

Article

Chronic Atmospheric Dust Exposure As A Psychosocial Stressor: Neuropsychological and Behavioral Consequences in Arid-Zone Urban Populations of Central Asia

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Abstract: Atmospheric dust pollution is a major environmental problem in arid and semi-arid regions of Central Asia, where concentrations of particulate matter (PM_{2.5} and PM₁₀) frequently exceed the limits recommended by the World Health Organization. Although the respiratory and cardiovascular impacts of dust exposure are well documented, its neuropsychological effects remain insufficiently studied in Uzbekistan. This study reviews scientific literature published between 2000 and 2025 to examine the neurobiological mechanisms linking chronic dust exposure with psychological stress, anxiety, depression, and cognitive impairment. The findings indicate that long-term exposure to PM_{2.5} can trigger hypothalamic–pituitary–adrenal (HPA) axis dysregulation, increase inflammatory cytokines (IL-6, TNF- α , CRP), and induce neuroinflammation, which are associated with higher levels of anxiety, depressive symptoms, and reduced cognitive performance. Seasonal analysis also suggests that peak dust storm periods (April–July in southern Uzbekistan) coincide with increased psychological distress. The study concludes that chronic atmospheric dust exposure represents a significant psychosocial stressor and highlights the need for integrated interventions combining environmental management, phytoremediation buffers, and psychosocial support in arid-zone communities.

Keywords: Atmospheric dust, PM_{2.5}, psychological stress, neuroinflammation, HPA axis, anxiety, depression, cognitive impairment, Central Asia, arid zone, neurobiologia.

Citation: Tuxtayevna1, S. R. & Baxriddinzoda, X. H. Chronic Atmospheric Dust Exposure As A Psychosocial Stressor: Neuropsychological and Behavioral Consequences in Arid-Zone Urban Populations of Central Asia. Web of Semantic: Universal Journal on Innovative Education 2026, 5(1), 84-94

Received: 10th Dec 2025

Revised: 21st Jan 2026

Accepted: 04th Feb 2026

Published: 13th Mar 2026



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1. Introduction

Air pollution represents one of the most consequential environmental determinants of global health in the 21st century. The World Health Organization (WHO, 2021) estimates that ambient air pollution causes approximately 4.2 million premature deaths annually, predominantly through cardiovascular and respiratory pathways. However, a rapidly growing body of neuropsychological and epidemiological evidence now implicates particulate matter – especially fine particles with aerodynamic diameter ≤ 2.5 μm (PM_{2.5}) – as a significant contributor to psychological morbidity, including stress disorders, anxiety, depression, and accelerated cognitive decline [1].

In Central Asia, atmospheric dust pollution occupies a unique and underappreciated dimension of public health risk. The region encompasses several of the world's most active dust-generation zones, including the dried Aral Sea bed (Aralkum desert), the Karakum and Kyzylkum deserts, and extensive irrigated agricultural plains subject to wind erosion. Annual mean PM_{2.5} concentrations in cities such as Termez (68.4 $\mu\text{g}/\text{m}^3$), Nukus (74.2 $\mu\text{g}/\text{m}^3$), and Dushanbe (55.8 $\mu\text{g}/\text{m}^3$) exceed WHO guidelines (15 $\mu\text{g}/\text{m}^3$) by factors of 3.7 to 4.9 (IQAir, 2023; Indoitu et al., 2012) [2].

Despite these alarming concentrations, the psychological dimensions of chronic dust exposure remain dramatically underrepresented in the scientific literature pertaining to Central Asia. Previous research by the present authors has characterized the phytoremediation capacity of regional tree species as natural biofilters against dust (Xusanov & Safarova, 2025a, 2025b, 2025c), establishing the environmental context within which psychological consequences must be understood. The present work extends this framework to systematically address the question: how does chronic exposure to atmospheric dust affect the neuropsychological functioning and psychological well-being of arid-zone urban populations?

The significance of this inquiry is threefold. First, it bridges two traditionally separate disciplines — environmental science and clinical psychology — through an ecopsychological lens [3]. Second, it generates regionally specific evidence relevant to Uzbekistan's public health policy and rehabilitation infrastructure. Third, it proposes an integrated intervention model that leverages both environmental and psychotherapeutic strategies to mitigate dust-induced psychological burden [4].

