

Childhood lead exposure in Bangladesh: a nationally representative biomonitoring survey of prevalence, determinants, and associations with undernutrition and anaemia

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Research Article

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Abstract

Background

Bangladesh is thought to bear one of the world's heaviest childhood lead burdens, but that view rests on modelled estimates rather than on measurement. We report the first nationally representative blood lead survey in Bangladesh.

Methods

In the 2025 Bangladesh Multiple Indicator Cluster Survey, a two-stage stratified cluster survey, venous blood from children aged 12–59 months was analysed for lead and haemoglobin, alongside paired household soil lead, concurrent anthropometry, and a dedicated lead-exposure questionnaire. Survey-weighted regression was used to identify determinants of elevated blood lead (at or above 5 µg/dL) and its associations with stunting, wasting, underweight, and anaemia.

Results

Among 10,622 children, the weighted geometric mean blood lead was 4.42 µg/dL (95% CI 4.35–4.49); 66.2% exceeded 3.5 µg/dL and 38.5% exceeded 5 µg/dL. District prevalence ranged from 5.0% to 93.7% and was concentrated in and around Dhaka (versus Barishal, adjusted odds ratio [AOR] 3.89, 3.06–4.96), with a quarter of the total variance in blood lead lying between districts. Prevalence rose with household wealth (richest versus poorest AOR 1.84, 1.35–2.49; concentration index + 0.127), whereas stunting in the same children concentrated among the poor (-0.134). Each doubling of blood lead was associated with stunting (AOR 1.17, 1.09–1.27), underweight (1.17, 1.08–1.26), and wasting (1.13, 1.03–1.24), but not anaemia (0.94, 0.88–1.01).

Conclusions

Childhood lead exposure in Bangladesh is widespread and geographically concentrated, and its socioeconomic gradient runs opposite to that of undernutrition, so programmes that target poor households are poorly placed to reach the most exposed children. Anaemia is not a useful screening proxy, and geographically targeted control of environmental sources merits priority.

Introduction

Lead (Pb; *sisa* in Bangla) is a heavy metal with no biological function in the human body. It is chemically similar to calcium and iron, is absorbed and stored in bone and soft tissue, and accumulates with repeated exposure. Blood lead concentration, expressed in micrograms per decilitre (µg/dL), is the standard measure of recent exposure. No blood lead concentration is known to be safe, and exposure in

early childhood causes permanent losses in learning ability and attention [1, 2]. In 2021 the US Centers for Disease Control and Prevention lowered its blood lead reference value from 5 to 3.5 µg/dL to better identify the most exposed children [3]. Global models attribute about 1.5 million deaths and more than 30 million disability-adjusted life-years each year to lead, almost all of them in low-income and middle-income countries [4, 5].

South Asia carries more than its share of this burden, and models have repeatedly placed Bangladesh among the countries with the highest estimated childhood blood lead concentrations in the world [6, 7]. Implicated pathways include informal recycling of used lead-acid batteries, lead chromate adulteration of turmeric, lead-glazed cookware, legally permitted leaded paint, and traditional cosmetics such as surma [8–11]. The existing Bangladeshi evidence, however, comes from small studies in hand-picked communities. It cannot support national estimates or show how exposure is spread across the population [12, 13].

This matters because environmental exposures do not have to follow the same social pattern as undernutrition. Where lead arises from industrial activity, urban contamination, or adulterated commodities, the burden may fall on wealthier and urban households rather than the poor, the reverse of the historical pattern in high-income countries, where deteriorating leaded paint in substandard housing concentrated exposure among the disadvantaged [14]. Which pattern holds in Bangladesh decides whether lead screening can be added to existing programmes aimed at poor households, or has to be run separately. A second open question concerns nutritional status: lead and iron are absorbed by the same pathways, which leads one to expect that high blood lead and anaemia occur together [15, 16], while lead has also been linked to impaired growth signalling [17], but because both conditions share social causes, simply seeing them together is an unreliable guide to whether one leads to the other.

The 2025 Bangladesh Multiple Indicator Cluster Survey (MICS) incorporated, for the first time, venous blood collection for heavy metals and haemoglobin, paired household soil sampling, and a dedicated lead-exposure questionnaire, alongside standard anthropometry. We used this survey to establish the national distribution of blood lead, how it varies by area and by social group, its environmental and behavioural determinants, and its association with concurrent undernutrition and anaemia, to inform whether and how lead should be integrated into national child health programming consistent with Sustainable Development Goal target 3.9.

Materials and methods

Study design and participants

We analysed the Bangladesh MICS 2025, implemented by the Bangladesh Bureau of Statistics with UNICEF support using a two-stage stratified cluster design drawn from the 2022 Population and Housing Census frame (3149 clusters, 62,980 households; fieldwork 24 February to 30 June 2025) [18]. Clusters were selected with probability proportional to size within strata defined by division and urban or rural

residence, and households were then selected systematically within each cluster. Of 17,132 children aged 12–59 months eligible for blood collection, 10,634 yielded a valid laboratory result and 10,622 comprised our analytic sample after excluding implausible values (2985 primary sampling units, 131 strata). This study followed the STROBE guideline for cross-sectional studies [19].

Specimen collection and laboratory analysis

The biomarker component was implemented with the Institute of Epidemiology, Disease Control and Research and the International Centre for Diarrhoeal Disease Research, Bangladesh. Venous specimens were collected by trained phlebotomists attached to each field team using lead-free collection materials, kept cool in the field, and transported to the central laboratory, where each specimen was returned with a laboratory validity code; only specimens flagged valid were retained for analysis. Haemoglobin was measured in the field at the time of collection. Household soil specimens were collected concurrently from the same households, from bare ground within the compound where children play, and analysed for the same suite of metals. The survey's laboratory protocol and its quality-control results are set out in its own technical documentation rather than derived here, so we did not re-assess assay performance and treat the released, validity-flagged concentrations as the measurement of record.

Blood lead was analysed continuously (natural log scale) and dichotomised at 5 µg/dL, the threshold applied in the MICS7 protocol, which retains comparability with the substantial literature published before 2021. Because the US Centers for Disease Control and Prevention lowered its blood lead reference value to 3.5 µg/dL in 2021 [3], all principal analyses were repeated at that threshold, and prevalence at 10 µg/dL, the former intervention level, is also reported. Stunting, wasting, and underweight were defined from concurrent anthropometry as height-for-age, weight-for-height, and weight-for-age z-scores below – 2 SD of the WHO Child Growth Standards median, excluding observations flagged as biologically implausible by the WHO algorithm. Anaemia was haemoglobin below 11.0 g/dL, the WHO threshold for children aged 6–59 months.

