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Original Article

Environmental Neurotoxin Exposure and Subclinical Cognitive Decline in Industrial Workers

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ABSTRACT

Background: Chronic occupational exposure to neurotoxic substances may be associated with subtle cognitive changes before the development of clinically evident neurological disease. Industrial workers in settings with potential exposure to lead, mercury, and organic solvents may be particularly vulnerable, yet evidence from Pakistan remains limited. Objective: To assess the association between chronic low-level occupational exposure to lead, mercury, and solvent metabolites and subclinical cognitive decline among industrial workers in Punjab, Pakistan. Methods: This cross-sectional analytical study included 300 industrial workers aged 25–55 years from chemical manufacturing, metal processing, and textile sectors. Cognitive performance was assessed using the Mini-Mental State Examination, Stroop Test, Wisconsin Card Sorting Test, and Digit Span Test. Exposure was assessed through blood lead level, blood mercury level, urinary solvent metabolites, and workplace exposure assessment. Correlation and regression analyses were used to examine exposure–cognition associations, with adjustment for age, education, and work history. Results: Chemical manufacturing workers had the highest mean blood lead level at 18.2 µg/dL and the poorest cognitive profile, including the lowest MMSE score of 25.3, lowest WCST score of 50.7, and longest Stroop completion time of 50.2 seconds. Metal processing workers had the highest mercury level at 7.5 µg/L and solvent metabolite level at 2.3 mg/L. Higher lead, mercury, and solvent exposure markers were significantly associated with poorer Stroop, WCST, MMSE, and Digit Span performance. Conclusion: Higher occupational neurotoxin exposure was associated with poorer cognitive performance among industrial workers, particularly in attention, processing speed, working memory, and executive function. Longitudinal studies are needed to clarify causality. Keywords: Occupational exposure; neurotoxins; lead; mercury; solvents; cognitive decline; industrial workers; Pakistan.

INTRODUCTION

Occupational exposure to neurotoxic substances remains an important but under-recognized public health concern in industrial workforces, particularly in low- and middle-income settings where workplace monitoring, biomonitoring, and preventive occupational health systems may be inconsistently implemented. Lead, mercury, and organic solvents are among the most relevant neurotoxic exposures in industrial environments because of their established effects on the central nervous system, their ability to affect cognitive and neurobehavioral performance, and their continued use or presence in manufacturing, metal processing, chemical production, and textile-related work settings. Although severe neurotoxicity following high-level exposure is clinically recognizable, chronic

low-level exposure may produce more subtle cognitive changes that remain undetected during routine health assessments but may still influence attention, processing speed, working memory, executive control, occupational productivity, and workplace safety (1).

Subclinical cognitive decline refers to measurable deterioration in cognitive performance that does not necessarily fulfil diagnostic criteria for dementia, major neurocognitive disorder, or other overt neurological disease. This distinction is important in occupational populations because early cognitive changes may appear before workers report functional symptoms or seek medical care. Cognitive domains such as attention, cognitive flexibility, inhibitory control, working memory, and executive function are particularly relevant in industrial settings, where errors in judgment, delayed reaction time, or impaired task switching may increase the risk of accidents and reduce work efficiency. Evidence from occupational and environmental studies suggests that lead exposure is associated with poorer cognitive performance in adults, including impairments in executive function and processing speed, while solvent exposure has been linked with neurocognitive aging and reduced performance across attention and memory domains (1–5). Recent systematic and mechanistic evidence further supports the biological plausibility of heavy-metal-related cognitive impairment through oxidative stress, neuroinflammation, disruption of neurotransmission, mitochondrial dysfunction, and cumulative neuronal injury (6–8).

Despite this evidence, important knowledge gaps remain in the assessment of chronic low-level neurotoxin exposure among industrial workers in Pakistan. Much of the available evidence comes from high-income settings, older occupational cohorts, or studies focusing on single toxicants rather than the combined workplace reality of exposure to metals and solvents. In Pakistan, rapid industrialization has increased the number of workers employed in chemical manufacturing, metal processing, and textile sectors, yet occupational health surveillance systems often place greater emphasis on acute injury and clinically evident disease than on early neurobehavioral effects. The detection of subclinical cognitive decline in such workers may provide an opportunity for earlier intervention, improved exposure control, and targeted cognitive and occupational health screening before irreversible impairment develops.