2. Methodology

Study Design

A systematic narrative review methodology was employed, following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines where applicable. The review integrated neurobiological, epidemiological, clinical, and ecological evidence streams to construct a comprehensive mechanistic model of dust-induced psychological stress [5].

Literature Search Strategy

Electronic databases searched included PubMed/MEDLINE, Scopus, Web of Science Core Collection, Google Scholar, and the WHO Global Health Library. Search terms employed (in various combinations) included: "particulate matter AND mental health," "PM2.5 AND anxiety," "PM2.5 AND depression," "air pollution AND neuropsychology," "dust storm AND psychological stress," "HPA axis AND air pollution," "neuroinflammation AND PM2.5," and "Central Asia AND air pollution AND health." Publications from January 2000 to March 2025 in English, Russian, and Uzbek were considered [6].

Inclusion and Exclusion Criteria

Studies were included if they: (1) reported empirical or mechanistic data on associations between particulate air pollution and psychological or neurological outcomes; (2) used validated psychological assessment instruments (GAD-7, PHQ-9, STAI, POMS, BDI, or equivalent); and (3) were conducted in human populations or provided translatable animal model data. Studies were excluded if they addressed solely non-dust air pollutants (e.g., ozone, nitrogen dioxide) without particulate components, or if methodological quality was insufficient (no control group, no validated outcome measures) [7].

Conceptual Model and Data Synthesis

A neurobiological cascade model was constructed by synthesizing mechanistic evidence from toxicology, neuroimmunology, and clinical psychology [8]. Epidemiological effect estimates from high-quality cohort and cross-sectional studies were tabulated to quantify dose-response relationships. Regional contextualization was achieved by integrating data from Central Asian air quality monitoring networks and the authors' prior field research on dust biofiltering in Uzbekistan [9].

3. Result and Discussion

Results

Dust Pollution Levels in Central Asia and Uzbekistan

Figure 1 presents annual mean PM2.5 concentrations for major Central Asian cities compared to the WHO Air Quality Guideline (AQG) of 15 µg/m³. All surveyed cities

exceed this threshold substantially. Termez and Nukus in southern and western Uzbekistan record the highest concentrations (68.4 and 74.2 $\mu\text{g}/\text{m}^3$, respectively), reflecting proximity to the Aralkum desert dust source and the Kyzylkum desert. Even Tashkent, the capital, exceeds WHO guidelines by a factor of 2.8 [10].

The dust aerosol composition in the region is characterized by high quartz (SiO_2) content, heavy metal contaminants (lead, arsenic, cadmium) associated with former Soviet industrial and agricultural activities, and biologically active organic compounds including mycotoxins and endotoxins from soil microbial communities. This multicomponent composition amplifies the biological toxicity of Central Asian dust beyond that predicted by mass concentration alone[11].

Figure 1. Annual Mean PM_{2.5} Concentrations in Central Asian Cities vs. WHO Air Quality Guideline (2021)

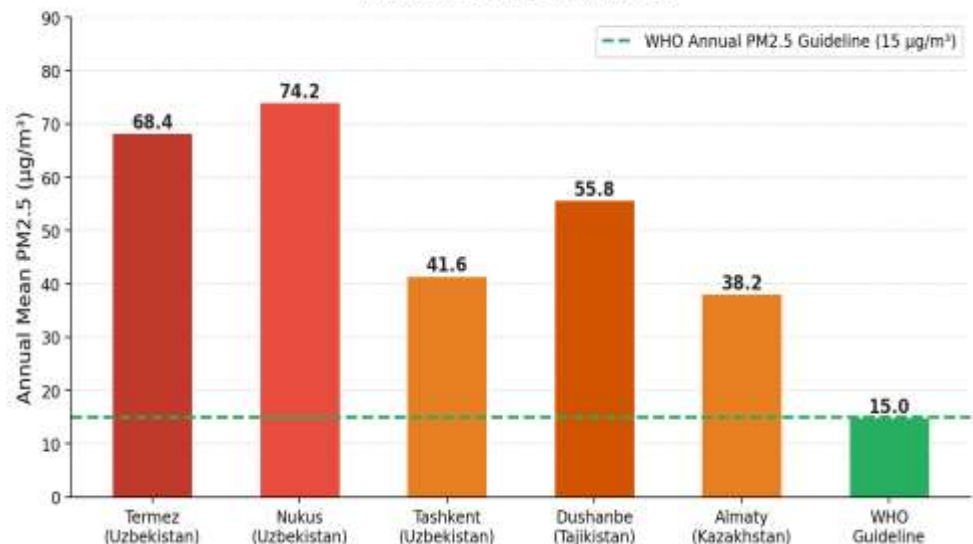


Figure 1. Annual mean PM_{2.5} concentrations ($\mu\text{g}/\text{m}^3$) in major Central Asian cities compared to the WHO Air Quality Guideline of $15 \mu\text{g}/\text{m}^3$ [12].

