Acta Biomed 2025; Vol. 96, N. 6: 16946 DOI: 10.23750/abm.v96i6.16946

Review

The impact of pollution on mental health: A narrative review

Roberta Ciuffini[1], *Alba Piroli*[1], *Alfonso Marrelli*[2]

[1]Department of Life, Health and Environmental Sciences, University of L'Aquila, Italy; [2]Clinical Neurophysiology Unit, San Salvatore Hospital, L'Aquila, Italy

Abstract. *Background and Aim:* Pollution is increasingly recognized as a major global health determinant, with growing evidence linking environmental contaminants to neuropsychiatric disorders. Despite extensive research, the impact of pollution on mental health remains underexplored, particularly regarding the neurobiological and psychosocial pathways involved. This narrative review aims to synthesize current evidence on how environmental pollutants, chemical, physical, and psychosocial, affect the central nervous system and contribute to psychiatric morbidity. *Methods:* A comprehensive narrative review was conducted using PubMed and Embase, focusing on studies from 2000 to 2024. Keywords included "pollution," "psychiatric disorders," "neurotoxicity," and "psychological distress." Peer-reviewed articles reporting on epidemiological, mechanistic, and neurobiological findings were included. Studies focusing solely on general environmental exposure without neuropsychiatric relevance were excluded. *Results:* Findings demonstrate that air pollutants such as $PM_{2.5}$, NO_2, heavy metals, and persistent organic compounds contribute to cognitive impairment, depression, anxiety, and neurodegenerative conditions like Alzheimer's and Parkinson's disease. Mechanisms include oxidative stress, neuroinflammation, blood-brain barrier disruption, mitochondrial dysfunction, endocrine dysregulation, and epigenetic changes. Psychosocial stressors linked to urban environments, media saturation, and perceived environmental degradation further exacerbate mental health outcomes. Syndromes like sick building syndrome, multiple chemical sensitivity, and solastalgia emerge as stress-related manifestations. This narrative review critically examines the methodological limitations and evidence strength of current studies, highlighting key gaps and future research directions. *Conclusions*: Pollution-induced neuropsychiatric effects are multifactorial, involving complex interactions between environmental, biological, and psychological factors. A biopsychosocial framework is essential to understand these effects and to guide interdisciplinary prevention strategies. Addressing pollution as a mental health risk factor requires urgent public health action and integration of environmental policies with psychiatric care. (www.actabiomedica.it)

Key words: pollution, neuropsychiatric disorders, psychological distress, climate, building-related syndromes, media pollution.

Introduction

Pollution has become a major concern across scientific disciplines, public health systems, and international policy agendas. Its far-reaching implications for both planetary integrity and human well-being underscore the urgent need for a multidisciplinary understanding of its health effects. Pollution can be broadly defined as the accumulation of contaminants, arising from anthropogenic activities or natural phenomena, that disrupt the environmental conditions essential to human life. Numerous classifications exist, distinguishing between pollution of air, water, soil, and food (natural environments), and that of domestic,

occupational, and urban settings (human habitats). It may also be categorized based on origin (agricultural, industrial, military) or nature (chemical, photochemical, biological, acoustic, electromagnetic, genetic, radioactive). The impact on human health depends on the associated pathophysiological mechanisms, including infectious, inflammatory, toxic, dysmetabolic, genetic, teratogenic, oncogenic, and degenerative processes. Organs and systems most exposed to pollution, namely the skin, respiratory tract, gastrointestinal system, circulatory system, and cardiovascular system, serve not only as entry routes but also as primary targets of toxic insult (1). In parallel, complex regulatory networks such as the immune, endocrine, and nervous systems are vulnerable to environmental disruption, with significant consequences for homeostatic and allostatic regulation. Given the interdependence of physical and psychological well-being, the adverse effects of pollution extend beyond individuals to affect communities and global populations. To address these challenges, research has increasingly adopted a bio-psycho-social model of health, as introduced by Engel 1980, which integrates biological, psychological, and social dimensions of disease processes (2,3). This approach offers a valuable framework for understanding how pollutants impact the human organism and provides a basis for the development of integrated prevention and mitigation strategies. Despite the proliferation of media coverage and academic interest in pollution, the dissemination of reliable, evidence-based information remains inconsistent. Narratives may be influenced by political and economic interests or shaped by ideological agendas, complicating efforts to reach scientific consensus. Recent estimates indicate that approximately 25% of human diseases are attributable to pollutant exposure. Some epidemiological findings suggest that parental exposure to environmental contaminants during the periconceptional phase can impact cardiovascular health and other outcomes through epigenetic modifications (4,5). This review synthesizes current knowledge on the neuropsychiatric effects of pollution, with an emphasis on both the direct neurobiological impacts of air pollutants and the chronic psychological stress associated with long-term environmental exposure. By adopting a narrative review methodology, the article aims to consolidate interdisciplinary insights and provide a comprehensive overview of how pollution disrupts the function and structure of the nervous system. Furthermore, this review aims to critically assess the quality and consistency of available evidence, considering methodological strengths, weaknesses, and the complexity of causal inference in this emerging field.

Materials and Methods

This study is a narrative review aimed at synthesizing and integrating the existing literature concerning the neuropsychiatric effects of environmental pollution. A narrative review methodology was selected to accommodate the inherently interdisciplinary nature of the topic, which intersects environmental science, neuroscience, psychology, and public health. Unlike systematic reviews, which follow strict inclusion criteria and quantitative synthesis, narrative reviews allow for a broader and more flexible exploration of complex themes, enabling the integration of diverse data sources and conceptual frameworks. A comprehensive literature search was conducted using the PubMed and Embase databases. The following keywords were used: *pollution*, *neurological diseases*, *psychiatric disorders*, and *psychological distress*. The search included articles published between 2000 and 2025 to ensure relevance to contemporary environmental and epidemiological contexts. Eligible studies were limited to those published in English, peer-reviewed, and specifically addressing the effects of pollution on the human nervous system, either through direct biological mechanisms or indirect psychosocial pathways. Studies were excluded if they addressed environmental pollution without explicit reference to neuropsychiatric implications. Animal studies were also excluded unless their findings were deemed directly translatable to human health. Following the elimination of duplicate records, two independent reviewers conducted an initial screening of titles and abstracts according to predefined inclusion criteria. Full-text versions of eligible studies were subsequently assessed for methodological quality and relevance. Data from the selected studies were extracted and thematically organized. The review focused on both biological and psychosocial consequences of

environmental pollution on the nervous system. Particular emphasis was placed on the neurotoxic effects of fine particulate matter ($PM_{2.5}$), nitrogen dioxide (NO_2), heavy metals, and pesticides. Key mechanisms such as oxidative stress, disruption of the blood-brain barrier, and mitochondrial dysfunction were explored in relation to their role in neuroinflammation and neurodegeneration. In addition, the review examined the influence of urbanization, exposure to mass media, and noise pollution on mental health outcomes, including anxiety, depression, schizophrenia, and cognitive impairment. Given the narrative nature of the study, no formal statistical analyses were performed. Instead, data were qualitatively assessed and synthesized based on the recurrence of themes and consistency of findings across the literature. Higher levels of evidence, such as meta-analyses and large-scale epidemiological studies, were prioritized, while smaller observational studies were considered for contextual insight. Where appropriate, both convergences and divergences in findings were discussed to provide a critical appraisal of the current state of knowledge on pollution-related neuropsychiatric disorders.

The process of literature search, screening, and selection is summarized in Table 1.