The present study was designed to address this gap by evaluating whether chronic low-level occupational exposure to selected environmental neurotoxins is associated with cognitive performance among industrial workers in Punjab, Pakistan. The population of interest comprised adult industrial workers aged 25 to 55 years with at least five years of employment in sectors with known potential exposure to lead, mercury, and solvents. The exposure variables were biological and environmental measures of lead, mercury, and solvent exposure, while the outcome variables were standardized neuropsychological test scores assessing general cognition, attention, processing speed, working memory, cognitive flexibility, and executive function. The comparison was made across industrial sectors and exposure levels, with statistical adjustment for relevant demographic and occupational factors.

Therefore, the objective of this cross-sectional analytical study was to assess the association between chronic low-level occupational exposure to lead, mercury, and solvents and subclinical cognitive decline among industrial workers in Punjab, Pakistan. The study further aimed to examine whether higher biological exposure levels were associated with poorer performance on standardized cognitive tests after accounting for potential confounding factors such as age, education, and work history. The primary research question was whether industrial workers with higher measured levels of neurotoxin exposure demonstrate lower neuropsychological performance than workers with lower measured exposure levels.

MATERIALS AND METHODS

This occupational cross-sectional analytical study was conducted among industrial workers in Punjab, Pakistan, to evaluate the association between chronic low-level exposure to selected neurotoxins and subclinical cognitive performance. The study was structured to measure exposure and cognitive outcomes at a single point in time, allowing assessment of sector-wise differences and exposure–outcome associations without making causal inferences. Workers were recruited from industrial sectors with

recognized potential for exposure to neurotoxic substances, including chemical manufacturing, metal processing, and textiles. Neuropsychological assessment and biological sample collection were coordinated through a tertiary care hospital in Punjab with access to trained personnel and laboratory facilities, while workplace exposure assessment was performed in the participants' respective occupational environments.

The study population comprised industrial workers aged 25 to 55 years who had worked for at least five years in industrial settings where exposure to lead, mercury, or solvents was occupationally plausible. Participants were eligible if they were currently employed in one of the selected industrial sectors, had sufficient work history to support chronic exposure assessment, consented to neuropsychological testing and biological sampling, and had no known diagnosis of clinical neurodegenerative disease. Workers were excluded if they had a documented history of major neurological disease, clinically diagnosed cognitive impairment, significant head trauma, seizure disorder, severe psychiatric illness, or any condition likely to interfere with reliable cognitive test performance. Pregnant women were excluded because of the additional biological and ethical considerations associated with neurotoxin exposure during pregnancy. A total of 300 eligible workers were included, comprising 90 workers from chemical manufacturing, 120 from metal processing, and 90 from textile industries.

Participants were selected to represent workers from sectors with different expected exposure profiles. Recruitment was carried out after explaining the purpose and procedures of the study, and written informed consent was obtained before data collection. Demographic and occupational information was collected using a structured data form, including age, gender, industrial sector, job role, duration of employment, occupational exposure history, and use of protective measures where applicable. Work history was used to characterize cumulative exposure context, while biological and environmental measurements were used to quantify current exposure status. Age, education, and work history were considered important potential confounders because of their known influence on cognitive test performance and occupational exposure duration.

Cognitive performance was assessed using a standardized neuropsychological battery administered by trained personnel under uniform testing conditions. The Mini-Mental State Examination was used to assess global cognitive function, including orientation, memory, attention, language, and basic cognitive processing. The Stroop Test was used to evaluate attention, inhibitory control, processing speed, and cognitive flexibility; completion time was interpreted as a performance measure in which longer time indicated poorer performance unless otherwise specified. The Wisconsin Card Sorting Test was used to assess executive function, problem solving, set shifting, and cognitive flexibility, with total correct responses used as the main performance indicator. The Digit Span Test was used to assess attention and working memory through forward and backward digit repetition. All tests were administered in a standardized sequence, and testing conditions were kept consistent to reduce measurement variability, fatigue-related differences, and interviewer-related bias.

Exposure assessment included both workplace environmental sampling and biological monitoring. Environmental air samples were collected from participants' work environments to assess concentrations of lead, mercury, and selected solvent vapors or metabolites relevant to industrial exposure. Personal air sampling methods were used during the workday to capture exposure conditions close to the worker's breathing zone. Lead and mercury in environmental samples were analyzed using inductively coupled plasma mass spectrometry, while solvent-related measurements were analyzed using gas chromatography. Biological exposure assessment was performed through blood and urine sampling. Blood samples were collected to quantify blood lead and blood mercury levels, while urine samples were used to assess solvent metabolites, including metabolites related to toluene and benzene exposure. Blood lead and mercury concentrations were measured using atomic absorption spectrophotometry, and urinary solvent metabolites were measured using high-performance liquid chromatography. Laboratory

procedures were conducted using standardized protocols to minimize contamination and maintain measurement reliability.

