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A Proposed Plastiglomerate Aerosol Framework for Assessing Pediatric Neurodevelopmental Risk Following Wildland-Urban Interface Fires

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ABSTRACT

Wildland-urban interface (WUI) fires are increasing in frequency, intensity, and geographic extent as human development expands into fire-prone landscapes. Unlike conventional wildfires that predominantly combust vegetation, WUI fires incinerate both biomass and synthetic materials, resulting in the emission of chemically complex byproducts, including micro- and nanoplastics (MNPs), heavy metals, polycyclic aromatic hydrocarbons, volatile organic compounds, and ultrafine particles. Contemporary health frameworks addressing wildfire impacts rely heavily on particulate matter mass (PM2.5) as the key metric of exposure, predicated on the assumption that particle mass sufficiently represents toxicological burden. Nonetheless, WUI fires produce chemically diverse aerosols. The biological effects of these particles may be influenced by composition, surface chemistry, and associated toxicants, rather than mass alone.

By integrating data from aerosol science, nanotoxicology, neurobiology, and environmental health, this review paper evaluates the potential neurodevelopmental implications of WUI-derived MNP exposure. This review proposes a conceptual framework termed "plastiglomerate aerosols" to describe hybrid plastic-toxicant exposure systems generated during WUI fires. This review posits that these hybrid particles should be evaluated as integrated exposure systems rather than as independent pollutants. Existing evidence from nanotoxicology and combustion aerosol research is examined to elucidate biologically plausible mechanisms through which these particles may affect the pediatric nervous system, encompassing oxidative stress, mitochondrial dysfunction, inflammation, epigenetic remodeling, and altered microRNA regulation. Potential routes of central nervous system exposure through the olfactory pathway are discussed alongside developmental factors that may increase pediatric susceptibility.

Several critical knowledge gaps are identified, including the absence of longitudinal pediatric cohorts, limited human evidence for nervous system translocation, challenges in nanoplastics exposure quantification, and difficulties translating experimental findings to real-world developmental outcomes. Existing approaches can quantify environmental exposure or detect downstream clinical abnormalities but provide limited insight into early neurophysiological dysfunction. The proposed multi-modal chemosensory and autonomic integration (MCAI) framework addresses this gap. Heart rate variability and chemosensory event-related potentials are

combined in MCAI as complementing indicators of autonomic control and the integrity of sensory pathways.
Collectively, this review argues that WUI-derived plastiglomerate aerosols may represent an emerging exposure class that is not adequately captured by conventional wildfire health frameworks. This review identifies biologically plausible pathways of pediatric neurodevelopmental vulnerability and proposes MCAI as a framework for investigating neurophysiological dysfunction following exposure.

Keywords: Wildland-Urban Interface Fires; Plastiglomerate Aerosols; Micro- and Nano-Plastics; Pediatric Neurodevelopment; Environmental Neurotoxicity; Neuroinflammation; Heart Rate Variability; Chemosensory Event-Related Potentials

1. INTRODUCTION

1.1 Expansion of WUI Fires

Wildland-urban interface (WUI) is the area where human civilization intersects with fire-prone natural vegetation. Because of population growth and urbanization of previously unhabitant areas, WUI regions have grown rapidly over the past several decades. WUI areas have increased by approximately 12.6% in the world between 1985 and 2020, and more than 43 million homes in the United States alone are now located in WUI regions.[1,2] As a result, wildfire activity has intensified. California alone has experienced an approximately fivefold increase in annual burned areas between 1972 and 2018.[3] These trends have increased the frequency and scale of human exposure to wildfire emissions altogether.

The public health implications of this expansion are significant, especially for children because pediatric populations experience greater pollutant exposure per unit body weight. Exposure to wildfire smoke has been linked to higher respiratory morbidity in children, including asthma exacerbations and hospitalizations.[4,5] Most health frameworks were developed around traditional biomass combustion. However, modern WUI fires involve the simultaneous burning of vegetation and synthetic urban materials, generating complex exposure environments. Thus, it is crucial to understand how these evolving combustion products affect public health.

1.2 Hidden Assumptions in Existing Wildfire Health Frameworks

Current wildfire health frameworks primarily assess exposure using bulk particulate matter with an aerodynamic diameter less than 2.5 μm (PM2.5). Numerous epidemiological studies have strongly associated PM2.5 concentrations with adverse respiratory, cardiovascular, and neurological outcomes.[6] Thus, this established particulate mass as a widely adopted indicator of health risks from wildfire.

The use of PM2.5 as a primary exposure metric, however, is built on the assumption that particulate mass is a reliable indicator of toxicological burden. In contrast to conventional wildfires that mostly burn vegetation, WUI fires produce diverse combinations of biomass, synthetic combustion products, heavy metals, volatile organic compounds (VOCs), and ultrafine particles (UFPs) with unique physicochemical properties. Evidence from aerosol toxicity research continues to grow, indicating that factors such as particle number concentration, surface area, chemical composition, and reactivity may shape biological responses independent of total particulate mass.

As a result, using PM2.5 as a primary exposure metric may underestimate exposure to emerging particle classes generated during WUI fires.[6] This limitation does not invalidate existing wildfire health frameworks but emphasizes the need to evaluate whether exposure models that incorporate particulate composition are necessary for assessing the health implications of WUI fire combustion products.

1.3 Objectives of This Review

Although recent studies have raised concerns regarding the increasingly complex chemical composition of WUI fire emissions, the potential implications of these emissions for pediatric neurodevelopment remain fragmented across aerosol science, environmental toxicology, neurobiology, and public health. This review aims to consolidate these disparate bodies of research into a coherent conceptual framework. This review (1) examines the formation and environmental behavior of micro- and nanoplastics (MNPs) derived from WUI fire combustion (2) synthesizes current evidence in describing potential pathways of central nervous system (CNS) exposure and neurotoxicity, (3) evaluates limitations in existing exposure assessment and diagnostic frameworks, and (4) proposes a neurophysiological framework for the early detection of neurodevelopmental dysfunction in children. By connecting these discrete research domains, this review seeks to identify critical gaps in knowledge and establish future research trajectories.

