgroup, whose participants were notably older and had a slightly higher average BMI compared to the GZ subgroup, a one-unit increase in ln-transformed EDPeB concentration was associated with a decrease in HDL-C concentration by 0.32 mmol/L (95% CI: −0.56, −0.09, $P = 0.009$). Similarly, a comparable association was observed between PFPT concentration and changes in TG levels (coefficient: 0.26, 95% CI: 0.02, 0.50, $P = 0.040$). Additionally, PFPT concentration was negatively associated with HDL-C levels at T3 (coefficient: −0.25, 95% CI: −0.49, −0.02, $P = 0.039$) (Supplementary Table S5). In the GZ subgroup, which comprised younger participants with a lower average BMI and a higher proportion of smokers, at T3 of BVBC, a positive association with TG levels was observed (coefficient: 0.41, 95% CI: 0.15, 0.67, $P = 0.004$). Furthermore, inverse associations were observed between BDPrB and both TC and LDL-C (Supplementary Table S6).

Table 2. Associations [Coef. (95% CIs)] between serum LCMs with blood lipids indices in all participants.

Compounds		TC		TG		HDL-C		LDL-C	
		Coef. (95% CI)	*P*-value	Coef. (95% CI)	*P*-value	Coef. (95% CI)	*P*-value	Coef. (95% CI)	*P*-value
EDPeB	T1	Reference	-	Reference	-	Reference	-	Reference	-
	T2	0.03	0.925	0.21	0.322	−0.16	0.112	0.21	0.418
		(−0.60, 0.66)		(−0.20, 0.62)		(−0.36, 0.04)		(−0.30, 0.72)	
	T3	−0.13	0.685	0.16	0.436	−0.18	0.082	0.11	0.666
		(−0.75, 0.49)		(−0.25, 0.57)		(−0.38, 0.02)		(−0.39, 0.61)	
	Per unit	0.12	0.466	0.03	0.762	−0.09	0.090	0.23	0.090
	increase	(−0.20,0.44)		(−0.18,0.25)		(−0.19,0.01)		(−0.03,0.48)	
MPB	T1	Reference	-	Reference	-	Reference	-	Reference	-
	T2	0.39	0.190	0.01	0.965	−0.07	0.475	0.47	0.048
		(−0.19, 0.97)		(−0.38, 0.39)		(−0.26, 0.12)		(0.01, 0.94)	
	T3	0.24	0.381	0.14	0.436	−0.09	0.326	0.23	0.291
		(−0.29, 0.77)		(−0.21, 0.49)		(−0.26, 0.09)		(−0.19, 0.65)	
	Per unit	0.01	0.938	0.07	0.336	−0.03	0.384	0.01	0.918
	increase	(−0.21, 0.22)		(−0.07, 0.21)		(−0.10, 0.04)		(−0.16, 0.18)	
PFPT	T1	Reference	-	Reference	-	Reference	-	Reference	-
	T2	−0.26	0.499	0.18	0.467	−0.11	0.369	−0.21	0.490
		(−1.01, 0.49)		(−0.30, 0.67)		(−0.36, 0.13)		(−0.82, 0.39)	
	T3	−0.26	0.288	0.30	0.062	−0.07	0.405	−0.24	0.219
		(−0.73, 0.22)		(−0.01, 0.61)		(-0.22, 0.09)		(-0.63, 0.14)	
	Per unit	−0.12	0.276	0.14	0.046	−0.03	0.346	−0.12	0.192
	increase	(−0.34, 0.09)		(0.01, 0.28)		(−0.10, 0.04)		(−0.29, 0.06)	
EtCBN	T1	Reference	-	Reference	-	Reference	-	Reference	-
	T2	−0.94	0.072	0.36	0.311	−0.22	0.201	−0.84	0.048
		(−1.95, 0.07)		(−0.33, 1.05)		(−0.56, 0.12)		(−1.67, −0.02)	
	T3	−0.58	0.020	−0.12	0.488	−0.14	0.100	−0.42	0.039
		(−1.06, −0.10)		(−0.44, 0.21)		(−0.29, 0.02)		(−0.80, −0.03)	
	Per unit	−0.29	0.013	−0.04	0.608	−0.07	0.066	−0.21	0.028
	increase	(−0.52, −0.07)		(−0.20,0.11)		(−0.15,0.00)		(−0.40, −0.03)	
EDPBB	T1	Reference	-	Reference	-	Reference	-	Reference	-
	T2	−0.40	0.592	0.00	0.999	−0.04	0.872	−0.25	0.675
		(−1.84, 1.04)		(−0.95, 0.95)		(−0.51, 0.43)		(−1.42, 0.92)	
	T3	−0.05	0.848	0.00	0.995	0.06	0.457	−0.13	0.542
		(−0.55, 0.45)		(−0.33, 0.33)		(−0.10, 0.23)		(−0.53, 0.28)	
	Per unit	−0.01	0.917	0.00	0.979	0.04	0.375	−0.05	0.578
	increase	(−0.25, 0.22)		(−0.16, 0.15)		(−0.04, 0.11)		(−0.25, 0.14)	
PPB	T1	Reference	-	Reference	-	Reference	-	Reference	-
	T2	−0.33	0.740	0.80	0.217	−0.14	0.670	−0.52	0.520
		(−2.28, 1.61)		(−0.46, 2.07)		(−0.77, 0.50)		(−2.10, 1.06)	
	T3	−0.18	0.447	−0.15	0.330	0.00	0.992	−0.15	0.437
		(−0.63, 0.27)		(−0.44, 0.15)		(−0.15, 0.15)		(−0.51, 0.22)	
	Per unit	−0.09	0.407	−0.05	0.463	0.01	0.856	−0.09	0.302
	increase	(−0.30, 0.12)		(−0.19, 0.09)		(−0.06, 0.08)		(−0.26, 0.08)	
TPrBB	T1	Reference	-	Reference	-	Reference	-	Reference	-
	T2	−0.20	0.435	0.29	0.076	−0.03	0.745	−0.22	0.275
		(−0.69, 0.29)		(−0.03, 0.61)		(−0.19, 0.13)		(−0.62, 0.17)	
	T3	-	-	-	-	-	-	-	-
	Per unit	−0.07	0.571	0.14	0.077	−0.01	0.876	−0.09	0.340
	increase	(−0.30,0.16)		(−0.01,0.28)		(−0.08,0.07)		(−0.28,0.09)	

