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Association between exposure to flame retardants, methylmercury, phthalates, and perfluoroalkyl mixtures and depression in US adults

Qiao Zhang[1,a], Qiancheng Zhao[1,b], Mimi Xiao[1,c,*]

[1] Research Center for Medical and Social Development, School of Public Health, Chongqing Medical University, Chongqing, China
[a]1225866557@qq.com, [b]2410514926@qq.com, [c]Xiaomimi@cqmu.edu.cn
[*]Corresponding author

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Abstract: Environmental pollutants often coexist in a mixed form and have neurotoxicity, but previous studies have mostly focused on the association between a single or a certain type of pollutant and depression. This study systematically assessed the individual and mixed effects of environmental chemical mixtures on depressive symptoms using data from 2,986 adults in the 2017–2018 U.S. NHANES. Depression prevalence was 11.3%. Weighted generalized linear regression and restricted cubic splines (RCS) revealed significant associations: MBzP (β=0.05, 95% CI: 0.01–0.08), BCEP (β=−0.04, 95% CI: −0.06–−0.01), and TBBA (β=0.12, 95% CI: 0.07–0.18) showed positive correlations with depressive symptoms, while PFNA (β=−0.04, 95% CI: −0.07–−0.01) exhibited negative correlations, CH_3Hg (β=−0.11, 95% CI: −0.18–−0.04) was shown a negative correlation in the overall mixed effects. Nonlinear relationships were identified for PFNA and CH_3Hg. Mixed-effect models (weighted quantile sum regression and Bayesian kernel machine regression) demonstrated a dose-dependent positive association between chemical mixtures and depressive symptoms, with MBzP, PFNA, and TBBA as key contributors. The exposure-response function indicated increasing trends for MBzP and TBBA, decreasing trends for PFNA and CH_3Hg, and a flat trend for BCEP. Subgroup analyses highlighted different groups of people's effects: PFNA showed stronger associations in older adults (p=0.003) and individuals with high-fasting blood glucose (p=0.027), CH_3Hg in smokers (interaction p=0.017), and BCEP in other race populations (p=0.005). Findings suggest that mixed environmental chemicals exert individual and joint effects on depression, with threshold effects observed for PFNA, CH_3Hg, and BCEP.

1. Introduction

Major depressive disorder (MDD), affecting 280 million people worldwide, is a leading cause of

disability and is projected to be the top contributor to the global disease burden by 2030. In the United States, the annual prevalence of MDD exceeds 9%, with lifetime rates of 17% in males and 30% in females[1]. MDD stems from intricate interactions among social, psychological, and biological factors, and mounting evidence identifies environmental chemicals as potential pathogenic contributors[2]. Neurotoxic environmental pollutants have been linked to structural brain abnormalities and elevated depressive symptoms[3]. Given the challenges of quantifying external pollutant exposure, internal exposure biomarkers (excreted compounds or metabolites) are widely adopted to assess real in vivo exposure. This study selected five neurotoxic chemicals associated with depression: methylmercury (CH_3Hg), MBzP (monobenzyl phthalate), PFNA (perfluorononanoic acid), organophosphorus flame retardant BCEP, and novel brominated flame retardant TBBA, to explore the impacts of chemical mixtures on depressive symptoms.

PFNA, a prevalent endocrine disruptor, has a longer half-life than PFOS and PFOA[4], yet its linear/non-linear and mixture effects on depression remain understudied. A 2022 cohort study found that each log-unit increase in serum PFNA was associated with a 34% rise in PHQ-9 scores[5]. MBzP, the main metabolite of BBzP, enters the human body via consumer products, triggers neuroinflammation, and impairs neuroprotective pathways linked to depression[6], with limited mechanistic research on its association with depression severity. BCEP is a traditional flame retardant, while TBBA is an emerging alternative[7]; TBBA disrupts endocrine function[8], while BCEP impairs neurological activity[9]. Methylmercury, a bioaccumulative neurotoxin, alters depression-related biomarkers in animal models[10], and co-occurs with other target chemicals in the human body.