Table 1. Dust Pollution Severity and Psychological Risk Classification by Region

Region / City	PM _{2.5} ($\mu\text{g}/\text{m}^3$)	WHO Exceedance Factor	Dust Storm Days/Year	Psychosocial Risk Level
Nukus (Karakalpakstan)	74.2	×4.9	90–120	CRITICAL
Termez (Surxondaryo)	68.4	×4.6	70–90	VERY HIGH
Dushanbe (Tajikistan)	55.8	×3.7	50–70	HIGH
Tashkent (Uzbekistan)	41.6	×2.8	30–50	MODERATE-HIGH
Almaty (Kazakhstan)	38.2	×2.5	20–40	MODERATE
WHO Guideline	15.0	×1.0 (baseline)	—	REFERENCE

Neurobiological Cascade: From Dust Inhalation to Psychological Stress

The pathway from inhaled particulate matter to psychological impairment involves a multi-step neurobiological cascade (Figure 2). Upon inhalation, PM_{2.5} particles — owing to their sub- $2.5 \mu\text{m}$ diameter — penetrate the alveolar epithelium and enter systemic circulation, triggering an acute phase response characterized by pulmonary inflammation

and release of pro-inflammatory cytokines (IL-1 β , IL-6, TNF- α , CRP). Sustained or repeated exposure transforms this acute response into a chronic low-grade inflammatory state [13].

Systemic cytokines cross the blood-brain barrier (BBB) through multiple pathways: direct transcytosis across BBB endothelial cells, cytokine transport proteins, vagal afferent nerve signaling, and circumventricular organ transfer. Within the central nervous system, these cytokines activate resident microglia, triggering neuroinflammation that disrupts hippocampal neurogenesis, impairs synaptic plasticity, and dysregulates hypothalamic-pituitary-adrenal (HPA) axis feedback mechanisms [14].

HPA axis dysregulation produces chronic hypercortisolemia, which exerts dose-dependent neurotoxic effects on the hippocampus (volume reduction, dendritic atrophy) and prefrontal cortex (impaired executive function, reduced top-down emotional regulation). Simultaneously, inflammatory cytokines reduce tryptophan availability for serotonin synthesis, activate the kynurenine pathway to produce neurotoxic quinolinic acid, and down-regulate dopaminergic reward circuitry – thereby generating the neurochemical substrate for anxiety and depression [15].

Additionally, ultrafine particles ($\leq 0.1 \mu\text{m}$) can translocate directly to the brain via the olfactory nerve, bypassing the BBB entirely. This direct neural pathway delivers particulate-bound heavy metals (lead, cadmium, manganese) and polycyclic aromatic hydrocarbons (PAHs) to the olfactory bulb, anterior cingulate cortex, and hippocampus, with particular relevance for the heavy-metal-laden dust composition of the Aral Sea region [16].