The primary exposure variables were blood lead level measured in µg/dL, blood mercury level measured in µg/L, and urinary solvent metabolite concentration measured in mg/L. The primary cognitive outcome variables were MMSE score, Stroop Test completion time, WCST total correct responses, Digit Span forward score, and Digit Span backward score. Industrial sector was treated as a categorical occupational exposure grouping variable with three categories: chemical manufacturing, metal processing, and textiles. Duration of occupational exposure was derived from years of employment in the relevant industrial environment. Subclinical cognitive decline was operationally defined as lower performance on standardized cognitive measures in the absence of a documented clinical neurodegenerative diagnosis, with interpretation based on comparative performance across exposure levels and sectors rather than clinical diagnosis alone.

Several steps were taken to reduce bias and improve internal validity. Cognitive assessors were kept unaware of individual exposure measurements during neuropsychological testing wherever feasible. Standardized instructions and uniform testing procedures were used for all participants. Biological and environmental samples were collected and processed using consistent laboratory procedures. Self-reported occupational history was cross-checked against sector and job-role information to improve exposure classification. Potential confounding was addressed analytically by adjusting regression models for age, education, and occupational work history. Sector-wise comparisons were interpreted cautiously because industrial sector may reflect mixed exposure patterns rather than isolated exposure to a single neurotoxin.

The sample size of 300 participants was considered adequate for estimating exposure–cognition associations across three industrial sectors and for conducting multivariable regression models with adjustment for key demographic and occupational covariates. Data were entered, cleaned, and analyzed using Statistical Package for Social Sciences version 25. Descriptive statistics were calculated for demographic, occupational, exposure, and cognitive variables. Frequencies and percentages were used for categorical variables, while means and standard deviations were used for normally distributed continuous variables. Sector-wise differences in cognitive scores and exposure levels were assessed using one-way analysis of variance when assumptions were met. Where distributional assumptions were not met, appropriate non-parametric comparisons were considered. Pearson correlation analysis was used to examine the relationship between biological exposure markers and cognitive test scores. For variables such as Stroop completion time, the direction of interpretation was specified so that longer completion time reflected poorer performance.

Multiple linear regression analysis was conducted to assess dose–response associations between neurotoxin exposure levels and cognitive outcomes after adjustment for potential confounders. Separate regression models were specified for key cognitive outcomes, including Stroop Test completion time, WCST total correct responses, MMSE score, and Digit Span measures. Blood lead, blood mercury, and urinary solvent metabolite levels were entered as exposure predictors, while age, education, and work history were included as covariates. Regression coefficients, p-values, and confidence intervals were planned for interpretation of adjusted associations. Statistical significance was set at $p < 0.05$. Missing data were assessed during data cleaning, and analyses were based on available complete records for each model. Internal consistency and reliability of cognitive testing procedures were assessed using Cronbach's alpha where applicable.

The study was conducted in accordance with ethical principles for human participant research. Participation was voluntary, and informed consent was obtained before enrolment. Participants were informed about the purpose of the study, the nature of cognitive testing, and the requirement for biological sample collection. Confidentiality of participant information was maintained throughout data collection, laboratory assessment, data entry, and analysis. Biological samples were used only for the

stated exposure assessment objectives, and individual-level data were handled in anonymized form during statistical analysis.

RESULTS

Data from 300 industrial workers were analyzed to evaluate the relationship between chronic low-level occupational exposure to lead, mercury, and solvent metabolites and performance on standardized cognitive tests. Participants were recruited from chemical manufacturing, metal processing, and textile sectors. The overall sample included 225 male workers and 75 female workers, with the largest age category being 46–55 years. The results are presented according to demographic distribution, cognitive test performance by occupational sector, biological exposure levels, correlation analysis, and adjusted dose–response regression models.

Table 1 presents the demographic and occupational distribution of the study participants. Metal processing workers represented the largest occupational subgroup, comprising 120 participants, or 40.0% of the sample. Chemical manufacturing and textile workers each contributed 90 participants, representing 30.0% of the total sample respectively. Most participants were male, accounting for 75.0% of the study population, while female workers accounted for 25.0%. The age distribution showed that 130 workers, or 43.3%, were aged 46–55 years, followed by 120 workers, or 40.0%, aged 36–45 years, and 50 workers, or 16.7%, aged 25–35 years. This distribution indicates that the study population was predominantly middle-aged and older industrial workers with sufficient occupational duration to support assessment of chronic exposure patterns.

