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Neurodegeneration and Environmental Nanoparticle Pollutants

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Abstract

Environmental nanoparticle pollutants have emerged as a significant environmental and public health concern due to their potential role in neurodegenerative diseases. Environmental nanoparticles, including ultrafine particulate matter, metal nanoparticles, and nanoplastics, originate from sources such as air pollution, industrial emissions, vehicular exhaust, and plastic waste. Due to their small size and high surface reactivity, these nanoparticles can enter the human body through inhalation, ingestion, or dermal exposure and translocate to the brain through the blood–brain barrier, olfactory nerve pathway, and gut–brain axis. Once in the brain, nanoparticles can induce oxidative stress, neuroinflammation, mitochondrial dysfunction, protein aggregation, and neuronal cell death, which are key mechanisms involved in neurodegeneration. Several experimental and epidemiological studies have reported an association between environmental nanoparticle exposure and neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. This review aims to examine the sources, exposure pathways, mechanisms of neurotoxicity, and the role of environmental nanoparticle pollutants in neurodegeneration. The study also identifies current research gaps and highlights the need for further research on long-term exposure, dose–response relationships, and combined nanoparticle toxicity. Understanding the role of environmental nanoparticles in neurodegeneration is important for developing preventive strategies, environmental regulations, and public health policies.

Keywords: *Environmental Nanoparticles; Neurodegeneration; Air Pollution; Nanotoxicology; Oxidative Stress; Neuroinflammation.*

1. Introduction

Neurodegenerative diseases are a group of progressive neurological disorders characterized by the gradual loss of structure and function of neurons, ultimately leading to cognitive and motor dysfunction. Among the most common neurodegenerative diseases are Alzheimer's disease, Parkinson's disease, amyotrophic lateral sclerosis (ALS), and Huntington's disease, which represent a growing global health burden due to aging populations and increasing environmental risk factors (Lane et al., 2025; Lee et al., 2023). While genetic factors contribute to the development of these disorders, increasing evidence suggests that environmental factors play a significant role in the onset and progression of neurodegeneration **(Kulcsárová et al., 2025).**

In recent years, environmental nanoparticle pollutants have emerged as an important environmental risk factor for neurological disorders. Environmental nanoparticles are ultrafine particles with a size less than 100 nm, originating from sources such as air pollution, industrial emissions, vehicle exhaust, metal wear particles, and nanoplastics **(Calderón-Garcidueñas & Ayala, 2022; Maher, 2019).** Due to their extremely small size and large surface area, these nanoparticles can easily enter the human body through inhalation, ingestion, or dermal exposure and may translocate to the brain through the blood–brain barrier, olfactory nerve pathway, or gut–brain axis **(Teleanu et al., 2018; Stone et al., 2017).**

Several studies have demonstrated that environmental nanoparticles can induce neurotoxicity through multiple biological mechanisms, including oxidative stress, neuroinflammation, mitochondrial dysfunction, DNA damage, and protein aggregation **(Hermosillo-Abundis et al., 2024; Kim et al., 2020).** These mechanisms are known to play a critical role in the pathogenesis of neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. For example, exposure to air pollution nanoparticles has been associated with increased amyloid-β deposition, α-synuclein accumulation, and neuroinflammatory responses in the brain, which are hallmark features of neurodegeneration **(Costa et al., 2020; Jankowska-Kieltyka et al., 2021).**

Furthermore, recent experimental studies have shown that metal-based nanoparticles, such as iron oxide and aluminum oxide nanoparticles, can induce neuronal cell damage through reactive oxygen species (ROS) production and inflammatory signaling pathways, leading to neuronal apoptosis and brain tissue damage **(Agarwal et al., 2024; Xue et al., 2024).** Long-term exposure to ultrafine particulate matter has also been linked to blood–brain barrier disruption and increased risk of neurodegenerative diseases **(Cory-Slechta et al., 2023; Kwon et al., 2025).**

Despite growing evidence linking environmental nanoparticle exposure to neurodegeneration, the exact mechanisms, exposure levels, and long-term effects of these particles on the human nervous system remain incompletely understood. Therefore, there is a need to comprehensively review and analyze the relationship between environmental nanoparticle pollutants and neurodegenerative diseases, focusing on exposure pathways, mechanisms of neurotoxicity, and their role in the development and progression of neurodegeneration.

This study aims to review and synthesize current scientific evidence on environmental nanoparticle pollutants and their contribution to neurodegenerative diseases, with particular emphasis on mechanisms such as oxidative stress, neuroinflammation, and blood–brain barrier disruption.