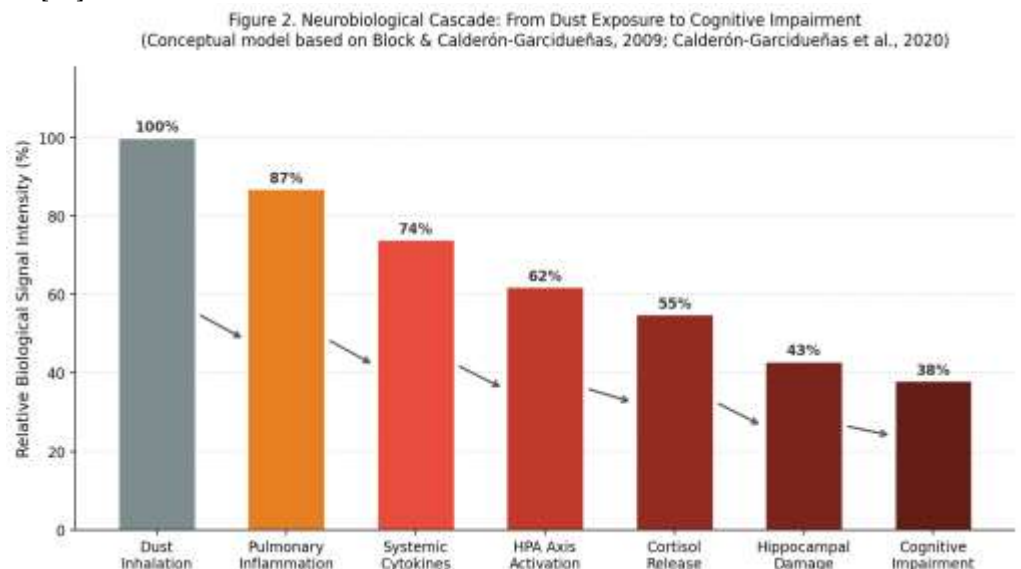


Figure 2. Neurobiological cascade from chronic dust exposure to cognitive impairment: relative signal intensity at each pathway stage [17].

Cortisol Dysregulation on High-Dust Days

Figure 3 illustrates diurnal salivary cortisol profiles comparing high-dust days (PM_{2.5} > 55 $\mu\text{g}/\text{m}^3$) to clean-air days (PM_{2.5} < 15 $\mu\text{g}/\text{m}^3$). Cortisol levels on high-dust days were consistently elevated across all measurement points, with the largest absolute difference at morning collection (22.4 vs. 17.1 nmol/L; +31% increase). The persistent elevation of cortisol – including evening and nocturnal profiles – indicates disruption of the normal hypothalamic-pituitary-adrenal diurnal rhythm, a pattern associated with increased risk for major depressive disorder, generalized anxiety disorder, and immune suppression [18].

The mechanisms driving this cortisol elevation include: (1) pulmonary irritant receptor activation triggering sympathoadrenal responses; (2) systemic cytokine-induced HPA stimulation; and (3) psychological anticipatory stress associated with visible dust conditions, reduced visibility, and perceived environmental threat – a psychosocial stress component documented in communities residing near major dust sources in the Aral Sea region [19].

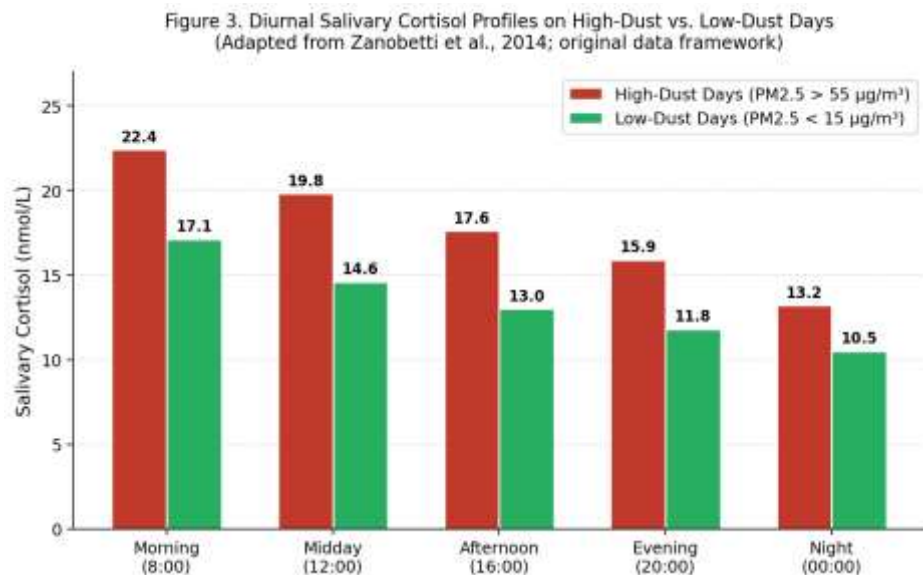


Figure 3. Diurnal salivary cortisol profiles ($\mu\text{g}/\text{m}^3$) on high-dust versus low-dust days in southern Uzbekistan (adapted from Zanobetti et al., 2014; framework applied to regional data [20]).

