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Research article

Associations between mixed exposure to phthalates and latent tuberculosis infection among the general U.S. population from NHANES 2011–2012

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ABSTRACT

Background: People are constantly exposed to phthalates, but few reliable studies have focused on the connection between phthalate exposure and latent tuberculosis infection (LTBI).

Methods: Data were obtained from the National Health and Nutrition Examination Survey (NHANES) database (2011–2012). The LTBI was assessed by QuantiFERON®-TB Gold-In-Tube (QFT) or tuberculin skin testing (TST). The odds ratios (ORs) and 95% confidence intervals (CIs) per log10 unit change in the concentration of phthalate metabolites were calculated using crude and adjusted logistic regression models. The relationships between mixed phthalate concentrations and LTBI were assessed using Bayesian kernel machine regression (BKMR) models.

Results: According to the results of the multivariable logistic regression, in a fully adjusted model, only monobenzyl phthalate (MBZP) was negatively associated with LTBI in Q3 (OR (95% CI): 0.485 (0.286,0.823), $P = 0.007$). According to the restricted cubic spline (RCS) model, there was a linear dose–response association between all 11 phthalate metabolites and LTBI (p for nonlinearity >0.05). We found a significant positive correlation between mixed phthalate metabolites and LTBI by using fully adjusted BKMR model.

Conclusions: Our analysis demonstrated that LTBI in the general U.S. population is linearly linked with exposure to single or combined phthalates.

1. Introduction

Tuberculosis (TB) is the main infectious illness and the cause of massive mortality worldwide. The Global Tuberculosis Report 2022 reveals that 10.6 million people worldwide infected with TB in 2021, or 134 cases per 100,000 people. Geographically, the Western Pacific (18%), Southeast Asia (45%), and Africa (23%) had the highest percentages of TB cases in 2021, followed by the Eastern Mediterranean (8.1%), the Americas (2.9%), and Europe (2.2%) [1]. According to several studies, 5–10% of those who have *Mycobacterium tuberculosis* (*M. tuberculosis, Mtb*) infection may develop active *TB*. The other people either will not have *Mtb* or will acquire a

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chronic immune response to *Mtb* antigens by staying asymptomatic and noncontagious [2]. However, the latter outcome, known as latent tuberculosis infection (LTBI), may result in the development of TB in certain cases in the future. Therefore, programmatic diagnosis and management of LTBI are crucial for TB control [3,4].

Phthalates are commonly used as plasticizers to soften and improve the flexibility of polyvinyl chloride (PVC) plastics and have been widely detected in food packaging and cosmetics in the U.S [5]. The applications of phthalates might vary depending on their molecular weight, which can be divided into high-molecular-weight (HMW) and low-molecular-weight (LMW) phthalates [6]. Because of their diverse structural makeup, phthalates have significantly varying toxicity characteristics [7]. Both *in vitro* and *in vivo* studies have shown that long-term exposure to phthalates causes oxidative stress in mammals, with detrimental consequences for their reproductive, respiratory, circulatory, and central nervous systems [8]. The coexistence of phthalates (PAEs) and perfluoroalkyl substances (PFASs) promotes the growth of potentially human pathogenic bacteria, including *Mtb* and *Klebsiella pneumoniae*, which increases the risk of related diseases in exposed populations [9]. The pro-oxidative and inflammatory qualities of phthalates, which can negatively influence the respiratory system, may be the cause of the relationship between phthalate exposure and low lung function [10,11]. Byrne et al. reported that pulmonary sequelae of TB may be an important factor in the risk of COPD, particularly in TB-endemic areas and young people [12]. Yii et al. reported that asthma or sinus disease modulates the immune response and reduces the incidence of active tuberculosis in adults [13]. Thus, we speculate that phthalates may damage the respiratory system and increase susceptibility to infection by *Mtb*. To further understand the connection between single or combined phthalate exposure and LTBI, studies in large populations may need to use a more flexible methodology.

