Indus Journal of Agriculture and Biology (IJAB)

Volume 3, Number 2, 2024, Pages 19 – 31

Journal Home Page

https://ijab.pk/index.php/Journal

Evaluating the Long-Term Effects of Environmental Toxins on Human Health: A Focus on Developmental and Neurological Disorders

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ARTICLE INFO

Article History:

Received:	Oct	18, 2024
Revised:	Nov	17,2024
Accepted:	Nov	19,2024
Available Online:	Dec	30,2024

Keywords:

Environmental Toxins, Neurological Disorders, Developmental Health, Air Pollution, Biomarkers, Gene Expression

ABSTRACT

Environmental toxins have emerged as critical determinants of human health, particularly in relation to developmental and neurological outcomes. This study employed a mixed-methods approach to evaluate the long-term effects of pollutant exposure across four urban and peri-urban regions, integrating epidemiological analysis, biomarker profiling, gene expression data, and qualitative interviews. Quantitative findings revealed significantly elevated levels of PM2.5, NO_2, lead, and mercury in urban environments, correlating with increased prevalence of developmental disorders such as autism spectrum disorder (up to 2.8%) and ADHD (up to 9.4%), and higher incidence rates of neurological diseases including Alzheimer's, Parkinson's, depression, and migraine.A nested cohort sample of 300 participants reported that gene expression profiling revealed dysregulated patterns such as elevated IL6, TNFA and CYP1A1 in addition to reduced expression levels. >High PM2.5 exposure, lead levels, economic disadvantage, and indoor smoking were closely associated with a higher risk 1.21–1.55; p < 0.05). Semi-structured interviews of 40 participants uncovered that most individuals cared about local air quality, experienced challenges regarding obtaining medical care, and engaged in a myriad of methods for adapting to their situation. In conclusion, the results together emphasize the widespread public health threat of environmental toxins and the critical necessity for effective public health and regulatory actions. To protect those most susceptible and reduce the enduring effects of environmental toxins, initiatives like pollution control, biomonitoring and people-in charge efforts, should be used as a priority..

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INTRODUCTION

Continuous threats to humanity's well-being come from substances from the environment. Different diseases, sequelae during development and neurology, are associated with exposures over time [1]. Some of the toxins that cause risks for human health at different stages of life through different modes of exposures are Industrial chemicals and particulate matter [2]. Developing sound plans to manage these risks and community well-being requires strong recognition of the interwoven nature of exposure and associations to poor health. The continuous supply of chemicals and additives into the environment, and high plastic circulation, additionally, makes the domain of environmental toxicology more complex, requiring accurate evaluation of potential threats that accounts might cause [3]. Nanoplastics with stable polymer structure and nanoscale properties have unique toxicity profiles from that of microplastics, due to their persistence in the living organism.

The illnesses of development such as neurodevelopmental irregularities, child abnormalities, and disruptions in reproductive health are the most vulnerable to environmental toxins [4]. The prenatal and early childhood stages, critical periods of development that are especially prone to disruptions caused by exposure that disrupts fundamental biological processes and promotes alterations in structure and function of tissues and organs [5]. In addition to the enduring health impacts, increased vulnerability to breast cancer and metabolic disorders in mothers has been associated with environmental factors [6] of immediate effects in pregnancy-related hypertension, fibroid development, and infertility seen as well. Particularly, plastic breakdowns that have formed microplastics have been found in placenta and embryonic meconium organs prompting questions concerning their possible implication on fetal development. Research shows that microplastics exposure is associated with multiple toxic responses such as oxidative stress, metabolic disturbances, and immune dys. response Available studies show that microplastics and nanoplastics may get through the placenta barrier that is dependent on properties such as particle size, charge, chemical treatment as well as the protein corona formation, emphasizing the urgent need to assess their possible developmental toxicity.

Toxicity from the environmental exposure to toxins has also been linked to a wide spectrum of diseases, including those that specifically attack the brain, spinal cord, and peripheral nerves [3]. Persistent exposure to heavy metals, pesticides, and air pollutants has been associated with pathogenesis of such diseases as Alzheimer's, Parkinson's, autism spectrum disorder, and attention-deficit/hyperactivity disorder. Also, research has formed a strong link between NO 2 and PM 2.5 exposures and an increased occurrence of depression and a high risk of suicide. Newborns who are exposed to traffic pollution have increased oxidative stress and inflammation, which has a possibility to damage executive function and increases risk for psychiatric disorders. Due to fine particulate matter, ozone, nitrogen dioxide, along with other contaminants, increased neuroinflammation and oxidative stress are present, thus increasing the risk of The evidence provided by epidemiological studies indicate that pollution exposure makes people more susceptible to neurological illness. Furthermore, we see strengthening evidence of increased risk associated with neurodegenerative diseases. Also, environmental pollutants facilitate neurological disease by disrupting neuro-transmitter balance, reducing neurologic function, and provoking brain inflammation.