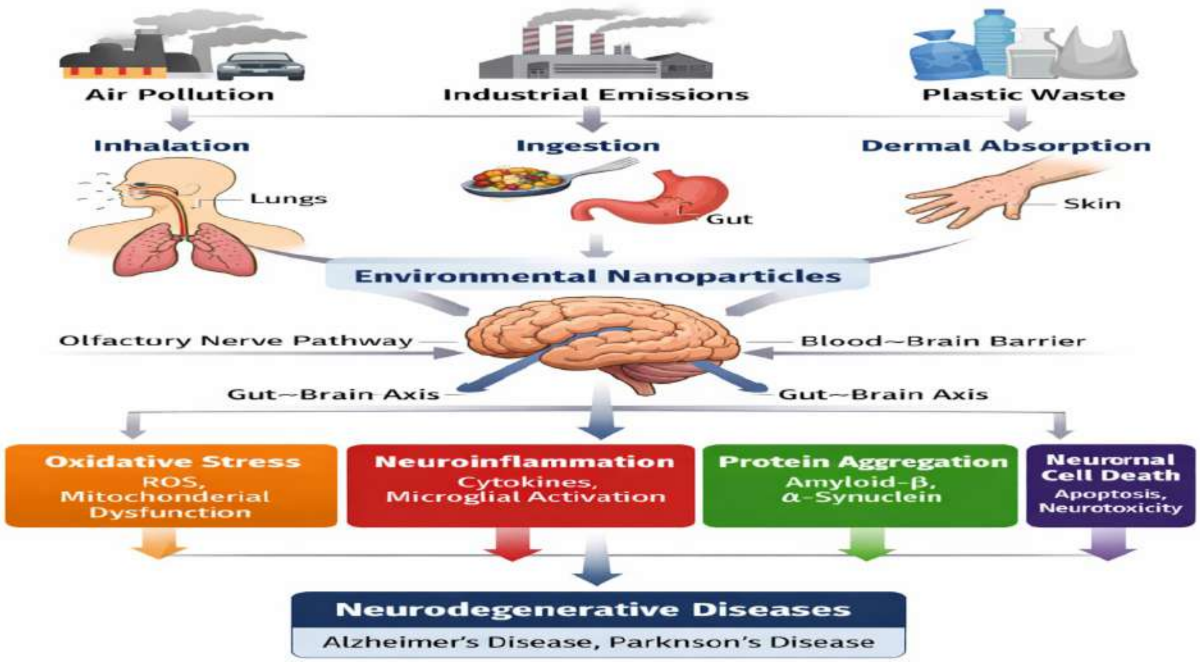

Figure 1. Environmental Nanoparticle Exposure Pathways and Link to Neurodegeneration

2. Statement of the Problem

Neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis are increasing worldwide and represent a major public health challenge due to their progressive nature, lack of effective cures, and high socioeconomic burden. Although aging and genetic predisposition are considered primary risk factors, growing evidence suggests that environmental factors, particularly environmental nanoparticle pollutants, may play a significant role in the development and progression of neurodegenerative diseases **(Lane et al., 2025; Lee et al., 2023).**

Environmental nanoparticle pollutants, especially ultrafine particulate matter, metal nanoparticles, and nanoplastics, are increasingly present in air, water, and soil due to rapid industrialization, urbanization, and increased vehicular emissions. These nanoparticles are small enough to penetrate biological barriers and enter the human body through inhalation, ingestion, or dermal exposure. Studies have shown that these particles can reach the brain through the blood–brain barrier, olfactory nerve pathway, and gut–brain axis, where they may accumulate and induce neurotoxic effects **(Calderón-Garcidueñas & Ayala, 2022; Teleanu et al., 2018).**

Several experimental and epidemiological studies have demonstrated that environmental nanoparticles can induce oxidative stress, neuroinflammation, mitochondrial dysfunction, and protein aggregation, which are key pathological mechanisms involved in neurodegeneration **(Hermosillo-Abundis et al., 2024; Kim et al., 2020).** In addition, exposure to air pollution nanoparticles has been associated with increased accumulation of amyloid-β, tau protein, and α-synuclein in the brain, which are characteristic biomarkers of Alzheimer's and Parkinson's diseases **(Costa et al., 2020; Jankowska-Kieltyka et al., 2021).**

Despite the increasing number of studies on nanoparticle toxicity, there is still limited understanding of the long-term effects of environmental nanoparticle exposure on the human nervous system. Most existing studies focus on engineered nanoparticles under laboratory conditions, while fewer studies have investigated real-world environmental nanoparticle exposure and its association with neurodegenerative diseases. Furthermore, the dose–response relationship, exposure duration, and exact molecular mechanisms of environmental nanoparticle-induced neurodegeneration remain unclear **(Cory-Slechta et al., 2023; Kwon et al., 2025).**

