Journal of Nutrition Explorations
Vol. 4, No. 4, November 2026, pp. 762~773
e-ISSN: 2987-761X
DOI: 10.36568/jone.v4i4.798
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Urinary Mercury in Toddlers with Nutritional Disorders or Thalassemia near Artisanal Gold Mining in South Aceh, Indonesia: An Exploratory Cross-Sectional Study

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ARTICLE INFO

Article History:
Received September 22, 2026
Accepted November 11, 2026
Published online November 15, 2026

Keywords:
Biomonitoring;
Gold mining;
Mercury;
Nutritional disorders;
Thalassemia;
Urine

ABSTRACT

Mercury amalgamation remains a common practice in artisanal and small-scale gold mining (ASGM) in South Aceh Regency, Indonesia, potentially exposing young children living in nearby communities. This study aimed to describe urinary mercury (U-Hg) concentrations among toddlers with nutritional disorders or thalassemia residing in an ASGM area. An observational cross-sectional study involving laboratory biomonitoring was conducted in April–May 2025 in the service area of UPTD Ujung Padang Rasian Community Health Center. Thirty children aged 0–60 months with undernutrition, malnutrition, stunting, or thalassemia were recruited through purposive sampling. Total mercury concentrations were measured in single urine samples and compared with the German Human Biomonitoring (HBM) reference values (HBM-I, 7 µg/L; HBM-II, 25 µg/L). Data were analyzed descriptively, with exact 95% confidence intervals (CIs) calculated for proportions. Mercury was detected in 10 children (33.3%), while the remaining 20 samples were reported as 0 µg/L. The median U-Hg concentration was 0 µg/L (interquartile range, 0–47.3 µg/L; range, 0–3,003 µg/L), with a mean of 164.3 µg/L (SD 555.5), reflecting several markedly elevated values. Nine children (30.0%; 95% CI, 14.7–49.4) exceeded both HBM-I and HBM-II thresholds, with concentrations ranging from 42 to 3,003 µg/L (median, 228 µg/L). The most frequently reported symptoms were cough (60.0%), followed by headache, weight loss, and loss of appetite (50.0% each). Nearly one-third of children in this selected sample had U-Hg concentrations exceeding the HBM-II threshold. However, the cross-sectional design and absence of a comparison group preclude causal inferences regarding mercury exposure, nutritional disorders, or reported symptoms. Children with confirmed elevated U-Hg concentrations require clinical follow-up and assessment of potential household exposure sources.

INTRODUCTION

Mercury (Hg) is used in artisanal and small-scale gold mining (ASGM) to bind gold particles into an amalgam that is later heated to recover the gold. During this process, mercury is released into air, water, soil, and sediment, and can accumulate in aquatic organisms [1,2]. The World Health Organization considers mercury one of the chemicals of major public health concern, and the developing nervous system is particularly sensitive to it [3].

Humans are exposed to elemental mercury vapor (Hg^0) mainly through inhalation, to inorganic mercury (Hg^{2+}) through ingestion and skin contact, and to methylmercury (MeHg) mainly through fish consumption. In mining communities, children may also be exposed through contaminated household environments, mercury carried home on the clothes of working adults, and accidental contact [4,5].

Infants and toddlers are especially vulnerable because their brain and kidneys are still developing, and both Hg^0 and MeHg readily cross the blood–brain barrier [4]. Urinary mercury mainly reflects exposure to elemental and inorganic mercury, whereas blood and hair are more suitable biomarkers of MeHg exposure [4,6,7].

