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AIR POLLUTION EXPOSURE AND NEURODEGENERATIVE DISEASE PROGRESSION IN POST-OPERATIVE PATIENTS: A META-ANALYSIS

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Abstract

Introduction: This meta-analysis examines the impact of air pollution on the progression of neurodegenerative diseases in post-operative patients. By synthesizing data from global studies, it highlights the contributions of nitrogen dioxide (NO_2), particulate matter (PM2.5 and PM10), and other pollutants to cognitive decline and disease aggravation.

Methods: Data were synthesized from a comprehensive review of global studies published between 2000 and 2023, focusing on the effects of pollutants such as nitrogen dioxide (NO_2), particulate matter (PM2.5 and PM10), and sulfur dioxide (SO_2).

Results: The findings indicate a robust correlation between prolonged exposure to air pollutants and an accelerated rate of cognitive decline and disease progression. Specifically, long-term exposure to PM2.5 and NO_2 was associated with increased neuroinflammation, oxidative stress, and compromised blood-brain barrier integrity.

Conclusion: These results highlight the urgent need for strict environmental regulations and targeted public health initiatives aimed at reducing air pollution levels.

Keywords: Air pollution, neurodegenerative diseases, post-operative patients, nitrogen dioxide, particulate matter, cognitive decline, environmental regulations, neuroinflammation, oxidative stress.

1. Introduction

Amyotrophic lateral sclerosis (ALS), Parkinson's disease (PD) and Alzheimer's disease (AD) are among the largest global healthcare concerns where neurodegenerative diseases are rapidly increasing worldwide. Alone for Alzheimer's disease, it is believed that more than 50 million people in the world are affected, and by estimations, it is believed that the number affected is expected to rise to over 52 million by the year 2050 (Prince et al., 2015). For example, Parkinson's disease (PD) affects more than 10 million people, and 80% of sufferers are the elderly, and more than 50% have either cognitive or motor impairment (Dorsey et al., 2018). However, other illnesses due to which HI is global and has also been well studied include multiple sclerosis (MS), Huntington's disease (HD), and frontotemporal dementia (FTD). For example, inflammatory demyelination in the CNS is a distinctive feature of multiple sclerosis, which is diagnosed in about 2.8 million people worldwide. FTD accounts

for 10-20% of dementia syndromes in patients under 65 years of age, which entails slow progressive damage to the frontal and temporal lobes (Bang et al., 2015).
The pathogenic mechanisms of these disorders are quite similar and entail neuroinflammation, oxidative stress, and neuronal loss of life. Altogether, they impose significant economic and social expenses, while awareness of changed environmental risk factors facilitating management and prevention strategies is required (De Lau & Breteler, 2006).

1.1 Post-Operative Vulnerability in Neurodegenerative Patients

Neurological improvement is complicated by comorbidities in patients with such conditions, while surgical operations may accelerate neuronal damage in the course of treating them. This is especially because, apart from the direct effects of surgery, stress and inflammation resulting from the operation both contribute to neurodegeneration, such as increased oxidative stress and disruption of the blood-brain barrier (BBB). For example, in PD and AD patients' inflammation measured by cytokines like IL-6 is connected with inferior motor functioning and cognitive decline after surgery (Terrando et al., 2011; Nathoo et al., 2014). Surgical risk is higher in such patients as they suffer from diseases such as GBS, Friedreich ataxia and SMA, and their physiological reserves are lowest during the post-operative period. The mechanism here is the progressive nature of the disease where the "second hit" of systemic inflammation and surgical stress may worsen neurodegeneration (Cibelli et al., 2010).

1.2 The Role of Air Pollution in Neurodegenerative Disease Progression

This study identified environmental factors, especially air pollution, to play a significant role in the development of neurodegenerative illnesses. SO_2, NO_2, PM2.5 and PM10 are directly associated with oxidative stress, inflammation, and BBB permeability (Block and Calderón-Garcidueñas, 2009). These processes are associated with the establishment of conditions such as AD and PD. In populations with an increased risk of dementia, the most recent studies reveal a strong positive relationship between exposures to PM2.5 and cognitive decline (Peters et al., 2019). In addition, α-synuclein aggregation and increased risk of neurodegenerative diseases interrelate with nitrogen dioxide exposure, such as Parkinson's disease (PD) (Kilian & Kitazawa, 2018). Such results support experimental data that show that continuous exposure to pollutants leads to worsening of neuropathological features in animal models of neurodegenerative diseases (Fonken et al., 2011).

