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Fetal Exposure To Mercury: Correlations Between Maternal Hair, Placental Mercury and Neonatal Outcome

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ABSTRACT

Background: Mercury contamination can cross cellular membranes and bioaccumulate in various tissues, including the placenta during pregnancy. This study evaluates the correlation between maternal hair and placental mercury levels and their impact on neonatal outcomes.

Methods: This cross-sectional study involved 98 pregnant women. Mercury levels in hair and placental were measured using atomic absorption Spectrophotometry. Statistical analyses, including Chi-Square and Pearson correlation tests, were performed using SPSS.

Results: Maternal hair mercury levels were positively correlated with placental blood mercury levels ($\rho = 0.26$, $p = 0.01$), indicating maternal-to-fetal transfer. Placental mercury levels were negatively correlated with placental weight ($\rho = -0.24$, $p = 0.02$) and head circumference ($\rho = -0.29$, $p = 0.01$). No significant associations were found between maternal hair mercury levels and neonatal outcomes.

Conclusion: Maternal hair mercury levels correlate with placental mercury levels, indicating transfer from mother to fetus. Placental mercury negatively impacts placental weight and head circumference, emphasizing its significance as an indicator of neonatal outcomes. Awareness campaigns about mercury exposure could help mitigate these risks

Keywords: Mercury, Neonatal Outcomes, Hair, Placental

1. INTRODUCTION

Mercury contamination, originating from both industrial activities and natural sources, predominantly accumulates within the food chain, especially in predatory fish and seafood. This accumulation presents significant health risks to vulnerable populations, such as pregnant women [1]. Mercury can cross cellular membranes, bind to thiol groups in proteins, and bioaccumulate in various tissues, including the placenta [2].

During pregnancy, mercury can traverse the placenta, leading to oxidative stress and inflammation that hinder the transfer of nutrients and oxygen, thereby adversely impacting neonatal outcomes, including birth weight, length, and head circumference [3]. Chronic exposure to mercury is particularly alarming, as it accumulates in keratinized tissues, such as mercury levels in maternal hair, reflecting long-term exposure patterns [4]. Prenatal exposure to mercury has been associated with low birth weight, preterm birth, and developmental deficits, especially in high fish-consuming populations [5,6].

This study aims to evaluate how chronic mercury exposure, reflected in mercury levels in maternal hair, and acute exposure, indicated by placental mercury levels, correlate with fetal growth and neonatal health.

Methods

This cross-sectional study was conducted at Khadijah Hospital and Health Laboratory Center for Makassar, in January-March 2023. The study was approved by Hasanuddin University Clinical Research Ethics Committee with the protocol number UH23070492 and informed consent was obtained from all participants

Variable such as demographic age (years), gestational age (weeks), placental weight (gram), birth weight (gram), birth length (cm), head circumference (cm), Hair and blood cord mercury level, and mercury level according to food recall were collected using a questionnaire through direct interviews and primary measurement with sample. Data was taken from pregnant women met the following inclusion criteria : single intrauterine pregnancy and a viable fetus, gestational age of 37 to 42 weeks, a hemoglobin level greater than 11 g/dL, and a mid-upper arm circumference of ≥23.5 cm. Sample with pregnant women with medical conditions associated with fetal growth restriction, such as diabetes mellitus, hypertensive disorders of pregnancy, maternal bleeding, infections, or fetal or placental anomalies,working in industries, mining, or agriculture involving mercury were excluded from the sample.

Blood cord samples were collected 5 cm from the placenta, with a volume of 1 cc in EDTA tubes. The blood was obtained after the cessation of pulsation in the cord. Hair samples at least 9 cm in length (Given the established growth rate of approximately 1 cm per month so corresponded to nine month of gestation prior to delivery) or approximately 30 mg, were collected from each participant. To address potential concerns regarding the formation of a bald spot, samples were obtained from the nape of the neck, with the cut made as close to the scalp as possible while avoiding the hair root. The samples were affixed to a piece of paper, with both the root and tip ends clearly labeled, then placed in ziplock bags for transportation to the laboratory. Upon arrival, the samples were processed for mercury analysis using Atonic Absorbstion Spectrophotometry.

All data collected were and accurate based on observations of laboratory results then the data coded were and entered into MS Excel then analyzed using IBM SPSS Statistic 25 using the Chi-Square test and Pearson correlation. The analysis result of P-value ≤ 0.05 was considered statistically significant.

Results

The study subjects in Table 1 had a median maternal age of 26 years (range: 16–43 years) and a median gestational age of 39 weeks (range: 37–42 weeks), indicating a young and term-pregnant population suitable for analyzing mercury exposure and birth outcomes (Table 1). Neonatal characteristics in Table 1 showed a mean birth weight of 2982 grams (SD 417), median birth length of 48 cm (range: 47–53 cm), head circumference of 33 cm (range: 28–39 cm), and a placental weight median of 435 grams (range: 300–475 grams), all within normal ranges and providing a reliable reference for evaluating maternal and fetal factors (Table 1).