2. WUI Fire Aerosol Transformation

2.1 Transition from Biomass to Synthetic Combustion

WUI fires differ from traditional wildfires because they burn both natural vegetation and anthropogenic materials. As fires spread through residential and commercial areas, combustion extends beyond biomass to include synthetic textiles, electronic waste, insulation foams, and petroleum-derived construction materials.[7] As a result, WUI emissions contain a broader and more chemically complex mixture of pollutants than vegetation-only wildfire smoke. This shift in fuel composition introduces combustion products that are not typically represented in conventional wildfire exposure frameworks, which have historically been developed around particulate matter generated from traditional wildfires.[8]

2.2 Formation of MNPs

High-temperature combustion can rapidly degrade synthetic polymers through thermal breakdown and oxidation, producing MNPs.[9] Combustion and thermal weathering of household plastics can produce large quantities of MNP fragments through melting, oxidation, and mechanical fragmentation processes.[10] Due to their small size, MNPs exhibit physicochemical behaviors that differ substantially from large particulate debris. Biological interactions are often influenced by particle number concentration, surface area, and surface reactivity in addition to particle mass.[11] As a result, particles that contribute minimally to PM2.5 mass may still represent a biologically relevant exposure pathway.[12]

2.3 Atmospheric Transport and Long-Range Dispersion

During active WUI fires, MNPs may become entrained within smoke plumes and transported through the atmosphere. The thermal energy of WUI fires may produce powerful pyro-convective columns that inject MNPs into higher atmospheric layers. Compared with larger particles, MNPs exhibit reduced settling velocities and may remain airborne for extended periods, increasing the probability of inhalation exposure beyond the immediate perimeter of the WUI fire. As a result, populations that are located far from active WUI fire zones may still experience exposure to PM derived from WUI fires.[13]

2.4 Surface Weathering and Secondary Toxin Adsorption

Once airborne, MNPs undergo physical and chemical ageing through exposure to heat, ultraviolet radiation, and atmospheric oxidants. These processes may incorporate polar oxygen-containing functional groups onto MNP surfaces, enhancing surface reactivity of the particles. Experimental studies have shown that these chemically weathered polymer surfaces have an increased adsorption affinity for environmental contaminants such as

polycyclic aromatic hydrocarbons (PAHs), heavy metals, VOCs, and halogenated byproducts.[14] Within WUI smoke plumes, these chemical modifications may facilitate the association of MNPs with co-existing WUI fire toxins. As a result, the toxicological properties of MNPs may depend not only on the base polymer but also on the chemical species adhered to their surface.

3. Plastiglomerate Aerosols

3.1 Limitations of Existing Aerosol Classification Systems

Current environmental health frameworks classify airborne pollutants using terms such as PM2.5, UFPs, wildfire smoke, or MNPs. These classifications provide useful information about particle size, source, and composition but do not capture the hybrid nature of particles generated during WUI fires.[15] PM2.5 is a mass-based metric and does not differentiate biologically inert particles from chemically reactive particles carrying adsorbed toxicants.[15] Similarly, classifications such as MNPs describe the polymeric size of the particles but do not take into account the secondary pollutants that may be acquired through combustion and atmospheric weathering.[16] As a result, existing terminology may overlook the combined physicochemical behavior of plastic particles associated with toxicants derived from WUI fires.

3.2 Defining Plastiglomerate Aerosols

To address this research gap, this review proposes the term “plastiglomerate aerosols” to describe airborne particles composed of MNPs associated with adsorbed toxicants from WUI fires. The term is inspired from the geological concept of plastiglomerates, which are composite materials formed by the amalgamation of plastic with surrounding environmental materials. In the context of WUI fires, this phrase refers to a potentially distinct aerosol system rather than a geological structure. This proposed classification also emphasizes that toxicological behavior may be governed by the interactions between the plastic surface and co-emitted combustion products, not just the polymer mass or composition. Consequently, plastiglomerate aerosols may be considered a conceptual framework for studying hybrid plastic-toxin exposures that are not adequately represented by existing environmental classifications.

3.3 Emergent Properties of Hybrid Plastic-Toxin Systems

The significance of plastiglomerate aerosols lies in the interactions between plastic particles and toxicants derived from WUI fires. The high surface area to volume ratio of MNPs provides numerous sites for the adsorption of contaminants derived from combustion, including PAHs, heavy metals, and other pollutants.[17] These toxicants may be transported alongside the plastic particle through the atmosphere once they are combined.[17,18] This may influence their biological availability and environmental persistence.

Research in nanotoxicology has shown that particle size, surface chemistry, and associated molecular species influence cellular uptake and biological responses.[19] Therefore, the toxicological behavior of these hybrid particles may not be adequately understood by studying plastics or combustion pollutants in isolation.[17,20] Although the health implications of plastiglomerate aerosols remain incompletely characterized, their hybrid nature suggests that they should be evaluated as integrated exposure systems rather than as independent pollutants.

3.4 A Systems-Level Framework for WUI Neurotoxicity

Existing wildfire health frameworks treat PM2.5 mass, nanoplastic exposure, and neurodevelopmental outcomes as largely independent domains. This review proposes that these concepts are components of a single coupled exposure system in which particle composition, toxicant adsorption, biological transport, and neurophysiological dysfunction interact across multiple spatial and temporal scales.

The potential public health significance of plastiglomerate aerosols surpass those of wildfire toxicity. WUI regions are expanding globally, increasing the number of children exposed to mixed-combustion environments where synthetic materials and biomass burn concurrently. If particle composition influences biological responses independently of particle mass, exposure frameworks centered on PM2.5 may overlook biologically significant pollutants. Thus, determining whether plastiglomerate aerosols constitute a separate exposure category has ramifications for environmental monitoring, risk evaluation, and pediatric health protection in fire-prone communities. The rationale for considering plastiglomerate aerosols as a distinct exposure class lies in the possible interactions between particle surfaces and adsorbed pollutants that alter transport, bioavailability, and biological responses compared to each component in isolation.