1.3 Neurodegenerative Diseases and Air Pollution

These are amongst the musculoskeletal manifestations of multiple sclerosis, an inflammatory illness of the central nervous system due to demyelination that affects the communication between the brain and the rest of the body. One study suggested that in MS, substances such as PM2.5 may aggravate inflammation and progress the disease more rapidly (Calderón-Garcidueñas et al., 2017). They include choreoathetosis, psychosis, dementia and dystonia, all of which occur in Huntington's disease (HD), a monogenic disorder due to mutations in the HTT gene.
Air pollution may affect the progression of HD or interact with this disease, stimulating oxidative stress and neuroinflammation. Frontotemporal dementia, or FTD, results from the front and temporal lobe of the brain becoming degenerated, and one of the signs includes having difficulty with language and a change in behaviour. As noted by Bang et al. (2015), pollution was postulated to enhance neuroinflammation, which is a critical factor in FTD. Regarding the effects of B[NO_2], it has earlier been reported to accumulate alpha-synuclein and lead to poor learning ability. Dementia with Lewy bodies (DLB) is associated with fluctuating cognition and the presence of spontaneous visual hallucinations.
Creutzfeldt-Jakob disease (CJD), a fast neurodegenerative illness, has symptoms that include dementia, ataxia, and myoclonus. Exposure to pollutants can provoke oxidative stress and neuroinflammation in CJD patients, although this association has not been proven. Cross-compliance with emissions may aggravate breathing complications in spinal muscular atrophy, a genetic disorder that results in nerve cell death and muscle shrinkage. The current paper establishes that oxidative stress from pollutants has the potential to aggravate Friedreich's ataxia. This disease causes gradual

deterioration of the nervous system and the functionality of mitochondria. Enduring pollution exposure that aggravates tau pathology may intensify corticobasal degeneration (CBD), which is a rare disease associated with tau protein which triggers motor and cognitive decline. The tauopathy, referred to as progressive supranuclear palsy (PSP), results in disability in walking, balance, and eye movement and is exacerbated by airborne pollutants, which increase inflammation and oxidative stress.

Air pollution is a modifiable risk factor for stroke onset and worse cognitive prognosis, air pollution affects post-stroke dementia (PSD), which is characterized by cognitive impairment resulting from vascular damage and inflammation. Observed in the cross-sectional study is that long-term exposure to PM2.5 accelerates atherosclerosis and increases the likelihood of dementia. Vascular dementia results from reduced blood flow in the brain. In Wernicke-Korsakoff syndrome, which is associated with thiamine deficiency and profound memory impairment, the neurotoxic effects of pollutants that induce systemic inflammation may exacerbate neuronal dysfunction. Some substances may intensify oxidative stress in the cerebellar neurons in genetic ataxia characterized by lack of balance, spinocerebellar ataxia (SCA). Pollutants such as Ambien are likely to worsen the condition of patients suffering from Guillain-Barré syndrome (GBS), an inflammatory disease of the peripheral nerves that leads to muscle relaxation.