In the working area of UPTD Ujung Padang Rasian Community Health Center, South Aceh Regency, gold ore is still processed traditionally. The ore is ground in ball mills and mixed with mercury, excess mercury is squeezed out of the amalgam through cloth, and the amalgam is then heated to release the gold. Slurry discharged from the mills, spilled liquid mercury, and vapor released during heating are potential sources of contamination for nearby homes. Elsewhere in Indonesia, about three-quarters of residents living around ASGM sites in Sukabumi had urinary mercury in the alert or high categories of the HBM values, with no significant difference between miners and non-miners [8]. In Aceh, mercury has been detected in soot around amalgam-burning sites [9], in the urine of residents living near traditional gold processing locations [10], and in stunted children in a small-scale mining area of Aceh Jaya Regency [11]. In contrast, a study of 14 wells in the present area did not detect mercury in well water [12], which does not exclude exposure through air, soil, or household routes.

Children under five years of age are the group most vulnerable to undernutrition, which impairs physical growth, brain development, and immunity and has long-term consequences for cognitive ability [13]. Children living in small-scale gold mining areas have been suggested to be at higher risk of stunting [14–16], and mercury exposure in such settings has been linked to lower hemoglobin levels and impaired iron status [17]. However, this evidence comes largely from reviews and small studies, and undernutrition is strongly influenced by diet, infection, and socioeconomic conditions.

Thalassemia is an inherited disorder caused by mutations or deletions in the globin genes, which reduce or abolish the synthesis of α- or β-globin chains and result in chronic hemolytic anemia [18]. Mercury exposure does not cause thalassemia. Because mercury can damage red blood cell membranes and alter hemoglobin function, it has been hypothesized that exposure might aggravate anemia in affected children [17], but this has not been established.

Few studies have measured mercury biomarkers in toddlers with nutritional disorders or thalassemia in ASGM areas of Aceh. This study therefore aimed to describe urinary mercury concentrations, the proportion of toddlers exceeding HBM values, and the pattern of parent-reported symptoms among toddlers with nutritional disorders or thalassemia in the working area of UPTD Ujung Padang Rasian Community Health Center, South Aceh Regency.

LITERATURE REVIEW

Mercury and Human Biomonitoring

Mercury exists in elemental, inorganic, and organic forms that differ in absorption, distribution, and toxicity. Inhaled Hg^0 is efficiently absorbed through the lungs and distributed to the brain and kidneys, whereas inorganic mercury accumulates mainly in the kidneys and is excreted in urine. Methylmercury is formed by microbial methylation in aquatic environments, accumulates in fish, and is excreted mainly in feces [4,6]. The central nervous system and the kidneys are the principal target organs, and early childhood exposure is a concern because of the vulnerability of the developing brain [3,4,19].

Human biomonitoring measures a chemical or its metabolite in body fluids or tissues to estimate internal exposure. Urine is the preferred matrix for elemental and inorganic mercury, but spot urine concentrations vary with hydration, so collection procedures, analytical quality control, and adjustment for creatinine or specific gravity affect interpretation [6,7,20]. The German Human Biomonitoring Commission defines two health-based values for mercury in urine. The HBM-I value (7 µg/L; 5 µg/g creatinine) is the concentration below which no adverse health effects are expected, and the HBM-II value (25 µg/L; 20 µg/g creatinine) is the concentration above which adverse health effects are possible and exposure reduction is required [21,22]. These values are screening and action values, not diagnostic criteria for mercury poisoning.

Nutritional Disorders in Toddlers

Nutritional status in children under five is assessed with anthropometric indices expressed as z-scores against the WHO Child Growth Standards [23]. In Indonesia, the Minister of Health Regulation No. 2 of 2020 classifies underweight as weight-for-age below −2 SD, wasting as weight-for-length or weight-for-height below −2 SD, severe wasting as below −3 SD, and stunting as length- or height-for-age below −2 SD [24]. These conditions have distinct causes and may coexist in the same child. Stunting reflects chronic or recurrent deprivation, while wasting reflects recent weight loss, and both are driven by inadequate dietary intake, infection, and household and socioeconomic factors [13].