Exposure and covariate definitions

Explanatory variables were chosen from established lead exposure pathways available in the MICS7 instrument: the child's age in 12-month bands, sex, residence, division, household wealth quintile from the survey's asset index, and mother's education; floor material and the presence and condition of interior paint as recorded by direct observation by the interviewer rather than by report; and, from the dedicated lead module, pica, use of surma or kohl, mouthing a worn amulet, contact with a household member using sindoor, household smoking, the reported presence near the residence of any of six lead-related trades, a specific indicator for the three battery-related trades, and occupational take-home exposure, defined as any individual in regular contact with the child having worked in one of these trades in the preceding three months. Paired household soil lead was analysed on the log scale. Items on behaviour and on nearby trades were reported by caregivers, so some children will be misclassified.

Because the outcome was measured in a laboratory, we would expect that misclassification to be unrelated to the outcome.

Statistical analysis

All analyses were survey-weighted (R version 4.5.0, survey package), accounting for stratification, clustering, and the blood-specimen sampling weight, with Taylor linearisation and single-PSU strata centred at the population mean. Because the blood module was administered to a subsample and participation was incomplete, the blood-specimen weight rather than the general child weight was applied throughout; ignoring the design would understate standard errors given the strong geographic clustering documented below. Central tendency of blood lead is summarised by the geometric mean given its right-skewed distribution. Determinants of elevated blood lead were modelled by multivariable logistic regression (adjusted odds ratios, AORs) and, in parallel, by a log-linear model yielding ratios of geometric means (GMRs). Associations with nutritional and haematological outcomes were modelled per doubling of blood lead (base-2 log) and by category, adjusted for age, sex, residence, division, wealth, and maternal education (nutritional models additionally adjusted for anaemia; the anaemia model for stunting), with tests for linear trend and for stability across four nested adjustment sets. We tested the log-linear assumption by comparing it with a natural spline (3 df) in blood lead, and quantified spatial clustering using a mixed model for log blood lead with random intercepts for survey cluster nested within district. Socioeconomic inequality was quantified with the relative concentration index and its Wagstaff normalisation [20]. Sensitivity analyses re-estimated determinants at the 3.5 µg/dL threshold, adjusted the lead-stunting association for household soil lead, tested effect modification by wealth quintile, and compared complete-case with missing-indicator specifications. Before inferential analysis we verified that our derivation code reproduced eight indicators published in the survey's preliminary report [21]; seven reproduced exactly (Supplementary Table S1).

Ethics

The survey and its biomarker component were approved by the Institutional Review Board of the International Centre for Diarrhoeal Disease Research, Bangladesh. Written informed consent was obtained from a caregiver for every child before specimen collection. This analysis used de-identified data released by the survey programme and involved no contact with participants.

Results

Distribution of blood lead

The weighted geometric mean blood lead concentration was 4.42 µg/dL (95% CI 4.35-4.49; arithmetic mean 5.21 µg/dL [SE 0.06]; median 4.32 µg/dL, IQR 3.09-6.20; Fig. 1A). Two-thirds of children, 66.2% (65.0-67.4), exceeded 3.5 µg/dL; 38.5% (37.2-39.8) exceeded 5 µg/dL; and 7.1% (6.3-7.9) exceeded 10

µg/dL. Design effects for these estimates (1.6-2.1) reflect strong geographic clustering. In the same children, 47.4% were anaemic, 25.9% stunted, 26.7% underweight, and 14.2% wasted.

Geographic distribution

Prevalence varied widely by area. Division prevalence ranged from 16.0% (Rajshahi) to 65.6% (Dhaka; Fig. 1B), and district prevalence from 5.0% (Natore) to 93.7% (Narayanganj; Fig. 2A, B, and Supplementary Table S5). The five highest-burden areas, Narayanganj (93.7%), Dhaka North (92.1%) and South (90.1%) City Corporations, Dhaka district (89.6%), and Munshiganj (65.7%), are contiguous and lie in and around Dhaka. In a mixed model for log blood lead with random intercepts for survey cluster nested within district, 25.2% of the total variance lay between districts and a further 14.2% between clusters within districts, giving an intraclass correlation of 0.39 at cluster level. These proportions were essentially unchanged after adjustment for child age, sex, household wealth, maternal education, and residence (district ICC 0.25), showing that the geographic pattern is not explained by differences in who lives where.

The burden was correspondingly concentrated. Ordering districts by prevalence, the five highest-burden districts contained 22.1% of all children with elevated blood lead while accounting for 9.5% of the child population; the highest 15 districts contained 48.4% of affected children in 28.1% of the population (Fig. 2C). Targeting by area would therefore reach affected children about 1.7 times more efficiently than treating the whole country alike. That is a real gain but not a large one, and it means a targeted programme would still leave about half of affected children outside the priority areas.

Socioeconomic gradient

The socioeconomic gradient ran in the opposite direction to that for undernutrition (Fig. 3). Prevalence rose from 30.2% in the poorest wealth quintile to 51.9% in the richest, and was higher in urban than rural areas (49.9% vs 33.8%). The concentration index was +0.127 (Wagstaff-normalised +0.207) for elevated blood lead, versus -0.134 (-0.180) for stunting and -0.042 (-0.079) for anaemia in the same children (Fig. 3B).

Determinants of elevated blood lead

In the fully adjusted model (Table 1, Fig. 4), division showed the largest associations: children in Dhaka had almost four times the odds of elevated blood lead relative to Barishal (AOR 3.89, 95% CI 3.06-4.96; GMR 1.46, 1.39-1.55), and Sylhet more than twice (AOR 2.13, 1.61-2.81). The positive wealth gradient persisted after adjustment (richest vs poorest AOR 1.84, 1.35-2.49), while maternal education was protective (higher secondary or above vs none AOR 0.42, 0.33-0.54), indicating that wealth and education were independently associated in opposite directions once geography was accounted for. Occupational take-home exposure was independently associated with elevated blood lead (AOR 1.22, 1.02-1.45); residence near battery-related activity approached significance (AOR 1.17, 0.98-1.38, $p=0.075$) and reached it at the 3.5 µg/dL threshold. Generalised variance inflation factors were below 1.6.

Table 1. Survey-weighted multivariable associations with elevated blood lead (at or above 5 µg/dL) and with blood lead on the logarithmic scale.