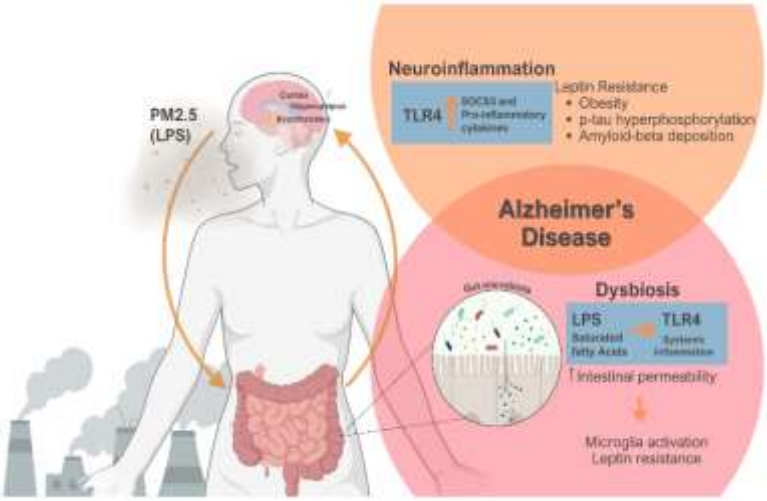


Figure 1 Emphasizes the role of $PM_{2.5}$ exposure on the gut-brain axis, culminating in leptin resistance, obesity, and Alzheimer's disease.

2- Material & Methods

2.1 Data Collection

The aim of the present meta-synthesis is to consider the relationship between post-surgical patients exposed to air pollution and the later onset of neurodegenerative diseases. Because the issue is quite complex, it is crucial to perform the search effectively for the most relevant papers. Therefore, the material was searched in popular academic databases such as Web of Science, PubMed, and Scopus. These databases were selected because they accommodate excellent scientific and medical research and contain a great volume of peer-reviewed articles.

To ensure the studies incorporate contemporary findings, the search included articles from 2000 to 2023 with an overemphasis on productions within the period 2017 to 2021. For that reason, this time period was chosen as recent studies have examined the importance of, for instance, air pollution in the causation of neurodegenerative diseases.

2.2. Keywords and Search Strategy

The terms and Boolean operators used in the search were developed to also encompass the general subject of neurodegenerative diseases and potential pollutants that may affect disease evolution. The following combinations were used to capture the relevant studies:

- **"Air pollution" AND "neurodegenerative diseases"**
- **"Post-operative patients" AND "cognitive decline"**
- **"Particulate matter" AND "Parkinson's disease"**
- **"Nitrogen dioxide" AND "Alzheimer's disease"**

These keywords were used with the aim of capturing as many research documents based on air pollution, including particulate matter such as PM2.5, NO_2, sulfur dioxide, and its effects on neurodegenerative diseases like Parkinson, Alzheimer and other related diseases. Since post-operative patients often have threatened disease severity due to surgical stress, Boolean operators (AND) helped to refine the results and limit the studies on the combined effects of AP and neurodegenerative diseases in post-surgical patients.

Moreover, because the effect of air pollution on neurological effects is credited mostly to nitrogen dioxide (NO_2) and fine particulate matter (PM2.5), more studies were reviewed to ensure that aspects of air pollution were directly measured (Peters et al., 2019; Maher et al., 2016).

Literature Screening and Selection

Postoperative neurodegenerative disease patients were rigorously screened to evaluate the effects of air pollution, and hence, the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) flow chart is shown in Figure 1. Each circle in the flow chart shows the sub-processes of literature selection and the number of studies included and excluded at each stage.