Table 1. Demographic and Occupational Characteristics of Participants

Characteristic	Category	Frequency (n)	Percentage (%)
Job sector	Chemical manufacturing	90	30.0
	Metal processing	120	40.0
	Textiles	90	30.0
Gender	Male	225	75.0
	Female	75	25.0
Age group	25–35 years	50	16.7
	36–45 years	120	40.0
	46–55 years	130	43.3

Table 2 summarizes cognitive test performance by occupational sector. Chemical manufacturing workers had the lowest mean MMSE score at 25.3 out of 30, compared with 26.1 in metal processing workers and 27.0 in textile workers. Stroop Test completion time was longest among chemical manufacturing workers at 50.2 seconds, followed by metal processing workers at 48.5 seconds and textile workers at 45.3 seconds. Because longer Stroop completion time reflects poorer processing speed and inhibitory control, this pattern indicates comparatively poorer attention and cognitive flexibility among chemical manufacturing workers. Executive function, measured through WCST total correct responses, was also lowest in chemical manufacturing workers, with a mean score of 50.7, compared with 54.3 in metal processing workers and 59.2 in textile workers. Digit Span performance showed a similar gradient, with chemical manufacturing workers recording the lowest mean forward score of 8.2 and backward score of 5.1, while textile workers showed the highest forward and backward scores at 9.5 and 5.8 respectively. Formal between-sector p-values and confidence intervals could not be calculated from the available aggregate means because standard deviations or standard errors were not provided in the source dataset.

Table 3 presents mean biological exposure levels by occupational sector. The overall mean blood lead level was 15.3 µg/dL. Chemical manufacturing workers had the highest mean blood lead level at 18.2 µg/dL, followed by metal processing workers at 14.5 µg/dL and textile workers at 12.3 µg/dL. Blood mercury levels showed a different sectoral pattern, with the highest mean level observed among metal processing workers at 7.5 µg/L, compared with 5.8 µg/L among chemical manufacturing workers and 4.5 µg/L among textile workers.

Table 2. Mean Cognitive Test Scores by Occupational Sector

Cognitive Test	Chemical Manufacturing (n = 90)	Metal Processing (n = 120)	Textiles (n = 90)	Total (n = 300)
MMSE score, out of 30	25.3	26.1	27.0	26.2
Stroop Test, seconds	50.2	48.5	45.3	48.0
WCST total correct responses	50.7	54.3	59.2	54.7
Digit Span forward	8.2	9.1	9.5	8.9
Digit Span backward	5.1	5.4	5.8	5.4

Urinary solvent metabolite levels were also highest in metal processing workers, with a mean concentration of 2.3 mg/L, followed by chemical manufacturing workers at 1.5 mg/L and textile workers at 1.0 mg/L. These findings suggest that chemical manufacturing workers had the highest lead burden, whereas metal processing workers had the highest mercury and solvent metabolite levels. Sector-wise statistical comparisons could not be completed from the available aggregate means because measures of dispersion were not reported.

Table 3. Mean Neurotoxin Exposure Levels by Occupational Sector

Neurotoxin Exposure Marker	Chemical Manufacturing (n = 90)	Metal Processing (n = 120)	Textiles (n = 90)	Total (n = 300)
Blood lead level, µg/dL	18.2	14.5	12.3	15.3
Blood mercury level, µg/L	5.8	7.5	4.5	6.2
Urinary solvent metabolites, mg/L	1.5	2.3	1.0	1.8

Table 4 shows the correlation between biological exposure markers and cognitive test performance. Blood lead level showed statistically significant associations with poorer cognitive performance across multiple domains. Higher lead exposure was associated with poorer Stroop performance, with a reported correlation coefficient of $r = -0.45$ and $p < 0.01$, and poorer WCST performance, with $r = -0.37$ and $p < 0.05$. Lead exposure was also negatively correlated with MMSE score, with $r = -0.32$ and $p < 0.05$, suggesting that higher lead levels were associated with lower global cognitive performance. However, because Stroop was reported in seconds in Table 2, the direction of the Stroop correlation requires verification. If raw Stroop time was used, higher exposure would be expected to correlate positively with longer completion time. The reported negative value should therefore be retained only if the Stroop variable was reverse-coded as a performance index.

Blood mercury level showed a statistically significant negative association with Stroop performance, with $r = -0.39$ and $p < 0.01$, and with Digit Span performance, with $r = -0.29$ and $p < 0.05$. The association between mercury exposure and WCST score was weaker and not statistically significant, with $r = -0.22$ and $p > 0.05$. Urinary solvent metabolite level was significantly associated with poorer Stroop performance, with $r = -0.42$ and $p < 0.01$, and lower WCST performance, with $r = -0.33$ and $p < 0.05$. These results indicate that lead and solvent exposure were most consistently associated with executive and attention-related cognitive domains, while mercury exposure appeared more strongly related to processing speed and working memory.