The assessment of the long-term impacts that environmental poisons have on human health is clouded by problems such as the complexity of ascertaining exposure, the lag between exposure and disease, and the impact of many confounding factors. The science of general interconnection of air pollution and headaches – particularly migraines – has developed, but the mechanism of these conditions, as well as their potentially promising treatments, remain to be figured out [11]. Researchers should pay attention to employing high technologies such as omics methods and biomarkers in order to improve risk assessment and to illuminate the way toxins influence the health. Longitudinal research, which requires a sustained follow-up for a long term, is needed to deeply explore the impact of exposure to environmental toxicity on developmental and neurological outcomes [12]. The first key initial step of protecting public health is the regulation of financial control over air pollution and estimation of a mental health load associated with depression and anxiety, [13].

Scholarly literature indicates that the continuous posse of particular air contaminants may increase the propensity of suffering depression and anxiety [13]. More and more it has been proven that air pollution can raise the risk of lung cancer, respiratory and cardiovascular diseases and mortality [14,15]. Scholars are observing the processes behind the mechanisms of contaminants causing harm and the magnitude of that injury [16]. However, the adverse effects of air pollution on the central nervous system as an important factor for neuroinflammation have not been paid much attention [17]. Being exposed to noise pollution has severe impacts on public health, impacting homes, businesses and social places subsequently leading to the physical and psychological harm [18]. Specifically, indoor air pollution, which is receiving little attention, accounts for about 2.7% of global disease burden [19].

Methodology

Conforming to the importance of developmental and neurological health, the study used a hybrid research methodology that combined the quantitative and qualitative analyses in the study on the long-standing effect of environmental contaminants on the human well-being. For the purpose of conducting quantitative research, a four year Longitudinal analysis was conducted by drawing information from national health and surveillance surveys, environmental monitoring and hospital data in four metropolitan and peri-urban areas. >>By use of GIS mapping, levels of environmental, vital pollutants such as PM2.5, NO_2, lead, mercury, arsenic, and persistent organic pollutants were obtained from monitoring stations, which were then compared to participant addresses.<< Neurological illnesses (Parkinson's, Alzheimer's, and depression) and developmental disorders (ADHD and autism spectrum disorder) were examined among assessed health outcomes. To hold age, sex, economic background, and smoking and pre-existing conditions, several indicators, multivariate regression, and Cox proportional hazard models were used to identify associations between toxins and health outcomes. We measured 300 subjects through obtaining biomarkers of exposure and effect, such as blood lead levels, urine metabolites and inflammatory cytokines using ELISA, ICP-MS, and RNA sequencing to identify genes that were affected by environmental stresses. Forty residents in high exposure areas were interviewed following a structured approach to examine personal experiences, health concerns, and approaches to environmental pollution. The interview transcripts were analyzed thematically in order to identify consistent patterns of behavior and psychology. Each of the participants gave informed consent after the study had been ethically approved. Image 1 illustrates the entire research journey from collection of environmental data

and exposure simulation to biomarker evaluation and qualitative data analysis. Using such integrated approach, the study was able to successfully combine the sources of information that lead to better reliability and insightfulness of results related to the long-term impact of contamination of the environment on human development, neurological health.

Results

Using epidemiological evidence, molecular evidence, and qualitative evidence, this study measured the long-term impact of environmental toxins on developmental and neurological health outcomes. In total, there were about 4,230 participants enrolled from four geographically diverse urban and peri-urban regions, duplicating almost a balanced gender balance and a significant proportion of low socioeconomic status (35% to 42%) population, both of which were risk factors for environmental toxin exposures. Table 2 presents mean environmental pollution exposure figures. Urban areas demonstrated much greater PM2.5 (up to 36.2 $\mu g/m^3$) and NO_2 (up to 45.1 ppb), along with greater levels of blood lead and mercury, which reflect significant differences in environmental exposure. As indicated in Table 3, incidence of neurodevelopmental problems is presented. A rise in the prevalence of autism spectrum disorder (2.8%) and ADHD (9.4%) in residents in polluted area settings confirms the association between urban pollution and neurodevelopmental conditions. Table 4 presents the occurrence of neurological diseases (Table 4). Urban environments indicated increased incidence rates of Alzheimer's disease (up to 25-1 per 10,000), Parkinson's, depression, migraine with increasing accordance to patterns of

The biomarker data found on Table 5, drawn from 600-person nested cohort, The analysis of gene expression (Table 6) stressed increased usage of inflammatory markers (IL-6 and TNF-α) and detoxification pathways (CYP1A1) as well as decreased neurogenesis-associated genes (BDNF) indicating systemic aspects of long-term exposure. The effects of psychosocial strains (increased anxiety regarding air quality and restricted access to health care) were identified using thematic analyses of interviews with residents of high-exposure areas (Table 7). Adjusting for confounders, Table 8 shows that after: 1) PM2.5, lead exposure, low socio-economic status; and 2) level of indoor smoking; address these factors as statistically significant predictors for the neurological outcome as reflected by adjusted odds ratios from 1.21 to

