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Association of maternal blood mercury concentration during the first trimester of pregnancy with birth outcomes

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Exposure to mercury has been associated with adverse effects on pregnancy outcomes. However, there is limited literature on mercury exposure and pregnancy outcomes in Chinese pregnant women. Our study was to investigate the possible association between maternal mercury exposure and spontaneous preterm birth and birth weight. This study was a nested case-control study. The association between blood mercury concentration and both spontaneous preterm birth and birth weight was analyzed using conditional logistic regression and linear regression adjusted for the potential confounding factors, respectively. The dose-response relationship between mercury concentration and birth outcomes was estimated using restricted cubic spline regression. The mean concentration of mercury was 2.8 ± 2.2 μg/L. A positive relationship was observed between maternal blood mercury concentration and SPB when analyzed as a continuous variable. However, it was not found to be statistically significant (adjusted OR = 1.10, 95% CI = 0.95–1.26, *P* = 0.202). Moderate mercury exposure was associated with a higher risk of SPB (Q3 vs. Q1: crude OR = 2.50, 95% CI = 1.16–5.41, *P* = 0.02; adjusted OR = 3.49, 95% CI = 1.33–9.11, *P* = 0.011). After considering the combined effects of chemicals other than mercury exposure (including lead, selenium, and cadmium), the results remained consistent. There was no statistically significant association between blood mercury levels and birth weight (adjusted coefficient = 18.64, P-value = 0.075). There were no statistically significant dose-response associations between mercury concentration and birth outcomes (SPB: *P* = 0.076; birth weight: *P* = 0.885). Public health policies should focus on reducing environmental releases of mercury, improving food safety standards, and providing education to pregnant women about the risks of mercury exposure and preventive measures.

Keywords Maternal blood mercury concentration, Spontaneous preterm birth, Low birth weight, Mercury exposure

Instruction

Preterm birth is the primary cause of perinatal death and infant mortality globally, accounting for 75% of cases[1,2]. Spontaneous preterm birth (SPB) is defined as a type of preterm birth that encompasses both spontaneous delivery with intact membranes and premature rupture of the fetal membranes (PROM). It is estimated that approximately 40–45% of all preterm births are spontaneous and that between 25% and 30% are PPROM[1,3]. Like preterm birth, birth weight is a critical indicator of pregnancy and fetal development. Low birth weight (< 2500 g, LBW) is significantly associated with neonatal mortality and neurobehavioral development[4]. In 2015,

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the global prevalence of LBW was 14.6%, with approximately 20.5 million LBW births, 91.0% of which occurred in developing countries[5]. Recent studies have reported a range of 2.77–6.1% LBW in China[6]. In addition to the well-established risk factors, such as maternal age, parity, multiple pregnancies, and smoking, studies have identified a potential association between prenatal exposure to toxic metals, including mercury (Hg), and adverse birth outcomes[1,7]. Mercury is listed as a priority hazardous substance by the Agency for Toxic Substances and Disease Registry (ATSDR)[8]. A number of studies have indicated that higher levels of mercury exposure during the first trimester of pregnancy may increase the risk of SPB in women. Furthermore, prenatal mercury exposure has been negatively associated with birth weight[9]. However, some studies have suggested that the relationship between mercury and preterm birth remains unclear or has yielded conflicting results[10,11]. It is evident that further discussion and research are required in order to reach a definitive conclusion regarding the divergent interpretations of these data.

Mercury exists in a variety of forms, including inorganic mercury (metallic Hg, mercury vapor, mercurous or mercuric salts) and organic mercury (mainly methylmercury))[12]. The different forms of mercury have distinct transport and deposition mechanisms, as well as varying ecological effects. Organic mercury is typically generated by microorganisms that transform inorganic mercury under specific circumstances. For instance, in aquatic ecosystems, microorganisms can convert inorganic mercury to methylmercury. Inorganic mercury is derived primarily from the natural environment and human activities, including volcanic eruptions, rock weathering, and the mining and smelting of mercury-containing ores[13]. Mercury pollution constitutes a significant challenge in China, where average annual mercury emissions reached 2,151 tons in 2012, representing the highest level globally[14]. Mercury concentrations in agricultural soils showed an increasing trend between 1979 and 2010, followed by a decrease. Mercury concentrations in soils were generally high in the west (e.g., Guizhou), south (e.g., Hunan), and northeast (e.g., Liaoning), where mining activities are concentrated[15]. It was also found that about 6.4% and 7.0% of the population exceeded the European Food Safety Authority (EFSA) health guidelines, with higher exposure risks in southwest and southern China[14]. However, the existing literature on the relationship between different forms of mercury exposure and pregnancy outcomes in Chinese pregnant women is limited. Accordingly, the objective of this investigation is to identify a potential association between maternal blood mercury concentration and birth outcomes.