Group	Level	AOR (95% CI)	P value	GMR (95% CI)
Child age (ref: 12–23 months)	24–35 months	0.88 (0.74–1.04)	0.131	0.96 (0.93–1.00)*
	36–47 months	0.85 (0.72–0.99)*	0.043	0.94 (0.91–0.98)**
	48–59 months	0.73 (0.62–0.86)***	<0.001	0.91 (0.88–0.95)***
Sex (ref: male)	Female	0.76 (0.68–0.85)***	<0.001	0.93 (0.91–0.95)***
Residence (ref: urban)	Rural	0.76 (0.65–0.89)***	<0.001	0.92 (0.89–0.96)***
Division (ref: Barishal)	Chattogram	1.60 (1.26–2.03)***	<0.001	1.13 (1.08–1.20)***
	Dhaka	3.89 (3.06–4.96)***	<0.001	1.46 (1.39–1.54)***
	Khulna	0.57 (0.44–0.74)***	<0.001	0.85 (0.81–0.90)***
	Mymensingh	0.99 (0.72–1.34)	0.929	1.00 (0.94–1.07)
	Rajshahi	0.47 (0.35–0.63)***	<0.001	0.83 (0.79–0.89)***
	Rangpur	0.69 (0.53–0.89)**	0.004	0.88 (0.83–0.92)***
	Sylhet	2.13 (1.61–2.81)***	<0.001	1.22 (1.15–1.30)***
	Second	1.06 (0.89–1.27)	0.494	1.01 (0.97–1.04)
	Middle	1.32 (1.07–1.63)*	0.010	1.04 (1.00–1.09)
Wealth quintile (ref: poorest)	Fourth	1.60 (1.24–2.06)***	<0.001	1.10 (1.04–1.16)***
	Richest	1.84 (1.35–2.49)***	<0.001	1.14 (1.06–1.21)***
Mother's education (ref: none/pre-primary)	Primary	0.97 (0.78–1.21)	0.807	1.03 (0.97–1.08)
	Lower secondary	0.74 (0.59–0.92)**	0.006	0.95 (0.90–0.99)*
	Secondary	0.56 (0.45–0.71)***	<0.001	0.90 (0.86–0.95)***
	Higher secondary or above	0.42 (0.33–0.54)***	<0.001	0.84 (0.80–0.89)***
Housing characteristics	Natural or rudimentary floor	1.00 (0.84–1.19)	0.988	1.00 (0.96–1.03)
	Interior paint, good condition	1.10 (0.95–1.28)	0.217	1.02 (0.98–1.05)
	Interior paint, deteriorating	1.15 (0.96–1.38)	0.126	1.05 (1.01–1.09)*
Behavioural exposures (ref: no)	Pica (ingests soil, clay or paint)	1.13 (0.93–1.37)	0.218	1.04 (0.99–1.08)
	Child uses surma or kohl	1.11 (0.97–1.27)	0.125	1.03 (1.00–1.06)*
	Child mouths an amulet	1.04 (0.88–1.23)	0.663	1.03 (0.99–1.08)
	Contact with sindoor user	0.90 (0.76–1.07)	0.223	0.98 (0.95–1.02)
Environmental exposures (ref: no)	Household member smokes	1.10 (0.98–1.24)	0.103	1.02 (0.99–1.04)
	Any lead-related industry nearby	1.04 (0.89–1.20)	0.629	1.01 (0.98–1.04)
	Caregiver contact works with lead	1.22 (1.02–1.45)*	0.028	1.03 (0.99–1.08)
	Battery manufacture or recycling nearby	1.17 (0.98–1.38)	0.075	1.04 (1.00–1.08)

Estimates are from a single survey-weighted model including all listed covariates simultaneously (n=10,260). AOR=adjusted odds ratio for blood lead at or above 5 µg/dL. GMR=ratio of geometric mean blood lead. CI=confidence interval. ref=reference category. ***p<0.0001, **p<0.01, *p<0.05.

Younger children and boys were more affected. Odds of elevated blood lead fell steadily with age, so that children aged 48-59 months had 27% lower odds than those aged 12-23 months (AOR 0.73, 95% CI 0.62–0.86), consistent with greater hand-to-mouth activity at younger ages, and girls had lower odds than boys (AOR 0.76, 0.68–0.85). Behavioural indicators weakened once geography was accounted for: pica, surma use, and amulet mouthing were each associated with higher crude prevalence but none reached significance in the adjusted model, although in the log-linear model surma use remained associated with a 3% higher geometric mean concentration (GMR 1.03, 1.00–1.06) and deteriorating interior paint with a 5% higher concentration (1.05, 1.01–1.09).

Associations with undernutrition and anaemia

Each doubling of blood lead was associated with higher odds of stunting (AOR 1.17, 95% CI 1.09-1.27), underweight (1.17, 1.08-1.26), and wasting (1.13, 1.03-1.24), and with a height-for-age z-score lower by 0.10 SD (95% CI -0.14 to -0.06); tests for linear trend were significant for all three anthropometric outcomes. These associations were stable across four nested adjustment sets and after additional adjustment for household soil lead, and did not vary by wealth quintile (interaction $p=0.21$) or by child age group ($p=0.33$). A natural spline in blood lead did not improve fit over the log-linear term ($p=0.21$), so the odds ratio per doubling is an adequate summary of the dose-response. The population attributable fraction for stunting was 7.0% (95% CI 2.3-11.7).

By contrast, no positive association was found between blood lead and anaemia. The adjusted odds ratio per doubling was 0.94 (95% CI 0.88-1.01), stable in direction across unadjusted, age-sex-adjusted, geography-adjusted, and fully adjusted specifications (range 0.92-0.97), and the association with haemoglobin measured continuously was null ($\beta=-0.0004$ g/dL per doubling, $p=0.985$). Because 47.4% of all children were anaemic, the 43.5% prevalence among children with elevated blood lead reported in the survey's preliminary report is in fact marginally below the population average rather than evidence of a positive association [21]. Jointly, 18.0% of children had both elevated blood lead and anaemia, 20.5% elevated blood lead alone, 29.3% anaemia alone, and 32.2% neither (Fig. 5).

Table 2. Adjusted associations between blood lead category and nutritional and haematological outcomes.

Outcome	Blood lead category ($\mu\text{g/dL}$)	AOR (95% CI)	P value
Stunting	<2.0 (reference)	1.00	
	2.0–3.4	1.11 (0.88–1.38)	0.383
	3.5–5.4	1.21 (0.97–1.51)	0.086
	≥ 5.5	1.38 (1.09–1.73)**	0.007
Wasting	<2.0 (reference)	1.00	
	2.0–3.4	1.49 (1.13–1.97)**	0.004
	3.5–5.4	1.70 (1.30–2.23)***	<0.001
	≥ 5.5	1.68 (1.26–2.24)***	<0.001
Underweight	<2.0 (reference)	1.00	
	2.0–3.4	1.24 (1.00–1.55)*	0.049
	3.5–5.4	1.34 (1.08–1.65)**	0.007
	≥ 5.5	1.50 (1.20–1.89)***	<0.001
Anaemia	<2.0 (reference)	1.00	
	2.0–3.4	0.77 (0.63–0.94)**	0.010
	3.5–5.4	0.71 (0.58–0.86)***	<0.001
	≥ 5.5	0.75 (0.61–0.93)**	0.008

Children with blood lead below 2.0 $\mu\text{g/dL}$ are the reference category throughout. All models adjust for child age group, sex, place of residence, division, household wealth quintile, and mother's education. Models for stunting, wasting, and underweight additionally adjust for anaemia; the model for anaemia

additionally adjusts for stunting. AOR=adjusted odds ratio. CI=confidence interval. *** $p<0.0001$, ** $p<0.01$, * $p<0.05$.