Note: Each LCM concentration was categorized into tertiles. T1: tertiles 1; T2: tertiles 2; T3: tertiles 3; -: No data available, the model cannot be fitted.

Adjusted model: adjusted for age, sex, BMI, smoking, alcohol and physical exercise.

For the dose-response association between lipid levels and serum levels of LCMs, we observed an inverted V-shaped relationship between MPB and TC, as well as between MPB and LDL-C in the total population (TC: *P*-nonlinear = 0.042; LDL-C: *P*-nonlinear = 0.015). Additionally, an inverted U-shaped relationship was found between PPB and LDL-C (*P*-overall = 0.034, *P*-nonlinear = 0.042) (Figure 2 and Supplementary Figure S1). In the QY subgroup, we observed an approximately U-shaped relationship between 8OCB and LDL-C, as well as between 8OCB and TC (LDL-C: *P*-nonlinear = 0.037; TC: *P*-nonlinear = 0.046). A similar relationship was also found between TPrBB and TC (*P*-nonlinear = 0.042). Furthermore, a similar trend was observed between EDPBB and both LDL-C and TC (LDL-C: *P*-nonlinear = 0.087; TC: *P*-nonlinear = 0.068). Additionally, the relationship between MPB and LDL-C, as well as TC, was consistent with that observed in the total population (LDL-C: *P*-nonlinear = 0.053; TC: *P*-nonlinear = 0.020) (Supplementary Figure S2). In the GZ subgroup, U-shaped relationships were identified between TePrB and TG (*P*-overall = 0.042, *P*-nonlinear = 0.024), as well as between PPB and TC (*P*-nonlinear = 0.063) (Supplementary Figure S3).