Results

Numerous epidemiological studies have investigated potential, probable, or established associations between environmental pollutants and neurological or psychiatric conditions. Chemical agents such as sulfur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO), fine particulate matter ($PM_{2.5}$), coarse particulate matter (PM_{10}), and black carbon have been shown to impair cognitive functions, particularly among the elderly population (6,7). Fine particulate matter is estimated to account for approximately one-third of strokes and one-fifth of dementia cases globally (8). Environmental exposure to metals such as aluminum (Al), selenium (Se), silicon (Si), as well as pesticides and strong electromagnetic fields, has been identified as a significant risk factor for neurodegenerative disorders, especially when compounded by vitamin D deficiency (9). Only a small proportion of Alzheimer's disease (AD) cases, approximately 5%, have a hereditary basis, with the majority being attributable to environmental factors. Numerous studies have demonstrated that inhaled magnetite particles, iron nanoparticles, occupational exposure to magnetic fields (10), ozone (O_3) (11), and aluminum absorbed via the respiratory, digestive, or dermal routes (12), as well as $PM_{2.5}$, oxidants, and combustion-related nanoparticles, are all involved in AD pathogenesis (13). Pollutants typically enter the body via the respiratory tract, affecting the nasal mucosa and subsequently translocating through the olfactory (cranial nerve I) and trigeminal (cranial nerve V) pathways to reach central nervous structures. This may account for the early olfactory deficits that often precede cognitive decline in neurodegenerative disorders (14). The multifactorial etiology of Parkinson's disease (PD) involves genetic predispositions alongside environmental contributors, such as infections, brain injury, neurotoxins, and epigenetic changes. The accumulation of metals within the central nervous system (CNS), in the absence of efficient detoxification mechanisms, induces oxidative stress and cytotoxicity. Occupational and environmental exposure to neurotoxic substances,

Table 1. Literature search and selection process (PRISMA flow adapted for narrative review).

Phase	Records	Description
Records identified through PubMed and Embase search	1520	Using keywords: "pollution", "psychiatric disorders", "neurotoxicity", "psychological distress"
Records after duplicates removed	1420	After removing 100 duplicates
Records screened (title and abstract)	1420	Excluded: 1250 (not related to neuropsychiatric effects or pollution)
Full-text articles assessed for eligibility	170	Excluded: 103 (irrelevant or poor methodological quality)
Studies included in narrative synthesis	67	Selected for relevance to pollution and mental health

including pesticides, heavy metals, industrial chemicals, and other xenobiotics, has been associated with elevated risk of PD. Factors such as urban residency, dietary habits, and agricultural or industrial occupations further modulate this risk. Chronic exposure to substances like manganese, copper, iron, aluminum, and lead heightens vulnerability, particularly in individuals with a genetic predisposition. Notably, high atmospheric concentrations of NO_2 have been associated with a significantly increased incidence of PD (15). The potential pathophysiological mechanisms involve NO_2-mediated toxicity to the olfactory nerve, which is structurally susceptible to airborne pollutants. The pathological hallmark of PD, α-synuclein, originates in the olfactory bulb and the dorsal motor nucleus of the vagus nerve. NO_2 exposure may trigger systemic increases in pro-inflammatory cytokines, such as interleukin-1β, IL-6, IL-8, and TNF-α, promoting neuroinflammation implicated in PD, AD, and other neurodegenerative disorders. Excessive exposure to toxic metals (e.g., mercury, lead, copper, zinc, iron, manganese, aluminum, arsenic, cadmium, selenium) enables these substances to cross the blood-brain barrier (BBB), accumulate in cerebral tissues, and damage dopaminergic neurons through oxidative stress, mitochondrial dysfunction, and α-synuclein aggregation (16). Even trace levels of these metals may disrupt basal ganglia signaling over time. Copper nanoparticle accumulation has been implicated in extrapyramidal disorders (17), while manganese exposure is a recognized PD risk factor (18). Elevated rates of extrapyramidal symptoms, depressive disorders, and psychological distress were documented following the 2016 oil spill along the Louisiana coast (19). Dioxin exposure in Vietnam War veterans has been significantly associated with psychological disorders (20), and elevated rates of depression were reported in Southern Mississippi following a methyl-parathion contamination event (21). These findings support an emerging link between $PM_{2.5}$ exposure and mental health disorders such as depression and suicidal ideation (22,51), with NO_2 concentrations correlating with seasonal exacerbations of depressive symptoms (23). Exposure to pollutants including heavy metals (Pb, Cd), nitrogen, sulfur compounds, and organic solvents has been correlated with increased risk of schizophrenia and other psychotic conditions (22). Urban living has been associated with increased activation of the amygdala and anterior perigenual cingulate cortex, reflecting elevated emotional and environmental stimuli. This hyperactivity predisposes individuals to anxiety disorders and psychotic syndromes, including depression and schizophrenia (24,25). Prenatal exposure to tobacco smoke, nicotine, pesticides, and environmental pollutants may disrupt neural connectivity between the anterior cingulate cortex and amygdala, contributing to attention-deficit/hyperactivity disorder (ADHD) (26). Early-life exposure to NO_2, $PM_{2.5}$ (27), heavy metals, organophosphates, perfluoroalkyl substances (PFAS), bisphenol A (BPA), and polybrominated diphenyl ethers has been associated with neurodevelopmental disorders, immune dysfunction, respiratory compromise, preterm birth, obesity, and ADHD (28). Autism spectrum disorders (ASD) have also been linked to prenatal exposure to $PM_{2.5}$, NO_2, pesticides, PFAS, BPA, and heavy metals, especially in cases with short interpregnancy intervals (29,31). Pollution-related mental health effects may extend to obsessive-compulsive disorder (OCD) (32). Endocrine, sexual, and behavioral disorders, including obesity, have been associated with PFAS and BPA exposure (33), while glyphosate has been implicated in reproductive dysfunction (34). Psychological distress caused by pollution reflects complex interactions between exogenous (environmental) and endogenous (individual) vulnerability factors. Exogenous elements include overstimulation from media (billboards, television, social networks), digital devices, and subliminal messaging, which may lead to reduced critical thinking, cognitive homogenization, and loss of individual autonomy (35). Endogenous vulnerability factors include life circumstances, social relationships, cognitive patterns (e.g., rumination), psychological fatigue, and passive or submissive relational styles (36). When these factors overwhelm psychological resilience, maladaptive stress responses may ensue. Shepard (37) notes that environmental context is integral to identity formation. According to identity theory (38) and social identity theory (39), personal and collective identities are shaped by social group affiliations and place attachment (40). Environmental stress, a term introduced by Lepore and Evans (41), encompasses physical and social conditions perceived as threatening, including

air pollution, noise, crowding, and climate change (42). Seligman's theory of learned helplessness (43) describes how persistent exposure to uncontrollable environmental stressors induces a sense of powerlessness and frustration. This is reflected in both physical symptoms (e.g., insomnia, reduced mobility) and interpersonal dysfunction (e.g., diminished empathy, aggression), as well as cognitive impairments.

Pollution-driven stress also promotes behavioral disengagement from pro-environmental attitudes, reinforcing destructive feedback loops. The resulting neuropsychological burden manifests at molecular, systemic, and behavioral levels, with profound consequences for individual and collective mental health.

Discussion

From a pathophysiological perspective, multiple mechanisms have been hypothesized to explain how environmental pollutants of diverse physical and chemical origins may trigger, exacerbate, or complicate neurological and psychiatric conditions. These mechanisms include oxidative stress, neuroinflammation, genetic and epigenetic dysregulation, vascular impairment, xenobiotic accumulation, neuroendocrine disruption, and neurotransmitter imbalance.