Soil lead and other metals

Paired household soil lead ($n=10,535$) had a weighted median of 21.0 mg/kg (mean 23.6), below the 400 mg/kg US EPA screening level for bare play areas [22], and was independently associated with children's blood lead (15.9% higher blood lead per doubling of soil lead, 95% CI 12.3-19.6, $p<0.0001$).

Among the other metals measured, elevated blood arsenic (>1.2 $\mu\text{g/dL}$) was found in 1.3% (95% CI 0.9-1.7) of children, similar in those with and without elevated blood lead (1.4% vs 1.2%), and blood arsenic was not positively correlated with blood lead. Lead is therefore the dominant metal exposure in this population by a wide margin. We could not reconcile the published cadmium and mercury prevalences with the released microdata under any consistent unit interpretation and do not report them (Supplementary Note 2).

Sensitivity analyses and participation

Re-fitting the determinants model at the 2021 CDC reference value of 3.5 $\mu\text{g/dL}$ gave essentially the same findings. The division effects retained their magnitude and ordering (Dhaka AOR 3.43, 95% CI 2.68-4.38; Sylhet 3.00, 2.19-4.10), the positive wealth gradient persisted (richest 1.82, 1.38-2.42), and the protective association with maternal education was preserved (higher secondary or above 0.60, 0.47-0.76). Two associations that fell marginally short of significance in the primary model reached it at the lower threshold, namely residence near battery-related activity and deteriorating interior paint, consistent with these pathways contributing across the exposure range rather than only at higher concentrations.

Participation in blood collection was itself socioeconomically patterned: 63.3% of children in the poorest quintile provided a valid specimen versus 42.3% in the richest ($p<0.0001$), and 60.7% among children of mothers with no education versus 46.9% for higher secondary or above ($p<0.0001$). Because non-participation was concentrated among the wealthier and urban households where exposure is highest, the reported wealth gradient is likely conservative rather than inflated. All analyses applied the blood-specimen weight to compensate for differential participation; item missingness was below 2.7% for every covariate, and a missing-indicator sensitivity analysis changed no adjusted odds ratio by more than 0.11 (Supplementary Table S3).

Discussion

To our knowledge this is the first nationally representative measurement of blood lead in Bangladeshi children, and it replaces modelled estimates with direct measurement: two-thirds of children aged 12–59 months had blood lead at or above the current CDC reference value. The national geometric mean of 4.42 $\mu\text{g/dL}$ is about five times the contemporary US average, where fewer than 3% of young children

exceed 3.5 µg/dL [23]. This is consistent with previous modelled analyses placing Bangladesh among the countries with the highest estimated childhood blood lead concentrations [6, 7].

The clearest implication for policy is geographic. District prevalence ranged from 5.0% to 93.7%, and a quarter of the total variance in log blood lead lay between districts, essentially unchanged after adjustment for household characteristics, indicating genuine geographic structure rather than differences in who lives where. The five highest-burden areas form a contiguous block in and around Dhaka in which informal lead-acid battery recycling has previously been documented [12, 13]. Where prevalence is above 89%, as it is in four of these five, screening to find individual affected children adds little, and surveying the environment to find and control sources is the more useful first step. The gain from targeting by area is real but limited: the 15 highest-burden districts hold 48% of affected children in 28% of the child population, so a targeted programme would reach affected children about 1.7 times more efficiently than an untargeted one while still leaving roughly half of affected children outside the priority areas. Targeting is therefore a way to order a national response, not a substitute for one.

The socioeconomic gradient has direct implications for programme design. Stunting and anaemia measured in these same children were concentrated among poorer households, whereas elevated blood lead was concentrated among wealthier and urban households, the opposite of the pattern described for leaded paint in substandard housing in high-income settings [14]. Lead services therefore cannot simply be bolted on to existing nutrition programmes aimed at poor households, because those programmes are built to reach the households at lower risk of this particular exposure. That maternal education was independently protective while wealth was a risk factor, with each adjusted for the other, fits the idea that wealth marks where a family sits within the industrial urban economy, while education reflects care practices that lower exposure within any given place, although we could not test these readings directly.

The graded association we observed between blood lead and anthropometric status, which persisted after adjustment for soil lead and did not vary across wealth quintiles, is consistent with experimental work on lead and growth-hormone signalling and bone mineralisation [17], but the cross-sectional design cannot separate this from the reverse explanation, that undernourished children absorb a greater share of the lead they swallow [15, 16]. Both mechanisms may operate. The anaemia result bears on an interpretation already in circulation: the preliminary survey report noted that 43.5% of children with elevated blood lead were anaemic and read this as supporting integration of lead and anaemia responses [21]. Because 47.4% of all children in this sample were anaemic, that figure is slightly below the population average, and we found no positive association after adjustment in any of four nested specifications. On this evidence anaemia is not a useful proxy for finding lead-exposed children, although it may still be practical to deliver both services through the same programme in priority districts.

Boys had higher blood lead than girls after adjustment, as reported in other paediatric populations and usually attributed to activity and hand-to-mouth behaviour rather than biology. We analysed sex as recorded by the survey; gender is not a meaningful construct at these ages. The finding is best read as

differential exposure opportunity rather than susceptibility, and does not alter the patterns driving our conclusions.

Limitations

Limitations include the cross-sectional design, which cannot establish cause; the roughly 30-day half-life of blood lead, which may understate cumulative exposure relevant to neurodevelopment [24]; uneven participation in blood collection, which survey weighting addresses but cannot rule out as a source of remaining bias, though its likely direction is to make our wealth gradient conservative; exposure pathways reported by caregivers, which will be misclassified for some children; the absence of product-level data on turmeric, cookware, or paint lead content, for which geographic terms in our models likely proxy; and a discrepancy between our anaemia estimate (47.4%) and the published figure (43.9%) that we could not reconcile despite testing thirteen alternative specifications, and consider most likely a tabulation error in the preliminary report given that both sources agree exactly on mean haemoglobin (Supplementary Note 1). This analysis also lacked the survey's geospatial coordinates, which were unavailable at the time of writing and would sharpen point-source identification beyond the district level used here.