Thalassemia

Thalassemia is a group of inherited hemoglobin disorders. β-thalassemia results from reduced (β^+) or absent (β^0) synthesis of β-globin chains, and α-thalassemia from deletion or mutation of α-globin genes. The imbalance of globin chains causes ineffective erythropoiesis and hemolysis, and severe forms require regular blood transfusion and iron chelation [18]. Children with thalassemia are prone to growth failure and undernutrition, which should be considered when interpreting nutritional status in this group [18].

METHOD

Study design and setting

This was an observational cross-sectional study with laboratory biomonitoring, reported following the STROBE guidelines [25]. No intervention or experimental manipulation was applied. The study was conducted in the working area of UPTD Ujung Padang Rasian Community Health Center, South Aceh Regency, Aceh Province, Indonesia, from April to May 2025. Gold ore in this area is processed traditionally by mercury amalgamation.

Participants

Toddlers aged 0–60 months living in the working area of the health center with a recorded diagnosis of undernutrition, malnutrition, stunting, or thalassemia were eligible if a parent or guardian agreed to their participation. Thirty toddlers were selected by purposive sampling. No a priori sample size calculation was performed; the study is therefore exploratory, and the results are presented descriptively. Each child was classified under one recorded diagnostic category, and the four categories together account for all 30 participants. No comparison group of toddlers without nutritional disorders or thalassemia was included.

Diagnostic categories

Nutritional and thalassemia diagnoses were used as recorded for each child. In Indonesia, the nutritional status of children under five is classified from z-scores against the WHO Child Growth Standards [23] according to Minister of Health Regulation No. 2 of 2020 [24], as summarized in Section 2.2. The individual anthropometric measurements and z-scores underlying each diagnosis, and the type and transfusion history of the children with thalassemia, were not available for analysis. The labels undernutrition and malnutrition are therefore reported as recorded and are not mapped to specific anthropometric indices.

Urine collection and mercury analysis

A single urine sample was collected from each child and analyzed for total mercury in the laboratory. Concentrations are expressed in µg/L without adjustment for urinary creatinine or specific gravity. Results reported by the laboratory as 0 µg/L were interpreted as not quantified, that is, below the limit of detection (LOD), rather than as absence of mercury.

Symptom assessment

Symptoms were recorded as present or absent for each child from parents or caregivers using a structured checklist of 32 items (31 distinct symptoms, as headache appeared in two symptom groups) covering the oral, respiratory, gastrointestinal, urinary, skin, neurological, and behavioral domains. These are parent-reported symptoms, not diagnoses of acute or chronic mercury toxicity. Subjective symptoms such as headache, depression, and memory loss cannot be reliably reported for infants and were interpreted with caution.

Reference values

U-Hg was interpreted against the HBM-I (7 µg/L) and HBM-II (25 µg/L) values of the German Human Biomonitoring Commission [21,22]. Concentrations above HBM-II indicate that exposure reduction and clinical and environmental follow-up are warranted; they do not by themselves establish mercury poisoning.

Statistical analysis

Categorical variables are presented as numbers and percentages with 30 children as the denominator. Because two-thirds of U-Hg results were not quantified and the distribution was highly skewed, U-Hg is summarized as the number of quantified results, median, interquartile range (IQR, linear interpolation), and range. The arithmetic mean and standard deviation (SD) are reported only for comparison with earlier studies. A geometric mean was not calculated because it is undefined for non-quantified results and no LOD value was available for substitution [26]. The proportions of children above HBM-I and HBM-II are presented with exact (Clopper–Pearson) 95% confidence intervals [27]. Given the small sample, the absence of a comparison group, and the large number of symptoms, no inferential tests of association between U-Hg and diagnoses or symptoms were performed, and symptom frequencies are reported descriptively.

Research ethics

The study protocol was approved by the Research Ethics Committee of Sari Mulia University, Banjarmasin (No. 076/KEP-UNISM/IX/2023, dated September 9, 2023). Parents or guardians were informed about the study and agreed to their child's participation before data collection, and the results were communicated to the parents and the health center.