Materials and methods

Study design

This study was a nested case-control study based on the Fujian Maternal and Child Health Hospital Birth Cohort Project. The study included women who met the following criteria: (1) Gestational age less than 14 weeks at enrollment. (2) Planned antenatal care and delivery at the Fujian Maternal and Child Health Hospital. (3) Signed informed consent form. After enrollment, questionnaires will be collected during different stages of pregnancy. This study mainly used questionnaire data from early pregnancy.

According to the definition of SPB, to exclude preterm birth for maternal or fetal indications (including birth defects, stillbirth, abortion, placental abruption, gestational hypertension and diabetes mellitus, placenta previa, and eclampsia) were excluded. Multiple births and participants with chronic hypertension and diabetes at baseline were also excluded[1,16]. The SPB group included participants with a single live birth and gestational age below 37 weeks. The control group was selected from other participants who met the criteria of being at least 37 weeks' gestation, and who were matched for maternal age, race, pre-pregnancy body mass index (BMI), gravidity, and parity. A total of 240 participants were recruited to take part in the study, comprising 120 controls and 120 subjects presenting a spontaneous preterm birth (Supplementary material). Our study was performed in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Fujian Maternity and Child Health Hospital (Approval Number: 2019–200). All participants were informed and signed an informed consent form before participating.

Covariates

All clinical information was obtained from the Electronic Health Record system, including maternal data (age, height, weight, method of delivery, gestational age, gravidity and parity), fetal data (birth weight, birth length and infant sex) and discharge diagnosis. The questionnaire was utilized to obtain information pertaining to social factors, including maternal education level, monthly family income, folate intake, passive smoking, and race. BMI was calculated by squaring the weight in kilograms to the height in meters. The term "passive smoking exposure" was defined as self-reported exposure to secondhand smoke at home and at work during the first three months of pregnancy. It should be noted that there were no smokers in this study.

Sample size

Sample size and power analysis: Sample size and power were determined based on the Logistic Regression. Sample size of 238 observations achieves 80% power at a 0.05 significance level to detect a change in probability of SBP from the value of 0.5 at the mean of Hg to 0.6 when Hg is increased to one standard deviation above the mean. This change corresponds to an odds ratio (ORs) of 1.5 (Estimated effect size based on previous study results).

Sample collection and determination

The blood sample was collected during the first trimester, following an eight-hour fast. The blood samples were subsequently stored at a temperature of -80 °C until they were analyzed. Quantification of blood metal concentrations was achieved through the use of the Agilent Technology 7800 Inductively Coupled Plasma Mass Spectrometry (ICP-MS) and the Agilent Technology SPS4 automated sampling systems.

A solution of 2.5 mL of 75% nitric acid and 0.5 mL of Triton X-100 was prepared in a 500mL volumetric flask by adding ultra-pure water until the volume was complete. This solution is referred to hereafter as the dilution solution. A 100-microliter aliquot of the calibration standards, quality control samples, and test samples was diluted with 3.9 mL of the diluent, shaken 20 times, and vortexed for 5 min using a multi-tube vortex mixer. The resulting solution was then injected for analysis. ICP-MS standard solutions were utilized as the reference materials, sourced from o2si smart solutions, USA. If the blood metal concentration was below the limit of detection (LOD), the calculated value was LOD/√2.

Statistical analysis

Blood mercury concentration was divided into four groups based on quartiles (Q1: < 25th quartile, Q2: 25th −50th quartile, Q3: 50th −75th quartile, Q4: > 75th quartile). Chi-squared test or analysis of variance was used to assess differences in demographic characteristics and lifestyle factors between quartiles of mercury concentration.

