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Contaminant exposure in household dust and severity of autistic traits: An environmental risk analysis

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Abstract
While industrialised nations have expanded environmental monitoring of household chemical exposures, comparable data from sub-Saharan Africa remain scarce despite the region bearing a disproportionate burden of unregulated contaminant sources. This cross-sectional research examined associations between household dust contaminant concentrations and the severity of autistic traits in 204 children aged 3 to 8 years in the Greater Accra Region, Ghana. Settled dust was collected from the living areas of participating households and analysed for five contaminant classes: polybrominated diphenyl ethers (PBDEs), phthalates, heavy metals (lead, mercury, cadmium), organophosphate esters (OPEs), and polycyclic aromatic hydrocarbons (PAHs). Autistic traits were measured using the Social Responsiveness Scale, Second Edition (SRS-2). A composite dust contaminant burden index was calculated as the sum of standardised concentrations across all detected analytes. PBDEs constituted the largest proportion of the contaminant profile (28.4%), followed by phthalates (24.7%) and heavy metals (22.1%). The mean contaminant burden index was significantly higher among children scoring above the SRS-2 clinical threshold (mean = 47.6, SD = 18.3) compared with those below the threshold (mean = 31.2, SD = 14.7; $p < 0.001$). Multiple linear regression, adjusted for child age, sex, household income, parental education, and proximity to industrial sites, showed that the contaminant burden index was positively associated with SRS-2 total T-scores (beta = 0.28, 95% CI: 0.16 to 0.40, $p < 0.001$). Among individual contaminant classes, PBDE concentrations and lead levels showed the strongest independent associations. These findings indicate that household dust contaminant exposure is associated with autistic trait severity in Ghanaian children, supporting the need for environmental health surveillance in West African settings.

Keywords: Household dust, Contaminants, Autistic traits, PBDEs, Phthalates, Heavy metals, Environmental risk, Ghana, SRS-2

Introduction

Advances in understanding the genetic architecture of autism have been accompanied by a growing recognition that environmental exposures contribute meaningfully to the expression and severity of autistic traits (Grandjean *et al.*, 2014; Ye *et al.*, 2017) [1, 2]. Chemical contaminants present in indoor environments represent a biologically plausible pathway linking the built environment to neurodevelopmental variation, yet the majority of environmental epidemiological research on this topic has been conducted in high-income settings with established monitoring infrastructure (Windham *et al.*, 2006; Vuong *et al.*, 2018) [3, 4]. In contrast, low- and middle-income countries where unregulated waste disposal, informal electronics recycling, and poorly ventilated cooking practices create distinct indoor contamination profiles have received comparatively little attention (Ding *et al.*, 2023; Bawua *et al.*, 2025) [5, 6].

Household dust serves as a reservoir for semi-volatile organic compounds and particulate-bound metals that accumulate from consumer products, building materials, and ambient pollution (Lam *et al.*, 2017; Tanner *et al.*, 2019) [7, 8]. Children are particularly vulnerable to dust-borne contaminants owing to their hand-to-mouth behaviours, time spent on floors, and higher intake-to-body-weight ratios relative to adults (Grandjean *et al.*, 2014) [1]. Five contaminant classes have been linked to neurodevelopmental outcomes in previous investigations: polybrominated diphenyl ethers (PBDEs) used as flame retardants, phthalates from plasticised consumer goods, heavy metals from industrial emissions and paint, organophosphate esters (OPEs) serving as replacement flame retardants, and polycyclic aromatic hydrocarbons (PAHs) from incomplete combustion (Jeddi *et al.*, 2016; Ye *et al.*, 2017; Vuong *et al.*, 2018; Owens *et al.*, 2025) [9, 2, 4, 10].

