

PAPER • OPEN ACCESS

Prenatal lead exposure and neonatal hearing screening outcomes in newborns from the PIPA cohort project, Rio de Janeiro

To cite this article: Anna Carolina Nascimento Waack Braga Villar *et al* 2026 *Environ. Res.: Health* **4** 025010

View the [article online](#) for updates and enhancements.

You may also like

- [Scenario-based modeling for exploring urban cooling pathways: a targeted, health-relevant approach toward just resilience](#)
Christos Giannaros, Vassiliki Kotroni and Konstantinos Lagouvardos
- [Effect modification of particulate matter air pollution on heat-related mortality in five provinces of Thailand: a two-stage time-series study](#)
Wei-Ling Hung, Masumi Sugeno, Surichai Bilheem *et al.*
- [Polycyclic aromatic hydrocarbons \(PAHs\) contribute to inflammation in a pregnancy cohort](#)
Yoojin Cho, Qi Meng, Kasey E Yu *et al.*

ENVIRONMENTAL RESEARCH HEALTH



PAPER

OPEN ACCESS

RECEIVED

12 November 2025

REVISED

6 March 2026

ACCEPTED FOR PUBLICATION

23 April 2026

PUBLISHED

8 May 2026

Original content from this work may be used under the terms of the [Creative Commons Attribution 4.0 licence](#).

Any further distribution of this work must maintain attribution to the author(s) and the title of the work, journal citation and DOI.



Prenatal lead exposure and neonatal hearing screening outcomes in newborns from the PIPA cohort project, Rio de Janeiro

Anna Carolina Nascimento Waack Braga Villar^{1,*} , Volney de Magalhaes Camara¹ , Ronir Raggio Luiz¹ , Maria Isabel Kós² and Carmen Ildes Rodrigues Fróes Asmus¹

¹ Public Health Institute, Federal University of Rio de Janeiro, Rio de Janeiro 21941-592, Brazil

² Faculty of Medicine, Federal University of Rio de Janeiro, Rio de Janeiro 22240-000, Brazil

* Author to whom any correspondence should be addressed.

E-mail: annacvillar@ippmg.ufrj.br, volney@iesc.ufrj.br, ronir@iesc.ufrj.br, bila@kos.med.br and carmenfroes@iesc.ufrj.br

Keywords: hearing, lead, newborn, environmental pollutants, environmental health

Supplementary material for this article is available [online](#)

Abstract

To investigate the association between prenatal lead (Pb) exposure and neonatal hearing screening (NHS) outcomes. This cross-sectional study included 434 newborns evaluated at a public university maternity hospital in Rio de Janeiro between 2021 and 2022. The NHS was performed using transient evoked otoacoustic emissions, analyzing amplitudes at 2, 3, and 4 kHz. Umbilical cord blood Pb concentrations were measured via inductively coupled plasma mass spectrometry. Participants were categorized as below-median Pb ($<0.80 \mu\text{g dl}^{-1}$) or at/above-median Pb ($\geq 0.80 \mu\text{g dl}^{-1}$). Median Pb concentration was $0.80 \mu\text{g dl}^{-1}$. A negative correlation was observed between Pb levels and 4 kHz right-ear amplitudes ($r = -0.104$, $p = 0.031$). In girls, higher Pb exposure was associated with significantly reduced 4 kHz right-ear amplitudes ($p = 0.008$; $d = 0.36$), which remained significant after multivariable adjustment ($p = 0.009$). Prenatal Pb exposure may impair cochlear function, particularly in girls, even at concentrations below the Centers for Disease Control and Prevention reference value ($3.5 \mu\text{g dl}^{-1}$). These findings highlight the need for environmental metal exposure prevention policies.

1. Introduction

Globally, hearing loss is a major public health problem, affecting people of all ages, including children. According to a recent survey by the World Health Organization (WHO), 1.5 billion people have some type of hearing impairment, with approximately 430 million needing greater assistance, including 34 million children. Without preventive measures being implemented, this number could exceed 2.5 billion by 2050 [1, 2]. A considerable portion of childhood hearing loss is preventable, which underscores the importance of identifying and controlling modifiable risk factors early in life, such as exposure to environmental factors.

Traditional neonatal hearing loss risk factors, such as prematurity, neonatal intensive care unit (NICU) admission, and ototoxic medication use, are well established, yet emerging environmental chemicals, such as heavy metals, represent modifiable contributors warranting early investigation.