The association between mercury and SPB was analyzed using conditional logistic regression, consisting of a base model and a model adjusted for the method of delivery, sampling gestational age, infant sex, maternal education level, monthly family income, passive smoking, and folate intake. A linear regression was conducted to analyze the association between mercury and birth weight, adjusting for the method of delivery, sampling gestational age, infant sex, maternal education level, monthly family income, passive smoking, folate intake, parity, gravidity, race, maternal age, gestational age, and pre-pregnant BMI. Trends in the concentration quartiles were tested by modeling each concentration quartile as a continuous variable. The reference value for the calculation of ORs was the group of Q1 mercury concentration. Concurrently, the combined effects of exposure to chemicals other than mercury (lead, selenium, cadmium) were assessed. Furthermore, the dose-response associations between mercury concentration and birth outcomes were estimated using restricted cubic spline (RCS) regression, with the results adjusted for the potential confounding factors.

Statistical analyses were performed with R version 4.3.1. A two-tailed P value of <0.05 and a 95% confidence interval (CI) excluding the null value were considered to be statistically significant in this study. Conditional logistic regression was conducted using the "survival" package (version 3.5-5) and RCS regression was conducted using the "rms" package (version 6.7-1).

Results

Table 1 summarizes the demographic characteristics of the participants. On average, the participants were 29.9 ± 4.2 years old. The majority of the participants were Han Chinese ($n = 238$, 99.2%). The mean pre-pregnancy BMI was 20.7 ± 3.0 kg/m^2. In terms of education, most women held a bachelor's degree ($n = 169$, 70.4%). 43.8% of the female participants were exposed to passive smoking during their pregnancy. The average gestational age was 37.8 ± 2.2 weeks, while the mean birth weight and length were 2984.8 ± 555.2 g and 48.1 ± 2.5 cm, respectively. Additionally, 130 (54.2%) of the infants were male. The mean concentration of mercury was 2.8 ± 2.2 µg/L. Except for gestational age, there were no significant differences among the four quartile groups ($P > 0.05$).

A positive relationship was observed between maternal blood mercury concentration and SPB when analyzed as a continuous variable. However, it was not found to be statistically significant (adjusted OR = 1.10, 95% CI = 0.95–1.26, $P = 0.202$) (Table 2). Moderate concentration of mercury was found to be associated with an increased risk of SPB (Q3 vs. Q1: crude OR = 2.50, 95% CI = 1.16–5.41, $P = 0.02$; adjusted OR = 3.49, 95% CI = 1.33–9.11, $P = 0.011$) (Table 2). After considering the combined effects of chemicals other than mercury exposure (including lead, selenium, and cadmium), the results remained consistent (Table 2). No statistically significant association was observed between maternal blood mercury concentration and birth weight in early pregnancy, regardless of whether the variable was considered continuous or divided into quartiles based on different concentration levels (adjusted coefficient = 18.64, P-value = 0.075) (Table 3). There were no statistically significant dose-response associations between mercury concentration and birth outcomes (SPB: $P = 0.076$; birth weight: $P = 0.885$) (Figs. 1 and 2).

Discussion

It is well established that exposure to mercury in the environment represents a significant public health concern. The major routes of mercury exposure for pregnant women include occupational, dental, and dietary sources. There is a paucity of research investigating the relationship between occupational and dental mercury exposure and preterm birth and birth weight. A study of occupational mercury exposure in the interior of Suriname concluded that low mercury exposure was associated with low birth weight[17]. The study of dental mercury exposure found no significant association between the number of amalgam fillings in the teeth or dental care received and birth weight or preterm birth[18,19]. While occupational and dental mercury exposure may not be the primary routes of exposure for pregnant women in Fujian Province, the potential effects of mercury exposure on pregnancy and fetal health cannot be entirely discounted. Further research is required to gain a more comprehensive understanding of this issue.

Dietary exposure, particularly seafood consumption, represents a significant source of methylmercury exposure[20]. Fuzhou City, situated within the Fujian Province, is among the regions with the highest rates of seafood consumption. Therefore, the main form of mercury exposure for pregnant women in Fujian Province may be methylmercury. Given that organic mercury is generally more toxic than inorganic mercury, methylmercury has the potential to cross the placenta and pose a threat to fetal neurodevelopment, which may result in adverse pregnancy outcomes[21]. Consequently, there is a current focus on further research into the relationship between methylmercury and adverse pregnancy outcomes. The national average exposure to methylmercury in aquatic products was 3.8 µg/day, with the highest exposure observed in the coastal province