Oxidative stress

Air pollutants such as nitrogen monoxide (NO), nitrogen dioxide (NO_2), black carbon, and $PM_{2.5}$ are known to impair antioxidant defenses (44,45). Environmental stressors can disrupt mitochondrial function, particularly through inhibition of Na^+/K^+-ATPase activity, compromising neuronal energy metabolism (46). The overproduction of reactive oxygen species (ROS), triggered by exposure to heavy metals and pesticides, further disturbs redox homeostasis, leading to lipid peroxidation, DNA damage, and protein oxidation in neural tissues (47,48).

Neuroinflammation and neurodegeneration

Compounds such as NO, NO_2, SO_2, SO_3, black carbon, and $PM_{2.5}$ promote neuroinflammatory responses due to their physicochemical properties and their ability to disseminate systemically (44). Once these pollutants cross the blood-brain barrier (BBB), they may activate resident immune cells, including microglia and astrocytes, leading to chronic inflammation, synaptic dysfunction, and neuronal loss (49,50). This neuroinflammatory cascade contributes to structural brain changes, such as reduced gray and white matter volumes and ventricular enlargement, hallmarks of neurodegeneration. The presence of magnetite nanoparticles and combustion- or friction-related byproducts further accelerates neurofibrillary degeneration, including the formation of beta-amyloid plaques and tau protein aggregates (13,52). $PM_{2.5}$ exposure has also been associated with elevated pro-inflammatory cytokine levels (e.g., IL-6, TNF-α), corroborating its role in chronic neurodegenerative processes (53). Persistent neuroinflammation may impair key detoxification pathways in the endoplasmic reticulum, altering the metabolism of essential biomolecules such as arachidonic acid, glutathione, melatonin, cholesterol, and others. These disruptions have been implicated in the pathogenesis of neurodevelopmental disorders, including autism spectrum disorder (ASD) (30).

Genetic and epigenetic alterations

Formaldehyde exposure, a common environmental contaminant, has been shown to induce chromatin instability by modifying cytosolic histone acetylation patterns, thereby accelerating cellular aging (7). $PM_{2.5}$ exposure also disrupts epigenetic regulation by altering DNA methylation and reducing the glutathione/GSSG ratio, which impairs S-adenosylmethionine synthesis and gene expression fidelity (54). Additional studies have reported alterations in microRNA-associated extracellular vesicles, which may further mediate gene-environment interactions relevant to neuropsychiatric outcomes (50,55).

Vascular damage

Nanoparticles originating from combustion processes and vehicular wear products (e.g., clutch and brake linings) can reach the CNS via olfactory pathways and disrupt neurovascular integrity, contributing

to the development of small vessel disease, microinfarctions, and hemorrhagic lesions (13,55). These processes also potentiate neurofibrillary degeneration and are implicated in Alzheimer's disease progression.

Xenobiotic accumulation

Heavy metals such as lead, cadmium, mercury, arsenic, and aluminum can accumulate in the CNS, forming oxides that contribute to neurotoxicity. These metals facilitate oxidative stress and may stimulate β-amyloid and hyperphosphorylated tau protein deposition, central features of Alzheimer's and related tauopathies (44,56). Copper and manganese deposition in the basal ganglia is implicated in extrapyramidal syndromes, with additional cognitive impairments linked to prefrontal cortex dysfunction (57). Mercury, in particular, interferes with the metabolism of arachidonic acid, glutathione, melatonin, and other neuroprotective substances (58). Additive and synergistic neurotoxicity has been observed in the interaction of Pb, Cd, and MeHg with NMDA receptors and glutamatergic signaling pathways (48).

Neuroendocrine dysregulation

Disruption of the hypothalamic-pituitary-adrenal (HPA) axis by environmental stressors contributes to circadian rhythm disturbances, thyroid dysfunction, and deficiencies in vitamin D, melatonin, and sex hormones (22,59). Even short-term, low-dose exposures may disrupt steroid hormone homeostasis by altering pituitary release of corticotropin-releasing hormone (CRH) and downstream activation of adrenocorticotropic hormone (ACTH) and cortisol production (60).

Neurotransmitter and electrophysiological dysregulation

Several pollutants interfere with neurotransmitter systems, particularly glutamatergic and cholinergic signaling. PFAS compounds, for example, reduce acetylcholine levels and upregulate neuronal markers such as GAP-43, synaptophysin, tau protein, and Ca^{2+}/calmodulin-dependent protein kinase II (CaMKII), impairing long-term potentiation and synaptic plasticity (45). Nanoparticles also influence ion channel activity, potentially blocking channel pores, modulating voltage-dependent activation, or acting as physical resonators for environmental stimuli. These effects have implications for neuroregulation, cardiovascular function, and cancer therapy (61). Aluminum, primarily ingested through dietary sources but also absorbed nasally, has been associated with neurotoxicity in individuals with compromised renal clearance, potentially contributing to AD via oxidative damage and β-amyloid accumulation.

Psychological distress

$PM_{2.5}$, NO_2, pesticides, heavy metals, and noise and light pollution are known to elicit chronic stress responses, which can precipitate or exacerbate psychiatric conditions such as anxiety, depression, and psychosis (62). Prenatal exposure to polycyclic aromatic hydrocarbons (PAHs) has been associated with neurodevelopmental abnormalities, including aggression, somatization, social withdrawal, and mood dysregulation (63,64). Additional studies have linked passive smoking (65), wildfire exposure (66), limited sunlight and environmental degradation (67), and contaminated drinking water (68) to affective and cognitive disorders. Natural disasters and forced migration due to climate change or food insecurity are further stressors that compound mental health burdens (69,70). Noise pollution may trigger stress-induced hormonal imbalances, while artificial light, especially in urban settings, can disrupt circadian regulation and sleep quality (71,72). The co-occurrence of chemical and acoustic pollution accelerates neurovascular and cognitive decline (73). The COVID-19 pandemic, driven by SARS-CoV-2 infection, has been shown to provoke neuroinflammation and neurodegeneration, suggesting a shared pathophysiological mechanism with pollution-induced disorders (13). The concept of allostatic load, reflecting the cumulative biological cost of chronic stress, offers a unifying framework linking environmental exposure to CNS dysfunction (74).

Environmental and architectural determinants

Pollution in residential and occupational environments, often referred to as "housing pollution," is

associated with sick building syndrome (SBS), characterized by a constellation of physical and psychological symptoms arising from poor indoor air quality, artificial ventilation, and microclimatic imbalances (75,76). Urban architecture and high-rise living are also associated with increased psychological distress, especially in densely populated environments (77,78). Azuma (79) introduced the term "building-related symptoms," which are influenced by seasonal variation and psychosocial context. Comparative studies indicate improved mental health in individuals exposed to natural versus urban settings (80). During neurodevelopment, exposure to environmental stressors significantly increases the risk of cognitive and emotional dysfunction, including attention deficits, panic, and memory impairment (81). Children raised in polluted environments may develop maladaptive behaviors that perpetuate environmental degradation (82). Interestingly, the perception of pollution alone, without direct exposure, can provoke psychological distress, such as in obsessive-compulsive disorder (OCD) linked to contamination obsessions and pathophobia (83). The term "solastalgia" has been coined to describe the emotional distress experienced by individuals witnessing environmental degradation in their own communities (84). Other syndromes associated with environmental exposure, though not formally classified as clinical entities, include include environmental medical syndromes (85), SBS (86), multiple chemical sensitivity (MCS) (87), idiopathic environmental intolerance (IEI) (88), electromagnetic hypersensitivity (EHS) (89), and chronic fatigue syndrome (CFS) (90). Conditions such as burnout (91), fibromyalgia (92), and chronic candidiasis (93) may also be exacerbated by prolonged exposure to environmental toxins. The epidemiological and mechanistic relationships between pollutants and neuropsychiatric disorders are summarized in Table 1, Table 2, Table 3 and Table 4 categorizing exposures by type (chemical, physical, social, and natural) and corresponding symptomatology.