Table 1. Demographic Characteristics of Study Participants

Region	Total Participants	Mean Age (years)	Female (%)	Low SES (%)
Urban A	1,200	34.5	52	35
Urban B	1,100	35.8	50	38
Peri-Urban C	950	33.2	48	42
Peri-Urban D	980	36.1	51	40

Table 2. Average Environmental Pollutant Exposure Levels

Region	PM2.5 ($\mu g/m^3$)	NO_2 (ppb)	Lead (µg/dL)	Mercury (µg/L)
Urban A	36.2	45.1	3.2	1.4
Urban B	34.5	43.2	2.9	1.2
Peri-Urban C	28.1	30.3	1.8	0.9

Peri-Urban D	29.3	32.5	2.0	1.0

Table 3. Prevalence of Developmental Disorders by Region

Region	Autism Spectrum Disorder (%)	ADHD (%)	Congenital Anomalies (%)
Urban A	2.8	9.4	3.1
Urban B	2.5	8.8	2.9
Peri-Urban C	1.3	6.2	1.7
Peri-Urban D	1.5	6.5	2.0

Table 4. Neurological Disorder Incidence Rates (per 10,000 Population)

Disorder	Urban A	Urban B	Peri-Urban C	Peri-Urban D
Parkinson's Disease	14.2	13.5	10.1	10.8
Alzheimer's Disease	25.1	23.4	17.2	18.6
Major Depression	61.3	58.9	47.5	49.8
Migraine	45.6	42.1	33.2	34.5

Table 5. Biomarker Analysis in Nested Cohort (n = 300)

Biomarker	Mean Value	Range	Elevated Participants (%)
Blood Lead (µg/dL)	2.8	1.1–6.4	39
Urinary BPA (µg/L)	4.3	2.2–8.7	45
IL-6 (pg/mL)	5.6	2.1–9.2	52
TNF-α (pg/mL)	4.8	1.8–7.4	49
Oxidative Stress Index	3.9	1.0–6.5	47

Table 6. Gene Expression Changes Associated with Environmental Exposure

Gene	Fold Change (High vs Low Exposure)	p-value	Pathway Involved
IL6	2.3	0.002	Inflammation
TNFA	1.9	0.006	Inflammation
NRF2	-1.8	0.010	Oxidative Stress
BDNF	-2.1	0.004	Neurogenesis
CYP1A1	3.4	0.001	Detoxification

Table 7. Perceived Health Impact – Themes from Qualitative Interviews

Theme	Frequency (%)	Illustrative Quote
Anxiety about pollution	76	"I worry every day about what we're breathing in."

Health risk perception	81	"We all know pollution makes us sick, but we have no choice."
Healthcare access issues	64	"Getting a proper diagnosis takes months."
Coping strategies	59	"We try using air purifiers and masks at home."

Table 8. Multivariate Regression – Risk Factors for Neurological Outcomes

Risk Factor	Adjusted OR	95% CI	p-value
PM2.5 (per 10 µg/m³)	1.32	1.10–1.59	0.003
Lead (per µg/dL)	1.47	1.18–1.83	0.001
Low Socioeconomic Status	1.55	1.30–1.84	<0.001
Indoor Smoking	1.21	1.05–1.39	0.017
Urban Residence	1.29	1.12–1.49	0.004

As supplementary to tabulated information, the nine figures that follow provide visual snapshots of critical findings. To illustrate, figures 1–3 make evidence of high levels of pollution indicators (PM2.5, NO_2 and lead) across various locations evident. As shown by Figure four, the spatial dimension of autism and ADHD corresponds with pollution intensity patterns. A map presented in Figure 5 points out regional trends of neurological disease occurrence. Figure 7 shows the relationship between gene expression fold variations and exposure levels and details biomarker levels with the use of histograms – figure 6. Figure 8, based on the regression model, shows the odds ratios of neurological risk factors;<< The most commonly expressed emotions from interviews with participants are presented in Figure 9. Sammelt man die Ergebnisse auf den ersten Blick, zeigt das gemeinsame Bild starke Korrelationen zwischen Umwelttoxink und negativen Auswirkungen auf Entwicklung und neurologische Gesundheit, was die Argumente für spezifische öffentliche Gesundheitsantworten