Beyond traditional risk factors such as ageing, noise exposure, infections, and ototoxic drugs, a growing body of epidemiological and experimental research has implicated environmental chemicals, particularly heavy metals, in auditory dysfunction [3–7]. Population-based studies and meta-analyses have reported associations between exposure to lead, cadmium, mercury, and other metals and elevated hearing thresholds, particularly at higher frequencies, in adolescents and adults, even at relatively low exposure levels [3–8]. In children, investigations in e-waste-exposed communities and other contaminated settings have linked early-life exposure to lead and cadmium with increased risk of hearing loss and sub-clinical alterations in otoacoustic emissions and brainstem auditory evoked potentials, indicating that the developing auditory system is particularly vulnerable to metal neurotoxicity [4,5].

Exposure to environmental pollutants in urban areas is increasing worldwide [9]. These substances can enter the human body through inhalation, dermal contact, or ingestion. Even during gestation, fetuses are exposed to these compounds due to the high permeability of the placental barrier [10]. Studies have shown that metal concentrations can be higher in infants than in their mothers [11, 12], and the harmful effects of such exposure may manifest at different stages of life, from early childhood to adulthood [12, 13]. Consequently, environmental contamination by hazardous chemical residues and the resulting increase in chemical exposure are recognized as imminent risks to child growth and development [14].

Lead (Pb) is among the ten chemicals of greatest concern to human health due to its widespread toxicity and adverse effects [15]. The relationship between Pb exposure and hearing function, however, remains poorly understood, particularly in newborns. Some studies have suggested an association between higher Pb concentrations and auditory alterations, including the development of chronic otitis media [16] and hearing loss [3–5, 7, 17]. The anatomical and functional integrity of the auditory system is essential for the acquisition of auditory skills, which support proper auditory processing and, consequently, the development of language and speech during the first years of life [18]. However, few studies have examined the association between Pb exposure and neonatal otoacoustic emissions, representing a critical gap in understanding early cochlear effects in newborns.

In this context, potential causal factors of auditory alterations should be further investigated to support prevention, early intervention, and healthy child development. This cross-sectional study, nested within a birth cohort, examined the influence of prenatal Pb exposure on peripheral auditory function through transient evoked otoacoustic emission (TEOAE) testing conducted as part of neonatal hearing screening (NHS) at a public university maternity hospital in Rio de Janeiro, Brazil (July 2021–July 2022).

2. Methods

2.1. Ethical approval

The study was approved by the Research Ethics Committee of the Maternidade Escola of the Federal University of Rio de Janeiro (Approval No. 7.318.857), in accordance with Resolution No. 466/2012 of the National Health Council.

2.2. Study area

This study is part of a research group within a birth cohort investigating exposure to environmental pollutants. The project was conducted at the Federal University of Rio de Janeiro Maternity School, a public university hospital in Rio de Janeiro's southern zone, Brazil. This facility provides prenatal and obstetric care for low-risk pregnant women from its coverage area and high-risk cases from across the city.

2.3. Study population

From July 2021 to July 2022, all pregnant women aged 16 years or older residing in this Brazilian capital and attending the maternity hospital for prenatal care and/or delivery were invited to participate in the birth cohort on exposure to environmental pollutants. Written informed consent was obtained from the mothers or legal guardians. A total of 901 pregnant women were enrolled, resulting in 844 births at the maternity hospital, including twin pregnancies. Thirteen fetal or neonatal deaths occurred (1.54% of eligible births) and 23 cases (2.72%) of address changes or withdrawal after birth took place. Thus, 808 infants were eligible for follow-up (95.73% of the total).

NHS using TEOAE amplitudes was the standard protocol for all newborns [19]. Due to operational limitations, NHS was performed on 572 of the 808 eligible infants (71%), and the results were recorded in the speech-language pathology service database. During the data review for the present study, records with incomplete information regarding birth or examination dates were excluded, resulting in 505 infants. Of these, 23 were excluded due to absence from retesting, which prevented confirmation of a 'failure' in the NHS. Among the 482 eligible newborns, 48 were excluded because an umbilical cord blood Pb assessment could not be performed, likely due to sampling issues.

Thus, the final sample of the present study consisted of 434 infants (figure 1). Perinatal characteristics (sex, gestational age, birth date) were comparable between included and non-evaluated infants.