Variables	Total ($n=240$)	Q1 ($n=60$)	Q2 ($n=60$)	Q3 ($n=59$)	Q4 ($n=61$)	p
Maternal age, years	29.9 ± 4.2	29.6 ± 4.4	29.4 ± 3.6	29.8 ± 4.1	30.7 ± 4.6	0.323
Race						0.733
Han	238 (99.2)	59 (98.3)	59 (98.3)	59 (100.0)	61 (100.0)	
Others	2 (0.8)	1 (1.7)	1 (1.7)	0 (0.0)	0 (0.0)	
Pre-pregnant BMI, kg/m^{2}SD)	20.7 ± 3.0	20.8 ± 3.5	20.5 ± 2.8	20.0 ± 2.3	21.3 ± 3.1	0.120
Gravidity						0.535
1	110 (45.8)	28 (46.7)	31 (51.7)	31 (52.5)	20 (32.8)	
2	66 (27.5)	17 (28.3)	15 (25)	14 (23.7)	20 (32.8)	
3	26 (10.8)	4 (6.7)	6 (10.0)	6 (10.2)	10 (16.4)	
4	38 (15.8)	11 (18.3)	8 (13.3)	8 (13.6)	11 (18.0)	
Parity						0.178
1	140 (58.3)	40 (66.7)	38 (63.3)	35 (59.3)	27 (44.3)	
2	78 (32.5)	16 (26.7)	19 (31.7)	17 (28.8)	26 (42.6)	
3	22 (9.2)	4 (6.7)	3 (5.0)	7 (11.9)	8 (13. 1)	
Gestational age, weeks	37.8 ± 2.2	38.2 ± 2.2	38. 1 ± 2.1	37.5 ± 2.1	37.2 ± 2.2	0.020
Infant sex						0.929
Male	130 (54.2)	31 (51.7)	32 (53.3)	34 (57.6)	33 (54. 1)	
Female	110 (45.8)	29 (48.3)	28 (46.7)	25 (42.4)	28 (45.9)	
Birth weight, g	2984.8 ± 555.2	3059.8 ± 511.1	3047.6 ± 579.3	2894. 1 ± 569.5	2936.8 ± 554.5	0.274
Birth length, cm	48. 1 ± 2.5	48.7 ± 2.2	48.3 ± 2.6	47.8 ± 2.3	47.7 ± 2.8	0.103
Maternal education						0.896
Primary school	2 (0.8)	1 (1.7)	1 (1.7)	0 (0.0)	0 (0.0)	
Middle school	27 (11.2)	9 (15.0)	7 (11.7)	6 (10.2)	5 (8.2)	
High school	29 (12. 1)	10 (16.7)	6 (10.0)	5 (8.5)	8 (13. 1)	
Undergraduate	169 (70.4)	38 (63.3)	43 (71.7)	44 (74.6)	44 (72. 1)	
Postgraduate and above	13 (5.4)	2 (3.3)	3 (5.0)	4 (6.8)	4 (6.6)	
Passive smoke						0.166
No	135 (56.2)	35 (58.3)	38 (63.3)	26 (44. 1)	36 (59.0)	
Yes	105 (43.8)	25 (41.7)	22 (36.7)	33 (55.9)	25 (41.0)	
Hg, μg/L	2.8 ± 2.2	1.0 ± 0.3	1.7 ± 0.2	2.7 ± 0.4	5.6 ± 2.6	< 0.001
SPB						0.077
No	120 (50.0)	36 (60.0)	34 (56.7)	24 (40.7)	26 (42.6)	
Yes	120 (50.0)	24 (40.0)	26 (43.3)	35 (59.3)	35 (57.4)	

Table 1. Demographic characteristics of study population. Data presented as mean ± SD, or percentage of participants. SD, standard deviation. BMI, body mass index. SPB, spontaneous preterm birth.

	Model 1[a]		Model 2[b]		Model 3[c]	
Hg (μg/L)	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Hg	1.09(0.97, 1.23)	0.143	1.10 (0.95, 1.26)	0.202	1.05 (0.90, 1.23)	0.536
Q1	1(Ref)		1(Ref)		1(Ref)	
Q2	1.09(0.55, 2.19)	0.8	1.31(0.58, 2.95)	0.52	1.24(0.53, 2.89)	0.619
Q3	2.50(1.16, 5.41)	0.02	3.49(1.33, 9.11)	0.011	3.11(1.11, 8.67)	0.03
Q4	1.96(0.95, 4.06)	0.069	2.43(0.97, 6.09)	0.058	2.38(0.83, 6.81)	0.107
P for trend trend		0.028		0.02		0.055

Table 2. Association between maternal blood hg concentrations during first trimester with SPB. SPB, spontaneous preterm birth. [a]It is an un-adjusted model. [b]Adjusted for the method of delivery, sampling gestational age, infant sex, maternal education level, monthly family income, passive smoking and folate intake. [C]Adjusted for the method of delivery, sampling gestational age, infant sex, maternal education level, monthly family income, passive smoking, folate intake, lead, selenium and cadmium.