Urinary phthalate metabolite levels are suitable proxies for daily human exposure to phthalates since phthalates are quickly eliminated through biotransformation and excretion (leading to minimal bioaccumulation) [14,15]. The National Health and Nutrition Examination Survey (NHANES) cycle data from 2011 to 2012 were used in this study to examine the association between LTBI and 11 phthalate metabolites in urine. We utilize restricted cubic spline (RCS) models, multivariate logistic regression and Bayesian nuclear machine regression (BKMR) to evaluate the nonlinear dose–response, single-effect and overall effects of phthalates, respectively. The above models thoroughly evaluate the effects of phthalate exposure on LTBI.

2. Materials and methods

2.1. Study population

All the data in our study are publicly available from the NHANES website (https://wwwn.cdc.gov/nchs/nhanes/Default.aspx). The NHANES is a cross-sectional programconducted by the Centers for Disease Control (CDC) in the U.S, which includes questionnaires, diet, physiological measurements and laboratory tests supervised by trained medical staff. All participants provided written informed consent, and institutional review board approval was obtained from the NCHS to conduct the surveys.

The present analyses were based on participants containing data on LTBI, which recruited from 2011 to 2012. We excluded 3560

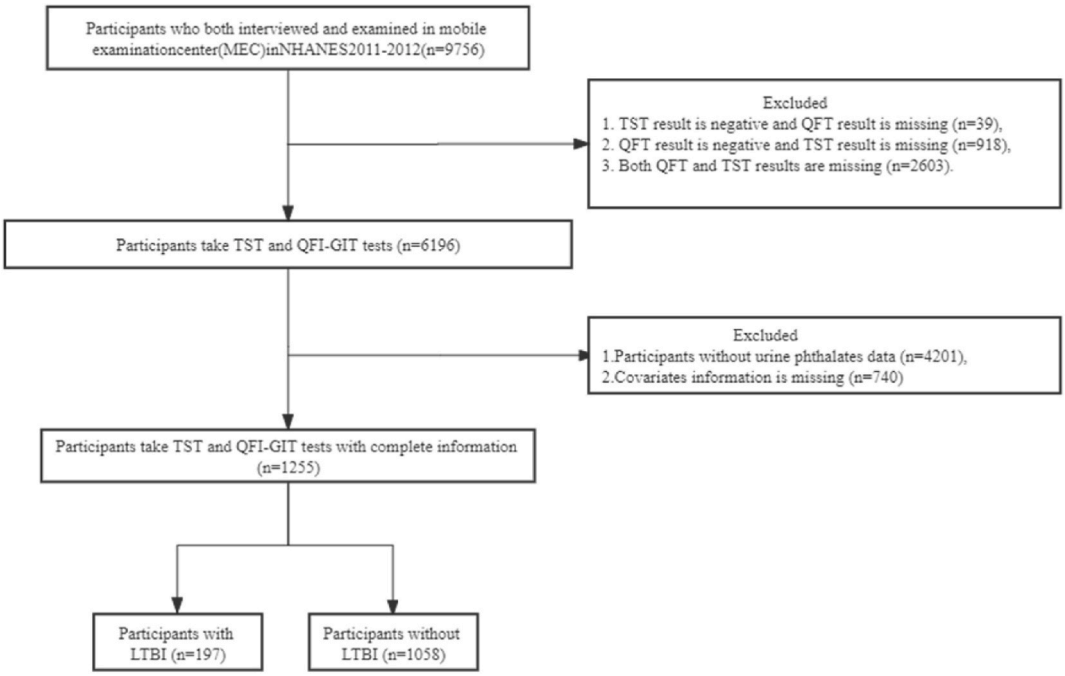

Fig. 1. Flow diagram of eligible study participants from NHANES 2011–2012.

participants without a QuantiFERON-TB® gold test (QFT) or tuberculin skin test (TST) data and 4201 participants without phthalate metabolites. 1255 participants who fit into our study were included in the final analysis, and the detailed screening process is shown in Fig. 1.