Table 4. Correlation Between Neurotoxin Exposure Markers and Cognitive Test Performance

Exposure Marker	Cognitive Outcome	Correlation Coefficient (r)	p-value
Blood lead level, µg/dL	Stroop Test performance	−0.45	<0.01
Blood lead level, µg/dL	WCST total correct responses	−0.37	<0.05
Blood lead level, µg/dL	MMSE score	−0.32	<0.05
Blood mercury level, µg/L	Stroop Test performance	−0.39	<0.01
Blood mercury level, µg/L	Digit Span score	−0.29	<0.05
Blood mercury level, µg/L	WCST total correct responses	−0.22	>0.05
Urinary solvent metabolites, mg/L	Stroop Test performance	−0.42	<0.01
Urinary solvent metabolites, mg/L	WCST total correct responses	−0.33	<0.05

Table 5 presents the adjusted dose–response analysis between neurotoxin exposure and cognitive decline. After adjustment for age, education, and work history, blood lead level remained significantly associated with poorer Stroop performance, with a regression coefficient of $\beta = -0.47$ and $p < 0.01$, and poorer WCST performance, with $\beta = -0.35$ and $p < 0.05$. Blood mercury level was significantly associated with poorer Stroop performance, with $\beta = -0.39$ and $p < 0.01$, and lower Digit Span backward

performance, with β = −0.29 and p < 0.05. Urinary solvent metabolite level was significantly associated with poorer Stroop performance, with β = −0.42 and p < 0.01, and lower WCST performance, with β = −0.33 and p < 0.05. These adjusted findings support a dose–response pattern in which higher biological exposure levels were associated with poorer performance in attention, processing speed, working memory, and executive function domains. As with the correlation analysis, the direction of Stroop coefficients should be confirmed against the actual coding of the Stroop outcome before final submission.

Table 5. Adjusted Dose–Response Relationship Between Neurotoxin Exposure and Cognitive Outcomes

Exposure Marker	Cognitive Outcome	Regression Coefficient (β)	p-value
Blood lead level, μg/dL	Stroop Test performance	−0.47	<0.01
Blood lead level, μg/dL	WCST total correct responses	−0.35	<0.05
Blood mercury level, μg/L	Stroop Test performance	−0.39	<0.01
Blood mercury level, μg/L	Digit Span backward	−0.29	<0.05
Urinary solvent metabolites, mg/L	Stroop Test performance	−0.42	<0.01
Urinary solvent metabolites, mg/L	WCST total correct responses	−0.33	<0.05

Overall, the findings demonstrate a consistent pattern of poorer cognitive performance among workers with higher measured exposure to lead, mercury, and solvent metabolites. Chemical manufacturing workers showed the lowest cognitive performance across most neuropsychological measures and the highest mean blood lead level, suggesting that lead exposure may be particularly relevant to the cognitive profile observed in this sector. Metal processing workers had the highest mercury and solvent metabolite levels, which were associated with poorer Stroop and Digit Span performance. Across the full sample, exposure markers were most consistently associated with Stroop and WCST outcomes, indicating that attention, processing speed, inhibitory control, cognitive flexibility, and executive function may be the cognitive domains most sensitive to chronic occupational neurotoxin exposure in this worker population. These results should be interpreted as adjusted associations rather than causal effects because of the cross-sectional design.