Variable	*n*	Crude Coefficient	Crude *P*-value *P*-value	Adjusted Coefficient[a] (95% CI)	Adjusted *P*-value
Hg (μg/L)	240	-4.29 (-36.22,27.64)	0.793	18.64 (-5.36,34.38)	0.075
Q1	60	0(Ref)		0(Ref)	
Q2	60	-12.25 (-210.56, 186.06)	0.904	15.67 (− 100.72, 137.96)	0.801
Q3	61	-179.01 (-364.91,33.38)	0.077	4.4 (− 132.79, 114.75)	0.945
Q4 P for	59	-107.88 (-320.52,74.46)	0.289	64.04 (-70.73, 174.61)	0.329
P for trend	240		0.125		0.390

Table 3. Association between maternal blood hg concentrations during first trimester with birth weight. [a]Adjusted for the method of delivery, sampling gestational age, infant sex, maternal education level, monthly family income, passive smoking, folate intake, parity, gravidity, race, maternal age, gestational age, and pre-pregnant BMI.

of Fujian[14]. Although the mean mercury exposure in our study was 2.8 μg/L, which does not exceed the U.S. Environmental Protection Agency's biomonitoring guideline of 5.8 μg/L in umbilical cord blood (equivalent to approximately 3.5 μg/L in maternal blood), the results of this nested case study suggest an association between mercury and an increased risk of SPB, especially at high concentrations[22]. The results of recent research have also provided corroboration for this conclusion. For instance, cohort studies in both China and the United States have indicated that maternal mercury levels are higher in the preterm birth group. However, the two articles have employed different definitions of preterm birth. China has focused on SPB, while the United States has referred to preterm birth (PTB)[23,24]. Although these studies provided clear evidence of an association between mercury and SPB, there are still inconsistent findings. Some studies had not found an association between SPB and mercury exposure, except at very high levels[25,26]. Regarding birth weight, our study did not find a significant effect of mercury exposure on low birth weight. However, other studies had suggested a negative association between mercury exposure and birth weight[27,28]. While our study also considered the combined effects of lead, cadmium, and selenium and found no significant association between mercury and SPB or birth weight, current research indicates that the combined effects of heavy metals may have adverse effects on pregnant women and fetuses[36,37]. In light of these findings, it is advisable for pregnant women to minimize their exposure to these heavy metals to mitigate the risk of premature birth and other adverse pregnancy outcomes.

The finding of this study that there is no significant association between low mercury exposure and SPB and birth weight may be related to the important relationship between the benefits and risks of consuming fish and seafood, as emphasized in other studies[25,26]. Studies have shown that pregnant women consuming fish rich in omega-3 fatty acids, such as salmon and mackerel, during pregnancy can reduce the risk of premature birth and also have a positive impact on birth weight. This may be because omega-3 fatty acids can reduce the effectiveness of hormones that cause premature birth, thereby prolonging pregnancy[29,30]. However, the presence of unsaturated fatty acids and mercury in fish is a complex issue that necessitates a comprehensive examination. Some studies have indicated that fish produced through aquaculture in seafood markets may also influence polyunsaturated fatty acid (PUFA) and mercury levels in fish. For example, a study of fish samples from the Wujiangdu (WJD) Reservoir in southwestern China demonstrated that the health benefits of n-3 PUFA were greater than the adverse effects of methylmercury in all farmed fish in the reservoir. Furthermore, the study revealed notable variations in PUFA concentrations across different fish species[31]. It is therefore imperative to conduct a comprehensive study of their food sources, growth rates, and position in the aquatic food chain. Furthermore, risk-benefit assessments should be conducted for different fish species. It is pertinent to mention that selenium, which is also present in marine products, has been demonstrated to possess detoxifying properties and can effectively counteract the toxicity of heavy metals[26,32]. In the interaction between selenium and mercury, selenium may reduce the toxicity of mercury by forming selenium-mercury complexes[33]. Nevertheless, some studies have demonstrated that there is no evidence to substantiate the notion that selenium has a detoxifying effect on methylmercury. Consequently, the emphasis should be on reducing exposure to methylmercury, rather than on increasing selenium intake[34]. In conclusion, the issue of how to ensure that pregnant women can safely consume fish nutritionally while minimizing fetal exposure to mercury represents a significant public health concern.