Dietary mercury intake assessed via 24-hour food recall had no significant relationship with mercury levels in hair or placenta (Table 2). Significant findings included a correlation between mercury in mercury levels in maternal hair and placental blood ($\rho = 0.26$, $p = 0.01$), suggesting maternal-to-fetal mercury transfer (Table 3). However, no significant relationships were found between hair mercury levels and neonatal outcomes (Table 3). Conversely, placental mercury showed significant negative correlations with placental weight ($\rho = -0.24$, $p = 0.02$) and head circumference ($\rho = -0.29$, $p = 0.01$), indicating potential adverse impacts (Table 4).

Table 1. Characteristics of Research Subjects

Mother Characteristic	Variable	Median (min-max)
	Age (years)	26.00 (16.00 – 43.00)
	Gestational age (weeks)	39.00 (37.00 – 42.00)
Neonates Characteristic	Variable	Mean (SD) or Median (Min-Max)
	Placental weight (gram)	435.00 (300.00 – 475.00)
	Birth weight (gram)	2982.00 (417.00)
	Birth length (cm)	48.00 (47.00 – 53.00)
	Head circumference (cm)	33.00 (28.00 – 39.00)

Table 2. Correlation between 24-hours Food Recall and Mercury Level

	ρ	p-value
24-hours food recall and hair mercury level	-0.03	0.77
24-hours food recall and placental mercury level	-0.01	0.99

Tabel 3. Correlation between Mercury in Hair and in Placental Mercury

Mercury Level (µg/L)	Median	ρ	p-value
Hair	0.0005	0.26	0.01*
Placental	0.00003		

*significant, ρ correlation coefficient

Table 4. Correlation between Hair Mercury, Placental Mercury, and Neonatal Outcome

	Mercury Level (µg/L)			
	Hair		Placental	
	ρ	p-value	ρ	p-value
Placental weight	-0.12	0.22	-0.24	0.02*
Birth weight	0.06	0.50	-0.16	0.12
Birth length	0.03	0.72	-0.11	0.29
Head circumference	-0.09	0.39	-0.29	0.01*

*significant, ρ correlation coefficient

2. DISCUSSION

Mercury deposition in hair occurs due to its affinity for thiol groups in keratin during hair formation. Methylmercury (MeHg) and inorganic mercury in maternal blood cross the placental barrier and integrate into fetal tissues and maternal hair. Studies demonstrate a significant correlation between hair mercury levels and placental mercury ($\rho = 0.26$, $p = 0.01$), making hair a valuable biomarker for prenatal mercury exposure.[4] Mercury accumulation in hair reflects chronic exposure and exhibits a strong correlation with mercury levels in fetal tissues, as evidenced by studies linking maternal and placental mercury levels.[7]. Furthermore, maternal hair mercury consistently functions as a reliable proxy for placental mercury in populations with elevated dietary fish intake, thereby affirming its use as a biomarker.[8]

Additional research supports these findings. The levels of mercury in maternal and blood cord exhibit a strong correlation with dietary fish intake, thereby underscoring the influence of dietary habits on maternal mercury levels[6,9]. Elevated mercury concentrations in hair and placenta are also associated with increased fish consumption, highlighting the bioaccumulation process [10]. Furthermore, placental mercury levels correlate with adverse neonatal outcomes, such as lower birth weight and head circumference, further underscoring its biological significance [11].

The relationship between mercury levels in maternal hair and neonatal outcomes, including placental weight, birth weight, birth length, and head circumference, showed no significant associations in this study (Table 4). This lack of statistical significance may be attributed to individual variability in mercury metabolism and distribution. Mercury accumulates in keratinized tissues such as hair due to its high affinity for thiol groups during systemic circulation, reflecting long-term exposure. However, this process may not accurately represent acute toxic effects directly impacting neonatal outcomes.[12] Mechanistically, mercury disrupts fetal development through oxidative stress, endothelial dysfunction, and alterations in placental function. These disruptions can reduce placental efficiency and fetal growth. High placental mercury levels have been linked to reduced head circumference and placental efficiency in previous studies.[11] However, in this study, low overall mercury exposure levels or confounding dietary factors, such as nutrient intake from fish, may have mitigated potential adverse effects. Nutrients like omega-3 fatty acids in fish are known to counteract some negative impacts of mercury exposure on fetal growth.[13]

Some studies have reported significant correlations between maternal mercury levels and neonatal head circumference, but findings on birth weight or length remain inconsistent.[14] Other research highlights that genetic variations in mercury metabolism, as well as dietary habits, may influence these outcomes and add complexity to the observed associations.[15] Additionally, systematic reviews suggest that while mercury exposure can impair fetal growth, its impact varies based on exposure levels and timing during pregnancy.[5]