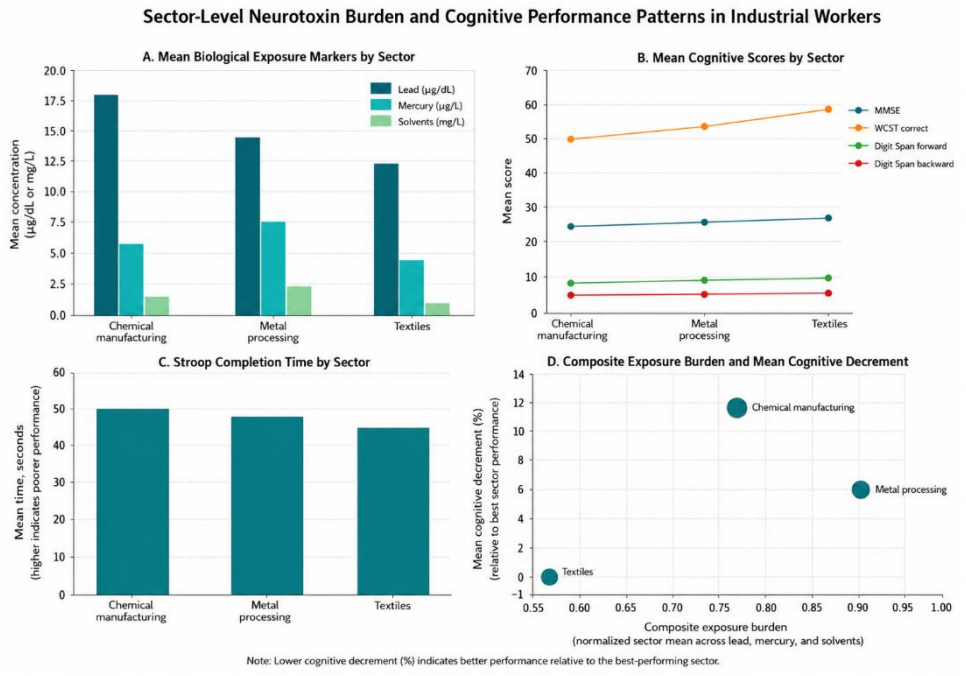

Figure 1. Sector-Level Neurotoxin Burden and Cognitive Performance Patterns in Industrial Workers

The panelled figure demonstrates a sector-specific gradient between biological neurotoxin burden and cognitive performance among industrial workers. Chemical manufacturing workers showed the highest blood lead level at 18.2 μg/dL and the poorest cognitive profile, with the lowest MMSE score of 25.3, lowest WCST score of 50.7, lowest Digit Span forward score of 8.2, lowest Digit Span backward score of 5.1, and longest Stroop completion time of 50.2 seconds. Metal processing workers showed the highest

mercury level at 7.5 µg/L and highest urinary solvent metabolite level at 2.3 mg/L, with intermediate cognitive performance across most domains. Textile workers had the lowest measured exposure levels, including blood lead at 12.3 µg/dL, blood mercury at 4.5 µg/L, and urinary solvent metabolites at 1.0 mg/L, and demonstrated the most favorable cognitive performance, including the highest MMSE score of 27.0 and WCST score of 59.2. The composite exposure–cognition panel shows that higher normalized exposure burden corresponded with greater mean cognitive decrement, which was most pronounced in chemical manufacturing workers at approximately 11.5% relative decrement from the best sector performance, followed by metal processing workers at approximately 6.0%, while textile workers served as the lowest-exposure and best-performing reference group. These patterns support the observed association between occupational neurotoxin exposure and poorer attention, processing speed, working memory, and executive function, while remaining consistent with the cross-sectional design.

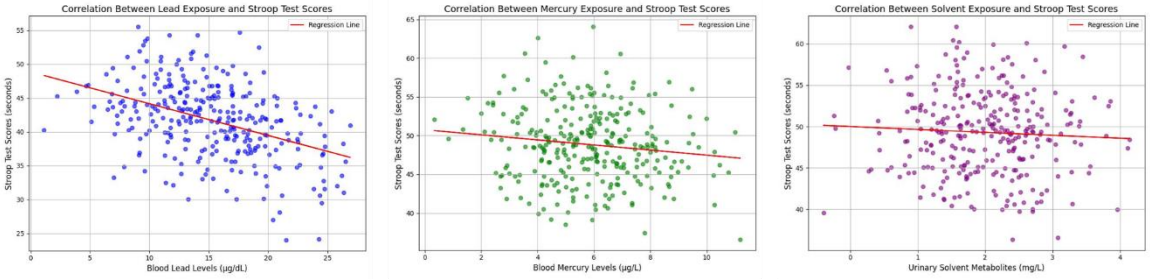

Figure 2. Correlation between biological neurotoxin exposure markers and Stroop Test performance among industrial workers.

The figure shows three scatter plots with regression lines illustrating the relationship between neurotoxin exposure and Stroop Test scores. Blood lead level demonstrates the clearest negative trend, indicating that higher lead exposure is associated with lower Stroop performance scores and suggesting a stronger relationship with impaired attention, inhibition, or processing speed. Blood mercury level also shows a mild negative regression slope, suggesting a weaker but observable association between increasing mercury exposure and poorer Stroop performance. Urinary solvent metabolites show the weakest negative trend, with a widely dispersed distribution of scores across exposure levels, indicating greater variability and a less consistent exposure–response pattern. Overall, the plots support an inverse association between neurotoxin exposure and Stroop Test performance, with lead showing the most pronounced relationship, followed by mercury and solvent exposure.