Most existing studies focus on single chemicals, ignoring real-world simultaneous mixture exposure, and findings on PFAS and methylmercury are inconsistent. This cross-sectional study used 2017–2018 NHANES data, combining biomonitoring and PHQ-9 assessments. We aimed to: (1) evaluate linear/nonlinear associations via weighted generalized linear regression and restricted cubic splines (RCS); (2) identify key mixture contributors using weighted quantile sum (WQS) regression; (3) explore population heterogeneity via subgroup analyses. All models were adjusted for covariates[11].

2. Materials and methods

2.1. Study population

This study analyzed associations between depressive symptoms and exposure to chemical mixtures among U.S. adults using 2017–2018 National Health and Nutrition Examination Survey (NHANES) data. The original sample included 9,254 participants; 3,761 individuals under 20 years old were excluded. We further excluded participants with missing data for marital status (n=16), education (n=31), alcohol intake (n=24), PHQ-9 scores (n=718), or target urinary/blood biomarkers (n=1717), yielding a final analytical sample of 2,986 U.S. adults with complete datasets (Figure 1).

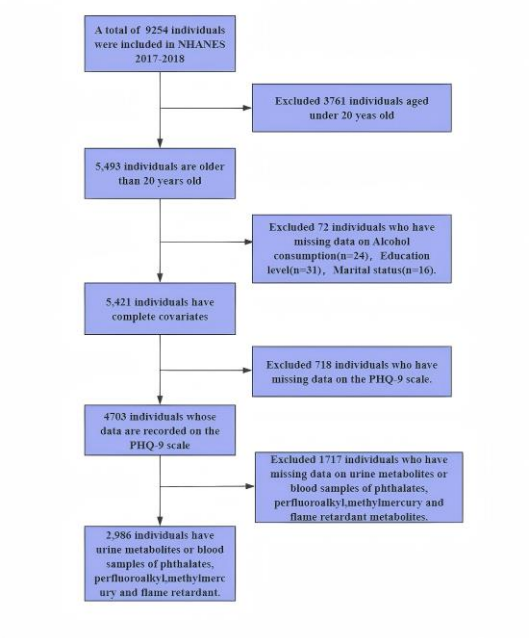

Figure 1. Flowchart of population included in our final analysis (N=2986).

2.2. Measurement of chemicals in urine and blood

Urine samples (for MBzP, BCEP, TBBA) and whole blood samples (for PFNA, methylmercury) were processed, stored, and shipped to the CDC National Center for Environmental Health (Atlanta, GA) for testing. Lower limits of detection (LLOD) were 0.1 ng/mL (PFNA, BCEP), 0.26 μg/L (CH_3Hg), 0.05 ng/mL (TBBA), and 0.3 ng/mL (MBzP). Concentrations below LLOD were imputed as LLOD/√2, consistent with NHANES protocols and standard environmental epidemiology method.

2.3. Measures of PHQ-9

Depressive symptoms were measured using the Patient Health Questionnaire-9 (PHQ-9), a 9-item scale assessing symptom frequency over the prior two weeks[12]. Severity was stratified into four groups: 0–4 (none), 5–9 (mild), 10–14 (moderate), and ≥15 (severe). Due to right skewness, continuous PHQ-9 scores were natural log-transformed (score + 2) to approximate normality for statistical analyses.

2.4. Covariates

Potential confounders were adjusted in all models, including sociodemographic factors (gender, age group, race/ethnicity, education, marital status) and lifestyle factors (serum cotinine-defined smoking status, alcohol consumption: non-drinkers, moderate drinkers, heavy drinkers).

2.5. Statistical analyses

Sample weights were applied per NHANES guidelines to account for complex survey design. Continuous variables were shown as means and categorical variables as counts/percentages; all five

chemical concentrations were log-transformed for skewness. We used weighted generalized linear regression, restricted cubic splines, and weighted quantile sum (WQS) regression (60% training/40% validation data) to assess single, nonlinear, and mixture effects. A priori subgroup analyses by race, age, gender, lifestyle, and hyperglycemia were conducted to explore population-specific vulnerability.