Mercury has been linked to suboptimal intrauterine environments, which may impair fetal growth. However, the biological mechanisms underlying this association remain unclear. One potential explanation for the association between mercury and adverse pregnancy outcomes, such as preterm labor and birth weight, is that it may be related to oxidative stress or an imbalance in oxidative/antioxidant activity in placental tissues[9]. Oxidative damage to deoxyribonucleic acid (DNA) refers to damage to DNA molecules caused by the attack of reactive oxygen species (ROS). A typical marker of oxidative damage is 8-hydroxydeoxyguanosine (8-OHdG), the quantitative detection of which reflects the extent of oxidative damage[35]. Although there is no direct evidence that mercury can cause oxidative DNA damage, given the toxicity of mercury to cells and the oxidative stress it can induce, it is theoretically possible that high levels of mercury exposure could indirectly cause oxidative DNA damage by increasing the production of reactive oxygen species. Some studies have demonstrated a positive correlation between serum and urine mercury concentrations and 8-OHdG concentrations in individuals

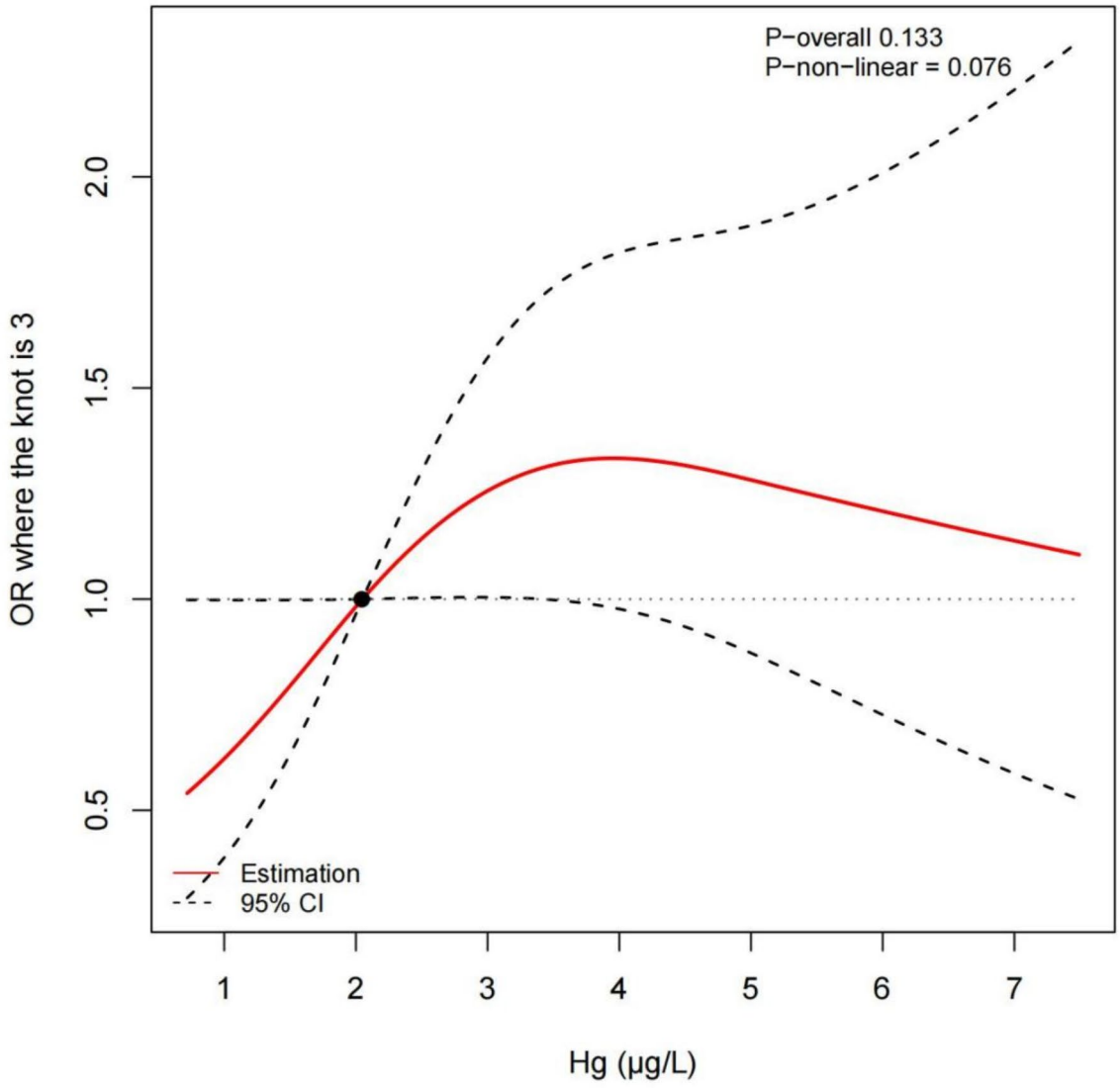

Fig. 1. The dose–response association between maternal blood concentration of Hg and SPB.

exposed to mercury, suggesting mercury-induced oxidative DNA damage[36]. Additionally, studies have been conducted to investigate the role of miRNAs in mercury-induced cellular responses. While these studies have primarily focused on cellular and plant models, they offer insights into the potential role of miRNAs in response to heavy