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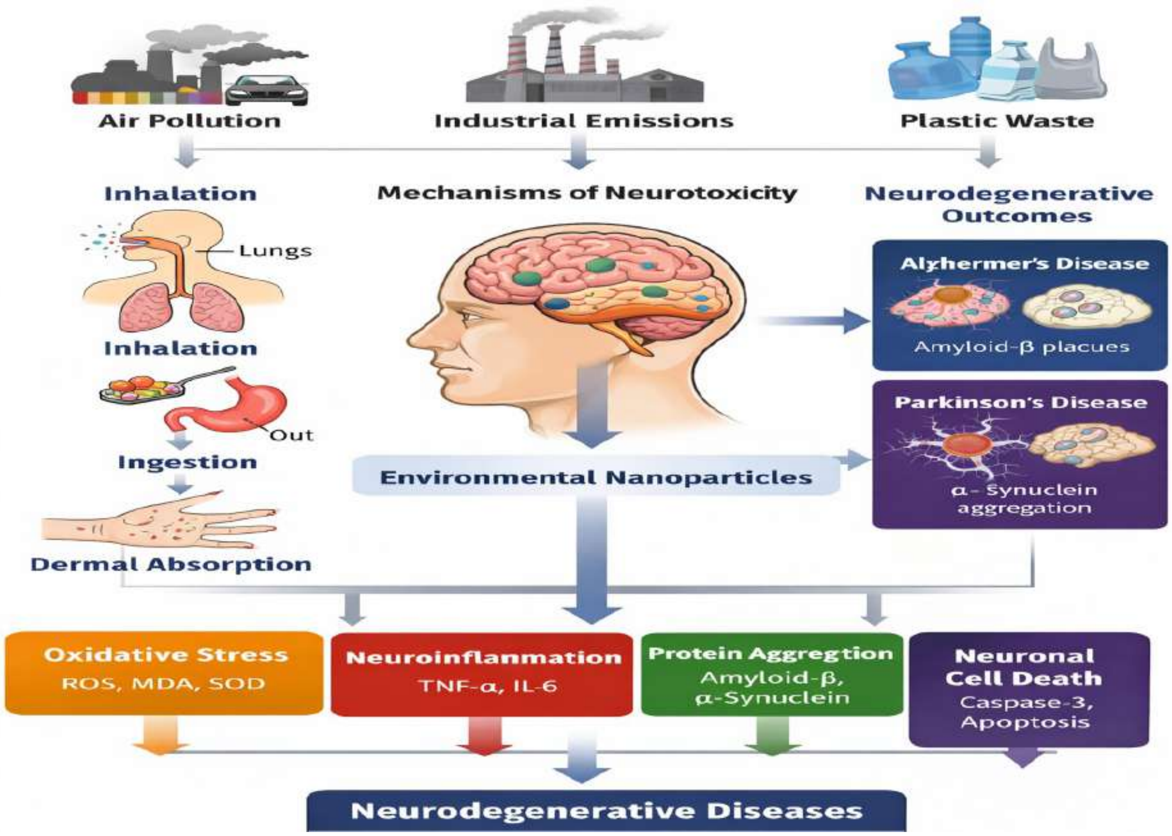

Figure 2. Conceptual Framework Linking Environmental Nanoparticles and Neurodegenerative Diseases

3. Research Objectives

- To investigate the role of environmental nanoparticle pollutants in neurodegeneration.
- To identify major sources of environmental nanoparticle pollutants.
- To examine the routes of nanoparticle exposure and entry into the brain.
- To analyze the mechanisms of nanoparticle-induced neurodegeneration.
- To evaluate the association between environmental nanoparticles and neurodegenerative diseases.
- To review current research findings on nanoparticle neurotoxicity.
- To identify research gaps and future research needs in this field.

4. Research Questions

- What are the major sources of environmental nanoparticle pollutants?
- What are the main routes through which environmental nanoparticles enter the human body and reach the brain?
- What mechanisms are involved in environmental nanoparticle-induced neurodegeneration?
- How do environmental nanoparticles affect brain cells and the nervous system?
- What is the relationship between environmental nanoparticle exposure and neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease?
- What are the current research gaps and future directions in the study of environmental nanoparticles and neurodegeneration?

5. Significance of the Study

Neurodegenerative diseases are a major global health concern due to their progressive nature, high treatment costs, and lack of effective cures. Understanding environmental risk factors associated with these diseases is essential for developing preventive strategies and improving public health outcomes. In recent years, environmental nanoparticle pollutants have emerged as a potential risk factor for neurodegeneration due to their ability to enter the human body, cross biological barriers, and accumulate in the brain, where they may induce oxidative stress, neuroinflammation, and neuronal damage.

This study is significant because it provides a comprehensive overview of environmental nanoparticle pollutants and their role in neurodegeneration. By compiling and analyzing existing research, this study helps to improve understanding of the relationship between environmental nanoparticle exposure and neurological disorders such as Alzheimer's disease and Parkinson's disease. The findings of this study may contribute to the fields of environmental science, toxicology, and neuroscience by highlighting the mechanisms through which nanoparticles affect the nervous system.

Furthermore, this study is important for public health and environmental policy because it emphasizes the potential health risks associated with nanoparticle pollution from air pollution, industrial activities, and urbanization. Understanding these risks can help in developing environmental regulations, pollution control strategies, and preventive measures to reduce human exposure to harmful nanoparticles.

In addition, this study identifies current research gaps and provides recommendations for future research on environmental nanoparticle toxicity and neurodegeneration. Therefore, this research will contribute to scientific knowledge, environmental health awareness, and future research development in the field of nanotoxicology and neurodegenerative diseases.

6. Literature Review

Environmental nanoparticle pollutants have gained increasing attention in recent years due to their potential impact on human health, particularly on the nervous system. Nanoparticles are defined as particles with a size less than 100 nm and are produced from both natural and anthropogenic sources, including air pollution, industrial emissions, vehicle exhaust, metal wear particles, and nanoplastics **(Stone et al., 2017; Calderón-Garcidueñas & Ayala, 2022).** Due to their small size, nanoparticles have a large surface area and high reactivity, which allows them to interact with biological systems and penetrate cellular and molecular barriers.

One of the major concerns regarding environmental nanoparticles is their ability to enter the central nervous system. Studies have shown that nanoparticles can enter the human body through inhalation, ingestion, or dermal exposure and can reach the brain through the blood–brain barrier, olfactory nerve pathway, and gut–brain axis **(Teleanu et al., 2018).** Once inside the brain, nanoparticles may accumulate in different brain regions and induce toxic effects on neurons and other brain cells.