2.2. Measurement and definitions

2.2.1. QFT-GIT and TST

On the palmar side of each participant's arm, NHANES-trained phlebotomists or doctors administered the tuberculin antigen injection. A doctor who was not familiar with the participant's medical history or had any history of exposure to TB assessed the TST reaction after 46–76 h, and a positive result was classified as an induration size ≥10 mm. The QuantiFERON-TB Gold test must meet the following criteria: the Nil value must be ≤ 8.0 IU gamma interferon (IF)/mL; the TB antigen value minus the Nil value must be ≥ 0.35 IU gamma interferon (IF)/mL. LTBI was diagnosed based on a positive result of TST or QFT [16].

2.2.2. Quantification of chemicals in urine phthalate metabolites

One-third of single spot urine samples were used to test phthalate metabolites. After being collected, these spot urine samples were frozen at −20 °C and sent to the National Center for Environmental Health for study of different phthalate metabolites. Phthalate analysis was conducted using tandem mass spectrometry, electrospray ionization, and high-performance liquid chromatography. Additional information on the laboratory protocols can be found elsewhere [17].

Based on a detection frequency >70%, we selected 11 phthalate metabolites, which included mono(carboxynonyl) phthalate (MCNP), monoethyl phthalate (MEP), monobutyl phthalate (MBP), mono(3-carboxypropyl) phthalate (MCPP), mono(2-ethylhexyl) phthalate (MEHP), monobenzyl phthalate (MBZP), mono-2-ethyl-5-carboxypentyl phthalate (MECPP), mono(2-ethyl-5-oxohexyl) phthalate (MEOHP), mono(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP) and monocarboxyoctyl phthalate (MCOP). MECPP, MEHHP, and MEOHP are metabolites of DEHP. MiBP, MCOP, MEP, and MBZP are metabolites of diisobutyl phthalate (DiBP), diisononyl phthalate (DNP), diethyl phthalate (DEP), and benzylbutyl phthalate (BzBP), respectively. MCPP is a metabolite of di-*n*-octyl phthalate (DOP), dibutyl phthalate (DBP), and other HMW phthalates [18].

2.3. Covariates

Using a directed acyclic graph (DAG), we were able to locate sociodemographic, lifestyle, medical history, and investigation-specific characteristics that could influence LTBI or urine phthalate metabolite concentrations (Fig. S1). Age, sex, body mass index (BMI), race, education, marital status, hypertension status, hyperlipidaemia status, and asthma status were all derived from NHANES data.

2.4. Statistical analysis

Due to the complexity of sample methods, NHANES weights was considered into statistical calculation. The Shapiro–Wilk statistical test was used to confirm normal distribution of continuous variable. The levels of eleven log10-transformed phthalate metabolites were calculated along with the Spearman correlation coefficient for all phthalate metabolite levels. Wilkoxon and Kruskal–Wallis tests were used to compare 11 phthalate metabolites between LTBI and nonraw patients. The mean (standard error) was used to represent continuous data, whereas n (%) was used to describe categorical variables. To examine continuous and categorical component differences, the sample weighted *t*-test and weighted chi-square test were employed, respectively.

2.5. Analysis of single phthalate exposure

To evaluate the correlation between single phthalate metabolites and LTBI, multivariate logistic regression models were applied. Levels of phthalate metabolite were divided into quartiles, and we conducted two models to evaluate the effects of phthalate metabolite levels. Model 1 was crude and fitted with LTBI and phthalate metabolites. In Model 2, we adjusted for age, sex, race, education, smoking status, hypertension, hyperlipidemia, asthma, BMI, and urine creatinine. The odds ratio (OR) and 95% confidence interval (CI) were used to determine the degree of the association.

RCS models were utilized to disclose the dose–response relationship between phthalate metabolites and LTBI. We selected knots based on the Akaike information criterion (AIC) and used the RCS by sex to explore the relationship between phthalate metabolites and LTBI.