metal stress. Although studies on miRNAs in mercury-induced cellular responses are still relatively limited, it has been demonstrated that miRNAs may be involved in cellular responses to the heavy metal mercury through a variety of mechanisms, including the regulation of processes such as cell cycle, apoptosis, and differentiation[37,38]. Given the highly spatiotemporal and tissue-specific nature of miRNA regulatory effects, future studies must further explore the specific mechanisms of miRNA action in different cell types and tissues in response to mercury induction. Given the intracellular regulatory network of miRNAs, investigating the role of miRNAs in mercury-induced cellular responses may help to identify new biomarkers and potential therapeutic targets, thereby providing new ideas for the prevention and treatment of diseases related to mercury exposure.

A significant advantage of this study is that the nested case-control design, derived from a large prospective birth cohort study, can provide a higher level of causal evidence. Secondly, we conducted a test on the blood samples taken during the early stages of pregnancy. There is a well-established consensus in the scientific community that the initial weeks of pregnancy are a particularly susceptible period of exposure to mercury. The greatest risk of fetal mercury exposure occurs during this period, and the probability of adverse outcomes may be higher than in subsequent stages of gestation[39]. Furthermore, in order to reduce any potential confounding effect, it is essential to distinguish SPB from iatrogenic preterm birth. However, this study also had several limitations.