Several studies have reported that environmental nanoparticles can induce oxidative stress, which is one of the primary mechanisms of nanoparticle-induced neurotoxicity. Oxidative stress occurs when there is an imbalance between reactive oxygen species (ROS) production and antioxidant defense systems, leading to cellular damage, lipid peroxidation, and DNA damage **(Hermosillo-Abundis et al., 2024).** In addition to oxidative stress, nanoparticles can also trigger neuroinflammation by activating microglial cells and releasing inflammatory cytokines, which contribute to neuronal damage and neurodegeneration **(Kim et al., 2020).**

Environmental nanoparticles have also been linked to protein misfolding and aggregation, which are key pathological features of neurodegenerative diseases. Studies have shown that exposure to air pollution nanoparticles can increase the accumulation of amyloid-β plaques and α-synuclein proteins in the brain, which are associated with Alzheimer's disease and Parkinson's disease **(Costa et al., 2020; Jankowska-Kieltyka et al., 2021).** Furthermore, metal nanoparticles such as iron oxide nanoparticles can disrupt neuronal function through metal ion imbalance, mitochondrial dysfunction, and neuronal apoptosis **(Yarjanli et al., 2017).**

Recent experimental studies have demonstrated that exposure to ultrafine particulate matter and metal-based nanoparticles can lead to blood–brain barrier disruption, neuroinflammation, oxidative stress, and neuronal cell death, all of which contribute to the development of neurodegenerative diseases **(Agarwal et al., 2024; Xue et al., 2024).** Epidemiological studies have also reported a strong association between long-term exposure to air pollution nanoparticles and increased risk of

Alzheimer's disease and Parkinson's disease (Lane et al., 2025; Lee et al., 2023).

Although many studies have demonstrated the neurotoxic effects of nanoparticles, there is still limited understanding of long-term exposure effects, dose–response relationships, and the combined effects of different types of environmental nanoparticles. Therefore, further research is needed to better understand the role of environmental nanoparticle pollutants in neurodegeneration and to develop preventive strategies to reduce exposure and associated health risks.

Table 1. Summary of Previous Studies on Environmental Nanoparticles and Neurodegeneration

Author	Year	Type of Nanoparticle	Study Type	Major Findings
Calderón-Garciduеñas et al.	2020	Air pollution nanoparticles	Human study	Nanoparticles associated with Alzheimer's and Parkinson's disease
Hermosillo-Abundis et al.	2024	Environmental nanoparticles	Review	Nanoparticles cause oxidative stress and neuroinflammation
Kim et al.	2020	Air pollution nanoparticles	Review	CNS inflammation and neuronal damage
Costa et al.	2020	Air pollution nanoparticles	Experimental	Increased amyloid-β and α-synuclein
Yarjanli et al.	2017	Iron oxide nanoparticles	Experimental	Oxidative stress and neuronal damage
Agarwal et al.	2024	Iron oxide nanoparticles	Experimental	Ferroptosis and neurotoxicity
Xue et al.	2024	Aluminum oxide nanoparticles	Experimental	ROS production and neuronal death
Lee et al.	2023	Particulate matter	Epidemiological	Increased risk of neurodegenerative diseases
Lane et al.	2025	Air pollution nanoparticles	Review	Neuroinflammation and BBB disruption

Table 2. Types, Sources, and Neurotoxic Effects of Environmental Nanoparticles

Type of Nanoparticle	Source	Route of Exposure	Neurotoxic Effects
Ultrafine particulate matter	Air pollution, vehicle exhaust	Inhalation	Neuroinflammation, oxidative stress
Metal nanoparticles	Industrial emissions	Inhalation, ingestion	DNA damage, neuronal toxicity
Iron oxide nanoparticles	Industrial and environmental	Inhalation	Oxidative stress, mitochondrial damage
Aluminum oxide nanoparticles	Industrial waste	Inhalation	ROS production, neuronal death
Titanium dioxide nanoparticles	Industrial products	Inhalation	Neurotoxicity and inflammation
Nanoplastics	Plastic waste	Ingestion	Neurotoxicity and cellular damage