RESULT

Participant characteristics

Table 1 summarizes the characteristics of the 30 participants. Most were female (22/30; 73.3%), the largest age group was 37–48 months (8/30; 26.7%), and senior high school was the most common level of parental education (11/30; 36.7%). Undernutrition was the most frequent diagnosis (11/30; 36.7%), followed by stunting (10/30; 33.3%), malnutrition (6/30; 20.0%), and thalassemia (3/30; 10.0%).

Table 1. Characteristics of participants (n = 30)

Characteristic	n	%
Sex		
Male	8	26.7
Female	22	73.3
Age (months)		
0–12	5	16.7
13–24	6	20.0
25–36	6	20.0
37–48	8	26.7
49–60	5	16.7
Parental education		
Elementary school	5	16.7
Junior high school	8	26.7
Senior high school	11	36.7
Bachelor's degree	6	20.0
Length of family residence near the mining area (years)[a]		
1–2	4	13.3
3–4	9	30.0
5–6	13	43.3
7–8	4	13.3
Diagnosis		
Undernutrition	11	36.7
Malnutrition	6	20.0
Stunting	10	33.3
Thalassemia	3	10.0

[a]Duration of residence of the household, which can exceed the child's age; it is not used as a measure of the child's personal exposure duration. Percentages may not total 100 because of rounding.

Urinary mercury concentrations

Mercury was quantified in 10 of 30 toddlers (33.3%), and the remaining 20 results (66.7%) were reported as 0 µg/L (Table 2, Figure 1). Nine toddlers (30.0%; 95% CI 14.7–49.4) had U-Hg above HBM-I, and all nine also exceeded HBM-II, with concentrations of 42–3,003 µg/L (median 228 µg/L), or 1.7 to 120 times the HBM-II value. The only other quantified result (2 µg/L) was below HBM-I. The median for all 30 children was 0 µg/L, whereas the arithmetic mean was 164.3 µg/L because of a few very high values, particularly one child with 3,003 µg/L.

Table 2. Distribution of urinary mercury concentrations (n = 30)

Parameter	Value
Quantified, n (%)	10 (33.3)
Not quantified (reported as 0 µg/L), n (%)	20 (66.7)
Median (IQR), µg/L	0 (0–47.3)
Range, µg/L	0–3,003
Arithmetic mean (SD), µg/L[a]	164.3 (555.5)
Above HBM-I (7 µg/L), n (%) [95% CI]	9 (30.0) [14.7–49.4]
Above HBM-II (25 µg/L), n (%) [95% CI]	9 (30.0) [14.7–49.4]
Children above HBM-II (n = 9)	
Median (range), µg/L	228 (42–3,003)
Arithmetic mean (SD), µg/L[a]	547.3 (939.6)

IQR, interquartile range; HBM, Human Biomonitoring value; CI, exact (Clopper–Pearson) confidence interval. [a]Shown for comparison only; strongly influenced by one value of 3,003 µg/L.