2.6. Analysis of mixed phthalate exposure

The BKMR flexibly and concisely estimates the multivariate exposure–response function and is therefore widely used in epidemiological studies of mixed environmental exposure [19]. BKMR was used to analyse the mixed effect of 11 phthalate mixtures on LTBI. Due to binary variables exist in this study, we adapted the variable selection model by a probit regression, which has been demonstrated to have great shrinkage properties in previous researches. In this study, we divided 11 phthalate metabolites into 4 groups based on Spearman correlations and their precursors. The specific BKMR model is:

Y^*_i = h[Group1=(MCOP, MCPP, MCNP), Group2=(MBP, MBZP, MiBP), Group3=(MEHHP, MEHP, MEOHP, MECPP), Group4

(MEP)]+ $x_i\beta+\epsilon_i$. where i = 1 … n is the individual in the participants, Y* stands for the potential normal random variable transformed by the health outcome (Y), and h [·] represents the exposure–response function, where x and β are the confounding factors and their coefficients, respectively, and ε is the residual that follows the normal distribution. A total of 10,000 iterations of Markov chain Monte Carlo (MCMC) sampling of BKMR were performed to scale all phthalate metabolites to improve sampling efficiency.

In order to adjust the results of the logistic regression, an environmental subset sample weight was used to illustrate complex of survey sampling. The statistical analysis was conducted via SPSS 26 software, and R 4.1.3 software was used to construct the model. $P < 0.05$ was considered to indicate a significant difference.

3. Results

3.1. Population characteristics

A total of 1255 participants were included in this study. The prevalence of LTBI was 18.6%. The mean age and urine creatinine level were 46.9 ± 16.8 years and 112.4 ± 74 mg/dL, respectively. Compared to participants with negative LTBI, positive participants were more likely to be older, male, married, have a low level of education and have a greater incidence of hyperlipidaemia ($P < 0.05$). There were no statistically significant differences in BMI, smoking status, or the incidence of hypertension or asthma between the two groups ($P > 0.05$) (Table 1).

3.2. Distribution and correlation of phthalate metabolites

The levels of phthalates in the urine of the population are shown in Table S1. According to the NHANES, if the phthalate metabolite levels were below the lower limit of detection (LLOD), the values were the square root of the lower limit of detection divided by 2 (LLOD/sqrt (2)). We also compared the concentrations among the participants with positive or negative LTBI, as shown in Table S2. MCOP, MBP, MCPP, MEHHP, MEHP, MEOHP, MBZP, MiBP, MCNP and MECPP were significantly lower in LTBI-positive patients than

Table 1
Baseline characteristics of participants by LTBI status, NHANES 2011–2012 (n = 1255).

	Total (n = 1255)	LTBI result		P value
		Negative (n = 1058)	Positive (n = 197)	
Age (years)	46.9 (16.8)	46.6 (16.9)	49.8 (15.7)	0.016
BMI (kg/m^2)				0.272
<25	410 (32.7)	339 (32.0)	71 (36.0)	
≥25	845 (67.3)	719 (68.0)	126 (64.0)	
Gender, n (%)				0.015
Male	633 (60.4)	518 (48.9)	115 (58.4)	
Female	622 (49.6)	540 (51.0)	82 (41.6)	
Race/ethnicity, n (%)				<0.001
Mexican American	116 (9.2)	84 (7.9)	32 (16.2)	
Other Hispanic	134 (10.7)	102 (9.6)	32 (16.2)	
Non-Hispanic White	482 (38.4)	457 (43.2)	25 (12.7)	
Non-Hispanic Black	317 (25.3)	268 (25.3)	49 (24.9)	
Other race	206 (16.4)	147 (13.9)	59 (29.9)	
Marital, n (%)				0.007
Married	616 (49.1)	506 (47.8)	110 (55.8)	
Widowed	93 (7.4)	73 (6.9)	20 (10.2)	
Divorced/separated	166 (13.2)	139 (13.1)	27 (13.7)	
Never married	380 (30.3)	340 (32.1)	40 (20.3)	
Education, n (%)				0.001
<High school graduate	270 (21.5)	208 (19.7)	62 (31.5)	
High school graduate/GED	252 (20.1)	219 (20.7)	33 (16.8)	
Some college or above	733 (58.4)	631 (59.6)	102 (51.8)	
Smoking status, n (%)				0.091
YES	524 (41.8)	431 (40.7)	93 (47.2)	
NO	731 (58.2)	627 (59.3)	104 (52.8)	
Hypertension, n (%)				0.400
YES	451 (35.9)	375 (35.4)	76 (38.6)	
NO	804 (64.1)	683 (64.6)	121 (61.4)	
Hyperlipidemia, n (%)				0.001
YES	439 (35.0)	349 (33.0)	90 (45.7)	
NO	816 (65.0)	709 (67.0)	107 (54.3)	
Asthma, n (%)				0.432
YES	182 (14.5)	157 (14.8)	25 (12.7)	
NO	1073 (85.5)	901 (85.2)	172 (87.3)	
Urine creatinine (mg/dL)	112.4 (74.0)	114.1 (74.4)	96.2(68.5)	0.049