3. Results

3.1. Baseline characteristics of the study participants

Baseline demographic data for 2,986 participants are summarized in Table 1. The overall depression prevalence was 11.3%, with severity stratification: 67.5% no depression (0–4 points), 21.3% mild (5–9), 9.3% moderate (10–14), and 1.9% severe (≥15). Significant between-group differences ($P < 0.001$) were observed for cotinine levels, education, marital status, and gender. Females, unmarried individuals, those with higher cotinine, and lower education were overrepresented in the higher depression severity groups.

Table 1. Characteristics of adult participants with depressive symptoms in NHANES (N=2986), USA, 2017-2018.

	No depression (0-4) (N=2015)	**Mild depression (5-9) (N=635)**	**Moderate depression (10-14) (N=279)**	**Severe depression (≥15) (N=57)**	**overall (N=2986)**	**P-value**
Gender[a]						
Male	991 (49.2%)	239 (37.6%)	100 (35.8%)	25 (43.9%)	1355 (45.4%)	**<0.001**
Female	1024 (50.8%)	396 (62.4%)	179 (64.2%)	32 (56.1%)	1631 (54.6%)	
Age[a]						
<40	772 (38.3%)	261 (41.1%)	107 (38.4%)	23 (40.4%)	1163 (38.9%)	0.956
≥40,<60	652 (32.4%)	204 (32.1%)	91 (32.6%)	16 (28.1%)	963 (32.3%)	
≥60	591 (29.3%)	170 (26.8%)	81 (29.0%)	18 (31.6%)	860 (28.8%)	
Ethnicity[a]						
Mexican American	262 (13.0%)	79 (12.4%)	36 (12.9%)	8 (14.0%)	385 (12.9%)	0.125
Non-Hispanic White	662 (32.9%)	228 (35.9%)	94 (33.7%)	29 (50.9%)	1013 (33.9%)	
Non-Hispanic Black	474 (23.5%)	168 (26.5%)	74 (26.5%)	9 (15.8%)	725 (24.3%)	
Other races	617 (30.6%)	160 (25.2%)	75 (26.9%)	11 (19.3%)	863 (28.9%)	
Alcohol consumption[a]						
Never	422 (20.9%)	148 (23.3%)	64 (22.9%)	14 (24.6%)	648 (21.7%)	0.682
Moderate	996 (49.4%)	295 (46.5%)	138 (49.5%)	32 (56.1%)	1461 (48.9%)	
Heavy	597 (29.6%)	192 (30.2%)	77 (27.6%)	11 (19.3%)	877 (29.4%)	
Marital status[a]						
Married	1137 (56.4%)	258 (40.6%)	135 (48.4%)	26 (45.6%)	1556 (52.1%)	**<0.001**
Unmarried	549 (27.2%)	214 (33.7%)	87 (31.2%)	18 (31.6%)	868 (29.1%)	
others	329 (16.3%)	163 (25.7%)	57 (20.4%)	13 (22.8%)	562 (18.8%)	
Cotinine level[a]						
Below LLOD	713 (35.4%)	186 (29.3%)	49 (17.6%)	15 (26.3%)	963 (32.3%)	**<0.001**
Above LLOD	1302 (64.6%)	449 (70.7%)	230 (82.4%)	42 (73.7%)	2023 (67.7%)	
Education level[a]						
<High school	389 (19.3%)	184 (29.0%)	74 (26.5%)	20 (35.1%)	667 (22.3%)	**<0.001**
High school	466 (23.1%)	154 (24.3%)	63 (22.6%)	17 (29.8%)	700 (23.4%)	