Implications for policy and surveillance

Four priorities follow. The first is to order the response by area. In the highest-burden districts the appropriate first step is environmental assessment to locate and control sources, not screening to identify individuals, because near-universal elevation makes case-finding uninformative. In lower-prevalence divisions, particularly Rajshahi, Khulna, and Rangpur, where prevalence is below 20%, targeted screening around identified point sources is the more efficient approach. This difference is worth stating plainly in national guidance, because the two settings call for different actions, not simply more or less of the same action.

The second is informal lead-acid battery recycling, implicated here through the association with occupational take-home exposure and, at the lower reference threshold, with residence near battery-related activity, and documented as a source in Bangladesh and elsewhere [10, 12, 25]. Practical measures include mapping and licensing of recycling sites in priority districts, prohibition of smelting in residential areas, provision of workplace changing and washing facilities to interrupt the take-home pathway, and remediation of contaminated soil at identified sites. This is time-sensitive: demand for battery recycling capacity is rising with the expanding electric two- and three-wheeler fleet, and capacity that forms informally is harder to regulate later than capacity that is licensed from the outset.

The third is enforcement of product standards for turmeric, cookware, cosmetics, and paint. This has the clearest precedent: regulatory action against lead chromate adulteration of turmeric from 2019 was followed by reductions in market turmeric lead concentrations, showing that action against a single commodity can produce measurable change at modest cost [9]. Our finding that surma use and deteriorating interior paint were each associated with modestly higher geometric mean blood lead is

consistent with these being secondary but real contributors. Regular market testing with published results is what turns a standard on paper into a real fall in exposure.

The fourth is sentinel biomonitoring in a set of high- and low-burden districts, reporting the geometric mean and the proportion above 3.5 µg/dL annually against an explicit target. The geometric mean is the more informative headline measure here, because the distribution is strongly right-skewed and threshold-based prevalence is insensitive to change among the most heavily exposed children. Keeping the paired soil sampling and exposure questionnaire used in this survey would allow change to be linked to particular actions rather than only observed. This survey provides a baseline that did not exist before, and its value depends on repeating it.

Conclusions

Direct measurement in a nationally representative sample shows that childhood lead exposure in Bangladesh is widespread, strongly clustered geographically, and distributed across the wealth distribution in the opposite direction to undernutrition. Together these two features mean that the programmes now used to reach undernourished children are poorly placed to reach the children most exposed to lead, and that an area-by-area response built on controlling environmental sources, rather than on screening individuals, is the more efficient starting point. Anaemia should not be used as a screening proxy. Finally, the reason lead exposure matters at this scale is neurodevelopmental rather than nutritional: pooled analyses report a loss of roughly 0.5 IQ points per 1 µg/dL, steepest at the lowest concentrations [2]. We did not estimate the loss in learning implied here, and carrying exposure-response relationships from one population to another is uncertain, but the concentrations we report fall where those analyses place the steepest effects. That matters for a country investing in schooling and child nutrition, because lead acts against the returns to those investments, and unlike most determinants of early child development it is preventable.

Declarations

Ethics approval and consent to participate

This study analysed de-identified secondary microdata from the 2025 Bangladesh Multiple Indicator Cluster Survey. The survey and its biomarker component were approved by the Institutional Review Board of the International Centre for Diarrhoeal Disease Research, Bangladesh (icddr,b), and written informed consent was obtained from caregivers during data collection. No additional ethical approval was required for this secondary analysis of anonymised data.

Clinical trial number

Not applicable.

Consent for publication

Not applicable.

Availability of data and materials

The Bangladesh MICS 2025 datasets analysed in this study are available from UNICEF at mics.unicef.org/surveys following registration and approval. The analysis code that reproduces every reported estimate is available from the author on reasonable request.

Competing interests

The author declares that he has no competing interests.

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Authors' contributions

ARR is the sole author. He conceived and designed the study, curated the data, performed the statistical analysis, produced the figures, and drafted and revised the manuscript. The author read and approved the final manuscript.

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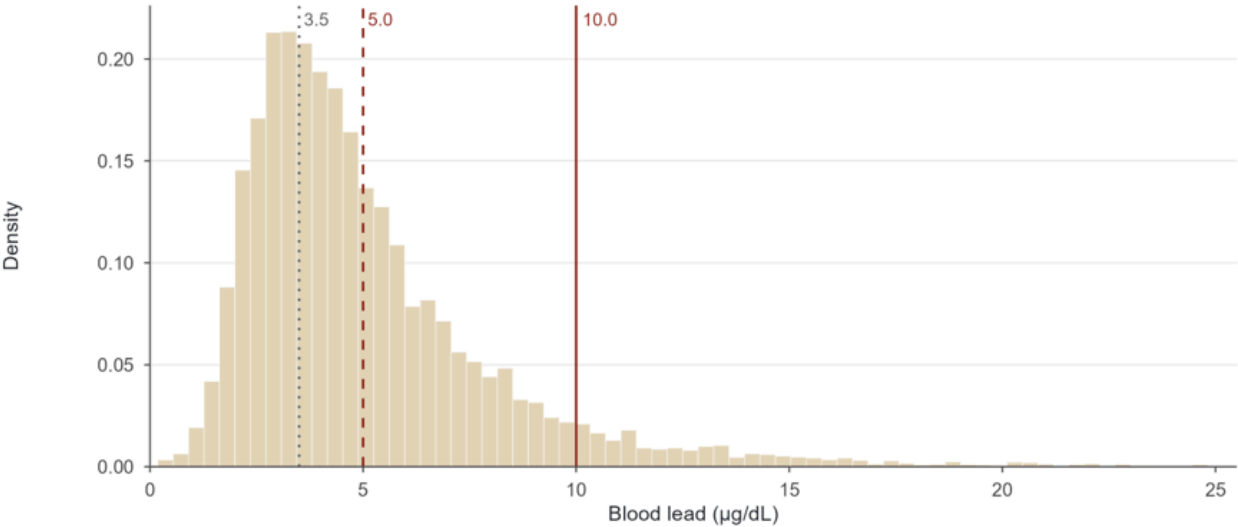
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Figures

A Distribution of blood lead concentration

Survey-weighted; reference lines at 3.5, 5 and 10 µg/dL



B Elevated blood lead by division

Children 12–59 months with blood lead ≥ 5 µg/dL (95% CI)