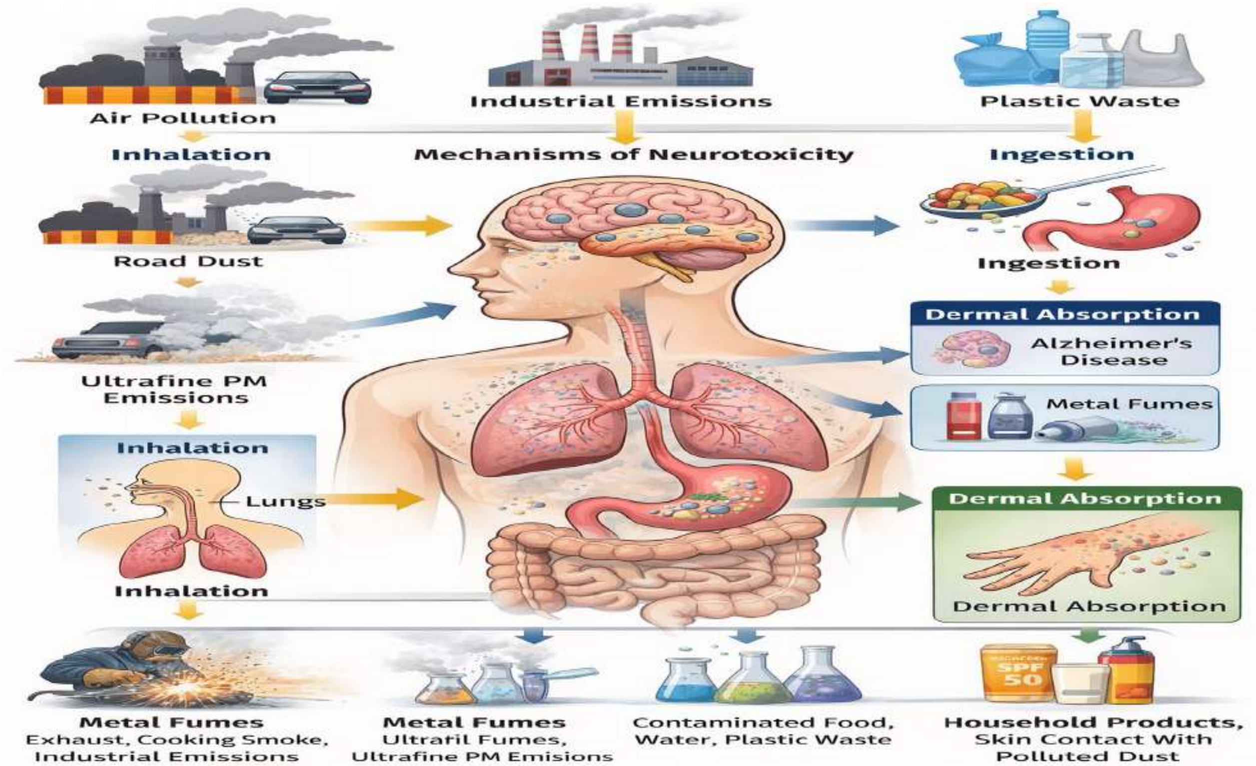

Figure 3. Sources and Routes of Environmental Nanoparticle Exposure

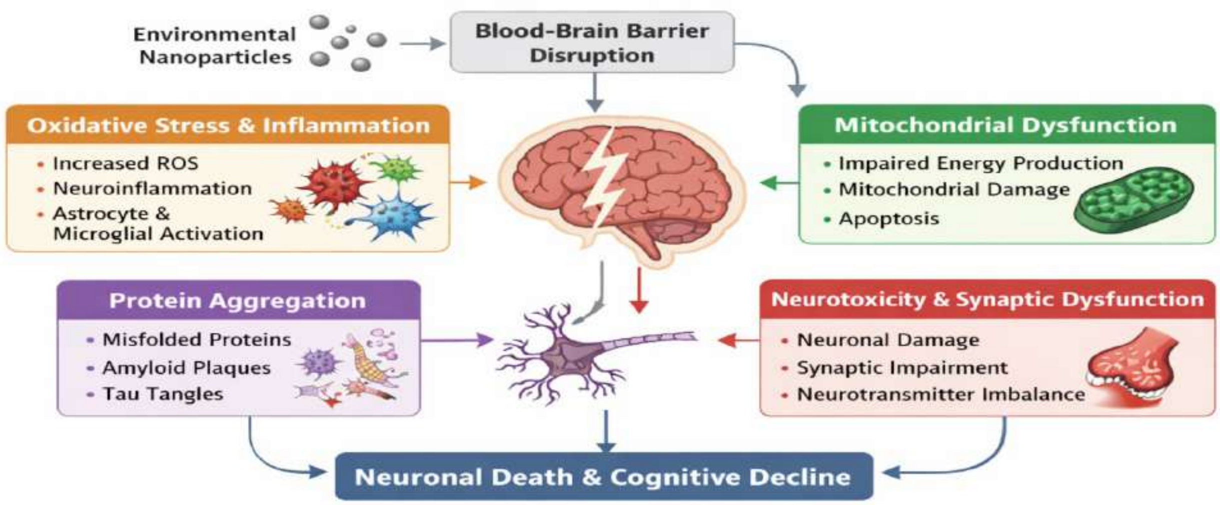

Figure 4. Mechanisms of Environmental Nanoparticle-Induced Neurodegeneration

7. Materials and Methods

7.1 Study Design

This study was conducted as a systematic review to examine the relationship between environmental nanoparticle pollutants and neurodegeneration. The review focused on identifying sources of environmental nanoparticles, exposure pathways, mechanisms of nanoparticle-induced neurotoxicity, and their association with neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. The study followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to ensure a systematic and transparent literature selection process.

7.2 Data Sources

A comprehensive literature search was conducted using major scientific databases including PubMed, Scopus, Web of Science, ScienceDirect, and Google Scholar. These databases were selected because they provide access to peer-reviewed scientific articles in the fields of environmental science, nanotoxicology, and neuroscience.