≥High school	1160 (57.6%)	297 (46.8%)	142 (50.9%)	20 (35.1%)	1619 (54.2%)	
PFNA[b]						
Mean (SD)	-1.05 (0.981)	-1.21 (0.942)	-1.14 (0.974)	-1.16 (0.859)	-1.09 (0.972)	0.007
BCEP[b]						
Mean (SD)	-1.01 (1.30)	-1.04 (1.29)	-0.611 (1.29)	-0.910 (1.26)	-0.980 (1.30)	**<0.001**
Mercury, methyl[b]						
Mean (SD)	-0.784 (1.06)	-1.04 (0.883)	-1.07 (0.889)	-1.01 (0.724)	-0.869 (1.01)	**<0.001**
TBBA[b]						
Mean (SD)	-3.28 (0.283)	-3.12 (0.561)	-3.22 (0.460)	-3.11 (0.646)	-3.24 (0.391)	**<0.001**
MBzP[b]						
Mean (SD)	1.12 (1.35)	1.65 (1.38)	1.91 (1.37)	1.98 (1.26)	1.32 (1.39)	**<0.001**
Glycated hemoglobin[a]						
Below LLOD	1780 (88.3%)	567 (89.3%)	230 (82.4%)	49 (86.0%)	2626 (87.9%)	**0.048**
Above LLOD	235 (11.7%)	68 (10.7%)	49 (17.6%)	8 (14.0%)	360 (12.1%)	

a Represented comparison by chi-square tests.
b Represented comparison by Kruskal-Wallis test.

3.2. Single chemical exposure and depression

The multicollinearity test indicates that multicollinearity is minimal (all variance inflation factors are less than 10). The results of the weighted generalized linear regression are shown in Table 2, In quartile regression, Q1 is always used as the reference point. PFNA showed significant inverse associations in crude and lifestyle-adjusted models, with a linear trend across quartiles (P-trend = 0.032). BCEP was inversely linked to depression in all adjusted models (Model 4: β = -0.04, P = 0.019). TBBA exhibited consistent positive associations (Model 4: β = 0.12, P = 0.006) with a clear dose-response pattern (P-trend < 0.001). CH_3Hg had an inverse crude association that weakened in adjusted models, though quartile comparison remained significant (Q4 vs Q1: β = -0.11, P = 0.003). MBzP was positively associated with depression in fully adjusted models (β = 0.05, P = 0.036), with a strong dose-response relationship. Restricted cubic spline models confirmed nonlinear inverted U-shaped associations for PFNA (P-nonlinear = 0.041) and CH_3Hg (P-nonlinear = 0.032), with inflection points at -0.92 and -1.2, respectively (Figure 2a-c).

Table 2. Association between exposure to a single compound and depression (N=2986), NHANES, USA, 2017–2018.

Variables	Model1		Model2		Model3		Model4	
	β (95%CI)	P-value	β (95%CI)	P-value	β (95%CI)	P-value	β (95%CI)	P-value
PFNA	-0.04 (-0.07 ~ -0.01)	**0.019**	-0.02 (-0.06 ~ 0.03)	0.397	-0.04 (-0.07 ~ -0.01)	**0.032**	-0.02 (-0.07 ~ 0.04)	0.392
Q2	-0.02 (-0.09 ~ 0.05)	0.655	0.03 (-0.03 ~ 0.10)	0.328	-0.01 (-0.08 ~ 0.06)	0.775	0.04 (-0.03 ~ 0.10)	0.319
Q3	-0.06 (-0.13 ~ 0.01)	0.09	0.02 (-0.05 ~ 0.09)	0.532	-0.06 (-0.13 ~ 0.01)	0.11	0.02 (-0.05 ~ 0.09)	0.57
Q4	-0.17 (-0.24 ~ -0.10)	**<.001**	-0.07 (-0.14 ~ 0.01)	0.073	-0.16 (-0.23 ~ -0.09)	**<.001**	-0.07 (-0.14 ~ 0.01)	0.074
P for trend	**<.001**		**0.046**		**<.001**		**0.042**	