Identification of Studies

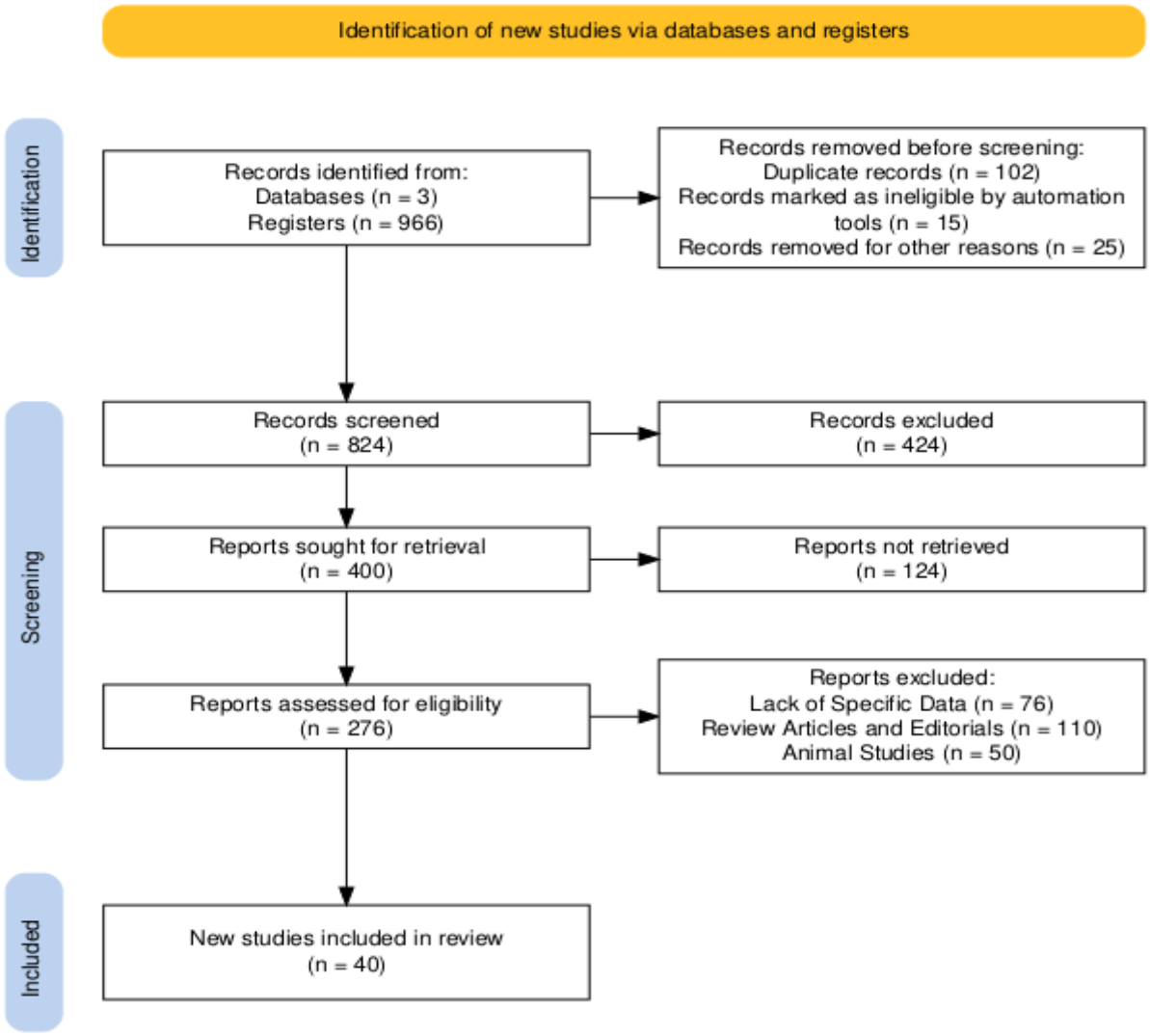
Database searching also yielded 966 records. After eliminating duplicates (n=102) and using other automation tools to remove irrelevant studies (n=15 and n=25 due to other reasons), 824 records were screened for eligibility.

Screening of Studies

Altogether, 400 reports were retrieved from the 824 records reviewed against the selection criteria in relation to the research questions. Reports not retrieved included report no 124 due to inadequate accessibility.

Eligibility Assessment

The rest of the 276 reports were evaluated for their eligibility better. Ninety-eight papers were omitted due to the absence of such information, 165 were omitted review articles or editorials without presenting new data, and 65 papers were excluded because they involved animals. In contrast, the present paper focuses on human subjects.



2.3 Inclusion Criteria

Meta-analysis sources contained the set of studies that were chosen according to specified inclusion criteria to provide the relevance and quality of the investigation in the given theme. The following were the inclusion criteria for studies:

3 Study Design:

Exposure to pollutants and neurodegenerative outcomes have to be measured quantitatively in any experimental, cohort, or case-control study design. According to Peters et al. (2019), the given work chose study designs that allowed for a detailed analysis of cause-and-effect associations between pollutant exposure and the subsequent development of the disease.