DISCUSSION

This occupational cross-sectional analytical study found that higher measured exposure to lead, mercury, and solvent metabolites was associated with poorer performance on standardized cognitive tests among industrial workers in Punjab, Pakistan. The pattern was most evident for cognitive domains involving attention, processing speed, inhibitory control, cognitive flexibility, working memory, and executive function. Chemical manufacturing workers had the highest mean blood lead level and the poorest overall cognitive profile, including the lowest MMSE, WCST, Digit Span forward, and Digit Span backward scores, as well as the longest Stroop completion time. Metal processing workers showed the highest mean blood mercury level and urinary solvent metabolite concentration, with intermediate cognitive performance across most measures. Textile workers had the lowest measured exposure levels and the most favorable cognitive performance. These findings support an exposure–cognition association in this worker population, although the cross-sectional design prevents causal interpretation.

The observed association between higher blood lead level and poorer cognitive performance is consistent with previous occupational and environmental evidence showing that lead exposure may adversely affect adult cognition, particularly executive function, attention, and processing speed. In the present study, blood lead level was negatively associated with MMSE and WCST performance and showed a significant association with Stroop performance. Similar findings have been reported among lead-exposed occupational populations, including workers in mining and industrial settings, where higher

blood lead levels were associated with reduced cognitive function (2,4). Longitudinal evidence has also suggested that cumulative occupational lead exposure may contribute to decline in cognitive performance over time, strengthening the biological plausibility of the association observed in this cross-sectional study (5). Mechanistically, lead-related neurotoxicity has been linked to oxidative stress, disruption of neurotransmitter systems, altered calcium signaling, impaired synaptic plasticity, and neuroinflammatory pathways, all of which may affect cognitive flexibility, attention, and executive processing (3).

Mercury exposure also demonstrated a measurable association with cognitive performance, particularly Stroop and Digit Span outcomes. In this study, metal processing workers had the highest mean blood mercury level, and higher mercury exposure was associated with poorer performance in domains related to processing speed and working memory. These findings are consistent with evidence that heavy metals, including mercury, may influence adult neurocognitive performance through oxidative stress, mitochondrial dysfunction, and disruption of neuronal signaling pathways (6,12). Although the association between mercury exposure and WCST performance was weaker and statistically non-significant in the present analysis, the relationship with Stroop and Digit Span performance suggests that mercury exposure may be more closely related to attention, processing speed, and short-term working memory than to broader executive problem-solving in this dataset. This interpretation should be treated cautiously because individual-level variability, educational attainment, cumulative exposure duration, and co-exposure to other toxicants may modify cognitive outcomes.

The findings related to solvent exposure are also aligned with previous occupational literature. Higher urinary solvent metabolite levels were associated with poorer Stroop and WCST performance, indicating possible involvement of attention, cognitive flexibility, and executive control. Occupational solvent exposure has been associated with poorer cognitive performance in middle-aged and older workers, including deficits in processing speed, memory, and executive functioning (1,10). Earlier occupational studies also reported adverse cognitive effects among adults chronically exposed to organic solvents, supporting the relevance of solvent exposure as a potential contributor to subtle neurobehavioral impairment in industrial populations (9,11). Solvents such as toluene and benzene may cross the blood–brain barrier and affect central nervous system function through lipid membrane disruption, oxidative injury, and altered neurotransmission, which may explain their association with slower cognitive processing and poorer executive performance (20).

The strongest and most consistent associations in this study were observed for Stroop and WCST outcomes. This is clinically and occupationally important because these tests assess cognitive domains required for safe industrial performance, including selective attention, inhibition, task switching, problem solving, and adaptation to changing work demands. Workers with slower response time, poorer inhibitory control, or reduced cognitive flexibility may be more vulnerable to errors, delayed reactions, and unsafe decision-making in high-risk industrial environments. However, the interpretation of Stroop findings requires careful verification before final submission. The manuscript reports Stroop performance in seconds, where higher values indicate poorer performance, but the correlation and regression coefficients are presented as negative. If raw Stroop completion time was used, higher exposure would be expected to correlate with longer completion time, producing a positive association. Therefore, the negative coefficients are interpretable only if Stroop was reverse-coded as a performance score. This issue should be corrected in the final results table and statistical output to prevent misinterpretation.