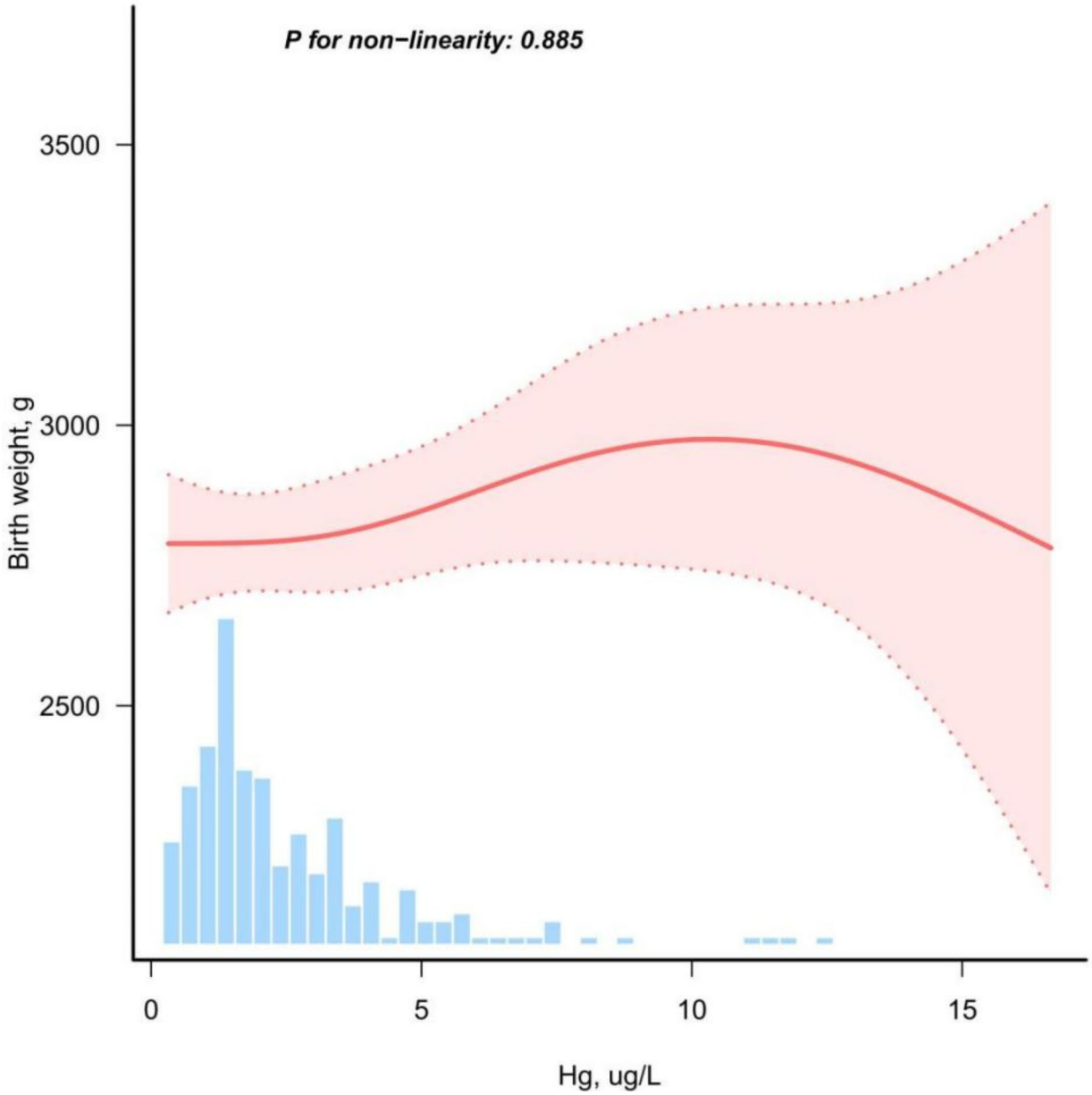

Fig. 2. The dose–response association between maternal blood concentration of Hg and the birth weight.

Firstly, it is important to note that the majority of participants in this study were Han Chinese. Consequently, the results may not be applicable to other populations. Secondly, the study did not consider certain potentially confounding factors, including detailed daily dietary intake, consumption of additional nutritional supplements, alcohol consumption, and environmental exposure. Research has shown that high levels of zinc may protect pregnant women from the adverse effects of mercury exposure[9]. Finally, this study excluded pregnant women with gestational diabetes mellitus (GDM), which may underestimate the effect of mercury exposure on SPB. Although there are conflicting findings regarding the relationship between mercury exposure and GDM[40,41].

Conclusion

Mercury, as an environmental contaminant, presents a potential risk to the health of pregnant women and fetuses. Reducing mercury exposure, especially for the pregnant population, is an important strategy for preventing preterm births and protecting the long-term health of children. Public health policies should focus on reducing environmental releases of mercury, improving food safety standards, and providing education to pregnant women about the risks of mercury exposure and preventive measures. In the future, it would be beneficial to investigate the relationship between seafood consumption habits (quantity and variety) and seafood

contamination and mercury exposure in Fujian Province. This would allow for the development of more accurate dietary advice for pregnant women.

Data availability

The data that support the findings of this study are not openly available due to reasons of sensitivity and are available from the corresponding author upon reasonable request.

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Author contributions

W.L.: Conceptualization, Investigation, Writing - Original draft, Writing - Review & editing. C.M.: Data curation, Project administration. Software. B.S.: Investigation, Writing - Review & editing. ZQ.W.: Formal analysis, Visualization. XR.W.: Investigation, Resources. HB.L.: Validation, Writing - Review & editing. HY.G.: Investigation, Resources. YB.Z.: Project administration, Writing - Review & editing, Funding acquisition. H.C.: Validation, Supervision, Funding acquisition. All authors reviewed and approved the final manuscript.

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Declarations

Competing interests

The authors declare no competing interests.

Ethical statement

Our study was performed in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Fujian Maternity and Child Health Hospital (Approval Number: 2019–200). All participants were informed and signed an informed consent form before participating.

Additional information

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