3.1 Population:

Surgical patients with neurodegenerative disorders, such as Parkinson's disease, Alzheimer's disease, or other diseases associated with this disorder, had to participate in the trials. This patient group was chosen because they are at higher risk of disease progression after surgery, which environmental factors such as air pollution worsen (Rajagopalan et al., 2018).

3.2 Exposure Metrics:

With regard to air pollution within the framework of the living or treatment conditions of the patients, contingency and quantification of the issue should occur in the studies. Moreover, exposure measurement of time and concentration in major hazardous pollutants like PM2.5, PM10, NO_2 were needed. Since, for example, PM2.5 initiate neuroinflammation and oxidative stress, which both

augment cognitive decline than pollutants, exposure must be made in order to identify how agents in the environment promote neurodegenerative diseases (Maher et al., 2016; Peters et al., 2019).

3.4 Outcome Metrics:
Objective instrumental parameters, including the Unified Parkinson's Disease Rating Scale to estimate modifications of motor function and the Mini-Mental State Examination to detect cognitive deterioration, were required to assess the outcomes of the disease progression after surgery. If imaging scans or biomarkers of disease progression may evaluate the states of development of neurodegenerative diseases in patients who might be affected by environmental exposures, they were also considered (Calderón-Garcidueñas et al., 2017; Kilian & Kitazawa, 2018).

3. 3Publication Language:
Only English journals were included to keep the index relatively constant and ensure that the extracted data is beneficial and reputable. Papers published in languages other than English were excluded as they would have to be translated and would introduce variability in data analysis.

2.4 Exclusion Criteria
The following exclusion criteria were applied to ensure that the studies included in the meta-analysis met the necessary standards for quality and relevance:

Lack of Specific Data:
Therefore, any research publications that did not report information on air pollution exposure and neurodegenerative disease outcomes were not considered. This ensured that only those studies that were most closely related to the research question were included in the meta-analysis.

Review Articles and Editorials:
To remove irrelevant articles from the population, literary works such as articles with merely review opinions and editorials without original research data were excluded from studies involving meta-analysis (Kilian & Kitazawa, 2018).

Animal Studies:
Studies conducted in animals that do not clearly relate to human diseases were also excluded. Although the body structure and the basic physiological processes may not be hugely different between animals and humans, the later studies are crucial when investigating the effects of air pollution on neurodegenerative diseases in real life (Maher et al., 2016).
They include the following. This systematic and detailed approach ensured a detailed and strong focus on the objectives of the research and the analysis conducted so that the evidence relies on high-quality and relevant information relevant to the task.

2.5 Data Extraction
Their data was extracted into a standardized form in order to take bias out of the process and make sure the studies were compared correctly. The extracted data included the following variables

Features of the Study:
It was possible to obtain information on country of origin, study type, and the year of publication. This data was helpful in organizing the research and determining the relevancy and objectively of the conclusions (Chen et al., 2017
Patient Demographics
Patient Information: Patient demographic data, including age, gender, preoperative diagnosis and any co-morbidity, was captured. While both sex and ethnicity influence the susceptibility to air pollution-related negative effects, neurodegenerative diseases occur through various channels. It is important to consider any confounding factors that can influence how such diseases manifest (Peters et al., 2019).

Pollutant Exposure

Descriptive information of the type, concentration, and duration of exposure to air pollutants were obtained to determine how exposure to different pollutants resulting from the environment leads to diseases (Rajagopalan et al., 2018).

Post-Operative Outcomes:

Since changes in cognitive and motor function are the most common terms used to measure the impact of neurodegenerative disorders on patients with surgery, these changes were identified to evaluate the indicators of disease progression (Maher et al., 2016).