4. The Olfactory Bypass and Pediatric Translocation

4.1 Olfactory Translocation Pathways

Inhaled MNPs may access the CNS through the olfactory pathway, a mechanism that has been proposed based on studies of engineered nanoparticles and UFPs.[21,22] Following inhalation, airborne MNPs can deposit in the upper nasal passage, where they meet the olfactory epithelium. The olfactory sensory neurons located within this tissue extend directly through the cribriform plate to the olfactory bulb.[23] This creates a potential anatomical route between the external environment and the CNS. Animal studies suggest that nanoparticles may undergo transport along these pathways and reach the olfactory bulb, bypassing systemic circulation and some of the protective functions of the blood-brain barrier (BBB).[22] The olfactory pathway may be particularly relevant in pediatric populations because children inhale a greater volume of air relative to their body weight compared to adults. This may result in higher exposure to airborne pollutants.[24] Developmental differences in respiratory anatomy and ventilation patterns in children may also influence particle deposition within the upper airway. As a result, if olfactory translocation occurs for airborne MNPs, children may experience greater exposure to particles that can interact with the CNS. However, direct evidence demonstrating olfactory transport in children is currently not available.

4.2 Biomolecular Corona Formation and BBB Interactions

Once MNPs enter biological fluids, their surfaces absorb proteins, lipids, and other biomolecules, forming a dense layer called biomolecular corona.[25] The composition of this corona can alter particle behavior by influencing cellular recognition, uptake, biodistribution, and immune responses. Cells may react to the adsorbed biological layer encircling the particle instead of reacting with the underlying polymer surface.

Experimental studies have shown that proteins associated with the corona, including apolipoproteins, can facilitate interactions between nanoparticles and receptor systems expressed on BBB endothelial cells.[26] These interactions may promote receptor-mediated transport processes that increase nanoparticle uptake across the BBB.[27] While several in vitro and animal studies suggest that nanoplastics are capable of crossing the BBB under certain experimental conditions, the extent to which biomolecular corona formation influences CNS accumulation following WUI fire exposures are not well understood yet.[28] As a result, biomolecular corona formation should be considered a potential mechanism that may influence CNS delivery of MNPs rather than a definitive pathway of BBB penetration.

4.3 Developmental Factors Influencing Pediatric Susceptibility

Physiological differences in children compared to adults may change how they are exposed to and react to airborne pollutants. Children have higher rates of inhalation relative to body mass, and during critical

stages of neurodevelopment, they may be exposed to greater environmental factors. Throughout infancy and youth, the immune system, transport systems, and neurovascular regulation all undergo ongoing developmental changes.

Although the BBB is present before birth, its molecular transport systems and neuroimmune interactions continue to mature throughout development. Age-related changes in BBB transport, particle clearance, and inflammatory responses may influence how environmental particles interact with the developing CNS.[29] Current evidence is not sufficient to conclude that MNPs cross the pediatric BBB more readily than the adult BBB.[30] However, developmental differences in neurovascular function may modify susceptibility to toxic particles during early life.

Since neurodevelopment depends on controlled processes such as neuronal migration, synapse formation, and circuit refinement, if these particles reach the developing CNS, their effects may differ from those observed in mature neural tissue. Disruption of these processes caused by environmental toxicants has been associated with adverse neurodevelopmental outcomes in multiple environmental health studies.[27] However, direct evidence linking exposure to MNPs generated from WUI fires to pediatric neurodevelopmental impairment is not yet established. This highlights an important gap in current research.

Additionally, environmental exposure is unlikely to be equal across pediatric populations. Individual exposure burdens may be influenced by several factors such as proximity to WUI regions, housing characteristics, indoor air filtration, time spent outdoors, school attendance during smoke events, and socioeconomic conditions. Therefore, variations in environmental exposure may define pediatric sensitivity in addition to biological susceptibility. Understanding how these factors interact will be important for identifying populations at greatest risk from WUI-derived plastiglomerate aerosol exposure.

5. Potential Neurodevelopmental Consequences of Plastiglomerate Aerosol Exposure

5.1 Mitochondrial Dysfunction and Oxidative Stress

Mitochondria play a pivotal role in cellular energy production, calcium regulation, and redox homeostasis, making them highly susceptible to cellular stress caused by nanoparticles. Exposure to nanoplastics has been associated with increased production of ROS, disruption of mitochondrial membrane potential, impaired ATP generation, and oxidative damage to lipids, proteins, and DNA.[25,26,31]

These observations are significant as neurodevelopment required metabolic energy to facilitate neuronal migration, proliferation, differentiation, axonal growth, and synapse formation. Excessive oxidative stress during these developmental stages is linked to altered cellular signaling, impaired neuronal maturation, and disruptions in normal neurodevelopmental trajectories.[32,35]

The potential significance of plastiglomerate aerosols arises from their hybrid composition. In addition to intrinsic biological activity of nanoplastics, adsorbed combustion-derived contaminants such as PAHs and heavy metals may contribute to additional oxidative stress through established redox-active mechanisms. Evidence from nanotoxicology also demonstrates that surface chemistry of such particles can influence biological responses independent of particle mass alone.

However, most evidence linking nanoplastic exposure to mitochondrial dysfunction comes from studies using engineered nanoplastics under controlled laboratory conditions instead of environmentally aged particles or plastiglomerate aerosols derived from WUI fires. As a result, while mitochondrial dysfunction presents a plausible mechanism, the magnitude of risk associated with WUI fire exposures remains uncertain.

5.2 Neuroinflammatory Signaling

Previous research has shown that exposure to nanoplastics can activate inflammatory responses, leading to increased production of pro-inflammatory cytokines, including interleukin-1b (IL-1b), interleukin-6 (IL-6), and tumor necrosis factor-a (TNF-a), while also modifying the activity of immune cells within neural tissues.[29,33,34,35] Comparable inflammatory responses have also been observed following exposure to UFPs and aerosols originating from combustion. This suggests that inflammation presents as a common biological response to inhaled particulate matter.

During development, inflammatory signaling must remain tightly regulated because microglia and astrocytes perform essential functions in synapse formation, synaptic pruning, neuron maturation, and neural circuit refinement.[35] Microglia eliminate excess synaptic connections during development, while astrocytes regulate synaptic organization and neurotransmitter homeostasis. Continuous activation of inflammatory pathways during critical developmental windows has been associated with altered new developmental trajectories and impaired neural circuit formation in both experimental and epidemiological studies.