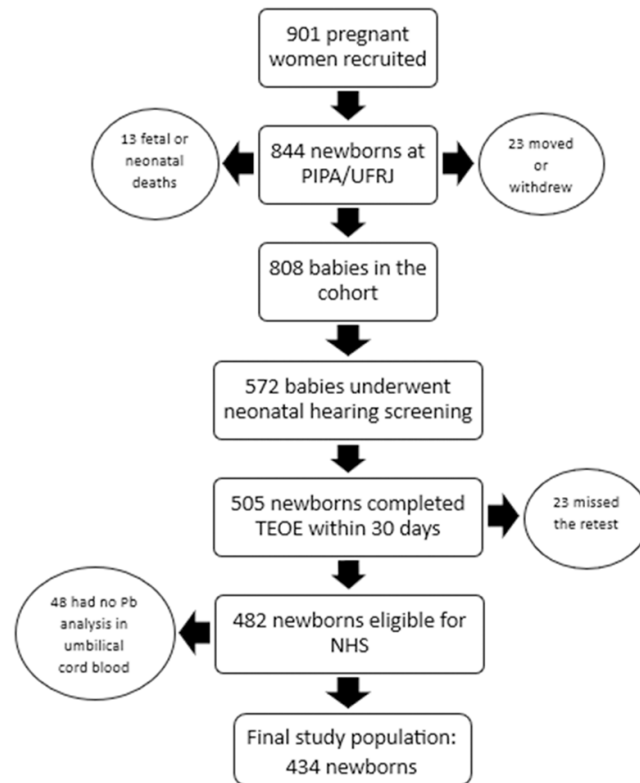


Figure 1. Derivation of the birth cohort sample, PIPA/UFRJ project, to the hearing/Pb study sample ($n = 434$).

2.4. Data sources

The following data sources and procedures were used:

- Birth cohort questionnaires:
 1. Clinical, environmental, and behavioral data (DAC): maternal education, ethnicity, active smoking, alcohol and drug use, household income, and number of residents for per capita income calculation;
 2. Pregnancy record card: date of birth for maternal age calculation;
 3. Newborn questionnaire: birth weight, gestational age, Apgar score, and prematurity.
 - Umbilical cord blood: measurement of lead (Pb) concentrations ($\mu\text{g dl}^{-1}$).
 - Audiological history and NHS: performed preferably within 24 h after birth.

According to the Multiprofessional Committee on Hearing Health, neonatal risk indicators for hearing loss (RIHL) include: stay in the NICU for more than five days; assisted or extracorporeal ventilation; use of ototoxic medications such as aminoglycosides and loop diuretics; hyperbilirubinemia requiring exchange transfusion; perinatal anoxia; Apgar score between 0–4 in the first minute or 0–6 in the fifth minute; birth weight below 1500 g; congenital infections (e.g. cytomegalovirus, rubella, toxoplasmosis, syphilis, herpes, HIV); craniofacial anomalies involving the ears or temporal bones; and family history of childhood hearing loss [19].

In this study, the presence of any of these RIHL was assessed using a dichotomous variable ('yes'/'no') to the question 'Does the newborn present any risk indicator for hearing loss?', classifying all listed indicators collectively as 'present' or 'absent' regardless of whether one or multiple were involved.

TEOAE are generated by rapid stimulation of the cochlea using clicks or tone bursts, covering a broad cochlear frequency range from 300 Hz to 5 kHz. In the NHS protocol of this maternity hospital, TEOAE were evaluated for pass/fail screening at 1 kHz, 1.5 kHz, 2 kHz, 3 kHz, and 4 kHz using click stimuli, preferably within 24 h post-birth. A 'pass' was defined as a response level (signal-to-noise ratio, SNR) of ≥ 6 dB in at least three of these frequencies per ear, indicating overall cochlear integrity and,

specifically, proper functioning of the outer hair cells; a ‘fail’ was recorded when these criteria were not met [20, 21].

TEOAE screening is a passive physiological test requiring no behavioral response from newborns. Pass/fail is determined objectively by detection of otoacoustic emissions (signal-to-noise ratio, SNR ≥ 6 dB) automatically generated by outer hair cells in response to click stimuli. No infant cooperation or audible reactions are required [21].

Otoacoustic emission amplitudes were recorded as SNR in decibels (dB). Values below 0 dB (SNR < 0 dB) were treated as 0 dB, corresponding clinically to the absence of detectable emission. This methodological decision follows clinical practice guidelines and specialized literature on otoacoustic emissions, which interpret negative SNRs as responses indistinguishable from background noise, equivalent to ‘no response’ in the context of NHS [22–24]. Both the pass/fail outcomes and response amplitudes at 2, 3, and 4 kHz per ear were analyzed. These frequency bands were selected because they are less affected by noise interference [25]. The device used for this screening was the Otoport DP + TE from Otodynamics.