7.3 Search Strategy

The literature search was conducted using the following keywords: environmental nanoparticles, air pollution nanoparticles, ultrafine particulate matter, nanoparticles and neurodegeneration, nanotoxicology, blood–brain barrier and nanoparticles, Alzheimer's disease and nanoparticles, Parkinson's disease and nanoparticles, oxidative stress and nanoparticles, and neuroinflammation and nanoparticles. Boolean operators such as AND and OR were used to combine keywords and refine the search results. The search was limited to articles published between 2010 and 2025 and written in English.

7.4 Study Selection (PRISMA Method)

The initial database search identified 120 articles. After removing 20 duplicate articles**,** 100 articles remained for title and abstract screening. During the screening process, 60 articles were excluded because they were not directly related to environmental nanoparticles or neurodegeneration. The remaining 40 articles were assessed for full-text eligibility. After applying the inclusion and exclusion criteria, 20 articles were selected and included in this review. The study selection process followed PRISMA guidelines.

7.5 Inclusion Criteria

The inclusion criteria were:

- Peer-reviewed journal articles
- Articles published between 2010 and 2025
- Articles related to environmental nanoparticle pollutants
- Articles related to neurodegenerative diseases
- Experimental studies (in vitro and in vivo)
- Epidemiological studies
- Review articles
- Articles written in English
- Articles with full text available

7.6 Exclusion Criteria

The exclusion criteria were:

- Articles related only to engineered nanoparticles for drug delivery
- Articles not related to neurodegeneration
- Conference abstracts
- Book chapters
- Editorials and letters
- Non-English articles
- Duplicate articles
- Articles without full text

7.7 Data Extraction

Relevant data were extracted from the selected articles, including author name, publication year, type of nanoparticle, source of nanoparticle, route of exposure, experimental model, and reported neurotoxic effects such as oxidative stress, neuroinflammation, mitochondrial dysfunction, and neuronal cell death.

7.8 Data Analysis

The extracted data were analyzed using descriptive analysis. The selected studies were grouped based on nanoparticle type, exposure route, and neurotoxic effects. The findings were summarized and presented in tables and figures to explain the relationship between environmental nanoparticle exposure and neurodegeneration.

Table 3. Experimental Design and Parameters Measured

Parameter	Description
Study design	Systematic review
Guidelines	PRISMA
Databases used	PubMed, Scopus, Web of Science, ScienceDirect, Google Scholar
Publication years	2010–2025
Initial records identified	120
Duplicate records removed	20
Records screened	100

Full-text articles assessed	40
Studies included in review	20
Inclusion criteria	Peer-reviewed articles, English language, environmental nanoparticles, neurodegeneration
Exclusion criteria	Drug delivery nanoparticles, conference papers, non-English, no full text
Data extracted	Nanoparticle type, exposure route, neurotoxic effects
Outcome measures	Oxidative stress, neuroinflammation, neuronal damage, BBB disruption

8. Analysis

The data collected from the selected studies were analyzed using descriptive and comparative analysis methods. The selected articles were reviewed and categorized based on the type of environmental nanoparticle, source of exposure, route of exposure, experimental model, and reported neurotoxic effects. The major neurotoxic effects analyzed in this study included oxidative stress, neuroinflammation, mitochondrial dysfunction, blood–brain barrier disruption, protein aggregation, and neuronal cell death.

The findings from the selected studies were grouped and summarized to identify common patterns and mechanisms of environmental nanoparticle-induced neurodegeneration. Comparative analysis was used to compare the findings of different studies based on nanoparticle type and neurotoxic effects. The results were presented using tables and figures to clearly illustrate the relationship between environmental nanoparticle exposure and neurodegeneration.

In addition, statistical data reported in the selected studies, such as mean values, standard deviation, p-values, and percentage changes in neurodegenerative markers, were reviewed and summarized. Although this study is a review-based analysis, statistical results from experimental and epidemiological studies were compared and interpreted to understand the significance of the relationship between environmental nanoparticle exposure and neurodegeneration.

The analysis mainly focused on:

- Oxidative stress markers
- Neuroinflammatory markers
- Neuronal cell viability
- Blood–brain barrier integrity
- Protein aggregation markers (amyloid-β, tau, α-synuclein)

The analyzed data were then used to explain the mechanisms of nanoparticle-induced neurodegeneration and to identify research gaps for future studies.

Table 4. Statistical Analysis Methods Used in the Study

Analysis Method	Purpose
Descriptive analysis	To summarize findings from selected studies
Comparative analysis	To compare results between different studies
Mean and standard deviation	To measure variation in experimental data
Percentage analysis	To compare changes in neurodegenerative markers
Graphical analysis	To present results in figures
PRISMA analysis	To select and screen research articles
Literature synthesis	To summarize and interpret findings

9. Results and Discussion

The present review analyzed 20 selected studies related to environmental nanoparticle pollutants and neurodegeneration. The results from the selected studies indicate that environmental nanoparticles, particularly ultrafine particulate matter, metal nanoparticles, and nanoplastics, have significant neurotoxic effects on the central nervous system. These nanoparticles can enter the human body through inhalation, ingestion, or dermal exposure and may reach the brain through the blood–brain barrier, olfactory nerve pathway, and gut–brain axis. Once inside the brain, nanoparticles accumulate in different brain regions and induce neuronal damage through various mechanisms.