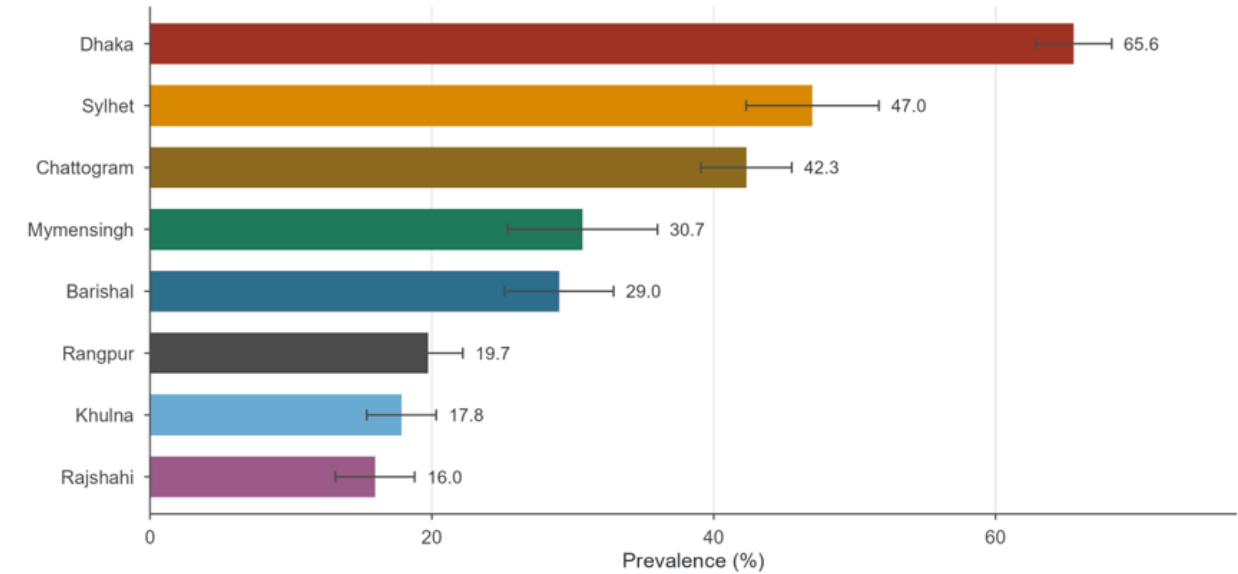


Figure 1

Distribution of blood lead concentration (A) and prevalence of elevated blood lead by division (B), with 95% confidence intervals.

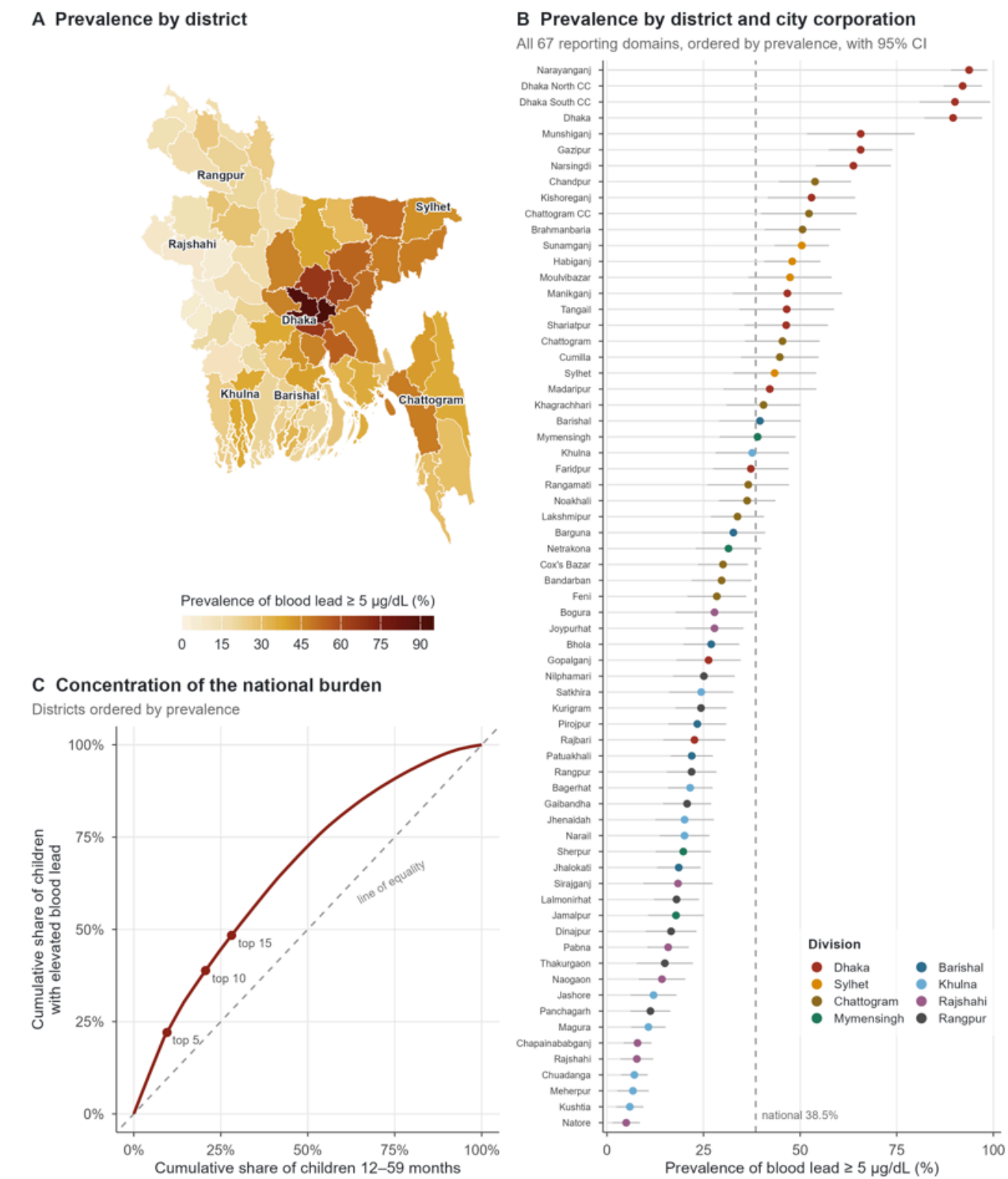
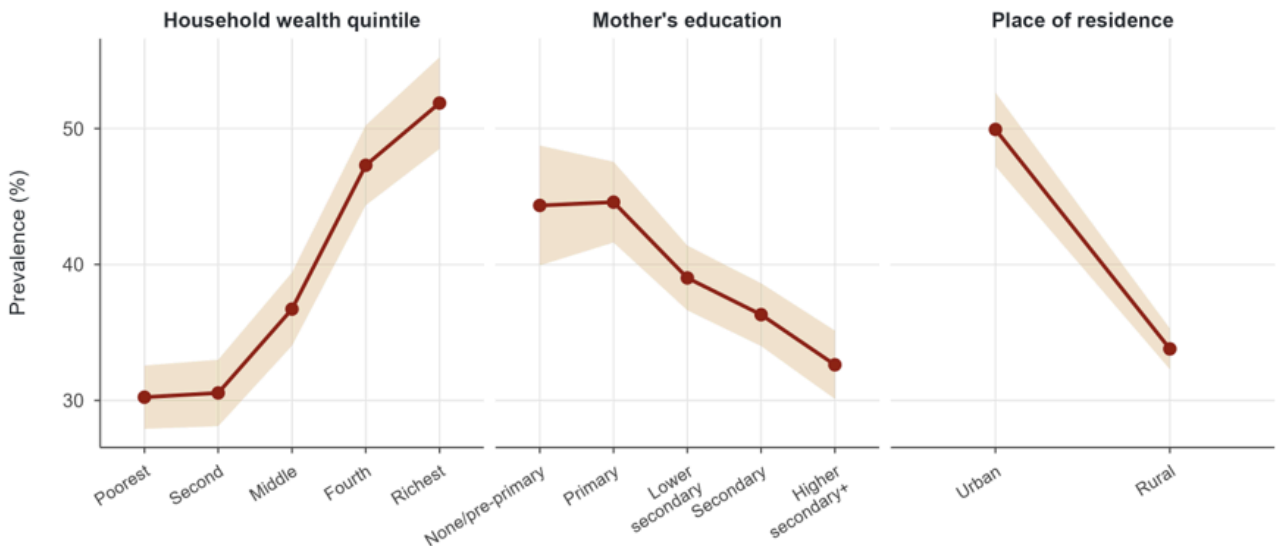