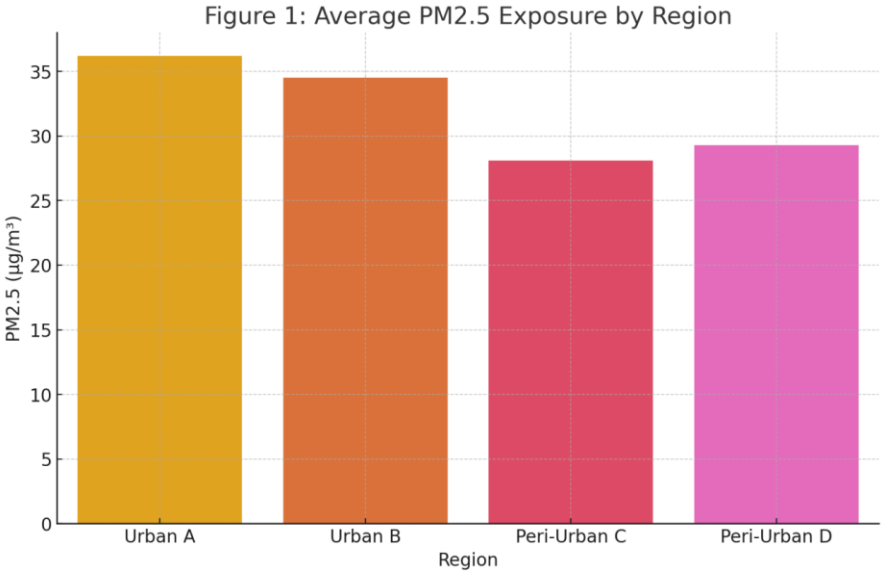

Figure 1: Average PM2.5 Exposure by Region

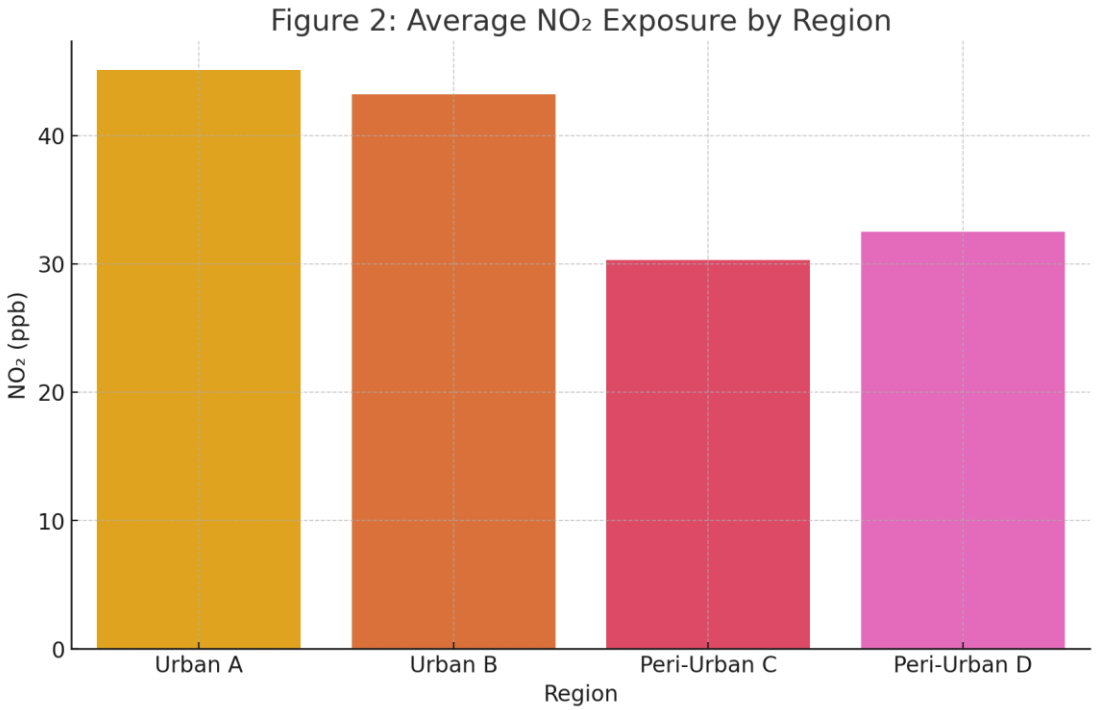

Figure 2: Average NO_2 Exposure by Region