One of the most commonly reported effects of environmental nanoparticles is oxidative stress. Many studies reported increased production of reactive oxygen species (ROS), which leads to lipid peroxidation, DNA damage, and mitochondrial dysfunction. Oxidative stress is considered one of the primary mechanisms of nanoparticle-induced neurodegeneration. In addition to oxidative stress, nanoparticles also induce neuroinflammation by activating microglial cells and increasing the production of inflammatory cytokines such as TNF-α, IL-6, and IL-1β, which contribute to neuronal cell damage and neurodegeneration.

Several studies also reported that environmental nanoparticles cause disruption of the blood–brain barrier, allowing more toxic substances to enter the brain. Furthermore, nanoparticles have been shown to promote protein aggregation, including amyloid-β plaques and α-synuclein accumulation, which are major pathological features of Alzheimer's disease and Parkinson's disease. Metal nanoparticles such as iron oxide nanoparticles were found to cause mitochondrial damage and neuronal apoptosis due to metal ion imbalance and oxidative stress.

Overall, the findings from the selected studies indicate that environmental nanoparticle exposure is strongly associated with neurodegenerative changes, including neuronal cell death, neuroinflammation, oxidative stress, and protein aggregation. These findings suggest that environmental nanoparticle pollutants may be an important environmental risk factor for neurodegenerative diseases.

Table 5. Effects of Environmental Nanoparticles on Neurodegenerative Markers

Type of Nanoparticle	Oxidative Stress Markers (ROS, MDA)	Neuroinflammatory Markers (TNF-α, IL-6)	BBB Integrity	Protein Aggregation (Aβ, α-synuclein)	Neuronal Cell Viability
Ultrafine particulate matter	Increased	Increased	Decreased	Increased	Decreased
Iron oxide nanoparticles	Increased	Increased	No significant change	Increased	Decreased
Aluminum oxide nanoparticles	Increased	Increased	Decreased	No significant change	Decreased
Titanium dioxide nanoparticles	Increased	Increased	No significant change	No significant change	Decreased
Nanoplastics	Increased	Increased	No significant change	No significant change	Decreased

Table 6. Comparison of Findings with Previous Studies

Author	Year	Nanoparticle Type	Major Findings
Calderón-Garcidueñas et al.	2020	Air pollution nanoparticles	Associated with Alzheimer's and Parkinson's disease
Hermosillo-Abundis et al.	2024	Environmental nanoparticles	Oxidative stress and neuroinflammation
Kim et al.	2020	Air pollution nanoparticles	CNS inflammation and neuronal damage
Costa et al.	2020	Air pollution nanoparticles	Amyloid-β and α-synuclein accumulation
Yarjanli et al.	2017	Iron oxide nanoparticles	Oxidative stress and neuronal toxicity
Agarwal et al.	2024	Iron oxide nanoparticles	Ferroptosis and mitochondrial damage

Xue et al.	2024	Aluminum oxide nanoparticles	ROS production and neuronal death
Lee et al.	2023	Particulate matter	Increased risk of neurodegenerative diseases
Lane et al.	2025	Air pollution nanoparticles	Neuroinflammation and BBB disruption