Figure 2

Geographic distribution and concentration of childhood lead exposure. (A) Prevalence of blood lead at or above 5 µg/dL by district; the three city corporations are combined with their parent districts (Dhaka, Chattogram). Boundaries from GADM v4.1. (B) Prevalence in each of the 67 reporting domains, ordered by prevalence and coloured by division, with 95% confidence intervals; the dashed line marks the national prevalence. (C) Cumulative share of children with elevated blood lead against cumulative share of the child population, with districts ordered by prevalence; the dashed line denotes proportionality. Map lines delineate study areas and do not necessarily depict accepted national boundaries.

A Socioeconomic gradients in elevated blood lead

Prevalence of blood lead ≥ 5 µg/dL with 95% confidence bands



B Concentration curves by household wealth

A curve below the diagonal means the burden falls more on wealthier children

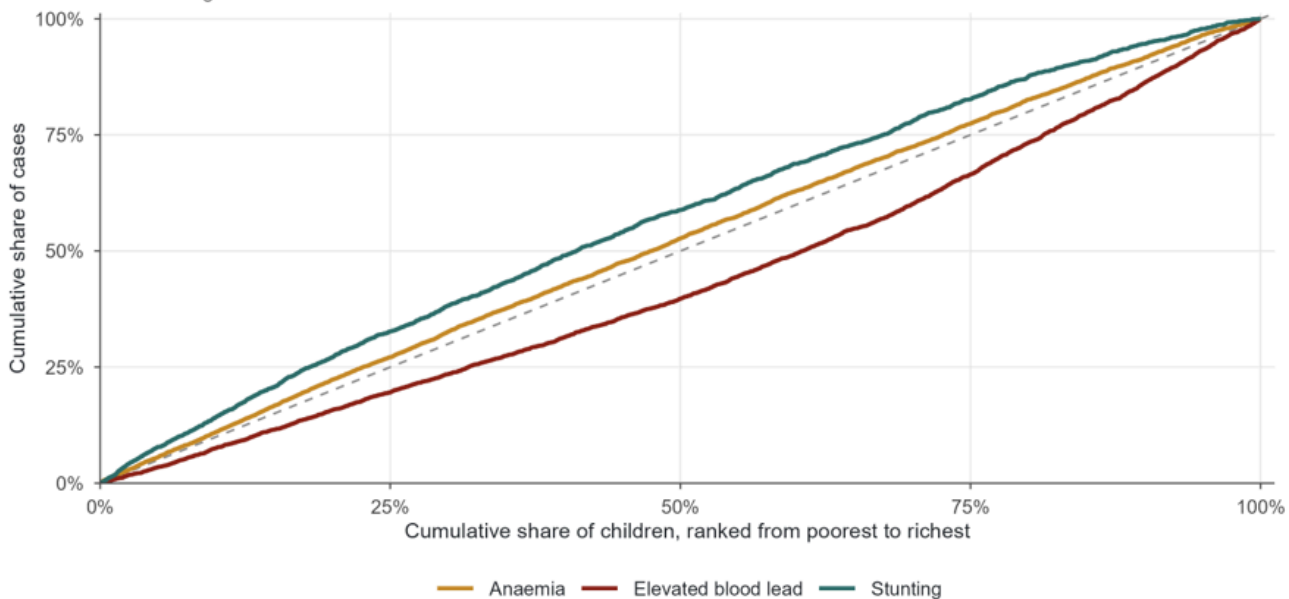


Figure 3

Socioeconomic patterning of childhood lead exposure. (A) Prevalence of blood lead at or above 5 µg/dL by household wealth quintile, mother's education, and place of residence, with 95% confidence bands. (B) Concentration curves for elevated blood lead, stunting, and anaemia against household wealth; a curve below the diagonal indicates a burden falling more on wealthier children, and one above it a burden falling more on poorer children.

Determinants of elevated blood lead (≥ 5 µg/dL)

Survey-weighted multivariable logistic regression; adjusted odds ratios with 95% CI

