Brief Report
Volume 7:02, 2024

Journal of Clinical Neurology and Neurosurgery

ISSN: 2684-6012 Open Access

Nanoplastics and their Association with ALS-related Neurodegeneration

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Introduction

Nanoplastics, minuscule fragments of plastic measuring less than 100 nanometers, have emerged as a profound environmental concern due to their ubiquity and potential adverse effects on various ecosystems and organisms, including humans. In recent years, research has increasingly focused on understanding the impacts of nanoplastics on human health, particularly their association with neurodegenerative diseases such as Amyotrophic Lateral Sclerosis (ALS). This comprehensive exploration aims to shed light on the intricate relationship between nanoplastics and ALS-related neurodegeneration, encompassing an introduction to nanoplastics, their routes of exposure and accumulation, mechanisms of toxicity and the emerging evidence linking nanoplastics to ALS pathogenesis [1]. Nanoplastics originate from the fragmentation and degradation of larger plastic debris, including microplastics, driven by environmental factors such as UV radiation, mechanical abrasion and biological processes. Their small size confers unique physicochemical properties, facilitating their dispersal in various environmental compartments, from oceans and freshwater bodies to soil and air. Despite their widespread distribution, nanoplastic research is still in its nascent stages and the full extent of their environmental presence and impact remains incompletely understood [2].

Description

Understanding the pathways through which nanoplastics enter and accumulate within living organisms is crucial for elucidating their potential health effects. Nanoplastics can enter the food chain at multiple points, from ingestion by aquatic organisms like plankton and fish to uptake by terrestrial plants. Once ingested, nanoplastics may translocate across biological barriers, including the gastrointestinal tract, blood-brain barrier and placental barrier, leading to systemic distribution and accumulation in various tissues and organs. Moreover, nanoplastics possess a high surface area-to-volume ratio, enhancing their capacity to adsorb and transport environmental pollutants and toxicants, which may exacerbate their adverse effects on biological systems [3]. The mechanisms underlying nanoplastic toxicity are multifaceted and involve physical, chemical and biological processes. Physically, nanoplastics can induce cellular damage through mechanical stress, membrane disruption and interference with cellular structures and organelles. Chemically, nanoplastics may release leachable additives and adsorbed contaminants, generating Reactive Oxygen Species (ROS) and eliciting oxidative stress, inflammation and genotoxicity. Furthermore, nanoplastics can interact with biomolecules, including proteins, lipids and nucleic acids, altering their structure, function and intracellular signaling pathways. The link between nanoplastics and ALS-related neurodegeneration has garnered increasing attention in recent years, fueled by emerging epidemiological, experimental and clinical evidence. ALS, also known as Lou Gehrig's disease, is a progressive neurodegenerative disorder characterized by the selective degeneration of motor neurons in the brain and spinal cord, leading to muscle weakness, paralysis and eventual respiratory failure [4].

While the exact etiology of ALS remains elusive, accumulating research suggests a multifactorial interplay of genetic predisposition, environmental factors and protein misfolding and aggregation. Several lines of evidence support the notion that nanoplastics may contribute to ALS pathogenesis through multiple mechanisms. First, nanoplastics have been shown to induce neuroinflammation, oxidative stress and mitochondrial dysfunction in various in vitro and in vivo models, processes implicated in ALS pathophysiology. Second, nanoplastics can cross the blood-brain barrier and accumulate in the central nervous system, where they may directly interact with neurons and glial cells, disrupting cellular homeostasis and neuronal function. Third, nanoplastics have the capacity to adsorb and transport neurotoxicants, such as heavy metals and organic pollutants, which have been implicated in ALS susceptibility and progression. Despite the growing body of evidence implicating nanoplastics in ALS-related neurodegeneration, several knowledge gaps and challenges remain. Further research is needed to elucidate the specific mechanisms by which nanoplastics contribute to ALS pathophysiology, including their interactions with key cellular pathways and protein aggregates associated with the disease. Additionally, epidemiological studies are warranted to investigate the potential association between nanoplastic exposure and ALS incidence and progression in human populations [5].

Conclusion

In conclusion, nanoplastics represent a pervasive environmental contaminant with profound implications for human health, including their potential association with ALS-related neurodegeneration. Understanding the pathways of nanoplastic exposure and accumulation, as well as their mechanisms of toxicity, is essential for developing effective mitigation strategies and interventions to protect public health and mitigate the burden of neurodegenerative diseases. Furthermore, interdisciplinary collaboration between scientists, policymakers and stakeholders is critical for advancing our understanding of nanoplastic pollution and its implications for human health and well-being in the face of this global environmental challenge.

Acknowledgement

None.

Conflict of Interest

None.

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Received: 01 April, 2024, Manuscript No. jcnn-24-134961; **Editor Assigned:** 03 April, 2024, PreQC No. P-134961; **Reviewed:** 15 April, 2024, QC No. Q-134961; **Revised:** 20 April 2024, Manuscript No. R-134961; **Published:** 27 April, 2024, DOI: 10.37421/2684-6012.2024.7.221

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How to cite this article: Tokathaknbe, Sigria. "Nanoplastics and their Association with ALS-related Neurodegeneration." *J Clin Neurol Neurosurg* 7 (2024): 221.