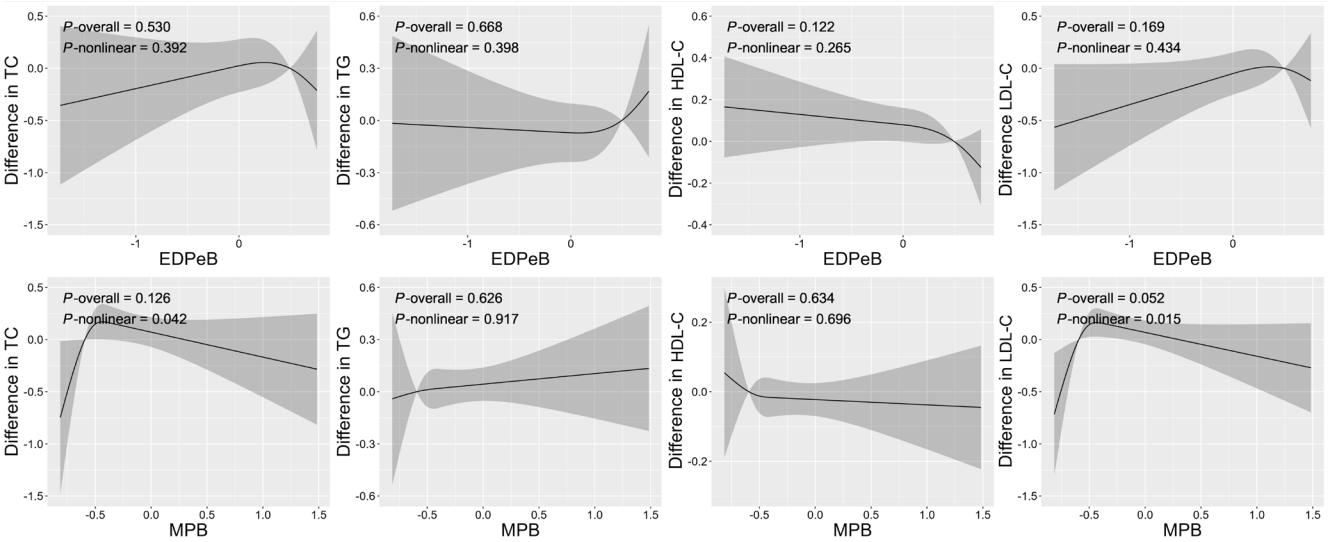

Figure 2. Dose-response relationships between LCMs (EDPeB and MPB) and blood lipid levels in all participants. Note: EDPeB and MPB are the LCMs with the highest DF. The RCS analysis results for the other five LCMs are presented in Supplementary Figure S1 The RCS model was adjusted for age, sex, BMI, smoking, alcohol and physical exercise.

Figure 3 depicts the overall dose effect of LCM mixtures on lipid level impairment and the contribution of individual chemicals to the joint associations. The WQS indices were statistically associated with HDL-C levels. A one-unit increase in the WQS index of the LCMs mixture was associated with a 0.44 mmol/L decrease in HDL-C (95% CI: −0.64, −0.25, *P* = 0.030), a 0.55 mmol/L increase in TC (95% CI: −0.31, 1.41, *P* = 0.524), a 0.36 mmol/L increase in TG (95% CI: −0.03, 0.75, *P* = 0.355), and a 0.41 mmol/L increase in LDL-C (95% CI: −0.37, 1.19, *P* = 0.603). In descending order, the most influential individual components in the WQS index for the significant decrease in HDL-C were EtCBN (w = 0.298), MPB (w = 0.222), PFPT (w = 0.210), and TPrBB (w = 0.156). The detailed contribution weights of each LCM are listed in Supplementary Table S7.

To account for potential effect modification by location and associated demographic characteristics (e.g., age and BMI), we analyzed the QY and GZ subgroups separately. For both subgroups, the negative association between the WQS index and HDL-C was consistent with that observed in the total population, though the magnitude varied slightly consistent with the demographic differences. A one-unit increase in the WQS index of LCM mixtures was associated with a decrease in HDL-C of 0.57 mmol/L (95% CI: −0.78, −0.35, *P* = 0.018) in the QY subgroup and 0.66 mmol/L (95% CI: −0.87, −0.45, *P* = 0.008) in the GZ subgroup. In both groups, TC, TG, and LDL-C exhibited an increasing trend with higher WQS index values, which is similar to the findings in the overall population. The specific confidence intervals and weights for each LCM are shown in Supplementary Figure S4, Supplementary Figure S5, and Supplementary Table S7.