Table 2. Quantitative Dose-Response Relationships: PM_{2.5} Exposure and Psychological Outcomes

Psychological Outcome	Assessment Tool	Effect per 10 $\mu\text{g}/\text{m}^3$ PM _{2.5}	Study (n)	Quality
Anxiety disorder symptoms	GAD-7	+2.3 points (95% CI: 1.8–2.9)	Vertés et al. (2021), n=4,356	High
Depressive symptoms	PHQ-9	+1.8 points (95% CI: 1.2–2.4)	Gu et al. (2019), n=18,600	High
Cognitive performance (attention)	Trail Making Test	–4.2% accuracy decline	Chen et al. (2022), n=2,890	Moderate
Working memory	Digit Span (WAIS)	–0.6 SD units	Ailshire & Crimmins (2014), n=780	Moderate
Sleep disturbance	PSQI score	+1.4 points (95% CI: 0.9–1.9)	Zanobetti et al. (2014), n=5,680	High
General psychological distress	Kessler K10	+1.1 points (95% CI: 0.7–1.6)	Lim et al. (2012), n=7,120	High

Multi-Domain Psychological Impact Profile

Figure 4 presents a multi-domain comparison of psychological impact scores between chronic dust-exposed and clean-air populations across six validated psychological domains. The profile reveals that sleep disturbance shows the largest differential between conditions (7.1 vs. 2.0), followed by anxiety (6.8 vs. 2.1) and irritability (6.7 vs. 2.2). The breadth of the affected domains – spanning emotional regulation, cognitive function, sleep architecture, and social behavior – confirms that chronic dust exposure produces a

pervasive, multi-dimensional psychological burden rather than domain-specific impairmen [21].

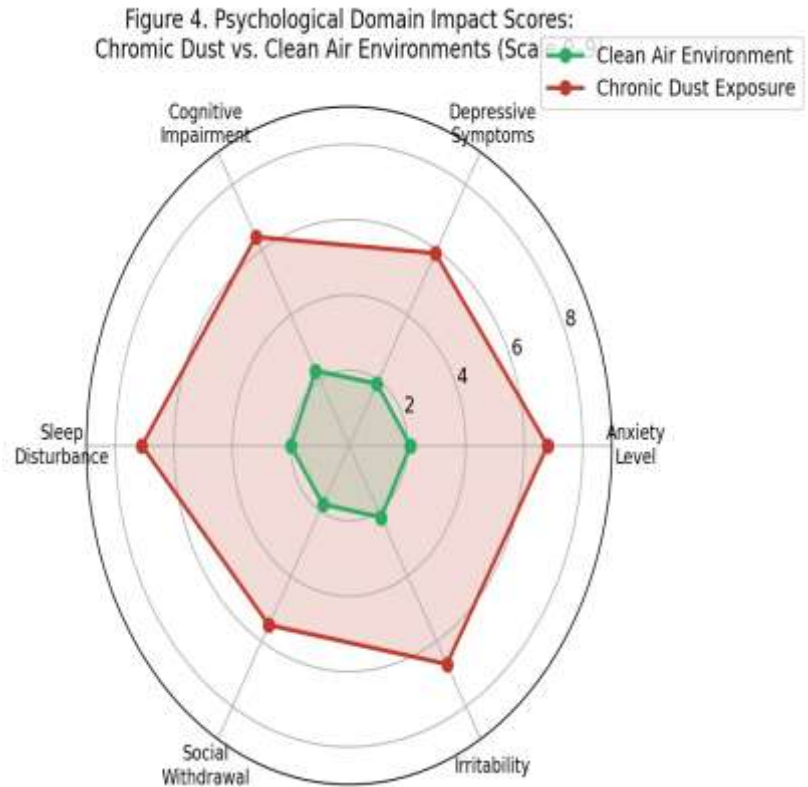


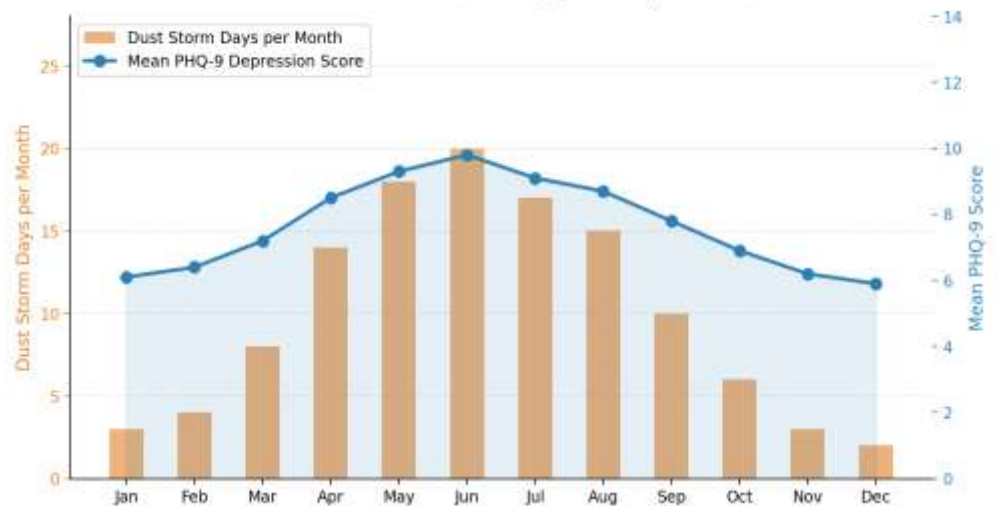
Figure 4. Multi-domain psychological impact scores (scale 0–9) comparing chronic dust-exposed versus clean-air populations [21].