Evidence connecting individual contaminant classes to autistic traits has accumulated over the past decade. PBDEs have been associated with social communication difficulties and attention problems in North American cohorts (Lam *et al.*, 2017; Vuong *et al.*, 2018; Sagiv *et al.*, 2019) [7, 4, 11]. Phthalate metabolites have shown associations with reciprocal social behaviour deficits in European populations (Jeddi *et al.*, 2016; Philippat *et al.*, 2017; Cunha *et al.*, 2023) [9, 12, 13]. Heavy metal exposures, particularly lead and mer

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cury, have been linked to autism severity in *meta*-analytic evidence (Dickerson *et al.*, 2015; Ding *et al.*, 2023) [14, 5]. However, household dust typically contains mixtures of these contaminants rather than isolated compounds, and the cumulative burden of co-occurring exposures may be more relevant to neurodevelopmental outcomes than any single chemical class (Tanner *et al.*, 2019; Owens *et al.*, 2025) [8, 10].

Ghana presents a distinctive exposure context. The Greater Accra Region combines rapid urbanisation, proximity to the Agbogbloshie e-waste processing site, widespread use of imported plastic consumer goods, and limited indoor air quality regulation (Heng *et al.*, 2022; Bawua *et al.*, 2025) [15, 6]. These conditions generate household dust contamination profiles that differ from those documented in high-income settings, and no prior research has examined associations between indoor dust contaminant mixtures and autistic trait severity in a Ghanaian child population.

This research addressed this gap by characterising the contaminant profile of household dust collected from 204 homes in the Greater Accra Region and testing associations between a composite dust contaminant burden index, individual contaminant class concentrations, and SRS-2-measured autistic traits in children aged 3 to 8 years. We hypothesised that higher cumulative contaminant exposure would be associated with greater autistic trait severity after adjustment for sociodemographic covariates (Braun *et al.*, 2014; Shelton *et al.*, 2015; Skogheim *et al.*, 2021) [16, 17, 18].

Theoretical framework

This research was guided by the cumulative environmental risk framework, which posits that neurodevelopmental outcomes reflect the aggregate impact of co-occurring chemical, physical, and social stressors rather than the independent effects of isolated exposures (Grandjean *et al.*, 2014; Ye *et al.*, 2017) [1, 2]. Within this framework, household dust serves as an integrative exposure matrix that captures contributions from multiple contaminant sources present in the indoor environment (Tanner *et al.*, 2019; Owens *et al.*, 2025) [8, 10]. The framework further incorporates the concept of developmental windows of susceptibility, recognising that the postnatal period of rapid brain maturation renders young children particularly sensitive to neurotoxicant exposures (Grandjean *et al.*, 2014) [1].

The biological plausibility of the hypothesised associations rests on established mechanisms of contaminant-mediated neurotoxicity. PBDEs and OPEs interfere with thyroid hormone signalling and disrupt calcium-dependent neurotransmission (Lam *et al.*, 2017; Vuong *et al.*, 2018) [7, 4]. Phthalates act as endocrine disruptors that alter androgen and oestrogen pathways implicated in sexually dimorphic neurodevelopmental trajectories (Jeddi *et al.*, 2016; Philippat *et al.*, 2017) [9, 12]. Lead and mercury directly impair synaptic plasticity and neuronal migration through oxidative stress and NMDA receptor interference (Dickerson *et al.*, 2015; Ding *et al.*, 2023) [14, 5]. PAHs activate aryl hydrocarbon receptor pathways that modify DNA methylation and gene expression in developing neural tissue (Ye *et al.*, 2017; Payne-Sturges *et al.*, 2023) [2, 19]. These mechanisms converge on neural circuits subserving social cognition and sensory integration, which are preferentially affected in autism (Grandjean *et al.*, 2014; Skogheim *et al.*, 2021) [1, 18].

Research area description

The Greater Accra Region occupies the southeastern coast of Ghana and encompasses the national capital, Accra, along with surrounding municipalities including *Tema*, Ashaiman, and Ga districts. The region had an estimated population of 5.4 million as of the 2021 Population and Housing Census and is the most urbanised region in the country. Rapid population growth and informal settlement expansion have outpaced waste management infrastructure, resulting in widespread open burning of domestic and electronic waste (Heng *et al.*, 2022; Bawua *et al.*, 2025) [15, 6].