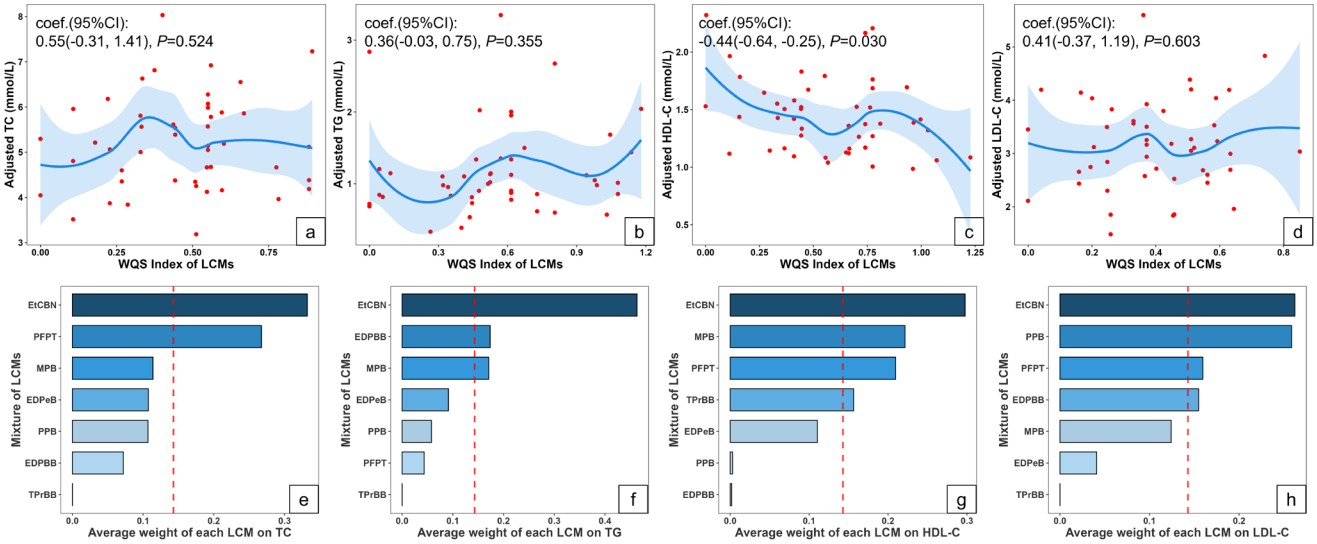

Figure 3. Overall dose-effect of exposure to LCM mixture on lipid levels and the contribution of individual pollutant in all participants. Note: Panels **(a–d)**: Dose-response relationships between the WQS index and: **(a)** Total cholesterol (TC), **(b)** Triglycerides (TG), **(c)** High-density lipoprotein cholesterol (HDL-C), **(d)** Low-density lipoprotein cholesterol (LDL-C). The blue lines represent smoothed fits with 95% confidence intervals (gray shaded areas). Red dots represent individual observations. Panels **(e–h)**: Average weights of individual LCMs contributing to the WQS index for: **(e)** TC, **(f)** TG, **(g)** HDL-C, **(h)** LDL-C. Average weights of individual LCMs contributing to the WQS index. Bars represent the mean weights calculated from 1000 bootstrap iterations. The red dashed line represents the reference weight. Chemicals with weights exceeding this threshold are considered to have substantial contributions to the mixture effect. The gWQS models were adjusted for age, sex, BMI, smoking, alcohol and physical exercise. Abbreviation: WQS, weighted quartile sum index for representing the overall exposure load of the LCM mixtures; Coef. 95 % CIs: Coefficients and 95 % Confidence Intervals.

4. Discussion

We conducted a cross-sectional study to investigate the association between LCMs and serum lipid levels in the general population. Our findings revealed that exposure to LCMs was associated with a decrease in HDL-C. Specifically, EtCBN, PFPT, and TPrECB were identified as major risk factors for dyslipidemia related to LCM pollutants.

Our study demonstrated the presence of LCM mixtures in the serum of the general population. The levels of $\sum_{51}$LCMs (ranging from 2.58 to 86.3 ng/mL) detected in this study were lower than those found in e-waste workers from South China (range: 7.78–276 ng/mL) 12 and in breast milk from the general population (median: 133.40 ng/g lipid weight) [11]. However, these values were significantly higher than the serum levels of LCMs in older adults from South China (range: 3.9–26.3 ng/mL) [8]. This discrepancy can be attributed to the limited sample size analyzed in that study [8].