Abbreviations: LTBI: latent tuberculosis infection, NHANES: National Health and Nutrition Examination Survey, BMI: body mass index.

in LTBI-negative patients ($P < 0.001$). However, the MEP of LTBI-positive patients was significantly greater than that of LTBI-negative patients ($P < 0.001$).

3.3. The correlation of phthalate metabolites

Fig. 2 shows a positive correlation between 11 phthalate metabolites. MEHHP was strongly correlated with MEOHP and MECPP (r = 0.97, $P < 0.001$; r = 0.90, $P < 0.001$, respectively). Moreover, MECPP was strongly correlated with MEOHP (r = 0.90, $P < 0.001$). MCOP was significantly correlated with MCPP (r = 0.74, $P < 0.001$) and MCNP (r = 0.70, $P < 0.001$), and MEHHP was correlated with MEHP (r = 0.78, $P < 0.001$), MBP (r = 0.67, $P < 0.001$) and MiBP (r = 0.65, $P < 0.001$). Furthermore, MiBP was strongly correlated with MBP (r = 0.81, $P < 0.001$), MBZP (r = 0.64, $P < 0.001$) and MEOHP (r = 0.66, $P < 0.001$).

3.4. The association of single phthalate metabolites with LTBI

The correlation between the single phthalate metabolites and LTBI is shown in Fig. 3. According to the fully adjusted model, only MBZP was negatively associated with LTBI in Q3 (OR (95% CI): 0.485 (0.286, 0.823), $P = 0.007$) (Fig. 3B). Moreover, we found that the MEP concentration in Q3 (OR (95% CI): 2.293 (1.071, 4.909), $P = 0.033$) was positively correlated with LTBI in females, while the MBZP concentration in Q3 (OR (95% CI): 0.437 (0.196, 0.974), $P = 0.043$) was negatively correlated with LTBI (Fig. S2B). The above conclusions were not reached for LTBI in males. However, we discovered a significant correlation between MCNP in Q3 and LTBI in males (OR (95% CI): 0.352 (0.171, 0.726), $P = 0.005$) (Fig. S3B).

We utilize adjusted RCS models to described the dose–response relationship between a single phthalate metabolite and LTBI (Fig. S4). The results showed that all phthalate metabolites were significantly correlated with LTBI ($P < 0.001$ for overall correlation; Table S3), and MBP ($P = 0.044$), MEP ($P = 0.034$), MEHP ($P = 0.009$), MBZP ($P = 0.049$), MiBP ($P = 0.042$) and MECPP ($P = 0.008$) were significantly correlated with LTBI ($P < 0.05$). We also described the dose–response relationship of a single phthalate metabolite to LTBI according to sex (Fig. S5). For all phthalate metabolites, males were more susceptible to LTBI than females were. No proof of nonlinear association was found between phthalate metabolites and the OR of LTBI other than MECPP ($P < 0.05$ for the nonlinear test). Table S3 provides more information about the RCS models.

3.5. Estimation of the joint effect of phthalate metabolites on LTBI

We found a significant positive correlation between mixed phthalate metabolites and LTBI compared to the 25th and 50th percentiles by using fully adjusted BKMR model (Fig. 4). The group posterior inclusion probability (groupPIP) and conditional inclusion probability (condPIP) for each phthalate metabolite are shown in Table S4. We found Group 2 to have the highest groupPIP (groupPIP = 0.795) since MBZP acted as the most critical role (condPIP = 0.657).