3. Laboratory analyses

The studied biological matrix was umbilical cord blood, collected by nursing technicians, nurses, and physicians in the maternity obstetric center immediately after birth. One milliliter of cord blood was transferred to a K2EDTA tube containing anticoagulant for Pb analysis. Samples were sent refrigerated (2 °C–8 °C) to a reference laboratory, where lead concentrations were measured using inductively coupled plasma mass spectrometry (ICP-MS). The limits of detection and quantification were both $0.1 \mu\text{g dl}^{-1}$.

4. Statistical analyses

Descriptive analyses consisted of the calculation of absolute and relative frequencies for categorical variables, while central tendency and dispersion measures (mean, median, standard deviation, minimum, maximum, and percentiles) were calculated for continuous variables. Data distribution normality was assessed using the Shapiro–Wilk test, which indicated non-normal distributions for TEOAE response amplitudes at 2, 3, and 4 kHz per ear, as well as for Pb concentrations.

Associations between Pb concentrations and TEOAE amplitudes were examined using Spearman’s rank correlation; Mann–Whitney U test assessed categorical NHS outcomes (pass/fail).

Pb concentrations were categorized as below-median ($< 0.80 \mu\text{g dl}^{-1}$) or at/above-median ($\geq 0.80 \mu\text{g dl}^{-1}$). Because minimal differences in TEOAE asymmetry were observed between these exposure groups and considering the sample size, parametric tests were used for outcome analysis. Comparisons of TEOAE amplitudes between Pb exposure groups were carried out using the Student’s t -test.

Additionally, the Student’s t -test was applied to assess associations between Pb groups categorized according to the centers for disease control and prevention (CDC) reference value of $3.5 \mu\text{g dl}^{-1}$ [26]. Cohen’s effect size was calculated to quantify the magnitude of TEOAE differences by frequency between exposure groups [27].

Thus, the presence of lead in umbilical cord blood was analyzed both continuously (using Spearman correlation) and categorically (using Student’s t -test and multivariable linear regression).

Finally, multivariable linear regression was performed, stratified by sex, using TEOAE amplitudes at 2, 3, and 4 kHz in each ear as dependent variables. Models adjusted for maternal education (socioeconomic status proxy), active smoking, and illicit drug use—identified *a priori* as potential confounders of the prenatal Pb exposure–neonatal auditory outcome association. Prematurity, birth weight, and RIHL were excluded, as they likely represent causal intermediates on the pathway from prenatal Pb exposure to neonatal hearing outcomes rather than classical confounders. Under this specification, the regression coefficients for Pb exposure should be interpreted as approximating the total effect of prenatal Pb exposure on neonatal auditory function.

A significance level of 5% ($p < 0.05$) was adopted, and all analyses were performed using the IBM SPSS Statistics software, version 21.0. Additional details on the statistical analyses are provided in the supplementary material.

Table 1. Characteristics of the study sample, neonatal hearing screening (NHS) profiles, and umbilical cord blood lead (Pb) exposure.

Sociodemographic, clinical, and gestational habits characteristics		
Variable	Category	<i>n</i> (%)
Maternal age (years)	Mean $\pm$ SD	29.63 $\pm$ 6.97
	<30	243 (56.0)
	$\geq$ 30	191 (44.0)
Per capita income (BRL)	Mean $\pm$ SD	915.20 $\pm$ 854.47
	<550	139 (32.0)
	$\geq$ 550	295 (68.0)
Maternal education (years)	$\leq$ 12	370 (85.3)
	>12	64 (14.7)
Ethnicity	White	113 (26.1)
	Non-white	320 (73.9)
Active smoking	Yes	30 (6.9)
	No	403 (93.1)
Drug use	Yes	19 (4.4)
	No	413 (95.6)
Alcohol consumption	Yes	177 (40.9)
	No	256 (59.1)
Birth data		
Birth weight (g)	Mean $\pm$ SD	3,203 $\pm$ 523
	<3,203	203 (47.4)
	$\geq$ 3,203	225 (52.6)
Sex	Male	218 (50.2)
	Female	216 (49.8)
Pre-term	Yes	51 (11.8)
	No	383 (88.2)
Apgar score at the 5th minute	<7	14 (3.2)
	$\geq$ 7	420 (96.8)
RIHL	Yes	53 (12.2)
	No	381 (87.8)
Lead (Pb) exposure profile		
Pb ($\mu\text{g}/\text{dl}^{-1}$)	Median (P25–P75)	0.80 (0.60–1.20)
	Min–Max	0.10–13.70
	P90	1.90
	P95	2.55
Categories	<0.80 $\mu\text{g}/\text{dl}^{-1}$	189 (43.5)
	$\geq$ 0.80 $\mu\text{g}/\text{dl}^{-1}$	245 (56.5)

Legend: SD = standard deviation; RIHL = risk indicator for hearing loss; Pb = lead in umbilical cord blood; *P* = percentile.