The high proportions of PVB, BVBC, EtCBN, EDPeB, PPB, BBDB, and TPrECB indicate that these chemicals are representative target analytes in this study. However, the main LCMs identified here differ slightly from those in previous studies, which may be attributed to variations in target analytes across different studies. The proportions of BAs and FBAs in serum samples from the QY population were comparable, with both showing significantly higher proportions compared to CBAs. This finding is consistent with results from previous studies [2]. It is noteworthy that the proportion of CBAs in serum was significantly higher in the GZ population compared to the QY population in this study. According to reports on LCMs, CBAs are predominantly used in twisted-nematic (TN) LCDs and have fewer applications in other types of LCDs [1]. It is possible that the disparity in the types of LCDs used in the two locations contributed to the significant variation in their composition. The negative correlation observed among several LCMs in Figure 1 supports the likelihood of distinct sources for these chemicals. Additionally, this discrepancy could be attributed to the gradual proliferation of CBA-type LCM usage in more recent generations, given that the LCDs used for screening target LCMs in the previous study were manufactured approximately 15 years ago [2]. The study conducted by [30] demonstrated that CBAs were the most abundant LCMs. Given the increasing number of studies detecting higher levels of CBAs, it is crucial to acknowledge the widespread environmental contamination with CBAs and the potential adverse effects of human exposure. Furthermore, lipid levels among the participants in this study (TC: 3.02–9.07 mmol/L, TG: 0.32–3.46 mmol/L, HDL-C: 0.91–2.56 mmol/L, LDL-C: 1.73–6.31 mmol/L) were slightly elevated compared to those in previous studies [31–34]. This difference may be partially attributed to age-related differences in exposure pathways and

behavioral habits. Unlike traditional lipid-associated pollutants, which primarily enter the body through dietary intake, LCMs are mainly released from liquid crystal display devices into indoor dust. Younger individuals are more prone to hand-to-mouth ingestion, whereas adults are primarily exposed through dermal absorption due to the lipophilic properties of LCMs [35].

To our knowledge, we are the first to elucidate the dose-response relationships and identify the key chemicals associated with lipid levels in LCMs using GLM and gWQS models. Regarding the relationship between lipids and LCMs, there are relatively few reports in the literature. In an animal experiment, it was observed that β-chlorides (a type of LCM) were absorbed by the intestine and accumulated in adipose tissue [36]. Furthermore, given the lack of epidemiological literature on the specific relationship between LCMs and lipids, we compared our findings with those related to other compounds. For instance, a cross-sectional study conducted in Guangzhou showed that TG and LDL-C levels increased with rising serum concentrations of phenanthrene (PHE) in the general population [37]. A study found a strong association between serum bisphenol A (BPA) concentrations and TG in healthy adults [38]. A birth cohort study in Mexico found that adolescent boys with higher prenatal exposure to phthalates had lower levels of TC and LDL-C [39]. Furthermore, a previous study has revealed a negative correlation between maternal PFAS exposure and TC levels in neonatal cord blood [32]. Conversely, a cross-sectional study conducted in Baltimore found no significant association between perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) exposure and TC, TG, or total lipid levels in cord serum [31]. The associations between serum concentrations of organic pollutants and lipid levels varied across regions and age groups in these studies.