To evaluate the single effect of the univariate exposure–response function, we fixed the trend of the univariate exposure–response function for 11 phthalate metabolites at their 50th percentile exposure levels, as shown in Fig. S6. Among them, MBP, MEHP, MECPP, and MEP increased with the risk of LTBI. In contrast, the trends in MBZP and MEOHP were negative. Moreover, interactions between 11 phthalate metabolites were also detected.

To detect interactions between each pair of phthalate metabolites, we fixed other phthalates at the 50th percentile and plotted the

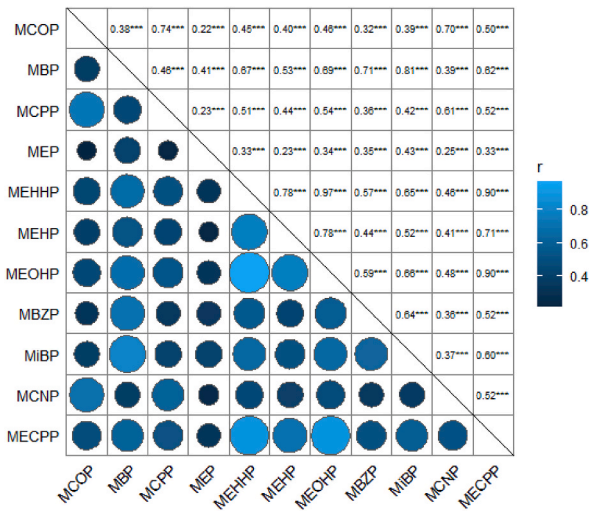

Fig. 2. Spearman correlation coeffifficients among 11 phthalate metabolites (N = 1255), NHANES, USA, 2011–2012. The size of the sphere indicates the *p*-value. When the *P*-value is smaller, the sphere will be larger.

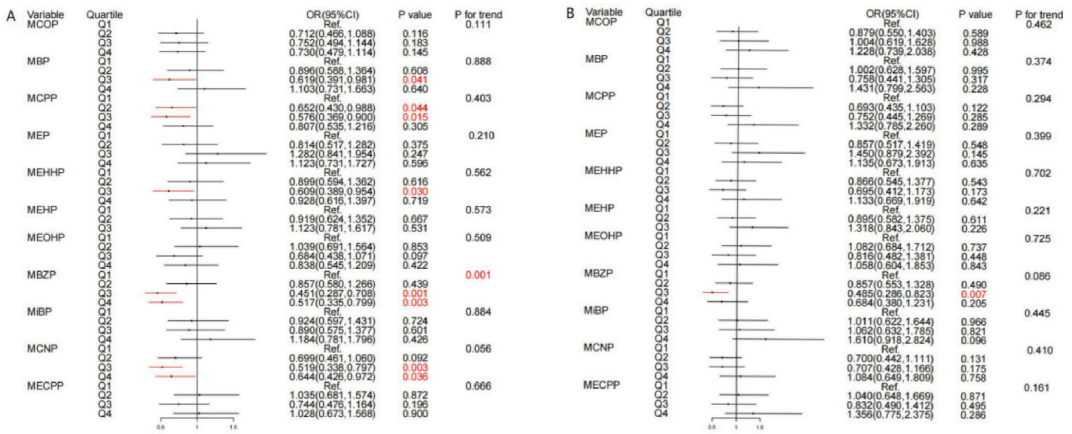

Fig. 3. Logistic regression result of LTBI and single phthalates. A represents a crude model, B represents a full adjusted model.