These findings indicate that while maternal hair mercury levels are a reliable biomarker of chronic exposure, their direct impact on neonatal outcomes remains unclear and context-dependent. Further studies exploring the interplay of environmental, genetic, and dietary factors are needed to clarify these associations.[6,12,16,17]

The study demonstrates a significant negative correlation between placental mercury levels and neonatal outcomes such as placental weight ($\rho = -0.24$, $p = 0.02$) and head circumference ($\rho = -0.29$, $p = 0.01$), emphasizing the adverse impact of mercury on fetal growth (Table 5). Mercury accumulates in the placenta due to its affinity for thiol groups in proteins, which facilitates its deposition and leads to oxidative stress and endothelial dysfunction. These disruptions impair placental vascularization, reduce nutrient and oxygen transfer, and negatively affect fetal growth parameters. These findings align with evidence indicating elevated placental mercury levels are linked to reduced placental efficiency and impaired fetal development.[11,12]

Although correlations between placental mercury levels and birth weight ($\rho = -0.16$, $p = 0.12$) or birth length ($\rho = -0.11$, $p = 0.29$) are not statistically significant, the trends support the hypothesis that mercury exposure hinders fetal growth. Mercury in maternal hair correlates with placental mercury due to systemic circulation, making hair a long-term biomarker of exposure. This relationship reinforces the biological plausibility of maternal mercury impacting neonatal outcomes through shared pathways.[5,16]

The findings are further supported by studies showing reduced fetal growth linked to placental metal concentrations, including mercury and prenatal exposure affecting head circumference and placental function.[9,15,18] Dietary mercury exposure through fish consumption could partly explain the observed variability, as fish also supply essential nutrients that may mitigate mercury toxicity.[13]

Maternal hair mercury levels may not directly correlate with neonatal outcomes due to individual differences in mercury metabolism, bioavailability, and placental transfer efficiency. Hair mercury reflects long-term exposure but does not necessarily capture acute or peak exposures that may be more critical for fetal development.[4] Additionally, genetic variations influence mercury detoxification and excretion, altering fetal vulnerability.[19] Factors such as maternal diet also play a role; omega-3 fatty acids in fish may counteract some negative effects of mercury on fetal growth.

Furthermore, mercury exposure contributes to oxidative stress and endothelial dysfunction, impairing placental function and fetal development.[20] Studies have linked oxidative stress markers in umbilical placental to adverse fetal outcomes in hypertensive pregnancies.[21] Mercury-induced oxidative stress can also disrupt nitric oxide signaling, damaging vascular and impaired fetal growth.[22] Future studies should explore how genetic and environmental factors interact with mercury exposure to refine risk assessments and preventive strategies.[23]

The use of 24-hour food recalls to assess mercury levels in the body is limited due to the chronic and cumulative nature of mercury exposure. Mercury primarily accumulates over time through repeated exposure, especially from dietary sources like fish, where it binds to thiol groups in tissues such as keratin and the placenta. This cumulative process means that a single 24-hour food recall cannot capture the long-term dietary patterns that contribute to mercury levels in hair and placental tissue. Studies confirm that short-term dietary recalls, such as those collected over 24 hours, are better suited for tracking immediate intake rather than chronic exposure biomarkers like mercury in biological tissues.[24,25] Research comparing dietary recall methods and biomarkers shows a disconnect between single-day dietary data and cumulative biomarkers like mercury. Mercury biomarkers reflect consistent dietary habits and long-term environmental exposure, requiring comprehensive or repeated dietary assessments to improve accuracy.[12,26]

Healthcare professionals should prioritize early screening for mercury exposure, particularly among high-risk populations with high fish consumption. Educating pregnant women on safer seafood choices, such as consuming low-mercury fish like salmon and sardines while avoiding high-mercury species, is essential. Regular biomonitoring using maternal hair mercury levels can aid in timely intervention. Future research should focus on longitudinal studies to assess long-term developmental effects of prenatal mercury exposure and explore protective dietary factors like omega-3 fatty acids.[5,19].

3. CONCLUSIONS

The study concludes that maternal hair mercury levels significantly correlate with mercury levels in placental blood, indicating the transfer of mercury from mother to fetus. However, hair mercury levels showed no significant direct association with neonatal outcomes, such as birth weight, birth length, and head circumference. Conversely, placental mercury levels negatively correlated with placental weight and head circumference, demonstrating its adverse impact on fetal growth. This finding highlights that placental mercury accumulation may be a more sensitive indicator of neonatal outcomes than maternal hair mercury levels.

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Authors' Contributions

N.F.J.: Protocol development, data management, data analysis, and manuscript preparation and editing; **E.C.J.**: Protocol development, manuscript editing, and supervision; **S.T.M.C.**: Protocol development, data analysis, and manuscript editing; **E.L.:** Protocol development, data collection, and manuscript editing; **L.L.:** Protocol development and supervision. All authors made substantial contribution to the conception of the work and the interpretation of the data, revised it critically for important intellectual content and approved the final version

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