In this study, because most LCMs are lipophilic chemicals, we anticipated positive associations with lipid levels. However, significant associations were observed between PFPT and TG (positive association) and between EtCBN and TC and LDL-C (negative associations) in the total population. In the QY subgroup, negative correlations were observed between EDPeB and HDL-C, and between EDPB and TG. In the GZ subgroup, BDPrB exhibited negative associations with TC and LDL-C. These results deviated from our expectations. Mounting mammalian evidence indicates that LCMs can disrupt hepatic lipid metabolism through nuclear receptor-mediated pathways. *In vivo* mouse studies demonstrated that three representative LCMs (5OCB, DTMDPB, MeO3bcH) disrupt hepatic lipid homeostasis through nuclear receptor pathways, including peroxisome proliferator-activated receptor alpha (PPARα), farnesoid X receptor (FXR), and aryl hydrocarbon receptor (AhR) [40] Specifically, 5OCB acted as a PPARα antagonist to induce triglyceride and cholesterol accumulation, whereas DTMDPB and MeO3bcH altered FXR/AhR signaling to reduce cholesterol and triglyceride levels, indicating structure-specific lipid effects across LCM congeners. Interestingly, a previous cellular study demonstrated a significant upregulation of FGF [19] gene expression in chicken embryonic hepatocytes following exposure to LCMs [17]. FGF [19] plays a crucial role in lipid digestion and absorption within the small intestine, as well as in regulating bile acid biosynthesis, gallbladder filling, and glucose and lipid metabolism [41]. Similar results were obtained in a study where combined metabolomic and transcriptomic profiling showed a reduction in fatty acids after LCM exposure [18]. Therefore, we hypothesize that exposure to LCMs induces the upregulation of FGF [19] gene expression and the activation of downstream genes involved in lipid metabolism [17,32], ultimately resulting in reduced lipid concentrations. Additionally, considering the low concentrations of target LCMs detected in this study, this could be explained by low-dose exposure to LCMs inducing a stress state in humans, which may lead to rapid fatty acid oxidation and a subsequent reduction in lipid accumulation [42]. However, further investigation is required to elucidate these underlying molecular mechanisms.

Interestingly, while minimal and non-significant negative correlations were observed between individual LCMs and HDL-C levels in both the total population and subgroups, a consistent and statistically significant negative association was found between the LCM mixture index and HDL-C levels across all subgroups. This key discrepancy underscores the critical importance of conducting mixture analysis for environmental contaminants such as LCMs. Individual LCM effects on lipid metabolism may be weak, subtle, or masked when assessed in isolation, whereas the combined effect of multiple coexisting LCMs, which likely reflects either additive contributions or nonlinear interactions among components, can drive a robust collective association that cannot be detected by assessing individual compounds alone. Given that LCMs are inherently encountered as complex mixtures in real-world scenarios [17], this combined effect likely plays a critical role in the observed reduction in HDL-C levels. It is important to note that HDL-C plays a crucial role in preventing atherosclerosis and reducing the risk of CVD [43]. Therefore, it is imperative to consider the potential adverse effects of LCM exposure on the cardiovascular system, particularly when evaluating real-world exposure scenarios involving complex chemical mixtures rather than isolated compounds.

Our RCS curves indicate a complex, nonlinear relationship between LCMs and dyslipidemia, rather than a simple monotonically increasing or decreasing pattern. This suggests that there may be threshold effects of LCMs on lipid levels, and further studies are needed to elucidate these relationships. Furthermore, based on the gWQS model, we identified EtCBN, PFPT, and TPrECB as the most active components in the LCM mixture. PFPT and TPrECB contain C-F bonds, which are highly stable, while EtCBN contains a cyano group, an electron-withdrawing group that can make the benzene ring less reactive. Consistent with screening studies indicating that fluorinated and structurally stable LCMs exhibit greater persistence and bioaccumulation potential (e.g., higher log Kow and BCF values) [44,45], these chemical properties likely enhance

their environmental stability and accumulation in human tissues. This increased bioaccumulation may result in higher internal doses relative to other LCMs within the mixture, thereby contributing to their dominant weights in the gWQS model and the observed associations with dyslipidemia. However, there is currently a lack of toxicological studies elucidating the mechanisms underlying the effects of LCM exposure on dyslipidemia. Therefore, further investigations are warranted to explore both the non-linearity between these LCMs and lipid levels and their effects on lipid metabolism. Such studies will contribute to reducing the health burden associated with LCM exposure. Our findings underscore the overall dose-dependent effect of LCM exposure and reinforce the evidence linking dyslipidemia to LCM mixtures.