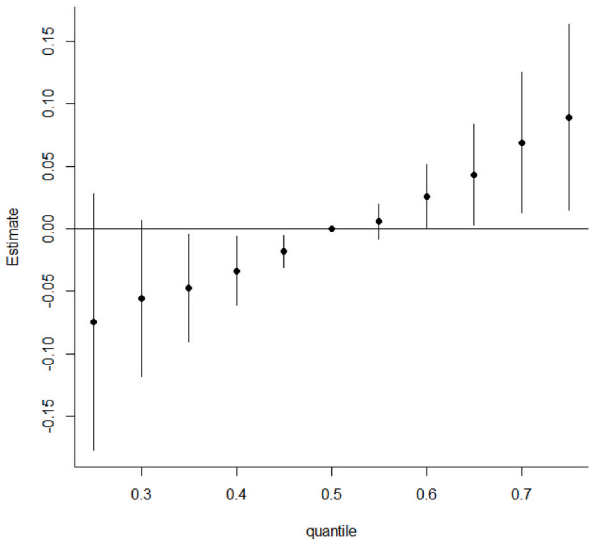

Fig. 4. Joint effects of the overall phthalates mixture on LTBI where graphs depict the effect estimated (with 95% credible interval) when all phthalate biomarkers are fixed at a particular percentile (x-axis) compared to when all biomarkers are at their respective median (reference).

bivariate exposure response function of a single phthalate metabolite at the 25th, 50th, and 75th percentiles (Fig. S7). The other slopes of the bivariate phthalate response function of one phthalate metabolite were identical at different quantiles of the other phthalate metabolite when other metabolite levels were fixed at the 50th quantile, indicating no interaction (Fig. S7). We further found that when other phthalate metabolites were fixed at the 25th, 50th, or 75th percentile, only MBZP had a significant effect on the risk of LTBI (Fig. S8).

4. Discussion

LTBI was defined as a state of persistent immune response to stimulation by *M. tuberculosis* antigens without evidence of clinical manifestations of active tuberculosis disease [20]. However, it is epidemiologically well recognized that only 5–10% of LTBIs will develop TB disease [21]. The duration from initial exposure to incipient TB or active disease is variable and depends on several host, mycobacterial, and environmental factors [22]. Thus, environmental factors may affect the occurrence and development of LTBI. Due to their widespread and continuous presence in the environment, phthalates and their metabolites are consistently detected in humans [23].

Humans are concurrently exposed to multiple phthalates, however most previous studies of phthalates have payed attention to a single phthalate rather than mixtures of phthalates. Thus, the connection between mixed exposure to phthalates in the American population and LTBI was investigated in our study using three statistical techniques. These three statistical models and various methods allowed them to provide more thorough results from various perspectives.

For the socioeconomic variables, LTBI was associated with a low level of education, older age, male sex, marital status and a history of hyperlipidemia. People who were married had a greater risk of LTBI in our research. Although there is no solid reason to explain this conclusion, our findings align with those of an earlier investigation [24].

Phthalate metabolites are closely related to human health and include those of the cardiovascular system and respiratory diseases [25]. Although there is mounting evidence that phthalates may have an impact on respiratory morbidity, no prior studies have been performed in patients with LTBI. According to our research, there is a direct correlation between phthalate exposure and the likelihood of LTBI. The results of the multivariable logistic regression showed that males and females responded differently to phthalate metabolites. Earlier research showed that women had greater quantities of phthalate metabolites than men did. This may be because women use more cosmetics and personal hygiene products, and their skin is more sensitive to phthalate exposure [26,27]. The RCS indicated that LTBI and phthalate metabolites had a linear relationship. In addition, exposure to several phthalates increased the likelihood of LTBI, with MBZP having the most noticeable effect. Quir'os-Alcala et al. reported a statistically significant positive correlation between the MBzP concentration and the SGRQ ($\beta = 3.48$, 95% CI 0.22, 6.75) and CCQ ($\beta = 0.25$, 95% CI 0.05, 0.44) scores in COPD patients [24].

The presence of PAEs and PFAS in water has been linked to the enrichment of several human pathogenic bacteria, which can cause TB [9]. Numerous studies have confirmed that phthalate exposure is associated with many respiratory diseases. Exposure to certain phthalates (DEHP and DBP) is linked to greater morbidity in chronic obstructive pulmonary disease (COPD) patients [28], possibly due to allergic irritability and oxidative stress in the airways [29]. Although phthalates may increase the incidence of preexisting respiratory system problems, most related studies have focused exclusively on childhood asthma [30]. Research suggests that exposure to certain phthalates may worsen the symptoms of asthma and the need for medical attention [31] through processes involving oxidative stress and immune responses [32]. Urinary levels of PAE metabolites were shown to be linked to a higher risk of reduced lung function in an older population in a replicated study [33].