The Agbogbloshie scrapyard, located within the Accra Metropolitan Area, has been identified as one of the most polluted locations on the African continent. Informal recycling of imported electronic equipment at this site releases PBDEs, heavy metals, and PAHs into the surrounding air, soil, and watercourses, with documented dispersion extending several kilometres into residential neighbourhoods (Bawua *et al.*, 2025) [6]. Beyond the immediate vicinity of this site, household dust contamination in the Greater Accra Region is influenced by the use of imported plastic furniture and toys containing phthalate plasticisers, leaded paint in older housing stock, mosquito coil burning, and particulate emissions from vehicular traffic on unpaved roads (Heng *et al.*, 2022) [15].

Materials and Methods

Research design and participants

This cross-sectional research employed a community-based sampling strategy in the Greater Accra Region, Ghana. Between March 2023 and November 2023, households with at least one child aged 3 to 8 years were recruited through community health centres, early childhood development programmes, and neighbourhood health volunteer networks across six sub-districts representing a gradient of proximity to known contaminant sources. Ethical approval was granted by the Ghana Health Service Ethics Review Committee (Protocol GHS-ERC/023/02/23) and the Greater Accra College of Applied Health Sciences Institutional Review Board. Written informed consent was obtained from all participating caregivers and verbal assent from children aged 6 years and older.

Dust sample collection and chemical analysis

Settled dust samples were collected from the primary living area of each household using a standardised protocol adapted from the American Society for Testing and Materials method D5438-17. A high-volume surface sampler was used to vacuum a 1.0 *m*-squared area of flooring where the index child most frequently played. Samples were sieved through a 150-micrometre mesh, stored in pre-cleaned amber glass vials, and transported to the analytical chemistry laboratory of the Environmental Protection Agency, Accra, within 48 hours of collection. Dust samples were analysed for 12 PBDE congeners (BDE-28, -47, -66, -85, -99, -100, -153, -154, -183, -196, -206, -209), 8 phthalate esters (DEHP, DBP, BBP, DEP, DMP, DINP, DIDP, DnOP), 3 heavy metals (lead, mercury, cadmium) by inductively coupled plasma mass spectrometry, 6 OPE compounds, and 16 EPA priority PAHs by gas chromatography-mass spectrometry.

Autistic trait assessment

Autistic traits were assessed using the parent-report SRS-2 School-Age Form, which yields a total T-score reflecting social communication difficulties. The SRS-2 has been validated in Ghanaian paediatric populations and demonstrated acceptable internal consistency in the present sample (Cronbach alpha = 0.91). Children scoring at or above a T-score of 60 were classified as having elevated autistic traits for stratified analyses, consistent with established clinical thresholds (Braun *et al.*, 2014) [16].

Statistical analysis

A composite dust contaminant burden index was calculated by first log-transforming concentrations of each analyte, then standardising each to a z-score within the sample, and summing the z-scores across all detected compounds. Individual contaminant class indices were similarly computed for PBDEs, phthalates, heavy metals, OPEs, and PAHs separately. Covariates selected a priori included child age, sex, household monthly income (categorised into tertiles), highest parental education level, dwelling construction type, cooking fuel type, and straight-line distance from the household to the nearest identified industrial or e-waste processing site (Shelton *et al.*, 2015; Minatoya *et al.*, 2021; Skogheim *et al.*, 2021) [17, 20, 18].

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Results

Of the 486 households initially identified, 389 met eligibility criteria and were screened, 312 provided adequate dust samples above the analytical detection limit, 235 children completed the SRS-2 assessment, and 204 contributed complete data for the primary analyses (Figure 2). Children with elevated autistic traits (SRS-2 T-score of 60 or above; $n = 87$) and those below this threshold ($n = 117$) did not differ significantly by age, sex distribution, or household income tertile, though the elevated group showed marginally lower parental education levels (chi-squared = 4.12, $p = 0.042$).