5. Strengths and limitations

This study is the first to analyze the overall impact of LCM mixtures on lipid levels and to identify the key chemicals responsible for dyslipidemia using the gWQS model. Furthermore, the RCS model was employed to assess the dose-response relationship between LCMs and lipid levels, providing fundamental data on the effects of LCM exposure on lipids. A key strength of this study is the application of advanced mixture modeling (gWQS) and non-linear modeling (RCS) to assess the associations between exposure to LCMs, an emerging class of environmental contaminants, and blood lipid levels. These methods are particularly well-suited for investigating the combined effects of complex chemical mixtures and capturing potential non-monotonic relationships, which would be overlooked by traditional single-chemical linear models. To our knowledge, this is the first study to apply these advanced analytical frameworks to LCMs, a class of emerging environmental contaminants with limited human health data.

However, this study has several limitations. First, as a cross-sectional study, it is difficult to establish a causal relationship between blood lipids and LCMs. Second, this study should be interpreted as a pilot investigation due to the relatively small sample size. While this limits statistical power and may increase the risk of false-negative or chance findings, it provides crucial preliminary human evidence for LCM-lipid associations. The low detection frequency of some LCMs further limits the generalizability of the results, highlighting the need for validation in larger cohorts. Third, previous studies have shown that the effects of compounds on lipid levels can vary across different age groups. the study population comprised two demographically distinct groups with significant differences in age and socioeconomic status. While we adjusted for these factors in multivariable models and observed consistent associations in subgroup analyses, the heterogeneity may still introduce residual confounding, particularly given that LCMs are persistent and bioaccumulative chemicals whose body burdens may vary with age and exposure history. Future studies with larger, more homogeneous cohorts are needed to disentangle the effects of age, location, and exposure sources. These studies could provide a basis for establishing exposure thresholds for LCMs at different ages in the future. Fourth, due to our limited understanding of the underlying mechanisms, further toxicological experiments are necessary to validate the results of this study. Finally, other chemicals associated with LCM exposure may influence the relationship between LCM exposure and changes in lipid levels. Therefore, future research should expand the screening to include other chemicals associated with LCM exposure to account for potential effects on lipid metabolism from co-existing substances.

6. Conclusions

In conclusion, we measured various LCM chemicals and blood lipid levels in serum samples from the general population in South of China. The high number of detected chemicals suggests that exposure to LCMs is widespread, and the potential exposure risks should not be ignored. Additionally, we are the first to report the dose-response effect of serum LCM chemical mixtures on dyslipidemia, particularly HDL-C, with EtCBN, PFPT, and TPrECB contributing the most to this relationship. These findings indicate that chronic exposure to low concentrations of LCM mixtures is associated with imbalance of blood lipids levels and may consequently increase the risk of cardiovascular disease. Our results provide a fundamental understanding the adverse health effects associated with LCMs and offer a scientific reference for developing preventive and intervention strategies against dyslipidemia induced by LCMs, thereby reducing the health burden associated with LCM exposure.

Supplementary data

The Supplementary Information contains detailed methodological descriptions for serum LCM quantification, including GC–MS/MS parameters for 57 target analytes, method performance metrics (recoveries, LODs, and LOQs), demographic characteristics of the study population, and extended statistical analyses of serum LCM concentrations and their associations with blood lipid.

Data availability statement

The data that support the findings of this study contain sensitive personal health information and precise GPS locations. To protect participant privacy and comply with ethical guidelines, these data are not publicly available. However, datasets may be available from the senior author upon reasonable request and subject to institutional review board approval.

Declaration of generative AI and AI-assisted technologies

During the preparation of this manuscript, the authors used generative AI tools only to improve language and readability. Specifically, the authors used ChatGPT and DeepSeek only for language polishing. The authors take full responsibility for the content of the manuscript.

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Authors' contribution

Lulu Huang: data curation, writing—original draft preparation; Zhengyang Yuan: writing—original draft preparation; Xue Cao: methodology, investigation; Lili Yao: formal analysis; Zulan Zhao: investigation; Li Li: methodology, data curation; Bairui Chen: data curation; Xinzhuo Hao:

data curation; Chun Xie: conceptualization, formal analysis, writing—review and editing; Qilong Liao: supervision, conceptualization, writing—review and editing. All authors have read and agreed to the published version of the manuscript.

Conflicts of interest

The authors declare no competing financial interests or personal relationships.

Ethical statement

The study was approved by the Medical Ethics Committee of Tongji Medical 557 College, Huazhong University of Science and Technology (Ethics Approval No. [2020] 558S284). All participants gave written informed consent.

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