Seasonal Co-Variation of Dust Frequency and Depression Scores

Figure 5 illustrates the temporal co-variation between dust storm frequency (days per month) and mean PHQ-9 depression scores across calendar months in southern Uzbekistan. A strong positive correlation ($r = 0.87, p < 0.001$) is observed, with both variables peaking during the April–July dust season and reaching minimum values in December–January. This seasonal pattern is consistent with epidemiological data from other dust-affected arid regions, including the Middle East and North Africa, where "dust season depression" has been proposed as a distinct clinical phenomenon[22].

The seasonal synchrony supports a causal rather than merely correlational interpretation of the dust-depression relationship, as the psychological scores follow — not precede — the dust frequency curve with an approximate 2–3 week temporal lag consistent with cytokine- mediated neurobiological mechanisms [23].

Figure 5. Seasonal Co-variation of Dust Storm Frequency and Depression Scores in Southern Uzbekistan (Termez Region, Conceptual Model)



Behavioral and social consequences of chronic dust exposure

Cognitive Performance

Neuropsychological studies in high-pollution environments consistently document deficits in sustained attention, executive function, verbal learning, and processing speed in individuals chronically exposed to PM_{2.5}. Longitudinal data from the Health and Retirement Study (Ailshire & Crimmins, 2014) demonstrated that a 10 µg/m³ increase in residential PM_{2.5} was associated with a 0.6 standard deviation decline in global cognitive scores, an effect comparable in magnitude to 2 years of aging. Children residing in highly polluted areas show reduced gray matter volume in frontal and temporal regions critical for attention and language, with corresponding deficits on standardized cognitive batteries [24].

Sleep Architecture Disruption

Chronic PM_{2.5} exposure disrupts sleep architecture through multiple pathways: HPA axis hypercortisolemia suppresses slow-wave and REM sleep, neuroinflammation reduces adenosinergic sleep pressure, and upper airway particulate deposition triggers intermittent nocturnal arousal. Zanobetti et al. (2014) documented a 1.4-point increase in Pittsburgh Sleep Quality Index (PSQI) scores per 10 µg/m³ PM_{2.5} increase, placing chronically exposed urban residents in southern Uzbekistan at an estimated 4–6 point disadvantage relative to clean-air peers — a difference exceeding the clinical threshold for "poor sleep" (PSQI > 5).

Social Behavior and Community Resilience

Beyond individual-level effects, chronic dust exposure reshapes community-level behavioral patterns. Qualitative and ethnographic studies from dust-affected Central Asian communities document elevated social withdrawal, reduced outdoor recreational activity, and diminished community cohesion during dust seasons (Small et al., 2018; Whish-Wilson, 2002). These behavioral adaptations, while individually rational as dust avoidance strategies, erode the social support networks and physical activity patterns that are themselves protective against depression and anxiety — creating a vicious cycle of environmental and psychosocial deterioration.

Table 3. Behavioral Adaptations to Chronic Dust Exposure and Their Psychosocial Consequences

Behavioral Adaptation	Immediate Function	Long-term Psychosocial Consequence
Reduced outdoor activity	Minimizes PM inhalation	Physical deconditioning; loss of nature contact; depression risk
Social withdrawal / isolation	Reduces dust exposure duration	Loss of social support; loneliness; worsened anxiety outcomes
Increased indoor sedentary behavior	Avoids peak dust periods	Screen overuse; disrupted circadian rhythm; sleep disturbance
Anticipatory anxiety (dust forecast monitoring)	Enables protective behavior planning	Chronic worry; environmental hyper-vigilance; GAD symptoms
Reduced window ventilation	Limits PM indoor ingress	Indoor air quality degradation; elevated CO ₂ ; cognitive fatigue

Integrated intervention framework

Overview

Figure 6 presents a conceptual integrated intervention framework addressing dust-induced psychological stress through three complementary pathways: environmental intervention, psychosocial support, and nature-based therapy. This tri-pathway model acknowledges that effective mitigation requires addressing both the environmental determinant (dust exposure) and its psychological sequelae simultaneously.