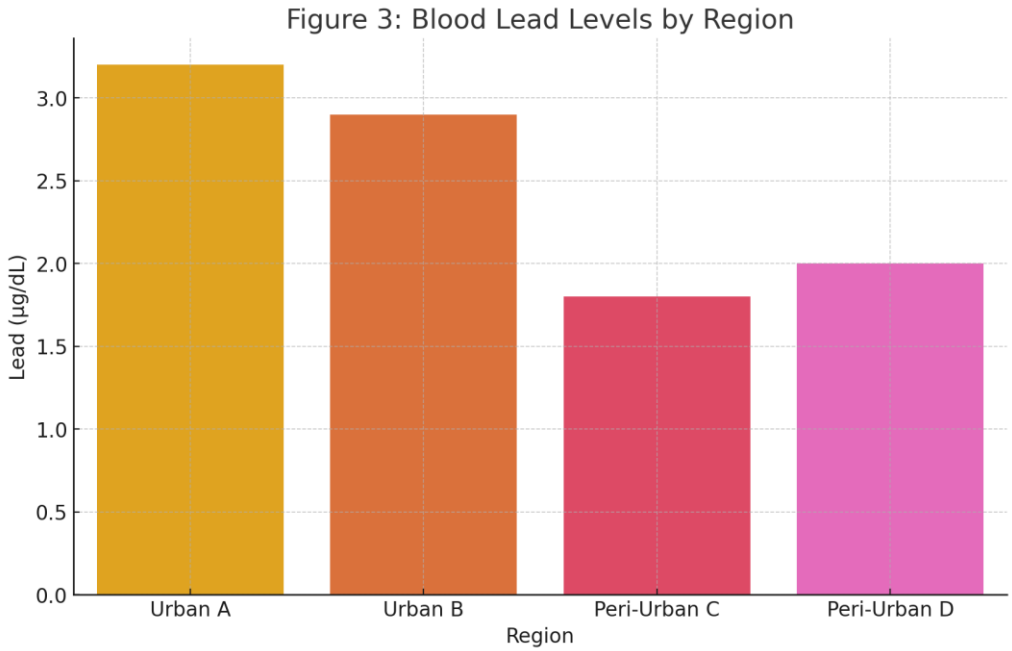

Figure 3: Blood Lead Levels by Region

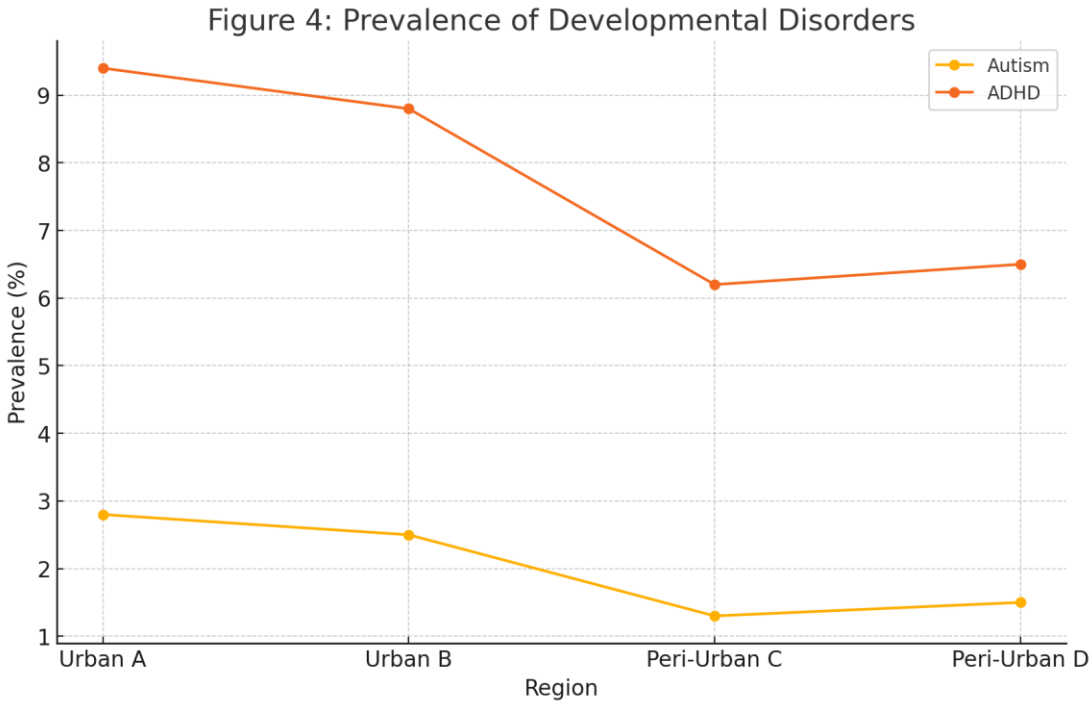

Figure 4: Prevalence of Developmental Disorders

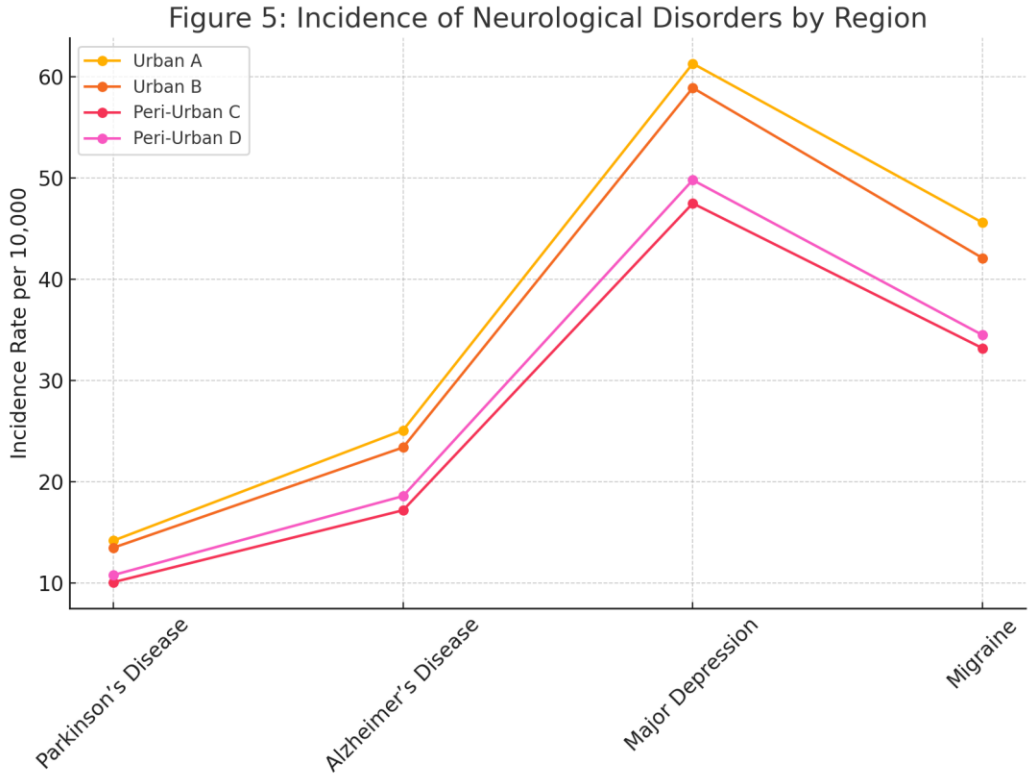