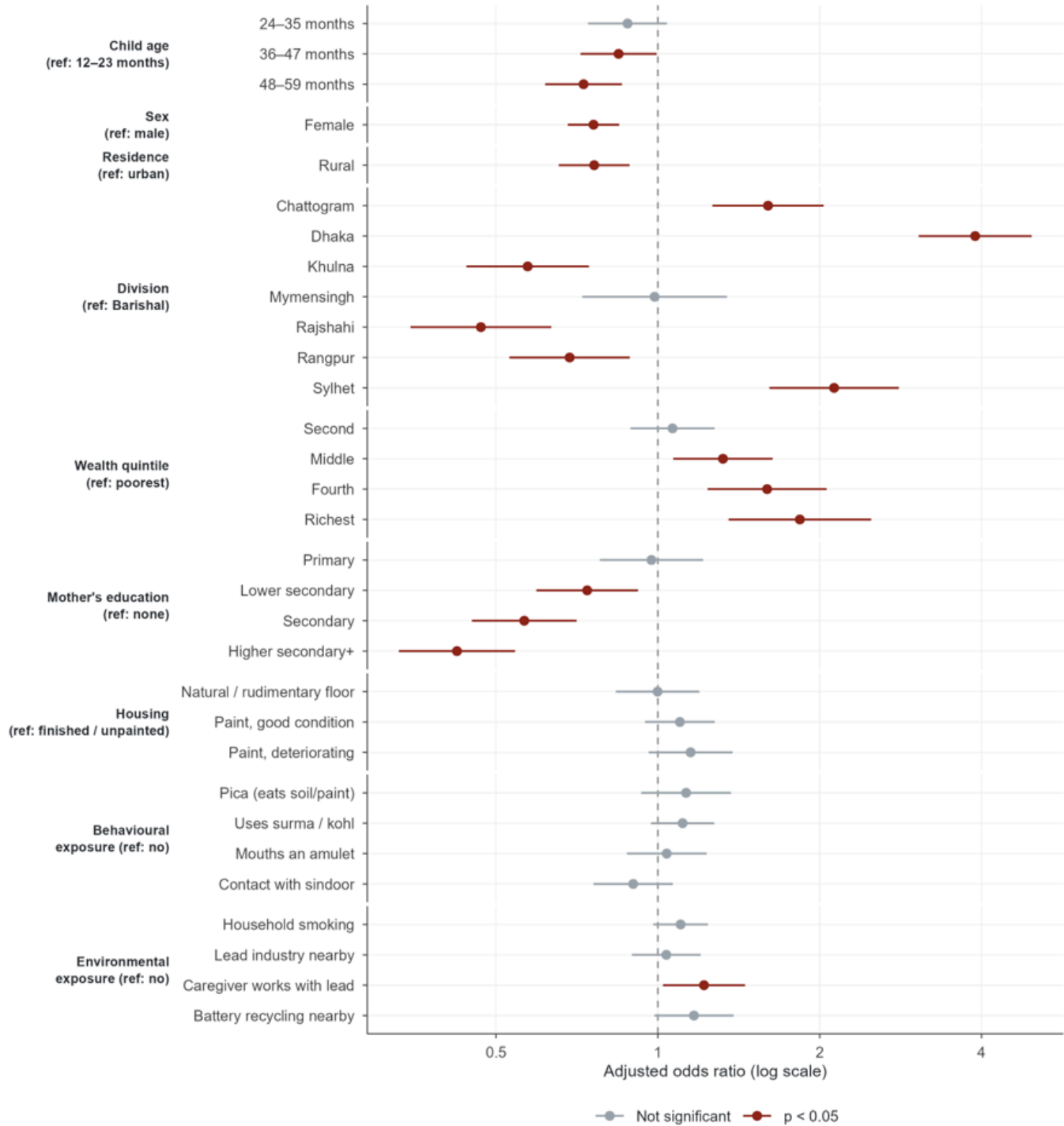


Figure 4

Adjusted odds ratios for elevated blood lead (at or above 5 µg/dL), survey-weighted multivariable logistic regression.

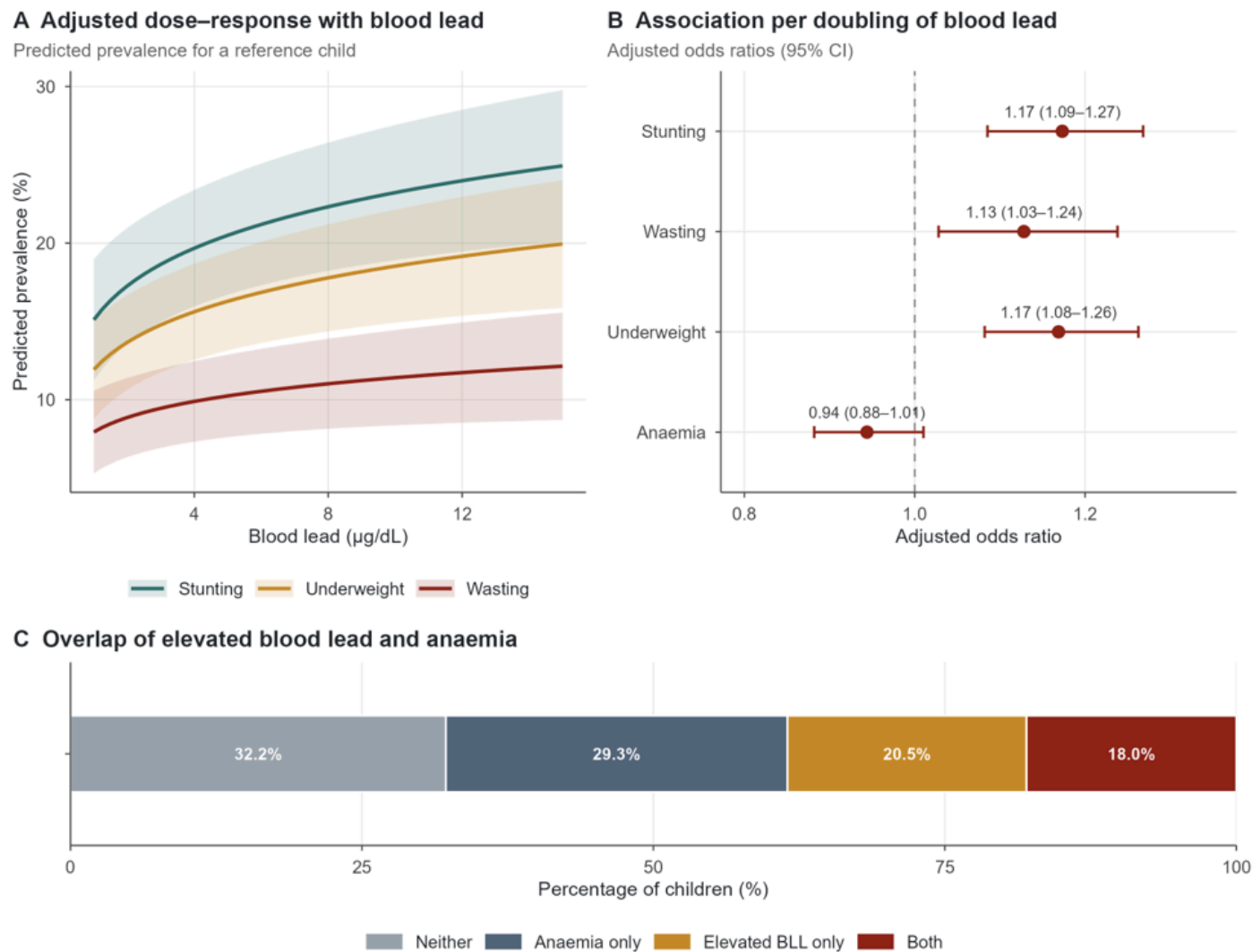


Figure 5

Blood lead and nutritional and haematological outcomes: dose-response (A), adjusted odds ratios per doubling of blood lead (B), and overlap with anaemia (C). Models in panels A and B use the same specification as Table 2.

Supplementary Files

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- [SupplementaryMaterial.docx](#)