The inflammatory potential of plastiglomerate aerosols may arises from their hybrid composition. As outlined in Section 3, contaminants derived from combustion such as PAHs, transition metals, and other ROS may adsorb onto nanoplastic surfaces, resulting in the formation of hybrid particles during atmospheric transport and environmental weathering. Research in particle toxicity indicates that inflammatory responses are affected by both particle load and physicochemical properties, including composition, surface chemistry, and adsorbed molecular species. Thus, plastiglomerate aerosols may induce biological effects different from those observed following exposure to isolated nanoplastics or conventional wildfire particulate matter.

Despite increasing concern over the health implications of wildfire emissions, the inflammatory reactions specifically linked to WUI-derived plastiglomerate aerosols remain uninvestigated. Hence, conclusions have been drawn from associated disciplines such as nanoplastic toxicology, UFP research, and combustion aerosol studies. Hence, neuroinflammation should be considered a biologically possible mechanism requiring further investigation rather than an established outcome of WUI fire exposure.

5.3 Epigenetic Remodeling

Environmental exposures can affect gene expression via epigenetic mechanisms without altering the fundamental DNA sequence. These processes encompass DNA methylation, histone modifications, chromatin remodeling, and non-coding RNA regulation. Collectively, they all coordinate cellular differentiation and several molecular systems that direct neurodevelopmental. In early development, regulation of gene expression is important for neuronal differentiation, sinus formation, and neural circuit maturation.

Past research indicates that environmental contaminants can interfere with these regulatory processes. Oxidative stress, neuroinflammation, and cellular stress signaling have been associated with environmentally induced epigenetic modifications. Exposure to airborne particulate matter has been linked to altered DNA methylation patterns in both adult and pediatric populations, raising concerns regarding the potential for environmental exposures to affect neurodevelopment via epigenetic pathways. Exposure to particulate air pollution has been associated with altered DNA methylation patterns in both adults and pediatric populations.[36,37] This suggests that inhaled environmental contaminants can influence molecular pathways involved in long-term cellular regulation.[36,37] Similarly, combustion-derived pollutants generated during wildfires have also been linked to

epigenetic changes associated with inflammatory and oxidative stress responses. An emerging body of evidence from nanoplastic toxicology also suggests that MNPs induce analogous molecular responses. These studies have indicated changes in DNA methylation, chromatin-related signaling pathways, and gene expression profiles following nanoplastic exposure, likely through mechanisms mediated by oxidative stress and inflammation. Because plastiglomerate aerosols combine nanoplastic particles with combustion-derived toxicants, they may influence epigenetic pathways through multiple conversion biological mechanisms.
However, most established evidence is derived from controlled experimental studies using engineered particles rather than environmentally weathered particles or WUI-derived plastiglomerate aerosols. Hence, epigenetic remodeling should be considered a biologically possible mechanism that requires further investigation rather than an established consequence of WUI fire exposure.

5.4 Alterations in MicroRNA Expression

MicroRNAs (miRNAs) are short non-coding RNA molecules that regulate post-transcriptional gene expression and are important for neuronal differentiation, synaptic plasticity, neuroimmune signaling, and cellular stress responses. Individual miRNAs can regulate multiple target genes simultaneously, so relatively small changes and miRNA expression may influence broad biological pathways involved in neurodevelopment and cellular homeostasis.
Environmental pollutants, including particulate air pollution and engineered nanoparticles, have been shown to alter miRNA expression profiles associated with oxidative stress, inflammation, and mitochondrial dysfunction.[39] Environmental studies have also reported differential expression of miRNAs involved in inflammatory signaling, apoptosis, and cellular stress responses following nanoplastic exposure.[38] This suggests a potential mechanism through which particle exposure may influence broader transcriptional networks beyond immediate toxicological effects.
Current evidence, however, is predominantly limited to in vitro and animal models, and the relevance of these findings to environmentally realistic human exposures remains ambiguous. No studies have directly investigated miRNA changes following exposure to WUI-derived plastiglomerate aerosols. Therefore, while altered miRNA expression represents a promising mechanistic pathway, its contribution to pediatric neurodevelopmental outcomes is yet to be determined.

5.5 Implications for Pediatric Neurodevelopment

The developing nervous system may be vulnerable to environmental stressors because neurodevelopment depends on tightly regulated metabolic, immune, and genetic signaling pathways. Mitochondrial dysfunction can impair cellular energy availability, neuroinflammation can alter developmental signaling networks, and epigenetic modifications may influence long-term patterns of gene expression.[39] These mechanisms provide biologically possible pathways through which plastiglomerate aerosols could affect pediatric neurodevelopment if exposure reaches the CNS.
Current evidence, however, is derived primarily from engineered nanoplastic studies, combustion toxicology research, and experimental exposure models.[40] Direct evidence linking WUI-derived plastiglomerate aerosol exposure to adverse neurodevelopmental outcomes in children is currently lacking. Hence, these mechanisms should be viewed as hypotheses that warrant further investigation rather than established causes of pediatric neurological disease.

6. Current Limitations in WUI Plastiglomerate Aerosol Neurotoxicity Research

6.1 Lack of Longitudinal Pediatric Cohorts

Despite growing concerns regarding the health impacts of wildfire smoke and MNP exposure, no longitudinal studies have specifically tracked neurodevelopmental outcomes in children exposed to aerosols from WUI plastiglomerates. Existing pediatric research primarily focuses on immediate respiratory health and healthcare utilization rather than long-term neurological development.[40,41]

Since neurodevelopment is a protracted process that extends from birth through adolescence, the effects of environmental exposure may not be detectable for several years. The current absence of studies following children through these critical developmental windows limits the ability to determine whether early-life exposure has lasting consequences on neurodevelopment. Consequently, future research should prioritize pediatric cohorts that integrate exposure measurements with clinical developmental assessments and biomarker-based monitoring.

6.2 Absence of Direct Human CNS Measurements

A major limitation of this field is the current inability to directly measure particle translocation, neuroinflammation, or neuronal dysfunction within living pediatric populations. Most evidence for CNS accumulation is derived from animal studies, in vitro models, or post-mortem human tissues. While recent reports have identified MNPs within human brain tissue samples, they do not establish the timing or source of exposure, nor do they prove a causal link to specific neurological outcomes.

Current hypotheses regarding CNS exposure thus rely largely on indirect evidence. Advancing this research requires the development of validated biomarkers, sophisticated neuroimaging techniques, and standardized analytical methods to determine if environmental exposures lead to significant CNS accumulation or dysfunction in humans.