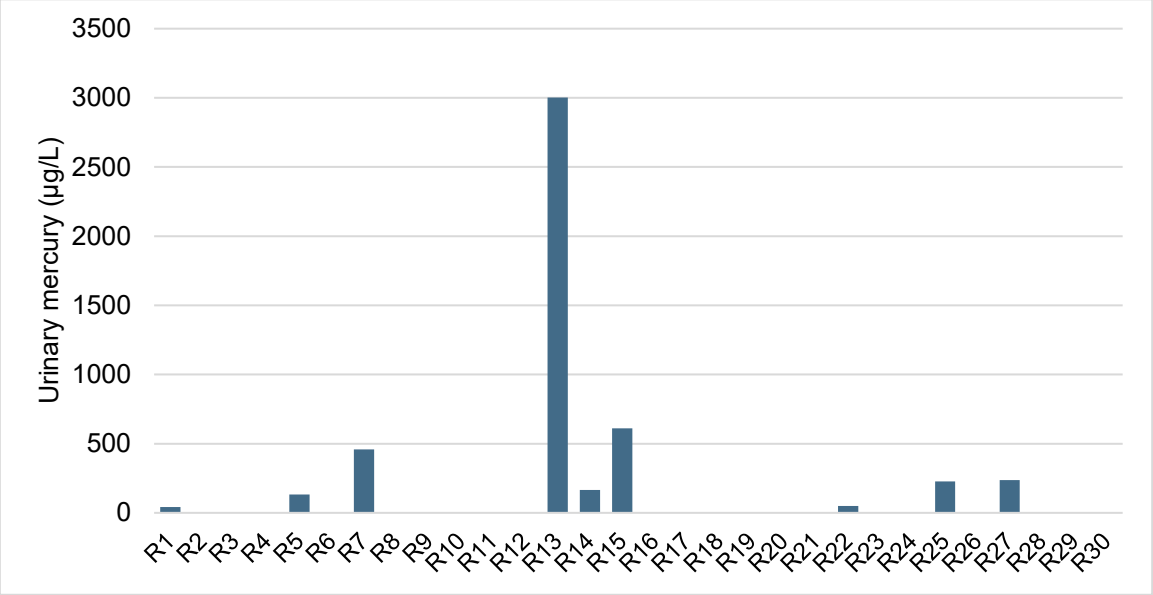


Figure 1. Urinary mercury concentrations of the 30 participants (R1–R30). Twenty results were reported as 0 µg/L. HBM-I, 7 µg/L; HBM-II, 25 µg/L.

Reported symptoms

Table 3 lists the 23 symptoms reported for at least one child. Cough was the most frequent (60.0%), followed by headache, weight loss, and anorexia (50.0% each), chest tightness and abdominal pain (46.7% each), and nausea, diarrhea, and difficulty sleeping (43.3% each). No child was reported to have salivary gland swelling, loss of smell, cloudy urine, blood in urine, pelvic pain, tremor, depression, or memory loss.

Table 3. Frequency of parent-reported symptoms (n = 30)

Symptom	n	%
Cough	18	60.0
Headache	15	50.0
Weight loss	15	50.0
Anorexia	15	50.0
Chest tightness	14	46.7
Abdominal pain	14	46.7
Nausea	13	43.3
Diarrhea	13	43.3
Difficulty sleeping	13	43.3
Chest pain	8	26.7
Vomiting	8	26.7
Aphthous ulcers	7	23.3
Erythema	6	20.0
Irritability	6	20.0
Loose teeth	5	16.7
Pain during urination	4	13.3
Difficulty breathing	3	10.0
Numbness in the mouth	2	6.7
Muscle cramps	2	6.7
Restlessness	2	6.7
Swollen gums	1	3.3
Dark gums	1	3.3
Hearing impairment	1	3.3

Symptoms with no reported cases (salivary gland swelling, loss of smell, cloudy urine, blood in urine, pelvic pain, tremor, depression, and memory loss) are not shown. Headache is counted once.

DISCUSSION

Nearly one-third of toddlers with nutritional disorders or thalassemia in this ASGM area had U-Hg above both HBM values. Under the HBM framework, concentrations above HBM-II call for exposure reduction and clinical and environmental follow-up [21,22]. The highest value (3,003 µg/L) was about 120 times the HBM-II value and is unusually high for a toddler and warrants repeat measurement, clinical evaluation, and investigation of the household exposure source. The finding is consistent with earlier biomonitoring in Aceh, where 36 of 91 residents (39.6%) living near traditional gold processing sites had U-Hg above 7 µg/L [10], and with reports of elevated mercury exposure in an urban ASGM area of Makassar [28] and with the high proportion of residents above HBM values in ASGM areas of Sukabumi, West Java [8]. Differences in age, sampling, and analytical methods limit direct comparison, and the present sample does not estimate the prevalence of elevated U-Hg among all toddlers in South Aceh.