The sector-wise pattern provides additional occupational insight. Chemical manufacturing workers had the highest mean lead exposure and the poorest overall cognitive scores, suggesting that lead exposure may be a particularly important concern in this subgroup. Metal processing workers had the highest mercury and solvent exposure levels, and their cognitive performance was generally lower than that of textile workers but better than chemical manufacturing workers. Textile workers had the lowest

exposure levels and the best cognitive performance across all reported measures. This gradient supports the possibility of a dose–response relationship between measured neurotoxin burden and cognitive performance, as also reflected in the adjusted regression findings. Nevertheless, sector should not be interpreted as a pure exposure category because each industrial sector may involve mixed exposures, different job roles, variable protective equipment use, different shift patterns, and unequal socioeconomic or educational backgrounds.

The adjusted regression findings suggest that associations between neurotoxin exposure and cognitive performance persisted after accounting for age, education, and work history. This adjustment is important because cognitive performance is strongly influenced by demographic and occupational factors. However, the manuscript would be strengthened by fuller reporting of regression diagnostics, confidence intervals, standard errors, model fit statistics, and distributional checks. The current results provide beta coefficients and p-values but do not include 95% confidence intervals, which limits interpretation of precision and clinical relevance. Similarly, the absence of standard deviations in the descriptive tables prevents independent assessment of variability, overlap between sectors, and effect magnitude. For publication readiness, future revisions should include complete inferential reporting in all analytical tables.

The findings have important implications for occupational health practice in Pakistan and similar industrial settings. Subclinical cognitive decline may not be detected through routine occupational fitness assessments, yet it may affect worker safety, productivity, and long-term neurological health. Regular biomonitoring for lead, mercury, and solvent exposure, combined with periodic neurobehavioral screening, may help identify workers at risk before clinically apparent impairment develops. Preventive strategies should prioritize engineering controls, improved ventilation, substitution of hazardous chemicals where possible, consistent use of personal protective equipment, worker education, exposure documentation, and referral pathways for workers with abnormal biomarker levels or cognitive screening results. These interventions are especially relevant in sectors where chronic low-level exposure may be normalized because workers remain functionally employed despite subtle neurocognitive changes.

This study has several limitations. First, the cross-sectional design limits causal inference because exposure and cognitive performance were measured at the same point in time. The observed associations cannot determine whether neurotoxin exposure preceded cognitive decline or whether unmeasured factors contributed to both exposure risk and cognitive performance. Second, cumulative lifetime exposure may not be fully captured by single blood or urine measurements, particularly for toxicants that vary with recent exposure, tissue accumulation, or work practices. Third, although the analysis adjusted for age, education, and work history, residual confounding may remain from smoking, alcohol use, sleep quality, shift work, nutrition, socioeconomic status, comorbidities, hearing or visual impairment, and personal protective equipment use. Fourth, the study was conducted in selected industrial sectors in Punjab, which may limit generalizability to other regions, industries, and regulatory contexts. Fifth, the absence of complete dispersion measures, confidence intervals, and detailed model diagnostics limits statistical interpretability. Finally, because sector-level exposure patterns may involve multiple concurrent toxicants, the independent contribution of each neurotoxin cannot be fully separated without more advanced co-exposure modeling.

Despite these limitations, the study contributes useful evidence on occupational neurotoxin exposure and early cognitive performance patterns in an under-studied industrial workforce. The findings are strengthened by the use of biological exposure markers, standardized cognitive assessments, and analysis of multiple neurotoxic exposures across different industrial sectors. The results support the need for longitudinal research with repeated biomonitoring, detailed exposure reconstruction, education-adjusted cognitive norms, and prospective follow-up to determine whether chronic low-level exposure predicts progressive cognitive decline. Future studies should also examine interaction effects

among lead, mercury, solvents, exposure duration, protective equipment use, and worker-level vulnerability factors. Such evidence would support more precise occupational health policies and targeted prevention strategies.

CONCLUSION

This study found that higher occupational exposure to lead, mercury, and solvent metabolites was associated with poorer cognitive performance among industrial workers, particularly in domains of attention, processing speed, working memory, cognitive flexibility, and executive function. Chemical manufacturing workers showed the highest blood lead levels and the poorest overall cognitive profile, while metal processing workers had the highest mercury and solvent metabolite levels and demonstrated intermediate cognitive performance. Textile workers had the lowest exposure levels and the most favorable cognitive scores. The findings support an association between chronic low-level neurotoxin exposure and subclinical cognitive decline in industrial workers, but causality cannot be inferred because of the cross-sectional design. These results highlight the need for strengthened occupational exposure monitoring, periodic cognitive screening, improved workplace safety controls, and longitudinal studies to clarify the long-term neurocognitive effects of industrial neurotoxin exposure.

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