6.3 Challenges in Exposure Quantification

Accurate qualification of plastiglomerate aerosol exposure remains a significant challenge. Standard air quality monitoring primarily relies on PM2.5 mass concentrations, which fails to capture critical information like particle count, surface area, chemical composition, shape, or adsorbed toxicants. These characteristics may influence biological health regardless of total particulate mass.

Furthermore, the lack of standardized protocols for detecting and measuring airborne nanoplastics makes it difficult to compare findings across different studies. This leads to high uncertainty in exposure estimates, which in turn complicates risk assessments.[42,43] Future monitoring frameworks must move beyond bulk mass to include multidimensional data on both particle characteristics and associated chemical contaminants.

6.4 Limitations in Animal-to-Human Translation

Current knowledge of how MNPs affect the nervous system is based on studies using rodents, zebrafish, and cell cultures. While these models are informative, they often fail to mimic human exposure environments, deposition patterns, or biological responses.

Many experiments use particle types and concentrations that do not reflect real-world WUI zones. Furthermore, inherent species differences, such as olfactory structures, BBB physiology, and developmental timelines, make it difficult to directly extrapolate findings to pediatric populations. Consequently, animal data should be viewed primarily as evidence of biological mechanisms rather than a precise predictor of human risk. Closing this gap requires human-focused research and exposure models that more closely resemble environmental reality.

6.5 Complexity of Mixed-Combustion Aerosols

Emissions from WUI are complex mixtures containing MNPs, PM2.5, UFPs, PAHs, heavy metals, and various combustion gases.

This makes it difficult to isolate the specific biological impact of plastiglomerate aerosols.

While most toxicological research examines individual pollutants in controlled laboratory conditions, actual WUI events expose people to multiple interacting contaminants simultaneously. These interactions can lead to additive or synergistic effects. This means that observed health responses cannot be pinned on plastic particles in isolation. This highlights the need for mixture-based models that treat these aerosols as integrated systems, which would better reflect the real-world conditions children face in these regions.

7. The Diagnostic Gap in Pediatric WUI Exposure Assessment

Collectively, current assessment approaches reveal a critical diagnostic gap in the evaluation of potential neurodevelopmental consequences associated with WUI-derived plastiglomerate aerosol exposure. Environmental monitoring can estimate external exposure but cannot determine whether it has resulted in biological effects in the developing nervous system. Behavioral screening questionnaires remain dependent on the emergence of observable symptoms, which may not appear until years after an environmental incident has occurred.[44,45] Neuroimaging techniques provide valuable information regarding brain structure and function but are often expensive, resource-intensive, and difficult to implement as population-level screening tools in young children.[46,47] Similarly, peripheral biomarkers may identify systemic biological responses, but they do not directly assess neural circuit function or early alterations in sensory processing pathways.[48] As a result, current approaches largely detect exposure, symptoms, or downstream biological responses rather than the earliest functional changes occurring within the developing brain. Hence, there remains a need for a diagnostic framework that can provide non-invasive and objective measures of neural function during the period between environmental exposure and the periods of clinically detectable neurodevelopmental abnormalities.

8. Addressing the Diagnostic Gap

8.1 Overview of Multi-Modal Chemosensory and Autonomic Integration (MCAI) Framework

To address the diagnostic gap identified in Section 7, this review proposes a conceptual framework termed Multi-modal Chemosensory and Autonomic Integration (MCAI). MCAI is not intended to diagnose plastic glomerate aerosol exposure directly. It is a hypothesis-generating neurophysiological framework designed to detect early functional abnormalities within biological systems that may be affected by environmental neurotoxins before overt neurodevelopmental symptoms emerge.

The framework integrates two established physiological measurements: Chemosensory Event-Related Potentials (CSERPs) and Heart Rate Variability (HRV). CSERPs assess signal transmission through the olfactory pathway by measuring electrophysiological responses to controlled olfactory stimulation, while HRV provides a non-invasive measure of autonomic nervous system regulation through analysis of beat-to-beat cardiac variability.[49] These modalities were selected because they involve two biological systems implicated throughout this review. The olfactory pathway represents a plausible route of CNS exposure for inhaled MNPs, whereas autonomic regulation is closely linked to neuroimmune signaling and inflammatory homeostasis.[50,51]

The central premise of MCAI is that neurophysiological dysfunction may precede structural abnormalities or clinically observable neurodevelopmental symptoms. Within this framework, CSERP data offers insights into the integrity of sensory pathways, while HRV measurements are used to assess autonomic regulation. MCAI should be viewed as a theoretical model rather than a validated diagnostic tool. To date, no research has applied this framework

to children exposed to WUI plastiglomerate aerosols. Therefore, its primary value currently lies in its potential to generate testable hypotheses for identifying early-stage neurological dysfunction caused by environmental factors.

Table 1. Conceptual Components of the MCAI Framework

Component	Physiological System	Measurement	Potential Relevance
CSERPs	Olfactory pathway	Electrophysiological responses to odor stimulation	Sensory pathway integrity
HRV	Autonomic Nervous System	Beat-to-beat heart rate variability	Autonomic Regulation
Combined MCAI	Integrated neurophysiology	CSERP and HRV assessment	Early neurophysiological dysfunction

8.2 Why Chemosensory Event-Related Potentials (CSERPs)?

The olfactory system is directly exposed to inhaled environmental contaminants and maintains anatomical connections between the nasal cavity and the CNS. As discussed in Section 4, the olfactory pathway has been proposed as a potential route through which inhaled nanoparticles may access neural tissues. As a result, physiological measurements that assess the integrity of olfactory signal transmission may provide a method for evaluating functional changes occurring along this pathway.[52]

CSERPs are electrophysiological responses generated following controlled olfactory stimulation and recorded using electroencephalography. Because CSERPs directly measure neural activity rather than subjective perception, they provide objective information regarding sensory processing within the olfactory system. Changes in CSERP latency, amplitude, or waveform morphology have previously been associated with alterations in olfactory function across multiple neurological disorders, suggesting their potential utility as indicators of neuron dysfunction.[53]

8.2.1 Extrapolating Conduction Velocity Decay from CSERP

As established in Section 4.1, inhaled MNPs may accumulate within the upper nasal cavity and potentially interact with tissues associated with the olfactory pathway. In established neurophysiological literature, the functional integrity of this pathway can be assessed using CSERPs by measuring the latency between odorant presentation and the appearance of characteristic cortical responses.[54]

Neurophysiological conduction velocity depends upon the coordinated function of axonal membranes, ion channels, synaptic transmission, and cellular energy metabolism. Consequently, processes that disrupt neural function, including oxidative stress, neuroinflammation, and mitochondrial dysfunction, may alter signal propagation and manifest as prolonged response latencies.[55] Because these mechanisms have been implicated in experimental nanoplastic toxicology studies, CSERP latency may provide an indirect measure of functional impairment occurring along the olfactory pathway.