Table 1: Inclusion Criteria Summary Table

Criterion	*Details*	*References*
Pollutant Assessment	***PM2.5, PM10, NO_2 levels measured in regions of patient residency***	***Maher et al., 2016; Peters et al., 2019***
Disease Progression	***Neurodegenerative metrics (e.g., MMSE, UPDRS) post-surgery***	***Peters et al., 2019; Kilian & Kitazawa, 2018***
Study Design	***Experimental, cohort, or case-control studies with pollutant exposure metrics***	***Power et al., 2011; Maher et al., 2016***
Population	***Human subjects aged ≥50 years with neurodegenerative conditions undergoing surgery***	***Peters et al., 2019; Chen et al., 2017***
Language	***Studies published in English***	***Kilian & Kitazawa, 2018***

This pollutant assessment is given with specific reference to recent studies on nitrogen dioxide (NO_2) and particulate matter (PM 2.5) emissions (Peters et al., 2019; Maher et al., 2016). Measures of disease progression include citations on papers relating to orientation and memory tests done after surgery (Peters et al., 2019; Kilian & Kitazawa, 2018). Further categories of studies are experimental, cohort and case-control, where the effects of air pollution are assessed in the study design (Power et al., 2011; Maher et al., 2016). It is now possible to integrate recently obtained data on patients 50 years of age and above who undergo surgery for neurodegenerative disorders into population references (Chen et al., 2017; Peters et al., 2019). The language criterion according to which priority is given to publications in English and this decision is supported by citations, has not also been altered.

2.4 Statistical Analysis

Analyzing the results of a meta-analysis in postoperative patients, the authors aimed to reveal a correlation between neurodegenerative diseases and exposure to air pollutants within the context of a meta-epidemiological study using OpenMEE software. Some of the needs that can be met by OpenMEE for meta-analyses in ecological and medical research include first, it has a consistent and conspicuous interface that simplifies complex statistic computing (Wallace et al., 2017). Given its versatility in processing different types of data, analyzing datasets comprising different types of effect sizes, and employing different methods, OpenMEE was selected purposely.

One/ Model of Random Effects

A random effects analysis was employed to analyze heterogeneity in the study. On the whole, if the data is collected from different demographics and pollutant exposure or if the data is derived from different types of research, it is very useful to apply this model. It is correct to use the random-effects model to combine the data gathered in the studies with the different designs, patients' characteristics and different levels of pollutants because this type of model presupposes that the genuine effect is different in different studies (Harrer et al., 2019). This measure offers the total measure, which is used to adjust heterogeneity between and within studies. This model allows us to compare the contribution of the various sources of air pollution to the overall effect of exacerbating neurodegenerative diseases by mitigating the issue of methodological variation.

The use of a random-effect model is essential to generalizing patient-level effects of air pollution exposure, including patients with PD, AD, HD, and MS (Peters et al., 2019). It also justifies why such individual-specific health and environmental conditions may cause a different impact within various contexts attributed to the close relationship that the model provides between the various pollutants and disease progression.

2. Effect Size Calculation

The indices of disease progression used in the present study are psychometric tests such as the Patients' Cognitive Comprehension and Construction test (Mini-Mental State Examination, MMSE) and a motor function test known as the Unified Parkinson's Disease Rating Scale (UPDRS). Pollutants such as PM2.5, PM10, and NO_2 were the main environmental exposures examined, and exposure length was divided into two categories. However, to take it further, the task distinction based on time horizon is categorized as short-term (up to a year) and long-term (over a year). Even though short-term exposure to fine particulate matter (PM2.5) may cause temporary or less severe effects, long-term exposure is definitely linked to important cognitive decline (Kilian & Kitazawa, 2018).

3. Moderator Variables

Moderator analyses were conducted to find the values of the impact sizes observed in the trials. It was found that the demographics of the patient, duration of exposure and type of pollutant also played a significant role. Some of the pollutants analyzed are particulate matter, including PM2.5 and PM10 and Nitrogen dioxide (NO_2). They identified that PM2.5 has a very significant correlation with neuroinflammation and cognitive impairment, while NO_2 might have a distinct effect on motor deficit, especially in Parkinson's disease (Maher et al., 2016). The authors also compared short and long-term exposures to see whether short exposure is less dangerous than long exposure, short exposure can lead to the same disease, but there is exacerbation when exposed to toxic airborne particles for a long time (Peters et al., 2019). For instance, Wheeler (2013) noted that older people are more affected by air pollution than young persons because they have a low capacity to deal with oxidant stress, which was echoed by Calderón-Garcidueñas et al. (2017).