BCEP	-0.03 (-0.05 ~ -0.01)	**0.004**	-0.03 (-0.06 ~ -0.01)	**0.008**	-0.03 (-0.05 ~ -0.02)	**0.004**	-0.04 (-0.06 ~ -0.01)	**0.022**
Q2	-0.02 (-0.09 ~ 0.05)	0.519	-0.02 (-0.08 ~ 0.05)	0.646	-0.05 (-0.12 ~ 0.03)	0.207	-0.03 (-0.10 ~ 0.03)	0.317
Q3	0.07 (-0.00 ~ 0.14)	0.057	0.05 (-0.02 ~ 0.11)	0.192	0.05 (-0.02 ~ 0.12)	0.195	0.03 (-0.04 ~ 0.10)	0.386
Q4	0.09 (0.02 ~ 0.16)	**0.013**	0.07 (-0.00 ~ 0.14)	0.052	0.06 (-0.01 ~ 0.13)	0.107	0.04 (-0.02 ~ 0.11)	0.207
P for trend	**<.001**		**0.009**		**0.003**		**0.042**	
TBBA	0.12 (0.08 ~ 0.16)	**<.001**	0.12 (0.08 ~ 0.17)	**<.001**	0.12 (0.08 ~ 0.17)	**<.001**	0.12 (0.07 ~ 0.18)	**0.006**
Q2	0.04 (-0.03 ~ 0.11)	0.283	0.03 (-0.04 ~ 0.10)	0.343	0.04 (-0.03 ~ 0.11)	0.29	0.03 (-0.03 ~ 0.10)	0.333
Q3	0.05 (-0.02 ~ 0.12)	0.194	0.05 (-0.02 ~ 0.12)	0.179	0.04 (-0.03 ~ 0.11)	0.233	0.05 (-0.02 ~ 0.11)	0.19
Q4	0.15 (0.08 ~ 0.22)	**<.001**	0.15 (0.08 ~ 0.22)	**<.001**	0.15 (0.08 ~ 0.22)	**<.001**	0.14 (0.08 ~ 0.21)	**<.001**
P for trend	**<.001**		**0.005**		**0.007**		**<.001**	
CH3HG	-0.03 (-0.06 ~ -0.01)	**0.028**	-0.02 (-0.05 ~ 0.01)	0.124	-0.02 (-0.05 ~ 0.01)	0.076	-0.02 (-0.05 ~ 0.02)	0.234
Q2	-0.03 (-0.10 ~ 0.04)	0.438	-0.03 (-0.09 ~ 0.04)	0.46	-0.03 (-0.10 ~ 0.04)	0.468	-0.02 (-0.09 ~ 0.05)	0.507
Q3	-0.03 (-0.10 ~ 0.04)	0.344	-0.01 (-0.08 ~ 0.06)	0.815	-0.03 (-0.10 ~ 0.04)	0.467	-0.00 (-0.07 ~ 0.07)	0.942
Q4	-0.19 (-0.26 ~ -0.12)	**<.001**	-0.12 (-0.19 ~ -0.05)	**<.001**	-0.17 (-0.24 ~ -0.10)	**<.001**	-0.11 (-0.18 ~ -0.04)	**0.003**
P for trend	**<.001**		0.062		**0.001**		0.133	
MBzP	0.05 (0.02 ~ 0.08)	**0.005**	0.05 (0.01 ~ 0.08)	**0.014**	0.05 (0.02 ~ 0.08)	**0.008**	0.05 (0.01 ~ 0.08)	**0.036**
Q2	0.09 (0.02 ~ 0.16)	**0.014**	0.10 (0.03 ~ 0.16)	**0.005**	0.08 (0.01 ~ 0.15)	**0.021**	0.09 (0.02 ~ 0.16)	**0.007**
Q3	0.21 (0.15 ~ 0.28)	**<.001**	0.20 (0.14 ~ 0.27)	**<.001**	0.20 (0.13 ~ 0.27)	**<.001**	0.19 (0.13 ~ 0.26)	**<.001**
Q4	0.37 (0.30 ~ 0.43)	**<.001**	0.33 (0.26 ~ 0.40)	**<.001**	0.34 (0.27 ~ 0.41)	**<.001**	0.31 (0.25 ~ 0.38)	**<.001**
P for trend	**<.001**		**<.001**		**<.001**		**<.001**	

The term "P for trend" indicates that the trend increases by quartiles. Statistically significant findings are shown in red.
Model1: Crude, Model2: Adjust: Gender, Age, Ethnicity, Marital status, Education level, Model3: Adjust: Alcohol consumption, Cotinine level, Model4: Adjust: Gender, Age, Ethnicity, Marital status, Education level, Alcohol consumption, Cotinine level.