This interpretation remains as a hypothesis. No studies have evaluated whether exposure to WUI-derived plastiglomerate aerosols produces measurable alterations in CSERP latency. Therefore, CSERP assessments should currently be viewed as a proposed research application rather than an established biomarker of environmental neurotoxicity.

8.2.2 Mapping Bioenergetic Limits Through Olfactory Habituation Kinetics

Beyond signal latency, repeated olfactory stimulation also gives insight into neural adaptations. Under normal physiological conditions, repeated presentation of an odorant leads to progressive attenuation of neural responses, known as habituation.

Habituation is an energy-dependent process involving synaptic transmission,

neurotransmitter recycling, ion transport, and network-level adaptation. Because mitochondrial dysfunction and oxidative stress can impair cellular energy production, changes in habituation kinetics may provide information regarding underlying bioenergetic capacity. Mitochondrial impairment and oxidative stress are closely linked to synaptic dysfunction as an early event in pathogenesis.[56] Reviews of oxidative stress and neurodegeneration emphasize that high brain metabolic demand makes neurons vulnerable to oxidative stress-mediated disruption of cellular homeostasis.[57]

Within the MCAI framework, repeated CSERP measurements could, therefore, be used to evaluate whether altered olfactory habitation patterns emerge following environmental exposure. Although the approach remains speculative, it provides a grounded hypothesis linking neurophysiological measurements to biological pathways discussed in Section 5.

8.3 Why Heart Rate Variability?

While olfactory measurements may reveal a proposed route of CNS exposure, they provide limited information regarding broader physiological consequences. Environmental toxicants induce biological responses involving inflammation, oxidative stress, autonomic dysregulation, and altered neuroimmune communication. As a result, assessing systemic physiological regulation requires a complementary approach.

HRV is a widely used measure of autonomic nervous system function that quantifies variation in successive cardiac cycles. Reduced HRV has been associated with chronic inflammation, oxidative stress, cardiovascular disease, environmental pollutant exposure, and numerous neurological disorders.[58] Because autonomic pathways participate in immune regulation and inflammatory homeostasis, HRV may give indirect insight into physiological processes extending beyond the olfactory system.

8.3.1 Mapping Neuroinflammation Through Glial-Vagal Desynchronization

Communication between the nervous system and immune systems is partially mediated through vagal pathways that are involved in the regulation of inflammatory responses. Experimental studies have demonstrated bidirectional interactions between inflammatory signaling, autonomic function, and vagal activity. This leads to the development of the neurovisceral integration framework and related models of autonomic regulation.

Neuroinflammatory processes discussed in Section 5 may therefore influence autonomic function indirectly through alterations in neuroimmune communication. Reduced vagal activity and diminished HRV have been observed in numerous inflammatory conditions, suggesting that autonomic dysfunction may reflect broader physiological dysregulation.[59]

In the context of MCAI, decreases in HRV are suggested as possible indicators of disrupted neuroimmune homeostasis. However, HRV is inherently non-specific and can be influenced by numerous physiological and environmental factors. As a result, HRV abnormalities alone cannot establish the presence of neuroinflammation and should be interpreted only within the broader context of complementary physiological measurements.

8.4 Why Combine Both Modalities?

CSERPs and HRV alone provide an insufficient assessment of the mechanisms proposed throughout this review. Each modality captures a distinct aspect of nervous system function and therefore proposes important limitations when interpreted independently.

CSERPs primarily assess sensory pathway integrity. Alterations in latency, amplitude, or habituation dynamics may reflect changes in olfactory receptor function, axonal conduction, synaptic transmission, or cortical processing. However, CSERPs provide limited information regarding

systemic physiological regulation or broader neuroimmune responses.

In contrast, HRV provides information regarding autonomic regulation and physiological homeostasis. HRV is sensitive to inflammation, stress, and autonomic dysfunction; however, it lacks anatomical specificity and cannot pinpoint the neural systems responsible for the observed abnormalities.

Combining both modalities may therefore improve biological specificity by integrating complementary physiological information. CSERPs evaluate a proposed route of CNS exposure, whereas HRV evaluates broader physiological regulation. The proposed benefit of multi-modal integration remains hypothetical and requires empirical validation. Future studies are necessary to determine whether combined measurements provide greater sensitivity, specificity, or predictive value than individual modalities.

8.5 Testable Predictions of the MCAI Framework

As a conceptual framework, MCAI derives scientific value from its ability to generate experimentally testable predictions.

1) Children residing in regions with higher estimated exposure to WUI-derived plastiglomerate aerosols should demonstrate longer CSERP latencies and reduced HRV relative to children from lower-exposure regions.[60]
2) Neurophysiological abnormalities identified through MCAI should emerge before clinically detectable neurodevelopmental abnormalities appear.[61]
3) Children exhibiting abnormalities in both CSERP and HRV measurements should demonstrate a greater likelihood of adverse neurodevelopmental outcomes than children exhibiting abnormalities and only one modality.[61]
4) Abnormalities derived from MCAI correlate with established biomarkers of inflammation and oxidative stress, providing support for the biological pathways discussed in Section 5.
5) If exposure is reduced or eliminated, at least partial normalization of MCAI measurements may occur over time, indicating that some physiological alterations are reversible during development.[62,63]

These predictions established a pathway through which MCAI can be experimentally evaluated. This ability to generate testable hypotheses represents a fundamental requirement of a scientifically useful framework and provides a foundation for future longitudinal studies of pediatric environmental neurotoxicity.