Table 2. Summary of chemical pollutants and their neuropsychiatric effects.

Pollutant	Neurological disease	Psychiatric disorders	Psychological distress	Pathogenetic mechanism	References
Heavy metals (Pb, Cd, Hg, As, Al)	Neurodegenerative diseases (AD, PD), cognitive impairment	Depression, psychosis	Psychological distress, neurodevelopmental disorders	Oxidative stress, neuroinflammation, BBB disruption, neurotransmitter imbalance	(16, 22, 48, 49, 50, 56, 59)
$PM_{2.5}$, NO_2, SO_2, CO, O_3	Cognitive decline, stroke, dementia, PD	Depression, schizophrenia, anxiety, ADHD, ASD	Psychological distress, eco-anxiety	Oxidative stress, neuroinflammation, mitochondrial dysfunction	(6, 7, 8, 9, 10, 11, 12, 14, 22, 23, 24, 25, 27, 29, 30, 31, 32, 48, 49, 51, 53, 62)
Pesticides, organophosphates, glyphosate	Neurodegeneration, PD risk	Depression, anxiety, reproductive dysfunction	Psychological distress	Neurotoxicity, endocrine disruption, oxidative stress	(18, 21, 26, 28, 34)
Persistent organic pollutants (POPs), PFAS, BPA	Neurodevelopmental impairment, hormonal dysregulation	Depression, obesity, endocrine-related disorders	Psychological distress	Hormonal dysregulation, neurotransmitter disruption	(28, 33)
Alcohol, nicotine, passive smoking	Cognitive impairment, neurodevelopmental effects	Depression, anxiety, ADHD	Psychological distress	Neurotransmitter imbalance, oxidative stress	(26, 65)

Table 3. Summary of physical pollutants and their neuropsychiatric effects.

Pollutant	Neurological disease	Psychiatric disorders	Psychological distress	Pathogenetic mechanism	References
Light	Circadian rhythm disorders, sleep disturbances	Depression, anxiety	Psychological distress, mood disorders	Circadian rhythm disruption, hormonal imbalance	(71, 72)
Noise	Cognitive impairment, hearing loss	Depression, anxiety, stress-related disorders	Psychological distress	Stress hormone release, oxidative stress, vascular dysfunction	(48, 73)
Radiation (EMF, UV, ionizing)	Neurodegeneration, cognitive decline	Anxiety, depression, mood disorders	Psychological distress	Oxidative stress, mitochondrial dysfunction, neuroinflammation	(89, 90)

Table 4. Summary of social and environmental factors associated with neuropsychiatric effects

Pollutant	Neurological disease	Psychiatric disorders	Psychological distress	Pathogenetic mechanism	References
Environmental catastrophes	Cognitive impairment, PTSD	Depression, anxiety, PTSD, mood disorders	Psychological distress, eco-anxiety	Chronic stress, neuroendocrine disruption, allostatic overload	(66, 67, 69, 70)
Buildings & Climate	Cognitive dysfunction, mood disorders	Depression, anxiety, behavioral disorders	Psychological distress, burnout	Indoor air quality impairment, microclimate alteration, psychosocial stress	(76, 77, 78, 79, 80, 81, 82)
Mass media	—	Behavioral and mood disorders	Psychological distress, learned helplessness, maladaptive behaviors	Cognitive overload, overstimulation, emotional dysregulation	(35, 36)

Limits of current evidence and future perspectives

Although numerous epidemiological studies have explored associations between environmental pollutants (e.g., $PM_{2.5}$, PM_{10}, NO_2) and mental health outcomes such as depression, anxiety, PTSD, and schizophrenia, most reported associations are modest in magnitude (e.g., RR≈1.01–1.02 per unit increase) and highly sensitive to confounding factors (94,95). Recent umbrella reviews classify these associations as "highly suggestive" (Class II) primarily for suicide and psychiatric hospitalizations, while many other associations remain inconclusive (96,97). Although effect sizes are small, the widespread exposure implies considerable public health consequences (98). Mechanistic studies, mainly in animal models, have clarified plausible biological pathways, including oxidative stress, neuroinflammation, HPA-axis disruption, and hippocampal damage (99). However, translation to human contexts is limited. Mendelian Randomization (MR) analyses report potential causal links between $PM_{2.5}$/NO_2 and mental disorders like schizophrenia and bipolar disorder, though statistical significance often attenuates after adjusting for genetic confounders (100,101). Methodological heterogeneity, across sample size, exposure assessment, study designs, and follow-up duration, hampers comparability and obstructs identification of vulnerable exposure windows, especially during perinatal and early childhood stages. Some studies on neurodevelopmental outcomes hint

at associations (autism, cognitive delays), but findings remain inconclusive (102,103). Furthermore, the combined impact of multiple environmental stressors, chemical, noise, light pollution, climate events, on mental health is underexplored. Psychological mechanisms triggered by extreme events (e.g., wildfires, heatwaves) such as PTSD and eco-anxiety are still fragmentarily studied (104,105).

Future research should prioritize:

1. Longitudinal clinical studies with individual-level exposure metrics and extended follow-up, targeting vulnerable populations (children, elderly, socially disadvantaged).
2. Meta-analyses rigorously assessing bias and heterogeneity (GRADE, sensitivity analyses, pre-registered protocols).
3. Multidisciplinary cohort integration of genetic, environmental, neuroimaging, and psychometric data to unravel causal mechanisms.
4. Intervention studies testing mental health outcomes alongside environmental remediation (e.g., traffic reduction, urban greening), as suggested by recent findings linking greener urban environments to decreased mental health medication use (106).
5. Standardization of methodology and transparent reporting (adherence to PRISMA, STROBE-MR, pre-registration).

However, the evidence discussed should be interpreted with caution due to inherent methodological limitations. Further high-quality, longitudinal, and multidisciplinary studies are required to clarify causal pathways and quantify the true mental health impact of environmental exposures.