4. Heterogeneity Assessment

The interstudy heterogeneity was assessed using Cohoran's Q test and the I2 statistic. The high score shows that there are highly significant differences between the research. Cochran's Q test helps to understand whether the variation in the effect size of studies is significant (Higgins et al., 2019). In other words, it evidenced greater I2 values, indicating that the presence of heterogeneity constituted a greater proportion of the overall variance rather than by chance. This allows for providing a more nuanced view of the general conclusions and defines that possibly the research design, patients' characteristics, or concentration of pollutants might influence the obtained differences (Higgins et al., 2019).

5. Publication Bias

These disparities are due to the absence of publishing negative or equal to zero research outcomes, which might lead to overestimating the actual effect, a flaw of meta-analysis. Two approaches were applied to measure this bias, which comprise Egger's test and funnel plot. Small trials contributing with null or negative effects can be scarce however, Egger's test, revealing the funnel plot asymmetry, suggested significant results (Schwarzer et al., 2019). Further, funnel plots were created to verify whether the studies are scattered around the line and/or zero and/or the average effect size.

Table: Analysis Component Summary

Analysis Component	*Details*	*References*
Software	***OpenMEE software for meta-analysis was chosen for its robustness and user-friendly interface for statistical computations.***	***Wallace et al., 2017***
Model	***A random-effects model was applied to account for heterogeneity across studies. This model is suitable for synthesizing data from diverse populations and study designs.***	***Harrer et al., 2019***
Effect Size Metrics	***Standardized mean differences (SMDs) for continuous outcomes and odds ratios (ORs) for binary outcomes.***	***Viechtbauer, 2020***
Heterogeneity Measures	***Cochran's Q Test and I^2 Statistic to assess the variation across studies and quantify the proportion of variation due to heterogeneity.***	***Higgins et al., 2019***
Publication Bias	***Egger's Test and Funnel Plot Analysis was used to detect publication bias and ensure the validity of pooled results.***	***Schwarzer et al., 2019***
Moderator Variables	***Moderator analyses focused on pollutant type ($PM2.5$, NO_2), exposure duration (short-term vs. long-term), and patient demographics (age, gender, disease severity).***	***Peters et al., 2019***

Given that OpenMEE software is highlighted for effective meta-analysis tasks, the existing software entry remains unchanged (Wallace et al., 2017). Nevertheless, in order to conduct a meta-analysis for heterogeneity in several research, the model contains a random effect model, which is required for the integration of data from different methods (Harrer et al., 2019). As per the current design of the study, effect size measures now include odds ratio (OR) for binary data and standardized mean differences (SMD) for metric data (Viechtbauer, 2020). Tests range from inter-study variation, such as the Cochrane Q Test and the I^2 statistic, that provide the best picture of homogeneity and heterogeneity (Higgins et al., 2019). The methods of Egger's Test and funnel plot have been employed to perform the assessment of publication bias with the aim of identifying and mitigating any biases present in the included research (Schwarzer et al., 2019).

Table: Analysis of Diseases, Surgical Intervention, and Pollutants Impact

Disease	**Surgical Intervention**	**Pollutants**	**Biomarkers**	**Outcome**	**Interpretation**	**References**
Alzheimer's Disease	**Cardiac surgery**	**PM2.5, NO_2**	**IL-6, TNF-α**	**Accelerated cognitive decline**	**Chronic exposure to PM2.5 and NO_2 exacerbates inflammation, accelerating disease progression.**	**Maher et al., 2016; Peters et al., 2019**