5. Results

Table 1 summarizes sample characteristics, NHS profile, and cord blood Pb exposure. Among 434 evaluated newborns, 218 (50.2%) were male and 216 (49.8%) female; 51 (11.8%) were preterm; and 39 (9.1%) had low birth weight (<2,500 g). Retesting was performed in 10.6% (*n* = 46) of the population. NHS failure rates were 1.4% (*n* = 6, right ear), 1.2% (*n* = 5, left ear), 0.9% (*n* = 4, bilateral).

Pb was detected in 98.85% (*n* = 429) of cord blood samples, with a median concentration of 0.80 $\mu\text{g}/\text{dl}^{-1}$, and 3.9% exceeded the CDC reference (3.5 $\mu\text{g}/\text{dl}^{-1}$) [26]. A weak negative correlation was observed between Pb concentrations and response amplitudes at 4 kHz in the right ear (ρ = −0.104; *p* = 0.031), with a similar weak negative trend at 3 kHz (ρ = −0.086; *p* = 0.074).

An analysis of the Pb exposure groups showed that the lower-exposure group exhibited higher response amplitudes (14.0 dB) compared to the higher-exposure group (12.8 dB) at 4 kHz in the right ear (*p* = 0.033; *d* = 0.20; table 2). Stratification by sex revealed that females in the ‘Pb below median’ group had higher response amplitudes at 3 kHz (15.0 dB) and 4 kHz (15.2 dB) in the right ear compared to those in the ‘Pb at or above median’ group (3 kHz: 13.7 dB; 4 kHz: 13.0 dB), with Cohen’s

No significant differences were detected across sexes and ears in groups with Pb $\geq 3.5 \mu\text{g dl}^{-1}$ [26], nor were there significant associations between Pb concentrations and dichotomous NHS outcomes ('pass'/'fail').

6. Discussion

This study identified a weak negative correlation between TEOAE response levels at 4 kHz in the right ear and cord blood Pb concentrations ($\rho = -0.104$; $p = 0.031$), as well as an association between mean response amplitudes at this frequency and ear by Pb exposure groups ($p = 0.033$; $d = 0.20$). When stratified by sex, this finding was confirmed in female newborns ($p = 0.008$; $d = 0.36$), with a trend also observed at 3 kHz in the same ear ($p = 0.084$; $d = 0.23$). The 4 kHz right-ear amplitude association among girls remained significant after adjustment for maternal education (socioeconomic marker), smoking, and drug use ($p = 0.009$), indicating that these maternal confounders do not fully explain the observed effect. Covariate selection was guided by biological plausibility and existing literature [3–8, 10–14]. Prematurity, low birth weight, and RIHL (neonatal ICU admission >5 d, ototoxic medications, hyperbilirubinemia, anoxia, low birth weight <1500 g, congenital infections, craniofacial anomalies, family history of hearing loss) were not included in the models because they likely represent intermediates on the causal pathway from prenatal Pb exposure to neonatal hearing outcomes. Therefore, our adjusted estimates approximate the total effect of Pb exposure on early cochlear function. No statistically significant differences were found for males.

These results indicate that, as Pb concentrations increase, TEOAE amplitudes decrease at these frequencies. Cohen's effect size ($d \geq 0.2$) confirmed meaningful differences in TEOAE means between exposure groups [27].

Environmental chemicals, including heavy metals, are widely detected in human populations, as demonstrated by large biomonitoring surveys such as NHANES and KNHANES, which consistently report measurable blood levels of lead, cadmium, and mercury in adults and adolescents [3, 7, 8, 17]. In the context of widespread low-level exposure, the high detection rate of Pb in cord blood in our sample, along with the observed reductions in TEOAE amplitudes, even below the CDC reference value of $3.5 \mu\text{g dl}^{-1}$ [26], supports concerns that no safe exposure threshold exists during fetal development [28, 29].

The specificity of the effects at 3–4 kHz aligns with the greater vulnerability of the basal regions of the cochlea, where the highest frequencies reside and which are typically the first affected by noise-induced, ototoxic, or metabolic damage, due to their greater susceptibility to mechanical stress and oxidative injury [30].