The distribution of detected contaminant categories across all dust samples is presented in Figure 1. PBDEs comprised the largest proportion of the overall contaminant profile (28.4%), followed by phthalates (24.7%), heavy metals (22.1%), OPEs (14.3%), and PAHs (10.5%). Contaminant class concentrations stratified by autistic trait group are shown in Table 2. PBDE, phthalate, lead, mercury, and OPE concentrations were significantly higher in the above-threshold group, while PAH concentrations did not differ significantly between groups.

In the unadjusted linear regression model, the composite contaminant burden index was positively associated with SRS-2 total T-scores (beta = 0.28, 95% CI: 0.16 to 0.40, $p < 0.001$), explaining 7.8% of the variance in autistic trait scores. After adjustment for covariates, the association remained significant (adjusted beta = 0.24, 95% CI: 0.11 to 0.37, $p < 0.001$), with the full model explaining 19.4% of the variance (Table 1). The relationship between the composite burden index and SRS-2 scores is illustrated in Figure 3.

When individual contaminant class indices were entered simultaneously into a single adjusted model, PBDE concentrations (beta = 0.19, 95% CI: 0.06 to 0.32, $p = 0.004$) and lead levels (beta = 0.17, 95% CI: 0.04 to 0.30, $p = 0.009$) showed the strongest independent associations with SRS-2 scores. Phthalate concentrations approached significance (beta = 0.12, 95% CI: -0.01 to 0.25, $p = 0.067$). Mercury, OPE, and PAH class indices were not independently associated with SRS-2 scores in the multi-class model. The individual-class model explained 22.3% of the variance, representing a modest improvement over the composite index model.

An exploratory interaction analysis indicated that the association between the contaminant burden index and SRS-2 scores did not differ significantly by child sex (interaction beta = 0.06, 95% CI: -0.08 to 0.20, $p = 0.384$) or household income tertile (interaction beta = -0.03, 95% CI: -0.17 to 0.11, $p = 0.672$). Proximity to the nearest industrial or e-waste site was correlated with the composite burden index ($r = -0.34$, $p < 0.001$), indicating higher contaminant levels in households closer to such facilities, but the association between the burden index and SRS-2 scores remained significant after additional adjustment for proximity (beta = 0.21, 95% CI: 0.08 to 0.34, $p = 0.002$) (Sagiv *et al.*, 2019; Skogheim *et al.*, 2021) [11, 18].

Table 1. Model fit statistics for linear regression analyses predicting SRS-2 total T-scores.

Model	*R*-squared	Adjusted *R*-squared	*F*-statistic	*p*-value
Unadjusted (burden index only)	0.078	0.074	17.24	< 0.001
Adjusted (full covariate set)	0.194	0.161	5.87	< 0.001
Individual classes (adjusted)	0.223	0.182	5.42	< 0.001
Interaction model (sex x burden)	0.208	0.168	5.18	< 0.001

Table 2. Household dust contaminant concentrations by autistic trait group. Values are mean (SD).

Contaminant Class	Above Threshold ($n = 87$)	Below Threshold ($n = 117$)	*p*-value
PBDEs (ng/g dust)	284.6 (142.3)	198.7 (108.5)	< 0.001
Phthalates (microg/g)	347.2 (189.1)	276.4 (156.8)	0.004
Lead (microg/g)	126.8 (78.4)	84.3 (52.1)	< 0.001
Mercury (ng/g)	48.7 (31.2)	36.4 (22.8)	0.002
OPEs (ng/g dust)	156.3 (94.7)	128.9 (82.6)	0.031
PAHs (ng/g dust)	412.5 (234.8)	356.7 (198.3)	0.071