Pollution: Antidotes and Remedies

Baker (107) emphasizes that environmental history plays a crucial role in reconstructing natural conditions preceding anthropogenic impact. Heim and Schwarzbauer (108) have contributed to this field through geochemical and geochronological investigations of sediment profiles, enabling the identification and temporal mapping of persistent organic and inorganic pollutants, such as heavy metals, polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), pesticides, pharmaceuticals, and personal care products. These studies allow for comprehensive source-tracking of contaminants and a better understanding of their ecological fate over time. Longitudinal environmental monitoring is essential to quantify pollutant accumulation trends from the early stages of industrialization to the present day. Chronostratigraphic and geochemical analyses of limnic, marine, and fluvial sediment archives serve not only as historical records but also as decision-making tools for environmental risk assessment and policy design. Mitigating the adverse health impacts of pollution necessitates coordinated actions at multiple levels, from individuals to communities to global governance systems. As early as 1967, the World Health Organization (WHO) emphasized the importance of multilevel collaboration for environmental protection (109). However, addressing modern environmental challenges requires more than reactive measures; it demands a proactive, strategic approach integrating sustainable development goals with technological innovation and resource optimization. The commonly perceived dichotomy between "nature" and "technology" is conceptually flawed. A regression to pre-industrial societal models is neither feasible nor beneficial, just as uncritical reliance on unchecked technological progress fails to ensure sustainability. Therefore, the reconciliation of environmental protection with socioeconomic advancement must be grounded in a rational and evidence-based framework. Given the inherent complexity of pollution-related health challenges, which involve intricate interactions among ecological, economic, and psychosocial domains, the biopsychosocial model (110) provides a robust theoretical foundation for addressing these multifactorial issues. In line with the WHO's definition of health as a state of complete physical, mental, and social well-being, we advocate for an integrated vision that acknowledges the interplay between neurobiological systems and environmental sustainability. Environmental stimuli play a central role in shaping neural plasticity, cognition, behavior, and social structure. Rapid anthropogenic changes, driven by population growth, technological expansion, and political

inertia, have introduced unprecedented environmental stressors. The increasing frequency, intensity, and unpredictability of such stimuli threaten the adaptive capacity of both individuals and ecosystems, raising the risk of global ecological and public health crises. As Haines and Kristie (111) point out, the cumulative effects of pollution and climate change are already impairing human health and are projected to worsen without urgent, coordinated intervention. Rising morbidity and mortality from heat-related illness, respiratory dysfunction, vector-borne disease, and food insecurity underscore the need for policies aimed at reducing air pollution and fostering social resilience. Combating misinformation and media manipulation is equally critical, as these amplify uncertainty and exacerbate psychological vulnerability. McMichael (112) further warns that globalization, while facilitating technological and economic growth, has exacerbated disparities in environmental exposures and health outcomes. Overcoming these interconnected challenges requires a paradigm shift in public health strategies, moving beyond reductionist models of causality toward systems-based, cooperative, and transdisciplinary solutions.

Conflict of Interest: Each author (RC, AP, and AM) declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

Authors Contribution: All authors (RC, AP, and AM) contributed equally to the conception, research, and writing of this manuscript.

Declaration on the use of AI: None.

Consent for Publication: All authors (RC, AP and AM) consent to the publication of this work.

References

1. Kasdagli MI, Katsouyanni K, de Hoogh K, Lagiou P, Samoli E. Investigating the association between long-term exposure to air pollution and greenness with mortality from neurological, cardio-metabolic and chronic obstructive pulmonary diseases in Greece. Environ Pollut. 2022 Jan 1;292(Pt B):118372. doi: 10.1016/j.envpol.2021.118372.
2. Brambilla F. Psychoneurendocrinology: A science of the past or a new pathway for the future? Eur J Pharmacol. 2000 Sep 29;405(1–3):341–9. doi: 10.1016/S0014-2999(00)00564-1.
3. Adler RH. Engel's biopsychosocial model is still relevant today. J Psychosom Res. 2009 Dec;67(6):607–11. doi: 10.1016/j.jpsychores.2009.08.008.
4. Vecoli C, Pulignani S, Andreassi MG. Genetic and epigenetic mechanisms linking air pollution and congenital heart disease. J Cardiovasc Dev Dis. 2016 Nov 29;3(4):32. doi: 10.3390/jcdd3040032.
5. Ventriglio A, Bellomo A, di Gioia I, et al. Environmental pollution and mental health: a narrative review of literature. CNS Spectr. 2021;26(1):51–61. doi: 10.1017/S1092852920001303.
6. Qiu X, Danesh-Yazdi M, Weisskopf M, et al. Association between air pollution and psychiatric symptoms in the Normative Aging Study. Environ Res Lett. 2022 Mar;17(3):034004. doi: 10.1088/1748-9326/ac47c5.
7. Wang JN, Wang Q, Li TT, Shi XM. [Association between air pollution and cognitive function in the elderly]. Zhonghua Yu Fang Yi Xue Za Zhi. 2017 Apr 6;51(4):364-368. Chinese. doi: 10.3760/cma.j.issn.0253-9624.2017.04.017.
8. Béjot Y, Reis J, Giroud M, Feigin V. A review of epidemiological research on stroke and dementia and exposure to air pollution. Int J Stroke. 2018 Oct;13(7):687–95. doi: 10.1177/1747493018772800.
9. Killin LO, Starr JM, Shiue IJ, Russ TC. Environmental risk factors for dementia: a systematic review. BMC Geriatr. 2016 Oct 12;16(1):175. doi: 10.1186/s12877-016-0342-y.
10. Maher BA. Airborne magnetite and iron-rich pollution nanoparticles: potential neurotoxicants and environmental risk factors for neurodegenerative disease, including Alzheimer's disease. J Alzheimers Dis. 2019;71(2):361–75. doi: 10.3233/JAD-190204.
11. Croze ML, Zimmer L. Ozone atmospheric pollution and Alzheimer's disease: from epidemiological facts to molecular mechanisms. J Alzheimers Dis. 2018;62(2):503–22. doi: 10.3233/JAD-170857.
12. Niu Q. Overview of the relationship between aluminum exposure and health of human being. Adv Exp Med Biol. 2018;1091:1–31. doi: 10.1007/978-981-13-1370-7_1.
13. Calderón-Garcidueñas L, Torres-Jardón R, Franco-Lira M, et al. Environmental nanoparticles, SARS-CoV-2 brain involvement, and potential acceleration of Alzheimer's and Parkinson's diseases in young urbanites exposed to air pollution. J Alzheimers Dis. 2020;78(2):479–503. doi: 10.3233/JAD-200891.
14. Jo S, Kim YJ, Park KW, et al. Association of NO_2 and other air pollution exposures with the risk of Parkinson disease. JAMA Neurol. 2021 Jul 1;78(7):800–8. doi: 10.1001/jamaneurol.2021.1335.
15. Lee JY, Kim H, Lee JS, Lee HJ, Kim Y, Park C, et al. Association between heavy metal exposure and Parkinson's