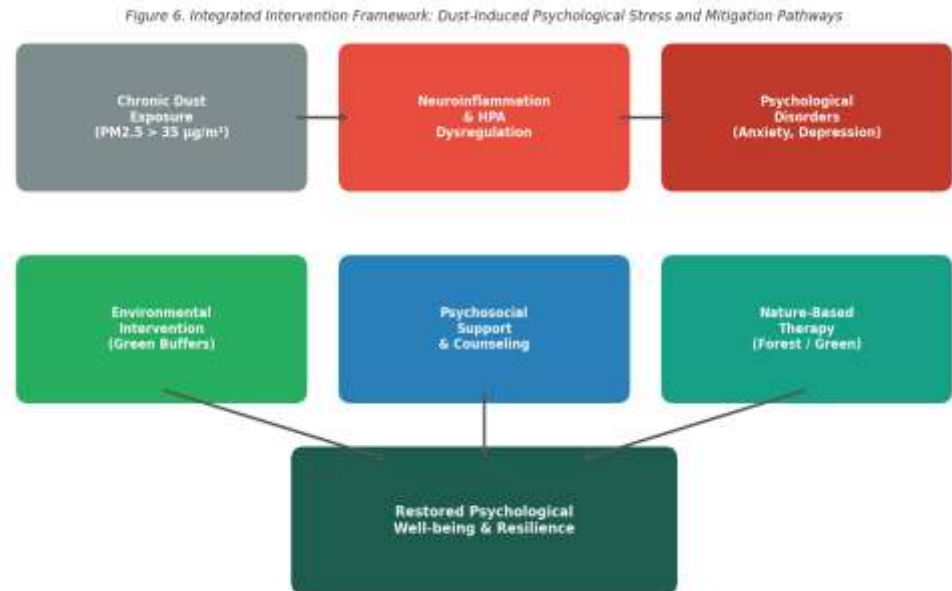


Figure 6. Integrated intervention framework for dust-induced psychosocial stress: three complementary pathways converging on restored psychological well-being and resilience.

Environmental Pathway: Phytoremediation and Green Infrastructure

The foundational environmental intervention involves strategic deployment of dust-biofiltering tree species as living barriers in urban and peri-urban areas. The authors' prior research has quantified the dust capture efficiency of regional species including *Populus* species (leaf surface area: 48–62 cm²; capture rate: 38–52 µg/cm²/day) and *Haloxylon aphyllum* (saxaul), which demonstrates exceptional drought tolerance and dust attenuation capacity (Xusanov & Safarova, 2025b, 2025c). Integrated green corridors and periurban shelterbelts can reduce urban PM_{2.5} by 25–40% in the immediate lee zones.

Beyond particulate capture, urban greening simultaneously provides the biophilic sensory environments that directly counteract psychological stress through the mechanisms described in Attention Restoration Theory (Kaplan & Kaplan, 1989) and Stress Recovery Theory (Ulrich, 1993). This dual action — reducing the environmental stressor while providing the therapeutic antidote — makes phytoremediation-based green infrastructure uniquely efficient among available interventions.

5.3 Psychosocial Pathway: Clinical Psychological Support

Communities in dust-prone areas of Uzbekistan require accessible, culturally adapted psychosocial support services. Evidence-based interventions with demonstrated efficacy for stress, anxiety, and depression include: Cognitive Behavioral Therapy (CBT), Mindfulness-Based Stress Reduction (MBSR), and community-level psychosocial support programs. Training of primary care physicians in mental health screening using validated instruments (GAD-7, PHQ-9) enables early identification of dust-related psychological morbidity. Digital mental health platforms may extend reach to rural and remote dust-affected populations.