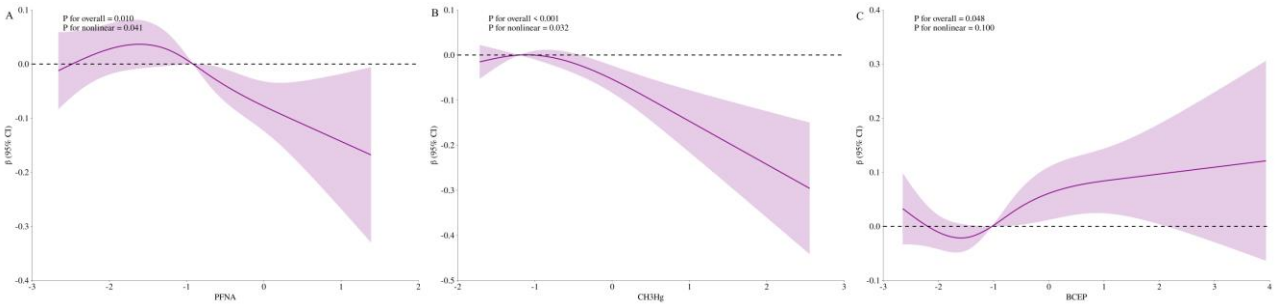

Figure 2a-c. Dose-response relationship between chemical compound and depression in RCS model. The purple area indicating the 95% CI.

3.3. Mixed chemical exposure and depression (WQS model)

As shown in Figure 3, the fully adjusted Weighted Sum of Quartiles (WQS) model indicates that MBzP (weighted at 0.55) and TBBA (weighted at 0.38) are the primary factors contributing to the risk of depression in the mixture.

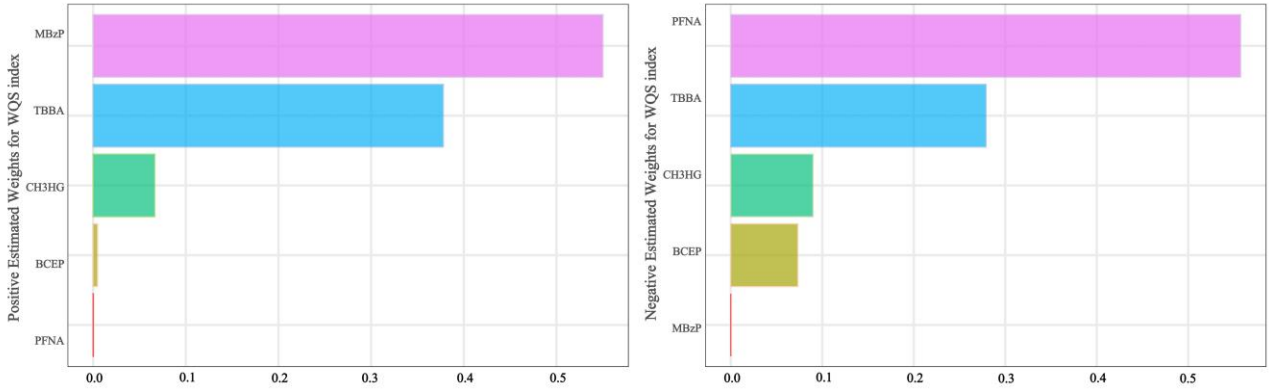

Figure 3. WQS model index weights for depression. Left illustrates the weights in the positive direction, while Right depicts those in the negative direction.