Disease. *Int J Environ Res Public Health*. 2022;19(23):15891. doi: 10.3390/ijerph192315891.
16. Raj K, Kaur P, Gupta GD, Singh S. Metals associated neurodegeneration in Parkinson's disease: Insight to physiological, pathological mechanisms and management. Neurosci Lett. 2021 May 14;753:135873. doi: 10.1016/j.neulet.2021.135873.
17. Pohanka M. Copper and copper nanoparticles toxicity and their impact on basic functions in the body. Bratisl Lek Listy. 2019;120(6):397-409. doi: 10.4149/BLL_2019_065.
18. Bouchard M, Mergler D, Baldwin M, Panisset M, Roels HA. Neuropsychiatric symptoms and past manganese exposure in a ferro-alloy plant. Neurotoxicology. 2007 Mar;28(2):290-7. doi: 10.1016/j.neuro.2006.08.002.
19. Rung AL, Gaston S, Oral E, et al. Depression, mental distress, and domestic conflict among Louisiana women exposed to the Deepwater Horizon Oil Spill in the WaTCH Study. Environ Health Perspect. 2016 Sep;124(9):1429-35. doi: 10.1289/EHP167.
20. Michalek JE, Barrett DH, Morris RD, Jackson WG Jr. Serum dioxin and psychological functioning in U.S. Air Force veterans of the Vietnam War. Mil Med. 2003 Feb;168(2):153-9. doi: 10.1093/milmed/168.2.153.
21. Rehner TA, Kolbo JR, Trump R, Smith C, Reid D. Depression among victims of south Mississippi's methyl parathion disaster. Health Soc Work. 2000 Feb;25(1):33-40. doi: 10.1093/hsw/25.1.33.
22. Buoli M, Grassi S, Caldiroli A, et al. Is there a link between air pollution and mental disorders? Environ Int. 2018 Sep;118:154-68. doi: 10.1016/j.envint.2018.05.044.
23. Liu Q, Wang W, Gu X, et al. Association between particulate matter air pollution and risk of depression and suicide: systematic review and meta-analysis. Environ Sci Pollut Res Int. 2021 Feb;28(8):9029-49. doi: 10.1007/s11356-021-12357-3.
24. Attademo L, Bernardini F, Garinella R, Compton MT. Environmental pollution and risk of psychotic disorders: A review of the science to date. Schizophr Res. 2017 Mar;181:55-9. doi: 10.1016/j.schres.2016.10.003.
25. Costa LG, Cole TB, Dao K, Chang YC, Garrick JM. Developmental impact of air pollution on brain function. Neurochem Int. 2019 Dec;131:104580. doi: 10.1016/j.neuint.2019.104580.
26. Vogt BA. Cingulate impairments in ADHD: Comorbidities, connections, and treatment. Handb Clin Neurol. 2019;166:297-314. doi: 10.1016/B978-0-444-64196-0.00016-9.
27. Thygesen M, Holst GJ, Hansen B, et al. Exposure to air pollution in early childhood and the association with Attention-Deficit Hyperactivity Disorder. Environ Res. 2020;183: 108930. doi: 10.1016/j.envres.2019.108930.
28. Vrijheid M, Casas M, Gascon M, Valvi D, Nieuwenhuijsen M. Environmental pollutants and child health – A review of recent concerns. Int J Hyg Environ Health. 2016;219(4-5):331–42. doi: 10.1016/j.ijheh.2016.05.001.
29. Flores-Pajot MC, Ofner M, Do MT, Lavigne E, Villeneuve PJ. Childhood autism spectrum disorders and exposure to nitrogen dioxide, and particulate matter air pollution: A review and meta-analysis. Environ Res. 2016 Nov;151:763-76. doi: 10.1016/j.envres.2016.07.030.
30. Fujiwara T, Morisaki N, Honda Y, Sampei M, Tani Y. Chemicals, Nutrition, and Autism Spectrum Disorder: A Mini-Review. Front Neurosci. 2016 Apr 20;10:174. doi: 10.3389/fnins.2016.00174.
31. Lyall K, Croen L, Daniels J, et al. The changing epidemiology of autism spectrum disorders. Annu Rev Public Health. 2017;38:81–102. doi: 10.1146/annurev-publhealth-031816-044318.
32. Cougle JR, Lee HJ, Horowitz JD, Wolitzky-Taylor KB, Telch MJ. An exploration of the relationship between mental pollution and OCD symptoms. J Behav Ther Exp Psychiatry. 2008;39(3):340–53. doi: 10.1016/j.jbtep.2007.08.007.
33. Braun JM. Early-life exposure to EDCs: role in childhood obesity and neurodevelopment. Nat Rev Endocrinol. 2017;13(3):161–73. doi: 10.1038/nrendo.2016.186.
34. Sanin LH, Carrasquilla G, Solomon KR, Cole DC, Marshall EJP. Regional differences in time to pregnancy among fertile women from five Colombian regions with different use of glyphosate. J Toxicol Environ Health A. 2009;72(15-16):949–60.doi:10.1080/15287390902929691.
35. Kanamori M, Beck KH, Carter-Pokras O. Association of school social networks' influence and mass media factors with cigarette smoking among asthmatic students. J Sch Health. 2015;85(3):155–62. doi: 10.1111/josh.12238.
36. Grunst AS, Grunst ML, Pinxten R, Eens M. Personality and plasticity in neophobia levels vary with anthropogenic disturbance but not toxic metal exposure in urban great tits: Urban disturbance, metal pollution and neophobia. Sci Total Environ. 2019 Mar 15;656:997-1009. doi: 10.1016/j.scitotenv.2018.11.383.
37. Shepard JD, Schulkin J, Myers DA. Chronically elevated corticosterone in the amygdala increases corticotropin releasing factor mRNA in the dorsolateral bed nucleus of stria terminalis following duress. Behav Brain Res. 2006;174(1):193–6. doi: 10.1016/j.bbr.2006.07.019.
38. Stryker S, Burke PJ. The past, present, and future of an identity theory. Soc Psychol Q. 2000;63(4):284–97. doi: 10.2307/2695840.
39. Tajfel H. Human Groups and Social Categories: Studies in Social Psychology. Cambridge: Cambridge University Press; 1981. doi: 10.1017/S0033291700041374.
40. Bonaiuto M, Ross CT, Breakwell GM. Teorie dell'identità e psicologia ambientale. In: Bonnes M, Bonaiuto M, Lee TR, editors. Pratica per la psicologia ambientale. Milano: Raffaello Cortina Editore; 2004. pp. 287–330.
41. Lepore SJ, Evans GW. Coping with multiple stressors in the environment. In: Zeidner M, Endler NS, editors. Handbook of Coping: Theory, Research, Applications. New York: Wiley; 1996. pp. 350–377.
42. Mainardi Peron E, Saporiti S. Stress ambientale: un approccio psicologico. Roma: La Nuova Italia Scientifica; 1995.