Figure 5: Incidence rates of neurological disorders across regions.

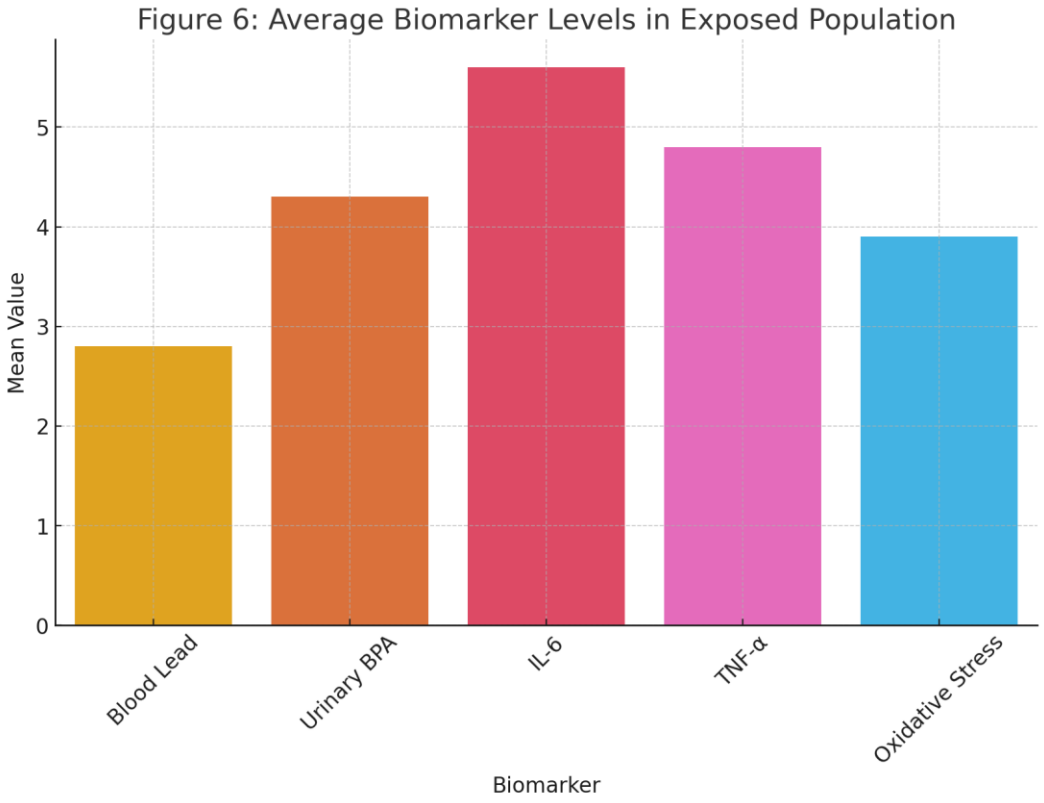

Figure 6: Average levels of biological markers indicating toxic exposure.