Moreover, several cellular and animal studies have been conducted on phthalate metabolites in the respiratory system. Human lung cancer tissue (A549) is more susceptible to the effects of DBP and DIBP than human vulvar epidermal carcinoma (A431), and exposing A549 and A431 cells to high concentrations of the DBP + DIBP mixture can result in death [34]. Camacho et al. reported that, compared with control rats, DEHP-treated rats exhibited multifocal granulomas and atelectasis as well as lung damage [35]. A study on rodents suggested that phthalates may be used as adjuvants to cause inflammatory and respiratory effects when allergens are present [36].

This study has several limitations. First, the cross-sectional design of the NHANES made it challenging to establish a causal link between phthalate exposure and LTBI, which can only preliminarily determine a correlation between the two. Thus, future longitudinal studies of the biological mechanisms of the effects of phthalates on LTBI are needed. Second, although frequently utilized in daily practice, metabolites have short half-lives [5], and one-time measured exposure levels are not always indicative of lifetime exposure levels. Significantly, phthalates exists in many daily consumer products, and human exposure to phthalates is ubiquitous, which causes thicker level of phthalates. In the future, the concentration of phthalates should be continuously monitored in a prospective cohort study, which is more conducive to elucidating the relationship of phthalates with LTBI. Third, due to the lack of a weighting technique in the R package, we did not utilize the NHANES to explore weights in the RCS and BKMR models. Fourth, due to limitations of the NHANES database, we cannot know whether people with LTBI and people with more phthalate exposure also have more risk factors for TB exposure (e.g., from endemic countries, incarceration, homelessness, etc.) than participants without LTBI.

In conclusion, our study enhanced confidence of the view that phthalates exposure are related to LTBI. In the future, we should be particularly autious in contact to products containing MBP, MEP, MEHP, MBZP, MiBP and MECPP for the prevention of LTBI.

5. Conclusion

Our analysis demonstrated that LTBI in the general U.S. population is linearly linked with exposure to single or combined phthalates. MBP, MEP, MEHP, MBZP, MiBP and MECPP were significantly correlated with LTBI.

Consent for publication

Not applicable.

Availability of data and materials

All the data included in this study are available upon request through contact with the corresponding author.

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CRediT authorship contribution statement

Bi Ran: Writing – original draft, Formal analysis, Conceptualization. **Jiangyue Qin:** Writing – review & editing, Conceptualization. **Yanqiu Wu:** Writing – review & editing, Validation. **Fuqiang Wen:** Writing – review & editing, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:Fuqiang Wen reports financial support was provided by West China Hospital of Sichuan University.

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Abbreviations

LTBI	latent tuberculosis infection
NHANES	National Health and Nutrition Examination Survey
QFT	QuantiFERON®-TB Gold-In-Tube
TST	tuberculin skin test
BKMR	Bayesian kernel machine regression
RCS	restricted cubic spline
TB	tuberculosis
M. tuberculosis (Mtb)	Mycobacterium tuberculosis
PAEs	phthalates
MCNP	mono (carboxynonyl) phthalate
MMP	monomethyl phthalate
MEP	monoethyl phthalate
MBP	monobutyl phthalate
MiBP	mono-isobutyl phthalate
MCPP	mono (3-carboxypropyl) phthalate
MEHP	mono(2-ethylhexyl) phthalate
MBZP	monobenzyl phthalate
MNP	monoisononyl phthalate
MEOHP	mono(2-ethyl-5-oxohexyl) phthalate
MEHHP	mono(2-ethyl-5-hydroxyhexyl) phthalate
MCOP	monocarboxyoctyl phthalate
MECPP	mono-2-ethyl-5-carboxypentyl phthalate
groupPIP	group posterior inclusion probability
condPIP	conditional inclusion probability

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e27958.

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