3.4. Subgroup analysis

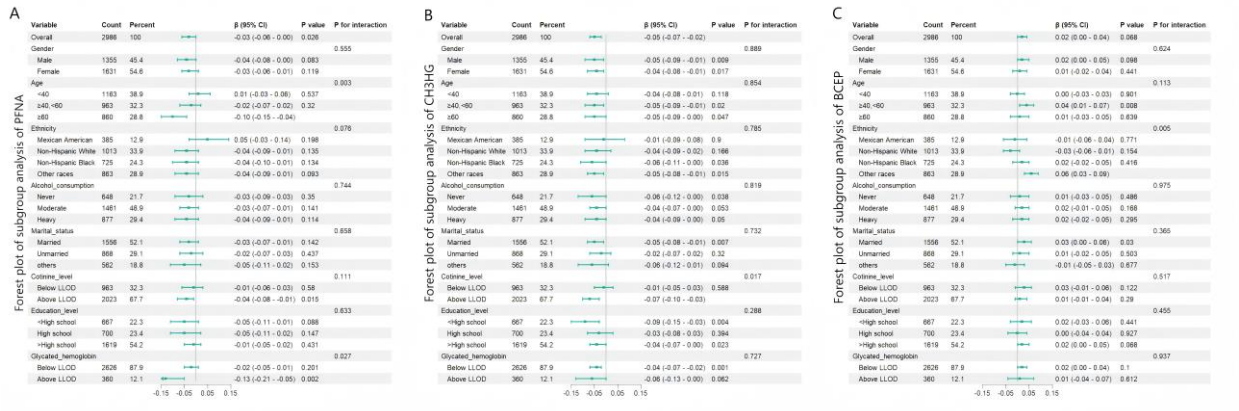

Figure 4a-c. Forest plot of subgroup analysis. HR: hazard ratio; 95% CI: 95% confidence interval.

Overall, PFNA and CH_3Hg were inversely associated with depression, while BCEP showed no overall significance (Figure 4a-c). Significant effect modifications were detected: CH_3Hg in smoking subgroups (interaction P = 0.017), BCEP across racial groups (interaction P = 0.005), and PFNA among older and hyperglycemic participants (age interaction P = 0.003; hyperglycemia interaction P = 0.027).

4. Discussions

This multi-model analysis clarifies environmental chemical mixture effects on depression, identifying MBzP and TBBA as core positive risk drivers, with PFNA exerting significant negative associations and CH_3Hg/BCEP exhibiting nonlinear U-shaped trends. PFNA's negative interactions resolve conflicting prior literature on PFAS-depression associations, as effect direction depends on mixture context and exposure thresholds[13]. Endocrine-disrupting chemicals (EDCs) including phthalates and flame retardants drive neuroinflammation and hormonal disruption[14], linking to depression pathogenesis[15]. Emerging flame retardants (TBBA, BCEP) are widely used but understudied[16]; we found TBBA strongly predicts depression risk, while BCEP shows population-specific effects (racial interaction P=0.005).

MBzP dominated mixture effects via oxidative stress and inflammatory pathways[17], consistent with prior single-chemical findings. CH_3Hg's inverted U-shape may reflect selenium antagonism from fish intake, a novel mechanism requiring further validation[18]. PFNA exerted protective effects in older/hyperglycemic subgroups, highlighting population heterogeneity[19]. Most prior studies focus on single compounds with linear models[20], missing complex interactions; our integrated RCS-WQS-BKMR framework captures nonlinearities and mixture contributions, overcoming WQS limitations via cross-validation[21].

Limitations include the cross-sectional design restricting causal inference, residual confounding, and self-reported depression scales. Despite this, this study advances environmental epidemiology by disentangling multi-class chemical effects, providing mechanistic insights and population-specific risk data for public health interventions.

5. Conclusions

This NHANES multi-model analysis links environmental chemical mixtures to depressive symptoms in U.S. adults. MBzP and TBBA are key positive risk drivers; PFNA and CH_3Hg exhibit negative nonlinear associations (particularly pronounced in older adults and those with hyperglycemia), while BCEP has race-specific effects. Mixed exposure increases the risk of depression, and public health interventions targeting mixed exposure should be implemented. Future longitudinal mechanistic studies are needed to confirm causality.

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