9. Future Research Priorities and Translational Roadmap

9.1 Validation of Plastiglomerate Aerosols as a Distinct Exposure Class

Before the health implications of plastiglomerate aerosols can be fully understood, future studies must first determine whether these particles represent a distinct exposure type. Controlled combustion experiments and environmental sampling during active WUI fires are needed to characterize their composition and associated toxicants. Toxicological studies should then compare hybrid plastiglomerate aerosols with isolated nanoplastics and conventional combustion aerosols to determine whether they produce different biological responses. Establishing whether these particles exhibit unique physicochemical or toxicological properties will allow researchers to clarify whether plastiglomerate aerosols should be considered a separate exposure category within environmental health research.

9.2 Standardized Nanoplastic Exposure Metrics

A major limitation of current WUI plastiglomerate aerosol research is the lack of standardized exposure metrics. Studies report MNP concentrations using different measures, including particle number, mass, and surface area, making cross-study comparison difficult.[64] Future exposure frameworks should move beyond PM2.5

mass alone by incorporating particle number concentration, size distribution, polymer composition, surface chemistry, and adsorbed contaminant information.[65] Standardized analytical workflows that distinguish particles derived from plastic from conventional combustion aerosols are required to improve comparability across studies and support quantitative risk assessment. Such approaches would also allow researchers to evaluate whether particle composition is a stronger predictor of biological response than particulate mass alone.

9.3 Longitudinal WUI Birth Cohorts

The absence of prospective pediatric cohorts remains a critical barrier to understanding the long-term developmental consequences of WUI fire exposure.[66] Future studies should recruit pregnant individuals residing in WUI regions and follow offspring through adolescence while integrating environmental monitoring, biological sampling, neurodevelopmental assessments, and longitudinal neurophysiological measurements, including CSERPs and HRV. Such cohorts could help identify vulnerable developmental windows and characterize exposure-response relationships across childhood. Such data would strengthen causal inference between WUI fire exposure and later neurodevelopmental outcomes.

9.4 Clinical Validation of MCAI in High-Risk Communities

The proposed MCAI framework requires future clinical validation. Initial studies should establish age-specific normative ranges for CSERP and HRV measurements in healthy pediatric populations. Subsequent studies should compare children residing in high-exposure WUI regions with matched controls while evaluating the sensitivity, specificity, and predictive value of MCAI-derived abnormalities for later neurodevelopmental outcomes. Longitudinal follow-up would be needed to determine whether combined CSERP and HRV measurements predict later neurodevelopmental outcomes more accurately than either component alone. Successful validation could establish MCAI as a non-invasive framework for the early identification of children at elevated risk of adverse neurodevelopmental outcomes.

9.5 Integration of Neurophysiology with Environmental Monitoring

Current environmental monitoring systems quantify pollutant concentrations but rarely assess concurrent biological responses. Future studies should integrate air quality monitoring with neurophysiological measurements, including CSERPs, HRV, and biomarkers of inflammation and oxidative stress, within the same study populations. Collecting environmental and physiological data simultaneously would allow researchers to establish exposure-response relationships across multiple biological scales and determine whether neurophysiological abnormalities emerge before detectable neurodevelopmental outcomes. Such integrated approaches may enable earlier identification of neurodevelopmental risk than environmental monitoring alone.

9.6 Alternative Explanations and Falsification Criteria

The plastiglomerate aerosol framework is intended as a testable conceptual model rather than a definitive explanation of WUI-derived neurodevelopmental risk. Observed biological effects may result from conventional wildfire pollutants, including PM2.5, PAHs, heavy metals, and other combustion-derived contaminants, rather than from plastiglomerate aerosols specifically. Future studies must therefore determine whether hybrid plastic-toxicant particles produce biological responses that differ from those of established wildfire exposure classes.

The framework should also be regarded as falsifiable. Evidence showing that WUI-derived MNPs occur at negligible concentrations, do not acquire substantial toxicant burdens, or exhibit toxicological

properties indistinguishable from conventional wildfire aerosols would weaken the rationale for considering plastiglomerate aerosols a distinct exposure class. Similarly, failure of MCAI-derived abnormalities to predict later neurodevelopmental outcomes would challenge the use of the MCAI framework.

Table 2. Summary of Major Knowledge Gaps Limiting Assessment of Pediatric Neurodevelopmental Risk Following WUI Fire Exposure

Knowledge Gap	Current Limitation	Why It Matters	Future Research Need
Exposure characterization	Limited measurement of MNPs during WUI fires	Unknown exposure burden	Standardized environmental monitoring
CNS translocation	Primarily animal and in vitro evidence	Human relevance uncertain	Human exposure studies
Pediatric susceptibility	Limited age-specific data	Vulnerable developmental windows unclear	Pediatric cohort studies
Neurodevelopmental outcomes	No longitudinal cohorts	Cannot establish causality	Long-term follow-up studies
Plastiglomerate aerosols	No direct toxicological studies	Distinct exposure class unvalidated	Controlled toxicology experiments
Early detection	Lack of functional biomarkers	Delayed identification of dysfunction	Validation of MCAI

10. CONCLUSION

WUI fires represent an increasingly important public health challenge as expanding human development exposes larger populations to complex combustion-derived aerosols. WUI fires, unlike traditional wildfires, produce emissions from both biomass and synthetic materials, resulting in aerosol mixtures that conventional frameworks do not fully capture. Emerging evidence from aerosol science, nanotoxicology, and environmental health suggests that combustion-generated MNPs may acquire toxicants during atmospheric transport, potentially forming hybrid exposure systems with biological properties distinct from either component alone. To describe this phenomenon, this review proposes the conceptual term "plastiglomerate aerosols."

Current evidence indicates several biologically plausible pathways through which plastiglomerate aerosols could influence the developing nervous system, including oxidative stress, mitochondrial dysfunction, neuroinflammation, epigenetic remodeling, and altered miRNA signaling. However, direct evidence linking WUI-derived plastiglomerate aerosol exposure to adverse pediatric neurodevelopment outcomes remains limited. Major barriers include the absence of longitudinal pediatric cohorts, difficulty quantifying airborne nanoplastic exposure, limited human evidence for central nervous system translocation, challenges distinguishing synthetic particle exposure from conventional wildfire smoke, and uncertainty regarding the translation of experimental findings to real-world pediatric development. This review further identifies a critical diagnostic gap between environmental exposure assessment and the detection of early neurophysiological dysfunction. Existing approaches largely measure pollutant concentrations, structural abnormalities, or clinically apparent symptoms, while little is known about the earliest functional changes that may occur during neurodevelopment. To address this limitation, the MCAI framework is proposed as a research model that integrates CSERPs and HRV to evaluate sensory pathway integrity and autonomic regulation. MCAI should not be interpreted as a validated diagnostic tool but as a framework for generating experimentally testable hypotheses regarding environmentally induced neurological dysfunction.