The predominance of the right ear in TEOAE reductions corresponds to established cochlear asymmetries, where click-evoked responses are stronger on the right due to hemispheric specialization, since the left hemisphere is for speech processing, and the right for tonal stimuli, as described by Slinger and Cone-Wesson [31].

The specific vulnerability of the female sex observed here likely reflects multiple factors, such as: hormonal/epigenetic influences on lead metabolism and auditory maturation [6, 32]; Sexual dimorphism in cochlear morphometry, as boys have a cochlear duct length approximately 1.2 mm longer and therefore a greater tissue reserve; and baseline TEOAE amplitudes that are significantly higher in women, making proportional ototoxic losses (even subclinical) more easily detectable, as evidenced by the larger effect size ($d = 0.36$) in 4 kHz RE in women [32–35].

The negative correlation between Pb exposure and TEOAE response aligns with previous research in pediatric populations, demonstrating Pb toxicity on the auditory system [3–5]. Those studies demonstrated that Pb can impair both neural conduction and sensory auditory function, even at levels previously considered below risk thresholds [36].

These findings carry significant public health implications and highlight the need for policies focused on monitoring environmental exposure during pregnancy, particularly in urban areas with higher vulnerability. NHS, beyond its clinical role, can serve as a tool for early detection of toxic effects from heavy metals such as Pb, as TEOAE amplitudes are highly sensitive for identifying cochlear dysfunction, often before hearing loss is detectable by pure-tone audiometry. Reductions in TEOAE amplitudes in Pb-exposed infants, even in the absence of clinical hearing loss, indicate subclinical impairment of outer hair cell function, highlighting Pb's impact on cochlear health at early or subtle stages.

Public health strategies aimed at preventing prenatal exposure to heavy metals, including environmental monitoring and health education for pregnant women, are essential to mitigate auditory and neurological development risks in children from urban and socioenvironmentally vulnerable contexts.

The observation of reduced auditory responses in newborns with low Pb exposure reinforces the notion that no safe threshold exists during fetal development [28, 29].

These findings support integrating environmental health actions with NHS programs and longitudinal follow-up of child development.

7. Strengths and limitations

One of the strengths of this study is the use of cord blood as a direct biomarker of exposure, providing more accurate prenatal Pb level estimates, and the analysis of TEOAE at specific frequencies, which allowed for a more detailed assessment of cochlear function.

However, some limitations should be noted. This was a convenience sample due to the equipment availability at the NHS. This may introduce selection bias if non-evaluated infants differ systematically from those included. However, the distribution of key perinatal characteristics (sex, gestational age, date of birth) was preserved.

Negative SNR values were treated as 0 dB, considered clinically equivalent to an absent emission, following clinical procedure guidelines and previous studies [25–27]. While this approach may slightly reduce data distribution, sensitivity analyses indicated that results were not substantially affected, supporting the validity of this strategy to represent clinically meaningful non-detection in NHS.

The absence of prior otoscopic examination could have influenced results in cases of transient conductive obstruction, and the lack of confirmatory tests, such as brainstem auditory evoked potentials, in newborns who failed screening limits the extent to which conclusions can be drawn regarding the true impact of Pb exposure on the auditory pathway.

8. Conclusions

This study identified a weak negative correlation between prenatal Pb exposure and TEOAE amplitudes at 4 kHz in the right ear, with reduced mean amplitudes in higher Pb exposure groups, particularly among female newborns.

The results highlight the importance of integrating environmental health surveillance with NHS and comprehensive childcare, as well as the need for further research encompassing these areas.

Acknowledgments

The authors thank the birth cohort team, the professionals at the maternity hospital where the study was conducted, and the participating families, whose collaboration made this study possible.

Data availability statement

All data that support the findings of this study are included within the article (and any supplementary information files).

Supplementary data 1 available at <https://doi.org/10.1088/2752-5309/ae63f0/data1>.

Statement on the Use of AI Technologies

The authors declare that artificial intelligence (AI) tools were used solely to assist with manuscript formatting (e.g. style, grammar, and text organization). No AI technology was employed for data generation, statistical analysis, result interpretation, or conclusion development. Full responsibility for the scientific content and interpretations presented rests solely with the authors.