43. Seligman ME, Beagley G. Learned helplessness in the rat. J Comp Physiol Psychol. 1975;88(2):534–41. doi: 10.1037/h0076430.
44. Kilian J, Kitazawa M. The emerging risk of exposure to air pollution on cognitive decline and Alzheimer's disease – Evidence from epidemiological and animal studies. Biomed J. 2018;41(3):141–62. doi: 10.1016/j.bj.2018.06.001.
45. Iqubal A, Ahmed M, Ahmad S, Sahoo CR, Iqubal MK, Haque SE. Environmental neurotoxic pollutants: review. Environ Sci Pollut Res Int. 2020;27(33):41175–98. doi: 10.1007/s11356-020-10539-z.
46. Marchetti C, Baranowska-Bosiacka I, Gavazzo P. Multiple effects of copper on NMDA receptor currents. Brain Res. 2014;1542:20–31. doi: 10.1016/j.brainres.2013.10.029.
47. Li H, Kilgallen AB, Münzel T, et al. Influence of mental stress and environmental toxins on circadian clocks: Implication for redox regulation of the heart and cardioprotection. Br J Pharmacol. 2020;177(23):5393–412. doi: 10.1111/bph.14949.
48. Hahad O, Prochaska JH, Daiber A, Muenzel T. Environmental noise-induced effects on stress hormones, oxidative stress, and vascular dysfunction: Key factors in the relationship between cerebrocardiovascular and psychological disorders. Oxid Med Cell Longev. 2019;2019:4623109. doi: 10.1155/2019/4623109.
49. Karri V, Schuhmacher M, Kumar V. Heavy metals (Pb, Cd, As and MeHg) as risk factors for cognitive dysfunction: A general review of metal mixture mechanism in brain. Environ Toxicol Pharmacol. 2016 Dec;48:203-13. doi: 10.1016/j.etap.2016.09.016.
50. Boda E, Rigamonti AE, Bollati V. Understanding the effects of air pollution on neurogenesis and gliogenesis in the growing and adult brain. Curr Opin Pharmacol. 2020 Feb;50:61-6. doi: 10.1016/j.coph.2019.12.003.
51. Gładka A, Rymaszewska J, Zatoński T. Impact of air pollution on depression and suicide. Int J Occup Med Environ Health. 2018;31(6):711–21. doi: 10.13075/ijomeh.1896.01277.
52. Costa E Silva JA, Steffen RE. Urban environment and psychiatric disorders: a review of the neuroscience and biology. Metabolism. 2019;100S:153940. doi: 10.1016/j.metabol.2019.07.004.
53. Jones JL, Thompson SAJ, Loh P, et al. Human autoimmunity after lymphocyte depletion is caused by homeostatic T-cell proliferation. Proc Natl Acad Sci U S A. 2013;110(50):20200–5. doi: 10.1073/pnas.1313654110.
54. Wei Y, Cao XN, Tang XL, et al. Urban fine particulate matter (PM2.5) exposure destroys blood-testis barrier (BTB) integrity through excessive ROS-mediated autophagy. Toxicol Mech Methods. 2018 May;28(4):302-319. doi: 10.1080/15376516.2017.1410743.
55. Miguel V, Cui JY, Daimiel L, et al. The role of microRNAs in environmental risk factors, noise-induced hearing loss, and mental stress. Antioxid Redox Signal. 2018;28(9):773–96. doi: 10.1089/ars.2017.7175.
56. Delgado-Saborit JM, Guercio V, Gowers AM, Shaddick G, Fox NC, Love S. A critical review of the epidemiological evidence of effects of air pollution on dementia, cognitive function, and cognitive decline in adult population. Sci Total Environ. 2021;757:143734. doi: 10.1016/j.scitotenv.2020.143734.
57. Tobore TO. On the Etiopathogenesis and Pathophysiology of Alzheimer's Disease: A Comprehensive Theoretical Review. J Alzheimers Dis. 2019;68(2):417-437. doi: 10.3233/JAD-181052.
58. Lucchini R, Placidi D, Cagna G, et al. Manganese and developmental neurotoxicity. Adv Neurobiol. 2017;18:13–34. doi: 10.1007/978-3-319-60189-2_2.
59. Cannon JR, Greenamyre JT. The role of environmental exposures in neurodegeneration and neurodegenerative diseases. Toxicol Sci. 2011 Dec;124(2):225-50. doi: 10.1093/toxsci/kfr239.
60. Lanoix D, Plusquellec P. Adverse effects of pollution on mental health: the stress hypothesis. OA Evid Based Med. 2013;1(1):6.
61. Huang Q, Zhu W, Gao X, Liu X, Zhang Z, Xing B. Nanoparticles-mediated ion channels manipulation: From their membrane interactions to bioapplications. Adv Drug Deliv Rev. 2023; 195:114763. doi: 10.1016/j.addr.2023.114763.
62. Yokel RA. The toxicology of aluminum in the brain: a review. Neurotoxicology. 2000 Oct;21(5):813-28. PMID: 11130287.
63. Pinault L, Thomson EM, Christidis T, et al. The association between ambient air pollution concentrations and psychological distress. Health Rep. 2020;31(7):3–11. doi: 10.25318/82-003-x202000700001-eng.
64. Perera FP, Wang S, Rauh V, et al. Prenatal exposure to air pollution, maternal psychological distress, and child behavior. Pediatrics. 2013;132(5):e1284–94. doi: 10.1542/peds.2012-3844.
65. Padrón A, Galán I, Rodríguez-Artalejo F. Second-hand smoke exposure and psychological distress in adolescents. A population-based study. Tob Control. 2014;23(4):302–7. doi: 10.1136/tobaccocontrol-2012-050548.
66. Maybery D, Jones R, Dipnall JF, et al. A mixed-methods study of psychological distress following an environmental catastrophe: the case of the Hazelwood open-cut coalmine fire in Australia. Anxiety Stress Coping. 2020;33(2):216–30. doi: 10.1080/10615806.2019.1695523.
67. Beecher ME, Eggett D, Erekson D, et al. Sunshine on my shoulders: Weather, pollution, and emotional distress. J Affect Disord. 2016;205:234–8. doi: 10.1016/j.jad.2016.07.021.
68. Schade CP, Gupta R, Jha A, Wright N. Persistent Distress after Water Contamination. W V Med J. 2016 Sep-Oct;112(5):40-6. PMID: 29368478.
69. Ahmad D, Afzal M. Flood hazards, human displacement and food insecurity in rural riverine areas of Punjab, Pakistan: policy implications. Environ Sci Pollut Res Int. 2021;28(8):10125–39. doi: 10.1007/s11356-020-11430-7.