The distribution of U-Hg was strongly uneven: two-thirds of results were not quantified, whereas all nine quantified results above HBM-I were markedly elevated. This pattern suggests that exposure is concentrated in particular households rather than being uniform across the community. Because urine mainly reflects elemental and inorganic mercury [4,6], plausible pathways include inhalation of vapor released when amalgam is heated near homes, mercury carried home by family members who

work in gold processing, and contaminated household surfaces and soil [5,8]. Mercury was not detected in local well water [12], so drinking water from wells is unlikely to explain these results, and methylmercury exposure from fish would require blood or hair measurements. These pathways were not assessed for individual children and need to be investigated.

All participants were selected because they already had a nutritional disorder or thalassemia, and no well-nourished comparison group was included. The study therefore cannot determine whether mercury exposure is associated with, or contributes to, these conditions. Mercury binds sulfhydryl groups of proteins [29] and has been linked to lower hemoglobin and impaired iron status [17], and children in mining areas may be at higher risk of stunting [14–16], but undernutrition is also driven by diet, infection, and socioeconomic disadvantage, which may confound any observed relationship [13].

Thalassemia is a genetic disorder [18] and is not caused by mercury exposure. Whether mercury aggravates hemolysis or anemia in affected children could not be evaluated, because the thalassemia group comprised only three children and hemoglobin, ferritin, and transfusion history were not analyzed. Cough, headache, weight loss, and anorexia were the most frequently reported symptoms. These complaints are nonspecific and common in early childhood infections, and anorexia and weight loss overlap with the nutritional disorders that defined the study population. Headache and other subjective complaints are also difficult to assess in infants. The symptom frequencies therefore cannot be attributed to mercury and should be regarded as hypothesis-generating.

Limitations

This study has several limitations. First, the sample was small (n = 30) and selected purposively from children with existing diagnoses, which limits precision and generalizability and may introduce selection bias. Second, the cross-sectional design and the absence of a comparison group of toddlers without nutritional disorders preclude conclusions about temporality or causality, and U-Hg was not compared across diagnostic groups. Third, U-Hg was measured in a single urine sample without creatinine or specific-gravity adjustment [20], two-thirds of results were not quantified, details of the laboratory LOD and quality control were not available, and urine does not capture methylmercury exposure [7]. Fourth, the anthropometric measurements underlying the recorded nutritional diagnoses were not available, so the diagnostic categories could not be verified against standard indices. Fifth, symptoms were reported by parents, were assessed for many outcomes, and include complaints that cannot be reliably reported for infants, which increases the risk of misclassification and chance findings. Sixth, information on potential confounders and exposure sources, including fish consumption, parental occupation, household mercury use, distance from processing sites, and water source, was not collected, and the sample size did not allow adjustment for confounding. Finally, the thalassemia group was too small for separate analysis.

Future studies should include a comparison group of well-nourished children, larger and representative samples, creatinine-adjusted urine and blood or hair mercury measurements, documented exposure histories, and environmental sampling of air, soil, and household dust.

CONCLUSION

Nine of 30 toddlers (30.0%) with nutritional disorders or thalassemia living near ASGM sites in South Aceh had urinary mercury above both the HBM-I and HBM-II values, with concentrations of 42–3,003 µg/L, whereas mercury was not quantified in two-thirds of the children. Cough, headache, weight loss, and anorexia were the most frequently reported symptoms, but these nonspecific complaints cannot be attributed to mercury. Because the study was cross-sectional and had no comparison group, it cannot determine whether mercury exposure contributes to nutritional disorders, and mercury is not a cause of thalassemia. Toddlers with confirmed elevated urinary mercury should receive clinical follow-up, household exposure sources should be identified and reduced, and larger comparative studies are recommended.

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