Author contributions

Anna Carolina Nascimento Waack Braga Villar  0009-0005-5612-4437

Data curation (lead), Formal analysis (equal), Investigation (equal), Methodology (equal), Software (equal), Visualization (equal), Writing – original draft (equal), Writing – review & editing (equal)

Volney de Magalhaes Camara  0000-0002-6596-6653

Formal analysis (supporting), Visualization (supporting), Writing – review & editing (supporting)

Ronir Raggio Luiz  0000-0002-7784-9905

Formal analysis (equal), Methodology (supporting), Validation (equal)

Maria Isabel Kós  0000-0002-0836-4234

Conceptualization (equal), Methodology (lead), Supervision (equal), Validation (equal), Writing – original draft (lead), Writing – review & editing (lead)

Carmen Ildes Rodrigues Fróes Asmus  0000-0002-9864-6656

Conceptualization (lead), Methodology (lead), Project administration (lead), Supervision (lead), Validation (lead), Writing – original draft (lead), Writing – review & editing (lead)

References

- [1] World Health Organization 2021 World report on hearing (WHO) (available at: www.who.int/publications/i/item/9789240020481) (Accessed 19 January 2026)
- [2] World Health Organization 2018 Addressing the rising prevalence of hearing loss (WHO) (available at: www.who.int/publications/i/item/addressing-the-rising-prevalence-of-hearing-loss) (Accessed 19 January 2026)
- [3] Choi Y H, Hu H, Mukherjee B, Miller J and Park S K 2012 Environmental cadmium and lead exposures and hearing loss in U.S. adults: the national health and nutrition examination survey, 1999–2004 *Environ. Health Perspect.* **120** 1544–50
- [4] Liu Y, Huo X, Xu L, Wei X, Wu W, Wu X and Xu X 2018 Hearing loss in children with e-waste lead and cadmium exposure *Sci. Total Environ.* **624** 621–7
- [5] Wang D H, Xu H, Zheng Y H, Gu D S, Zhu Y J, Ren Y, Wang S-C, Yang L and Xu L-W 2020 Environmental exposure to lead and cadmium and hearing loss in Chinese adults: a case-control study *PLoS One* **15** e0233165
- [6] Llop S, Lopez-Espinosa M J, Rebagliato M and Ballester F 2013 Gender differences in the neurotoxicity of metals in children *Toxicology* **311** 3–12
- [7] Choi Y H and Park S K 2017 Environmental exposures to lead, mercury, and cadmium and hearing loss in adults and adolescents: KNHANES 2010–2012 *Environ. Health Perspect.* **125** 067007
- [8] Shargorodsky J, Curhan S G, Henderson E, Eavey R and Curhan G C 2011 Heavy metals exposure and hearing loss in US adolescents *Arch. Otolaryngol. Head Neck Surg.* **137** 1183–9
- [9] Svoboda E 2018 When skin's defence against pollution fails *Nature* **563** S89–90
- [10] Mazoto M L, Filhote M I F, Câmara V M and Asmus C I R F 2011 Saúde ambiental infantil: uma revisão de propostas e perspectivas *Cad. Saúde Colet.* **19** 41–50 (available at: <https://pesquisa.bvsalud.org/porta1/resource/pt/lil-593698>)
- [11] Santos E O, Jesus I M, Câmara V M, Brabo E S, Jesus M I, Fayal K F and Asmus C I R F 2007 Correlation between blood mercury levels in mothers and newborns in Itaituba, Pará State, Brazil *Cad. Saúde Pública* **23** S622–9
- [12] Asmus C I R F *et al* 2020 Rio birth cohort study on environmental exposure and childhood development—PIPA project *Ann. Glob. Health* **86** 59
- [13] Fróes-Asmus C I R and Câmara V M 2022 Gravidez e poluição *Revista de obstetria* 14th edn, ed J Rezende Filho (Guanabara Koogan) pp 900–28
- [14] World Health Organization 2011 Summary of principles for evaluating health risks in children associated with exposure to chemicals (WHO) (available at: www.who.int/publications/i/item/summary-of-principles-for-evaluating-health-risks-in-children-associated-with-exposure-to-chemicals) (Accessed 19 January 2026)
- [15] World Health Organization 2020 10 chemicals of public health concern (WHO) (available at: www.who.int/news-room/photo-story/detail/10-chemicals-of-public-health-concern) (Accessed 19 January 2026)
- [16] Park M, Lee J S, Lee J H, Oh S H, Park M K and Feng Y 2015 Prevalence and risk factors of chronic otitis media: the Korean national health and nutrition examination survey 2010–2012 *PLoS One* **10** e0125905
- [17] Kang G H, Uhm J Y, Choi Y G, Kang E K, Kim S Y, Choo W O and Chang S S 2018 Environmental exposure of heavy metal (lead and cadmium) and hearing loss: data from the Korea national health and nutrition examination survey (KNHANES 2010–2013) *Ann. Occup. Environ. Med.* **30** 22
- [18] Cabrera L and Lau B K 2022 The development of auditory temporal processing during the first year of life *Hear. Balance Commun.* **20** 155–65
- [19] Comitê Multiprofissional em Saúde Auditiva 2020 Nota técnica—triagem auditiva neonatal universal em tempos de pandemia (COMUSA) (available at: www.sbp.com.br/fileadmin/user_upload/NOTA_COMUSA_2020.pdf) (Accessed 19 January 2026)
- [20] Durante A S 2013 Emissões otoacústicas *Tratado de audiologia* ed M C Bevilacqua, M A N Martínez, S A Balen, A C Pupo, A C M B Reis and S Frota (Santos) pp 145–58
- [21] Azevedo M F 2009 Triagem auditiva neonatal *Tratado de fonoaudiologia* 2nd edn, ed F D M Fernandes, B C A Mendes and N Alpgp (Roca) pp 81–94
- [22] Yang L P, Young S T and Kuo T S 2002 Effects of noise on transient-evoked oto-acoustic emission pass/fail criteria *Med. Biol. Eng. Comput.* **40** 278–81