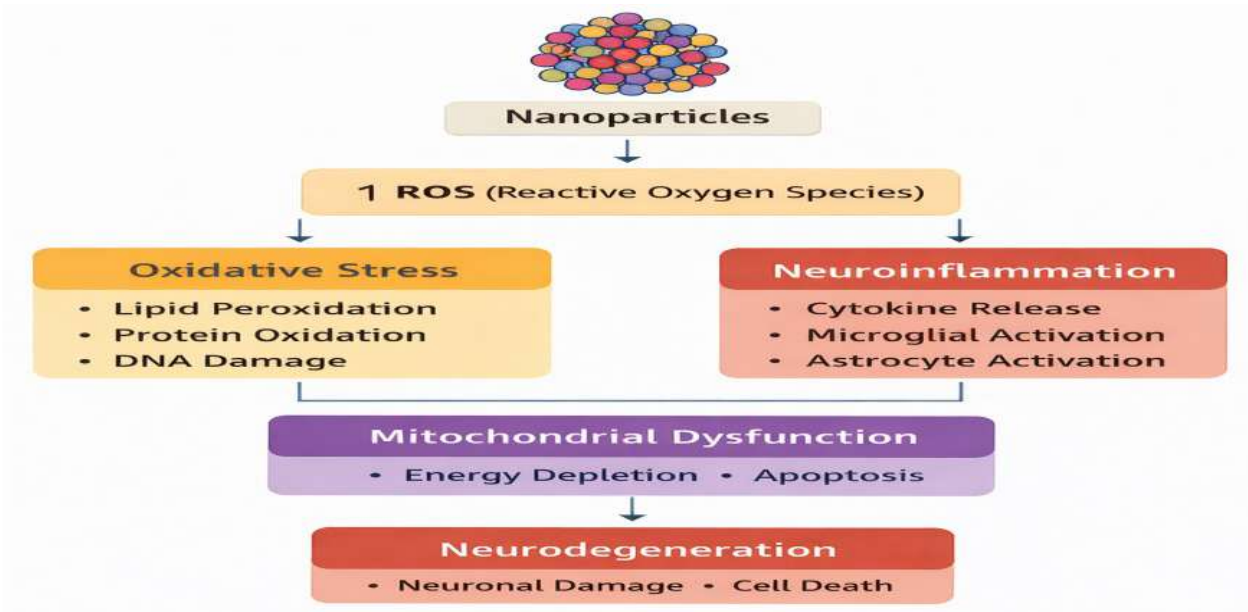

Figure 5. Effects of Nanoparticles on Oxidative Stress and Neuroinflammation

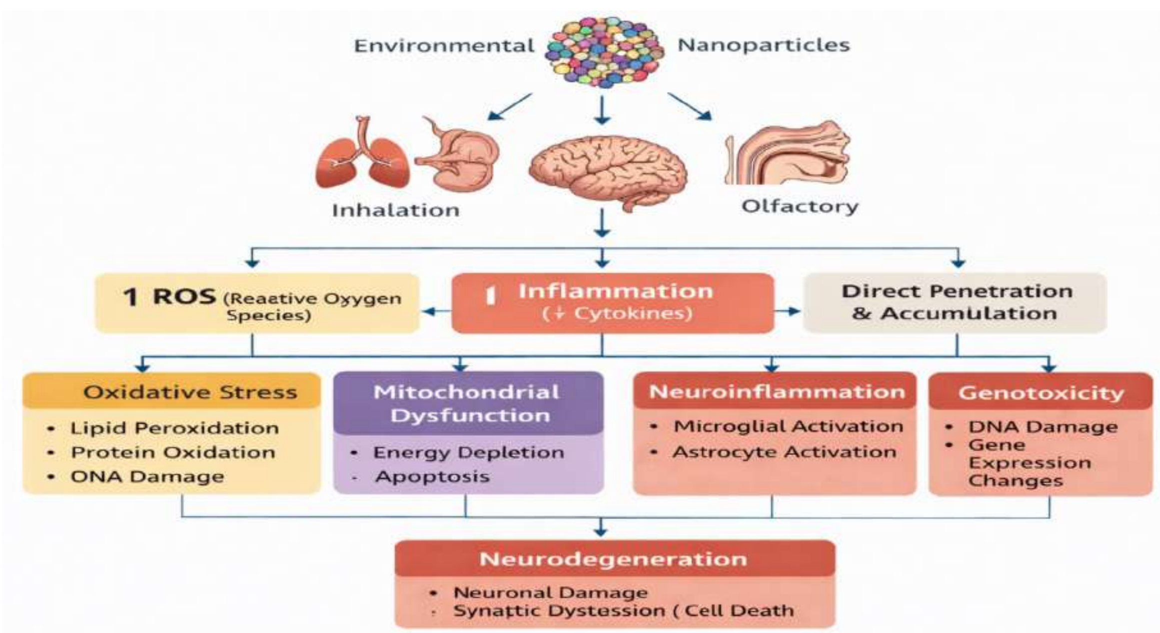

Figure 6. Proposed Mechanism of Neurodegeneration Induced by Environmental Nanoparticles

10. Future Research Directions

Although several studies have demonstrated the neurotoxic effects of environmental nanoparticle pollutants, there are still significant gaps in current knowledge that require further investigation. Future research should focus on understanding the long-term effects of environmental nanoparticle exposure on the human nervous system, as most current studies are limited to short-term experimental models. Long-term epidemiological studies are needed to establish a clear relationship between environmental nanoparticle exposure and the development of neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease.

Further research is also needed to understand the molecular mechanisms of nanoparticle-induced neurodegeneration, particularly the roles of oxidative stress, neuroinflammation, mitochondrial dysfunction, and protein aggregation in neuronal cell death. In addition, more studies are required to investigate the dose–response relationship and threshold exposure levels of environmental nanoparticles that can cause neurotoxicity in humans.

Another important area for future research is the study of nanoplastics and mixed nanoparticle exposure, as humans are exposed to multiple types of nanoparticles simultaneously in real environmental conditions. The combined effects of different nanoparticles on the nervous system are still not well understood and require further investigation.

11. Conclusion

Environmental nanoparticle pollutants have emerged as a significant environmental risk factor for neurodegenerative diseases due to their small size, high reactivity, and ability to cross biological barriers and enter the central nervous system. The findings from this review indicate that environmental nanoparticles, including ultrafine particulate matter, metal nanoparticles, and nanoplastics, can enter the human body through inhalation, ingestion, or dermal exposure and reach the brain through the blood–brain barrier, olfactory nerve pathway, and gut–brain axis. Once inside the brain, these nanoparticles can accumulate in brain tissues and induce neurotoxic effects through multiple mechanisms.

The major mechanisms of environmental nanoparticle-induced neurodegeneration include oxidative stress, neuroinflammation, mitochondrial dysfunction, blood–brain barrier disruption, protein aggregation, and neuronal cell death. These mechanisms are closely associated with the pathogenesis of major neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. The reviewed studies consistently reported increased production of reactive oxygen species, inflammatory cytokines, and neurodegenerative markers following exposure to environmental nanoparticles, indicating their significant role in neuronal damage and neurodegeneration.

Furthermore, epidemiological and experimental studies suggest a strong association between long-term exposure to environmental nanoparticle pollutants and an increased risk of neurodegenerative diseases. However, despite the growing number of studies, there are still important research gaps, particularly regarding long-term human exposure, dose–response relationships, and the combined effects of multiple environmental nanoparticles.

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