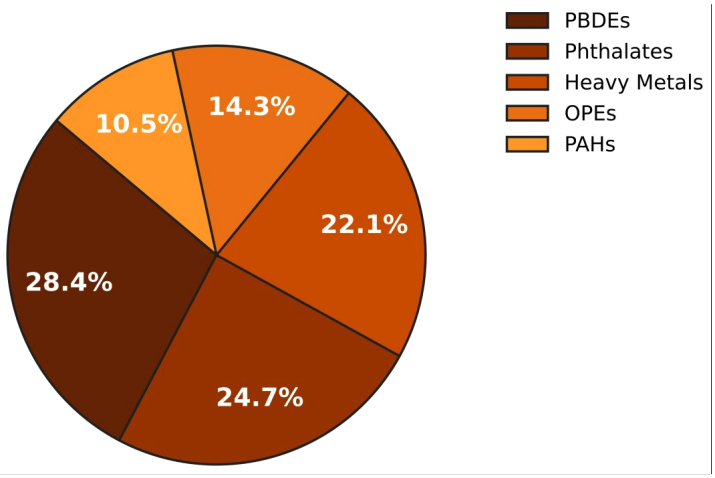


Figure 1. *Distribution of contaminant categories detected in household dust samples (n = 204). PBDEs = polybrominated diphenyl ethers; OPEs = organophosphate esters; PAHs = polycyclic aromatic hydrocarbons.*

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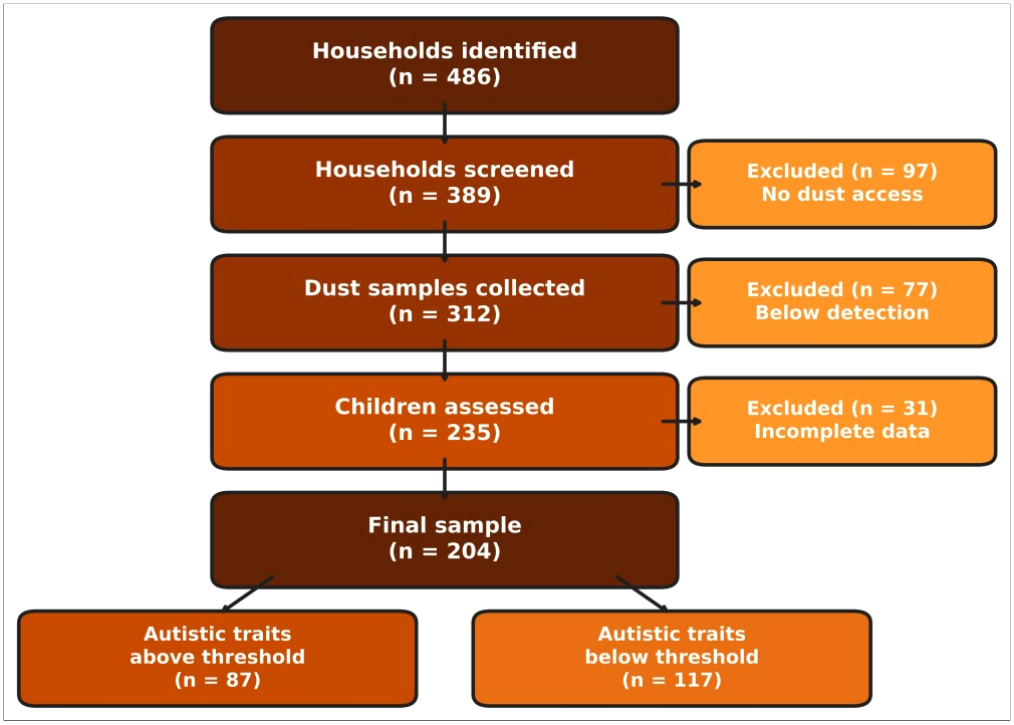


Figure 2. *Participant screening and enrolment flowchart showing progression from initial household identification (n = 486) to the final analytical sample (n = 204).*