- [23] Norton S J, Gorga M P, Widen J E, Vohr B R, Folsom R C, Sininger Y S, Cone-Wesson B and Fletcher K A 2000 Identification of neonatal hearing impairment: transient evoked otoacoustic emissions during the perinatal period *Ear Hear.* **21** 425–42
- [24] Interacoustics 2023 Otoacoustic emissions (OAEs): a complete guide (Interacoustics) Accessed 19 Jan 2026 (available at: www.interacoustics.com/academy/oae-training)
- [25] Norton S J, Gorga M P, Widen J E, Folsom R C, Sininger Y, Cone-Wesson B, Vohr B R, Mascher K and Fletcher A K 2000 Identification of neonatal hearing impairment: evaluation of transient evoked otoacoustic emission, distortion product otoacoustic emission, and auditory brain stem response test performance *Ear Hear.* **21** 508–28
- [26] Centers for Disease Control and Prevention (CDC) 2022 Recommended actions based on blood lead level (CDC) (Accessed 19 January 2026) (available at: www.cdc.gov/lead-prevention/hcp/clinical-guidance/index.html)
- [27] Espírito Santo H and Daniel F 2015 Calcular e apresentar tamanhos do efeito em trabalhos científicos (1): as limitações do $p < 0,05$ na análise de diferenças de médias de dois grupos *Rev. Port. Investig. Comport. Soc.* **1** 3–16
- [28] World Health Organization 2021 WHO guidance to reduce illness due to lead exposure (WHO) (available at: www.who.int/news/item/27-10-2021-who-guidance-to-reduce-illness-due-to-lead-exposure) (Accessed 19 January 2026)
- [29] Kasten-Jolly J and Lawrence D A 2017 Sex-specific effects of developmental lead exposure on the immune-neuroendocrine network *Toxicol. Appl. Pharmacol.* **334** 142–57
- [30] Natarajan N, Batts S and Stankovic K M 2023 Noise-induced hearing loss *J. Clin. Med.* **12** 2347 Erratum in: 2024 *J Clin Med.* **13** 944
- [31] Sininger Y S and Cone-Wesson B 2004 Asymmetric cochlear processing mimics hemispheric specialization *Science* **305** 1581
- [32] Baguant A, Cole A, Vilotitch A, Quatre R and Schmerber S 2022 Difference in cochlear length between male and female patients *Cochlear Implants Int.* **23** 326–31
- [33] Cassidy J W and Ditty K M 2001 Gender differences among newborns on a transient otoacoustic emissions test for hearing *J. Music. Ther.* **38** 28–35
- [34] Curtis D P, Baumann A N and Jeyakumar A 2023 Variation in cochlear size: a systematic review *Int. J. Pediatr. Otorhinolaryngol.* **171** 111659
- [35] Mishra S K, Zambrano S and Rodrigo H 2021 Sexual dimorphism in the functional development of the cochlear amplifier in humans *Ear Hear.* **42** 860–9
- [36] Silver M K, Li X, Liu Y, Li M, Mai X, Kaciroti N, Kileny P, Tardif T, Meeker J D and Lozoff B 2016 Low-level prenatal lead exposure and infant sensory function *Environ. Health* **15** 64