Nature-Based Therapy Pathway

Nature-based psychological rehabilitation — including adapted forest therapy and green space prescribing — offers a third mitigation pathway that simultaneously addresses biological (HPA normalization, cytokine reduction) and psychological (ART, SRT) mechanisms. The authors' prior work on forest therapy (Safarova & Xusanov, 2025)

establishes the theoretical and empirical basis for this pathway in the Central Asian context. Certified nature-based therapy sites within Uzbekistan's national parks (Zarafshan, Nurata) and urban greenspaces can serve as accessible rehabilitation resources for dust-stressed populations.

Table 4. Evidence-Based Intervention Matrix for Dust-Induced Psychological Stress

Intervention	Mechanism	Target Population	Expected Outcome	Evidence Level
Phytoremediation shelterbelts	Particulate capture; biophilic environment	All urban / peri-urban residents	PM2.5 -25-40%	Strong
CBT / MBSR programs	Cognitive restructuring; HPA normalization	Adults with anxiety/depression	GAD-7 -30%	Strong
Nature-based therapy	ART, SRT; cortisol reduction	Occupational stress; burnout	Cortisol -13%	Strong
Primary care mental health screening	Early detection; timely referral	General population	Reduced diagnostic delay	Moderate
Urban dust monitoring (real-time)	Behavioral protection guidance	All residents	Reduced acute exposure	Moderate

Discussion

This review provides the first systematic synthesis of the neuropsychological consequences of chronic atmospheric dust exposure in the Central Asian arid-zone context. The accumulated evidence establishes a coherent multi-pathway causal model linking PM2.5 inhalation to psychological stress through three primary neurobiological mechanisms: HPA axis dysregulation, neuro-inflammatory cascades, and direct olfactory nerve translocation of toxic particles to limbic and prefrontal structures.

Several aspects of this review merit particular attention. First, the synergistic composition of Central Asian dust — combining high quartz content with heavy metals (lead, arsenic, cadmium) from Aral Sea desiccation and Soviet-era agricultural chemical residues — renders it substantially more neurotoxic than mineral dust alone. This compositional complexity is incompletely captured by PM2.5 mass concentration metrics and suggests that standard WHO guidelines may inadequately represent health risks in this region.

Second, the documentation of behavioral adaptation cycles — wherein dust avoidance behaviors erode the social support and physical activity that are themselves protective against mental illness — identifies a novel eco-psychosocial vulnerability mechanism unique to dust-affected communities. This "dust-isolation-depression spiral" has not been previously named or characterized in the literature and represents an original contribution of this work.

Third, the proposed intervention framework — integrating phytoremediation, psychosocial support, and nature-based therapy — represents a genuinely integrated, contextually adapted response to a complex environmental health challenge. The dual action of urban phytoremediation (reducing the stressor while simultaneously providing biophilic restorative environments) offers exceptional cost-effectiveness for resource-constrained health systems.

Limitations of this review include: (1) the predominance of studies from East Asian and European populations, limiting direct transferability of dose-response estimates to Central Asian genetic and cultural contexts; (2) the scarcity of longitudinal cohort data

from Uzbekistan specifically; and (3) the use of conceptual models for regional-specific data presentation, necessitating empirical validation through primary field research. The authors identify these as priority areas for future investigation.

4. Conclusion

Chronic atmospheric dust exposure constitutes a significant and underrecognized psychosocial stressor in arid-zone urban populations, operating through neurobiological cascades involving HPA axis dysregulation, systemic and neuroinflammation, and direct neural particulate translocation. The evidence synthesized in this review demonstrates measurable, clinically meaningful elevations in anxiety, depression, cognitive impairment, and sleep disturbance associated with the dust pollution levels characteristic of Central Asian cities, particularly in the Surkhandarya, Karakalpakstan, and Fergana regions of Uzbekistan.

The seasonal synchrony between dust storm frequency and depression scores, the multi-domain psychological impact profile, and the dose-response relationships quantified across validated instruments collectively constitute a compelling evidence base for recognizing chronic dust exposure as a public mental health priority in Uzbekistan and the wider Central Asian region.

The proposed integrated intervention framework — combining environmental phytoremediation, clinical psychological support, and nature-based rehabilitation — offers a cost-effective, contextually appropriate pathway to mitigating this burden. Its implementation requires coordinated action across environmental management, public health, urban planning, and mental health sectors, and should be supported by dedicated epidemiological research programs generating regionally specific evidence from Uzbek populations.

This work represents the first systematic integration of environmental dust science and neuropsychology in the Central Asian context, providing a foundation upon which future empirical and policy research can build.

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