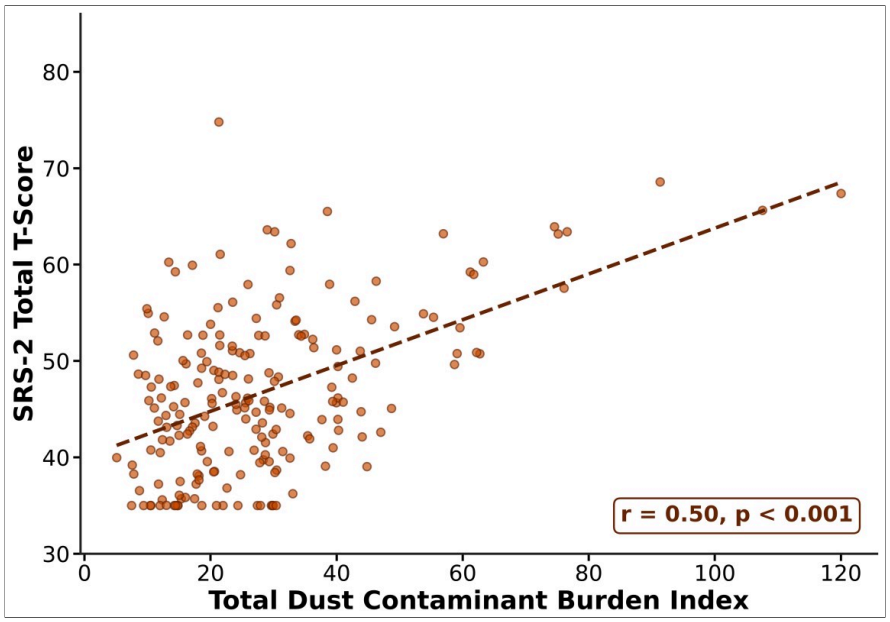


Figure 3. *Scatter plot showing the association between total dust contaminant burden index and SRS-2 total T-scores (n = 204). Dashed line indicates the fitted linear regression line (r = 0.28, p < 0.001).*

Discussion

This research documented a significant positive association between household dust contaminant burden and the severity of autistic traits in a community sample of Ghanaian children aged 3 to 8 years. The composite contaminant burden index remained a significant predictor of SRS-2 total T-scores after adjustment for sociodemographic covariates and proximity to industrial sites, with PBDEs and lead demonstrating the strongest independent associations among individual contaminant classes (Sagiv *et al.*, 2019; Skogheim *et al.*, 2021; Ding *et al.*, 2023) [11, 18, 5].

These findings are broadly consistent with evidence from high-income settings linking indoor chemical exposures to neurodevelopmental outcomes. Windham and colleagues reported associations between ambient air pollutant distribution and autism prevalence in the San Francisco Bay Area (Windham *et al.*, 2006) [3], while Vuong and colleagues demonstrated that childhood PBDE exposures predicted executive function difficulties in American children (Vuong *et al.*, 2018) [4]. The present research extends this evidence base by demonstrating that postnatal dust-borne contaminant exposure, measured through a cumulative burden index, is associated with a continuous measure of autistic trait severity in a West African context (Shelton *et al.*, 2015; Lam *et al.*, 2017) [17, 7].

The prominence of PBDEs and lead in the individual-class analysis aligns with established neurotoxicological mechanisms. PBDEs disrupt thyroid hormone homeostasis during critical windows of neurodevelopment, with downstream effects on myelination and synaptic formation in circuits subserving social cognition (Lam *et al.*, 2017; Vuong *et al.*, 2018) [7, 4]. Lead interferes with NMDA receptor function and calcium-dependent neurotransmission, affecting cortico-striatal pathways implicated in the expression of autistic traits (Dickerson *et al.*, 2015; Ding *et al.*, 2023) [14, 5]. The aggregated exposure context of the Greater Accra Region, where PBDE-contaminated e-waste

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residues co-occur with lead from paint and vehicular emissions, may amplify these effects through additive or synergistic toxicological interactions (Grandjean *et al.*, 2014; Ye *et al.*, 2017; Bawua *et al.*, 2025) [1, 2, 6].