Ultimately, the greatest contribution of this review is not the assertion of already established environmental effects but the identification of critical assumptions and knowledge gaps that may limit current understanding of WUI fire health impacts. Addressing these gaps will require interdisciplinary collaboration across aerosol science, environmental monitoring, toxicology, neuroscience, pediatrics, and public health. As WUI Fires continue to increase in frequency and intensity worldwide, developing exposure metrics and early detection strategies capable of evaluating emerging pollutants such as plastiglomerate aerosols may become increasingly important for protecting future generations from environmental neurodevelopmental risk. If validated, the concepts proposed in this review could inform future approaches to wildfire exposure assessment by incorporating particle composition alongside particle mass and support the development of earlier methods by identifying children at risk of adverse neurodevelopmental outcomes following WUI fire exposure.

Appendix A: Glossary of Terms and Abbreviations

Term/Abbreviation	Definition
BBB	Blood-Brain Barrier; a highly selective, semipermeable membrane that separates circulating blood from the brain's extracellular fluid. It shields the central nervous system from pathogens and toxins while regulating the essential transport of nutrients and gases.
CNS	Central Nervous System; the body's primary processing center consisting of the brain and spinal cord. It integrates sensory input, coordinates motor output, and regulates higher cognitive processing.
CSERP	Chemosensory Event-Related Potential; an electrophysiological response elicited by controlled olfactory stimulation and recorded using electroencephalography.
HRV	Heart Rate Variability; variation in the time interval between consecutive heartbeats used as a measure of autonomic nervous system regulation.
MCAI	Multi-Modal Chemosensory and Autonomic Integration; a conceptual neurophysiological framework proposed in this review integrating CSERP and HRV measurements.
MNPs	Micro- and Nanoplastics; plastic particles spanning the micrometer and nanometer size ranges.
Neuroinflammation	Activation of immune and inflammatory pathways within the nervous system, involving microglia, astrocytes, and inflammatory cytokines.
Oxidative Stress	Cellular imbalance between reactive oxygen species production and antioxidant defense mechanisms, resulting in molecular damage.
PAHs	Polycyclic Aromatic Hydrocarbons; organic compounds derived by combustion. They are frequently associated with environmental and wildfire pollution.
Plastiglomerate Aerosols	A conceptual aerosol class proposed in this review describing airborne hybrid particles composed of MNPs associated with toxicants derived from combustion that are acquired during wildland-urban interface (WUI) fires and subsequent atmospheric weathering.
PM2.5	Particulate matter with an aerodynamic diameter ≤ 2.5 μm. Most commonly used metric for assessing ambient particulate air pollution exposure.
UFPs	Ultrafine Particles; airborne particulate matter with a diameter of less than $0.1\mu m$. They are significantly smaller than regulated pollutants like PM2.5, make up very little physical mass, and are measured by particle count rather than mass.
VOCs	Volatile Oxidized Compounds; airborne carbon-containing chemicals bound with oxygen, such as aldehydes, ketones, and alcohols.
WUI	Wildland-Urban Interface; the region where human development directly intersects with fire-prone natural vegetation.
WUI Fires	Fires occurring within the WUI that involve simultaneous combustion of biomass and anthropogenic materials.

Appendix B: Critical Assumptions and Uncertainties in the Plastiglomerate Aerosol Framework

Assumption	Supporting Evidence	Quantitative Uncertainty	Critical Measurements Needed for Validation
WUI fires generate airborne MNPs in biologically relevant quantities	Combustion and degradation studies demonstrate MNP formation during thermal degradation of plastics	Ambient concentrations during active WUI fires remain largely unknown	Airborne MNP number concentration, size distribution, and polymer composition during active WUI fire events
MNPs undergo atmospheric transport and weathering	Atmospheric microplastic studies demonstrate regional and long-distance transport	Maximum transport distances and persistence under wildfire plume conditions remain uncertain	Transport distance, atmospheric residence time, weathering rates, and physicochemical changes under wildfire plume conditions
Combustion-derived toxicants adsorb onto MNP surfaces	Laboratory adsorption studies show adsorption of PAHs and metals to plastic surfaces	Adsorption kinetics under realistic WUI fire conditions are not well characterized	Adsorption kinetics, toxicant loading capacity, desorption behavior, and particle-bound contaminant composition under realistic WUI conditions
Plastiglomerate aerosols behave differently from isolated particles or toxicants	Nanotoxicology studies show particle-contaminant interactions can impact biological responses	Magnitude of the effect to biological responses is unknown	Comparative toxicological responses between plastiglomerate aerosols, isolated MNPs, and conventional combustion aerosols using standardized exposure models
Inhaled MNPs may access the CNS through olfactory pathways	Animal studies demonstrate nanoparticle translocation along olfactory pathways	Human pediatric transport rates remain unknown	Rates of olfactory translocation, CNS deposition, regional brain distribution, and particle clearance following inhalation exposure
Oxidative stress, neuroinflammation, epigenetic remodeling, and miRNA dysregulation may contribute to neurotoxicity	Toxicological studies show that toxicity is dependent on composition	Relative contribution of composition versus mass remains unclear	Biomarkers of oxidative stress, inflammatory signaling, epigenetic modifications, miRNA expression profiles, and their relationship to neurodevelopmental outcomes
Neurophysiological dysfunction may precede clinically detectable symptoms	Observed in several neurodevelopmental and neurodegenerative conditions	Timing between dysfunction and symptom emergence is unknown	Longitudinal CSERP and HRV measurements alongside neurodevelopmental assessments to determine whether functional abnormalities emerge before clinical manifestations.
MCAI may detect early dysfunction before symptom onset	CSERP and HRV are established physiological measures	Sensitivity, specificity, and predictive value remain untested	Age-specific normative CSERP and HRV ranges, sensitivity, specificity, predictive value, and longitudinal associations with later neurodevelopmental outcomes.

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