70. Kim Y, Tsutsumi A, Izutsu T, Kawamura N, Miyazaki T, Kikkawa T. Persistent distress after psychological exposure to the Nagasaki atomic bomb explosion. Br J Psychiatry. 2011;199(5):411–6. doi: 10.1192/bjp.bp.110.085472.
71. Aulsebrook AE, Jones TM, Mulder RA, Lesku JA. Impacts of artificial light at night on sleep: A review and prospectus. J Exp Zool A Ecol Integr Physiol. 2018;329(8-9):409-18. doi: 10.1002/jez.2189.
72. Cao B, Chen Y, McIntyre RS. Comprehensive review of the current literature on impact of ambient air pollution and sleep quality. Sleep Med. 2021 Mar;79:211-219. doi: 10.1016/j.sleep.2020.04.009.
73. Paul KC, Haan M, Mayeda ER, Ritz BR. Ambient air pollution, noise, and late-life cognitive decline and dementia risk. Annu Rev Public Health. 2019;40:203–20. doi: 10.1146/annurev-publhealth-040218-044058.
74. Thomson EM. Air pollution, stress, and allostatic load: linking systemic and central nervous system impacts. J Alzheimers Dis. 2019;69(3):597–614. doi: 10.3233/JAD-190015.
75. Niitsu T, Takaoka K, Uemura S, et al. The psychological impact of a dual disaster caused by earthquakes and radioactive contamination in Ichinoseki after the Great East Japan Earthquake. BMC Res Notes. 2014;7:307. doi: 10.1186/1756-0500-7-307.
76. Selinheimo S, Vuokko A, Hublin C, et al. Health-related quality of life among employees with persistent nonspecific indoor-air-associated health complaints. J Psychosom Res. 2019; 122:112-20. doi: 10.1016/j.jpsychores.2019.03.181.
77. Evans GW. The built environment and mental health. J Urban Health. 2003 Dec;80(4):536-55. doi: 10.1093/jurban/jtg063.
78. Reichert M, Braun U, Lautenbach S, et al. Studying the impact of built environments on human mental health in everyday life: methodological developments, state-of-the-art and technological frontiers. Curr Opin Psychol. 2020;32:158-64. doi: 10.1016/j.copsyc.2019.08.026.
79. Azuma K, Ikeda K, Kagi N, Yanagi U, Osawa H. Evaluating prevalence and risk factors of building-related symptoms among office workers: seasonal characteristics of symptoms and psychosocial and physical environmental factors. Environ Health Prev Med. 2017;22(1):38. doi: 10.1186/s12199-017-0645-4.
80. Triguero-Mas M, Gidlow CJ, Martínez D, et al. The effect of randomised exposure to different types of natural outdoor environments compared to exposure to an urban environment on people with indications of psychological distress in Catalonia. PLoS One. 2017;12(3):e0172200. doi: 10.1371/journal.pone.0172200.
81. Lin WW, Chen ZX, Kong ML, Xie YQ, Zeng XW. Air Pollution and Children's Health in Chinese. Adv Exp Med Biol. 2017;1017:153-180. doi: 10.1007/978-981-10-5657-4_7.
82. Loftus CT, Ni Y, Szpiro AA, et al. Exposure to ambient air pollution and early childhood behavior: A longitudinal cohort study. Environ Res. 2020;183:109075. doi: 10.1016/j.envres.2019.109075.
83. Coughtrey AE, Shafran R, Lee M, Rachman SJ. It's the feeling inside my head: a qualitative analysis of mental contamination in obsessive-compulsive disorder. Behav Cogn Psychother. 2012;40(2):163-73. doi: 10.1017/S1352465811000658.
84. Albrecht G, Sartore GM, Connor L, et al. Solastalgia: the distress caused by environmental change. Australas Psychiatry. 2007;15 Suppl 1:S95-8. doi: 10.1080/10398560701701288.
85. Wiesmüller GA, Hornberg C. Umweltmedizinische Syndrome [Environmental medical syndromes]. Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz. 2017;60(6):597-604. [German]. doi: 10.1007/s00103-017-2546-5.
86. Redlich CA, Sparer J, Cullen MR. Sick-building syndrome. Lancet. 1997;349(9057):1013-6. doi: 10.1016/S0140-6736(96)07220-0.
87. Katoh T. [Multiple chemical sensitivity (MCS): history, epidemiology and mechanism]. Nihon Eiseigaku Zasshi. 2018;73(1):1-8. [Japanese]. doi: 10.1265/jjh.73.1.
88. Carpenter DO, Belpomme D. Idiopathic environmental intolerance. Rev Environ Health. 2015;30(4):207. doi: 10.1515/reveh-2015-0065.
89. Dieudonné M. Electromagnetic hypersensitivity: a critical review of explanatory hypotheses. Environ Health. 2020;19(1):48. doi: 10.1186/s12940-020-00602-0.
90. Rollnik JD. Das chronische Müdigkeitssyndrom: ein kritischer Diskurs [Chronic Fatigue Syndrome: A Critical Review]. Fortschr Neurol Psychiatr. 2017;85(2):79-85. [German]. doi: 10.1055/s-0042-121259.
91. Lu Y, Zhang Z, Gao S, Yan H, Zhang L, Liu J. Association of occupational burnout and occupational exposure factors on psychological health among factory workers and miners: a propensity score analysis. Int Arch Occup Environ Health. 2021 Apr;94(3):441-450. doi: 10.1007/s00420-020-01587-6.
92. Bell IR, Baldwin CM, Schwartz GE. Illness from low levels of environmental chemicals: relevance to chronic fatigue syndrome and fibromyalgia. Am J Med. 1998;105(3A):74S-82S. doi: 10.1016/S0002-9343(98)00162-4.
93. Lacour M, Zunder T, Huber R, Sander A, Daschner F, Frank U. The pathogenetic significance of intestinal Candida colonization: a systematic review from an interdisciplinary and environmental medical point of view. Int J Hyg Environ Health. 2002;205(4):257-68. doi: 10.1078/1438-4639-00159.
94. Zhang X, Jiang Y, Li S, Wang H, Li H, Deng H, et al. Ambient air pollution and mental health: an umbrella review of systematic reviews and meta-analyses. Environ Res. 2024;240:116422. doi: 10.1016/j.envres.2023.116422.
95. Wang Y, Yang J, Li Y, Li X, Wu Y. The association between air pollution and psychiatric disorders: a systematic review and meta-analysis. J Affect Disord. 2024;345:157-168. doi: 10.1016/j.jad.2023.11.002.
96. Li W, Song Y, Fang F, Guo Y. The mental health effects of long-term exposure to air pollution: a systematic review and meta-analysis. Environ Pollut. 2024;328:122624. doi: 10.1016/j.envpol.2023.122624.

97. Marra G, Molteni F, Adorni F, Esposito F, Dalla Vecchia L, Gori D. Effects of environmental exposures on psychiatric hospitalizations: an umbrella review. Int J Environ Res Public Health. 2023;20(7):5356. doi: 10.3390/ijerph20075356.
98. WHO. Air pollution and mental health. Policy brief. Geneva: World Health Organization; 2024.
99. Chen Z, Hu R, Zhao J, Li H, Wang W. Mechanisms linking air pollution to mental disorders: insights from animal and human studies. Neurosci Biobehav Rev. 2023;153:105274. doi: 10.1016/j.neubiorev.2023.105274.
100. Wu X, Zhou Y, Tian Y, Wang Y, Zhang Y, Yang X, et al. Mendelian randomization analysis of air pollution and risk of mental disorders: evidence from large-scale biobank data. Transl Psychiatry. 2024;14(1):89. doi: 10.1038/s41398-024-03196-0.
101. Jansen PR, Wendt FR, Muniz-Terrera G, Hohmann C, Frei O, Sullivan PF, et al. Air pollution and mental health: causal inference from genetic data. Mol Psychiatry. 2023;28(6):2473-2481. doi: 10.1038/s41380-023-02104-4.
102. Lee YL, Chang YH, Wang YH. Early-life exposure to air pollution and neurodevelopment: a comprehensive review. Environ Int. 2023;179:108099. doi:10.1016/j.envint.2023.108099.
103. Fong KC, Gao X, Lin Y, Kouznetsova M. Environmental exposures, social vulnerability, and mental health in children: a review. Int J Environ Res Public Health. 2024;21(2):187. doi: 10.3390/ijerph21020187.
104. Clayton S, Karazsia BT. Eco-anxiety: a review of psychological responses to climate change. Curr Opin Psychol. 2024;56:101163. doi: 10.1016/j.copsyc.2023.101163.
105. American Psychological Association. Mental health and climate change: a policy statement. Washington (DC): APA; 2023.
106. Weimann H, Rylander L, Albin M, Persson RÅ, Engström K, Andersson E, et al. Urban greening and mental health: reduced medication use after residential relocation. Sci Total Environ. 2025;916:169489. doi: 10.1016/j.scitotenv.2024.169489.
107. Baker ARH. Geography and History: Bridging the Divide. Cambridge: Cambridge University Press; 2003.
108. Heim S, Schwarzbauer J. Pollution history revealed by sedimentary records: a review. Environ Chem Lett. 2013;11(3):255–70. doi: 10.1007/s10311-013-0409-3.
109. United Nations Environment Programme. Making Peace with Nature: A Scientific Blueprint to Tackle the Climate, Biodiversity and Pollution Emergencies. Nairobi: UNEP; 2021.
110. Engel GL. The clinical application of the biopsychosocial model. Am J Psychiatry. 1980;137(5):535-44. doi: 10.1176/ajp.137.5.535.
111. Haines A, Ebi K. The imperative for climate action to protect health. N Engl J Med. 2019;380(3):263-73. doi: 10.1056/NEJMra1807873.
112. McMichael AJ. Globalization, climate change, and human health. N Engl J Med. 2013;368(14):1335-43. doi: 10.1056/NEJMra1109341.

Correspondence:
Received: 7 March 2025
Accepted: 27 July 2025
Roberta Ciuffini
Dipartimento MeSVA Università dell'Aquila
Edificio D6 stanza 4B Via Vetoio 67010
Coppito (L'Aquila) - Italy
E-mail: roberta.ciuffini@univaq.it
ORCID: 0000-0002-3926-2249