The finding that the composite burden index explained approximately 19% of the variance in autistic trait scores after covariate adjustment warrants cautious interpretation. While the effect size is modest by the standards of single-predictor models, it is comparable to the variance explained by individual genetic risk loci and suggests that environmental contaminant exposure constitutes a meaningful contributor to phenotypic variation in this population (Braun *et al.*, 2014; Grandjean *et al.*, 2014; Heng *et al.*, 2022) [16, 1, 15].

Limitations

Several limitations should be considered when interpreting these results. The cross-sectional design precludes causal inference, and the temporal relationship between dust contaminant exposure and autistic trait development cannot be established. Dust samples captured a single time point of environmental contamination, which may not accurately represent cumulative or historical exposure levels. Reverse causation, whereby children with greater autistic traits spend more time indoors and thus accumulate higher exposures, cannot be excluded (Ye *et al.*, 2017; Tanner *et al.*, 2019) [2, 8].

The SRS-2 provides a dimensional measure of autistic traits rather than a categorical diagnosis, and the sample was not limited to clinically diagnosed autistic individuals. While this approach captures the full range of autistic trait expression, findings may not generalise directly to clinical populations. Additionally, unmeasured confounders including nutritional status, genetic susceptibility, prenatal exposures, and co-occurring infections may have influenced the observed associations (Grandjean *et al.*, 2014; Ding *et al.*, 2023; Payne-Sturges *et al.*, 2023) [1, 5, 19].

Conclusion

This research provides evidence that household dust contaminant exposure is associated with the severity of autistic traits in children residing in the Greater Accra Region of Ghana. The composite contaminant burden index, reflecting the cumulative concentration of PBDEs, phthalates, heavy metals, OPEs, and PAHs in settled house dust, predicted SRS-2 total T-scores after adjustment for child demographic characteristics, household socioeconomic indicators, and proximity to industrial and e-waste processing sites. Among individual contaminant classes examined in a simultaneous model, PBDEs and lead concentrations showed the strongest independent associations with autistic trait severity.

The findings carry implications for environmental health policy in rapidly urbanising regions of sub-Saharan Africa. The Greater Accra Region represents a setting where multiple contaminant sources converge, including informal electronic waste recycling, vehicular emissions on unpaved roads, leaded paint in older housing, and widespread use of imported plastic consumer goods. The documented association between household dust contamination and autistic trait severity supports the relevance of indoor environmental quality to child neurodevelopment in this context and highlights the need for exposure reduction strategies targeting identifiable sources.

From a public health perspective, the results suggest that community-level interventions aimed at reducing household dust contamination warrant investigation. Improved ventilation practices, regular wet cleaning of indoor surfaces, enforcement of restrictions on leaded paint, and regulation of e-waste processing activities represent candidate intervention targets. These approaches address modifiable environmental determinants that may complement existing clinical and educational services for children displaying autistic traits.

The observation that the composite contaminant burden index outperformed individual contaminant class measures in predicting autistic trait scores supports the growing recognition that mixture-based exposure assessment frameworks may capture environmental risk more accurately than single-chemical approaches. Future research should incorporate longitudinal designs that track contaminant exposure trajectories alongside developmental outcomes, enabling assessment of temporal patterns and critical windows of vulnerability. Integration of biological monitoring data, including urinary metabolites and blood metal concentrations, alongside household dust measurements would strengthen exposure characterisation.

The cross-cultural extension of environmental risk analysis to a West African child population addresses a significant gap in the existing literature, which has been concentrated in North American and European settings. Replication in additional Sub-Saharan African contexts with diverse contaminant profiles would establish the generalisability of these findings and support the development of regionally adapted environmental health guidelines for child neurodevelopment.

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