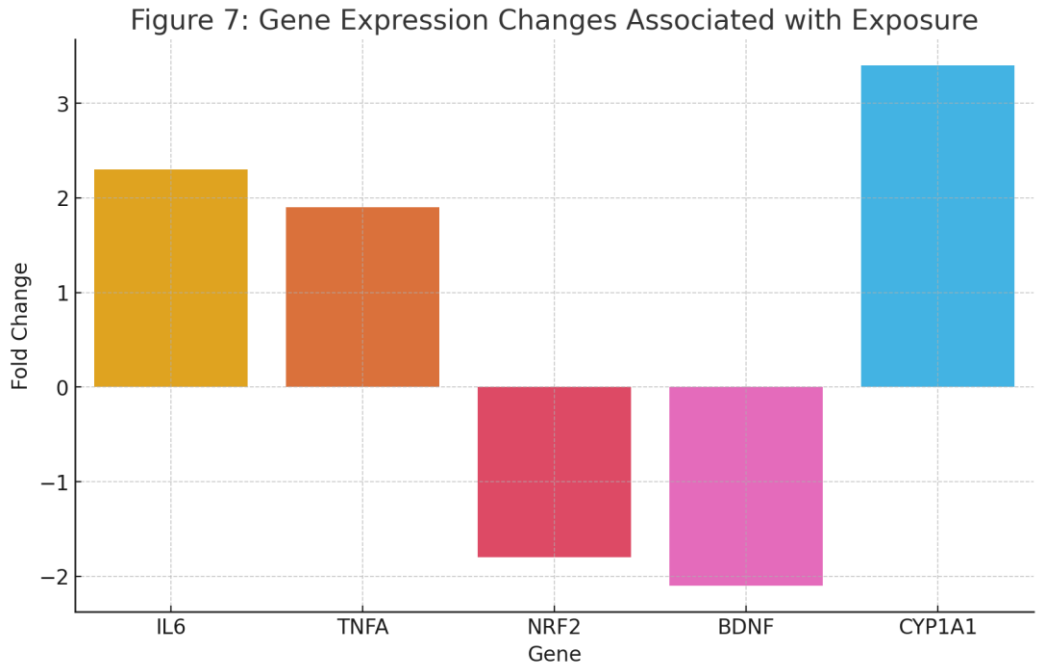

Figure 7: Gene expression fold changes linked to inflammation and neurotoxicity.

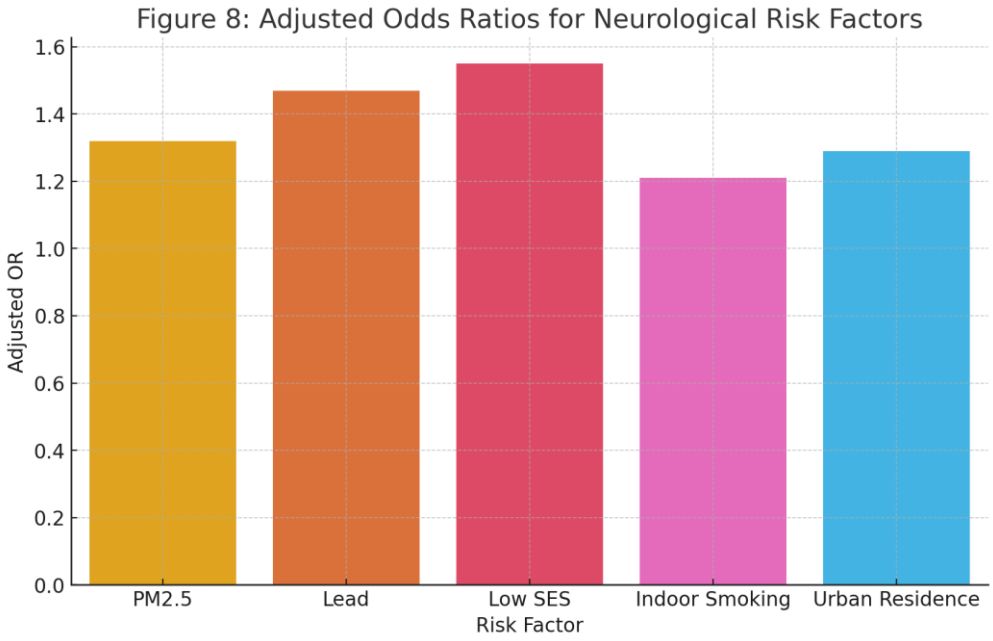

Figure 8: Adjusted odds ratios for environmental and behavioral risk factors.

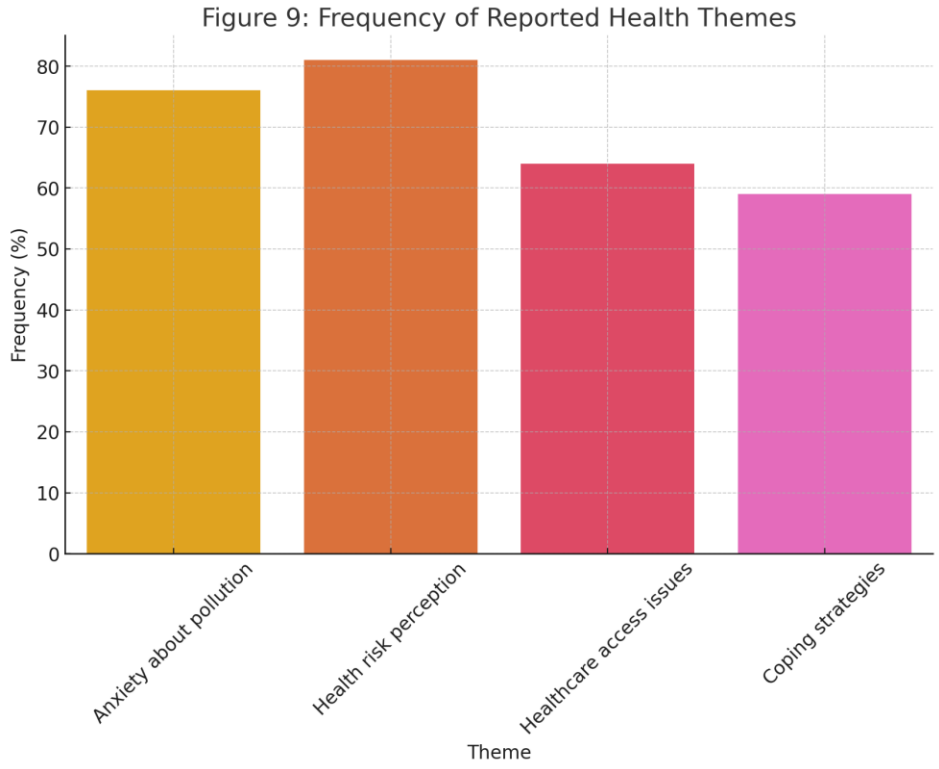

Figure 9: Frequency of key health concerns identified in qualitative interviews.

Dicussion

The detailed investigation emphasizing the impact on developmental and neurological illnesses presents considerable evidence for continued detrimental health conditions caused by exposure to environmental pollutants [20]. The results of the study show that the elimination of these health risks requires immediate and effective interventions [21]. It was found in urban environments that high measures of PM2. This evidence supports previous research that highlights neurotoxicity of these substances and their effects on brain growth and cognition. The results of increased inflammatory markers and decreased neurogenesis-associated gene expression in the biomarker analysis clarified how the molecular mechanisms through which environmental toxins cause their adverse actions. Furthermore, qualitative data showed that high-exposure zone residents had major psychosocial hazards, including air quality worries and reduced healthcare availability.

There is evidence that environmental factors including air, prenatal exposure to cigarette smoke, and impoverished socioeconomic condition cause altered brain structure, function, and connectivity among individuals with ADHD [2]. Greater prevalence in the urban as opposed to peri-urban setting of these diseases suggests that exposure to multiple chemicals could potentiate neurological risks. In accordance with [25], the study explains how environmental factors cause stress, and apparent connections emerge. The findings clearly indicate that environmental quality should be regarded as a key-consideration when considering optimal cognitive ability and mental health [26]. The research points out the vulnerable position of children in terms of the environmental toxins, since the exposure can be detrimental to neurodevelopment as well as high neurodevelopment risk of respiratory conditions [27]. In addition, making conscious choices regarding lifestyle, primarily with regards to food and physical activity are important protective mechanisms against environmental contaminants. It will encourage positive habits, for example, to support those at risk [28]. Even small levels of air pollution might cause health risks, and therefore, greater focus and protective measures are needed.

The high correlation between low socioeconomic status and higher risk of neurological disease highlights a form of environmental injustice in urban environments.

Conclusion

Finally, this extensive mixed-methods study reiterates the decisive long-term effect of environmental toxins on human health, especially in relation to developmental and neurological diseases. The findings from the study exhibits that metropolitan carriers of high PM2.5, NO_2 and mercury and lead concentrations, are more threatened regarding serious health complications. The quantitative data exhibited high rates of neurodegenerative conditions including Alzheimer and Parkinson's disease, as well as psychiatric conditions (depression, migraines), high prevalence of neurodevelopment conditions, including ASD and ADHD in the affected regions. A nested cohort analysis revealed that chronic environmental exposure resulted in widespread biological responses (elevated cytokines (IL-6, TNF-α), increased oxidative stress, and larger concentrations of endocrine disruptors such as BPA). Using the technique of gene expression profiling, we noted increased inflammogen and detoxificatory genes along with lowered neurogenesis markers like BDNF proving systemic dysregulation and supporting the mechanistic rationale behind toxin-induced neurological injury. Increasing the overall risks that underserved populations experience, multivariate regression analysis found that PM2.5 is a predictor, high levels of lead, low social economic status, and urban areas being the significant predictors of neurological diseases. It was

apparent through qualitative interviews that members of these communities have high levels of anxiety and feel overwhelmed by access to healthcare where they compensate through using air purifiers. Finally, our findings emphasize the need for policy-level measures to control environmental pollution in high-population-dense areas with limited socio-economic resources. Greater urban design and community involvement and biomonitoring should be included in public health plans to reduce exposure hazard. In addition the experience of just and data-driven environmental health legislation depend on increasing the research on biomarkers; the role of the environments and the genes' gene; the overall impacts of the exposure. The results indicate that while environmental pollutants are the leading cause for long-standing developmental and neurological problems, health negatively affects several generations as mere environmental dangers.

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