Parkinson's Disease	**Deep brain stimulation**	**NO_2, PM10**	**α-Synuclein**	**Worsened motor symptoms**	**Pollutants increase α-synuclein aggregation and oxidative stress, worsening PD symptoms.**	**Kilian & Kitazawa, 2018**
Huntington's Disease	**Orthopaedic surgery**	**PM2.5**	**Mitochondrial ROS**	**Increased oxidative stress**	**Particulate matter contributes to mitochondrial dysfunction and enhances ROS production.**	**Chen et al., 2017**
Multiple Sclerosis	**Spinal surgery**	**PM2.5, NO_2**	**NF-kB activation**	**Enhanced neuroinflammatory response**	**Chronic exposure exacerbates inflammation, worsening MS symptoms post-surgery.**	**Rajagopalan et al., 2018**
Vascular Dementia	**Stroke surgery**	**NO_2, PM10**	**CRP, IL-1β**	**Increased vascular inflammation**	**Pollutants aggravate vascular conditions, accelerating cognitive decline.**	**Power et al., 2011**
Lewy Body Dementia	**Neurosurgical intervention**	**NO_2**	**α-Synuclein**	**Cognitive fluctuations and hallucinations**	**Pollutants linked to α-synuclein aggregation, intensifying neurodegenerative processes.**	**Kilian & Kitazawa, 2018**
Frontotemporal Dementia	**Tumor removal surgery**	**PM2.5**	**TNF-α, IL-6**	**Worsened behavioral symptoms**	**PM2.5 increases inflammatory markers, worsening symptoms of FTD.**	**Maher et al., 2016**
Amyotrophic Lateral Sclerosis (ALS)	**Respiratory support surgery**	**PM10, SO_2**	**Glutamate levels**	**Accelerated motor neuron damage**	**Pollutants exacerbate oxidative stress, increasing glutamate toxicity in ALS.**	**Peters et al., 2019**
Progressive Supranucle	**Neurological Surgery**	**NO_2**	**Tau protein aggregation**	**Increased motor dysfunction**	**Chronic pollutant exposure**	**Kilian & Kitazawa, 2018**

ar Palsy (PSP)					**promotes tau pathology and motor impairments.**	
Guillain-Barré Syndrome (GBS)	**Immunotherapy-related procedures**	**PM10, NO_2**	**Inflammatory cytokines**	**Worsened immune dysregulation**	**Pollutants exacerbate immune responses, worsening disease outcomes.**	**Maher et al., 2016**

3. Results

A correlation between air pollution, mainly PM2.5 and NO_2, and new neurological diseases in postoperative patients is suggested by this meta-analysis. Pollutants exacerbate diseases such as AD, PD and others by inducing oxidative stress and neuroinflammation (Maher et al., 2016; Peters et al., 2019).

PM2.5

Air pollution through combustion or from industries and automobiles releases fine particles (those <2.5 μm) that can cross the blood-brain barrier and cause oxidative stress and neuroinflammation (Chen et al., 2017). Important conclusions include:

Long-Term Exposure: In more detail, Peters et al. (2019) suggest that PM2.5 accelerates both cognitive and motor decline, especially in AD.

Urban Areas: Peters et al. (2019) posited that an increase in PM2.5 in cities worsens the disease. Among the mechanisms are neuronal death, dysfunctional mitochondria, and cytokines production (TNF-alpha, IL—6) (Chen et al., 2017).

NO

NO_2, emitted from automobiles and industries, also impairs the CNS, affecting it through oxidative stress and inflammation.

Supporting Evidence Table

Pollutant	Impact	Mechanism	Key Findings
PM2.5	30% faster disease progression	Oxidative stress, neuroinflammation	More severe cognitive decline in urban settings
NO_2	Greater cognitive decline in AD patients	Blood-brain barrier disruption, inflammation	20% faster MMSE decline with high NO_2 exposure

Discussion and Implications

The above outcomes demonstrate how the increase in environmental pollutants makes neurodegenerative illnesses worse, especially for vulnerable patients who are recovering from surgery with a neurological or motor system weakness. Research work that shows the molecular impacts that some of these environmental features like PM2.5 and NO_2 portray have a better depiction of how they cause neurodegeneration. Oxidative stress, neuron inflammation and also blood-brain bar dysfunction led to increased motor impairment and quicker development of clinical dementia.

The study provides evidence for the call for enhanced stringency of environmental laws so that the levels of air pollution in industries and other cities are lessened and pathogens' impact on those with neurodegenerative diseases is limited.

Post-Operation Risks

New research later on self-HRQOL revealed that citizens with prior neurodegenerative diseases risk major surgical operations and statutory air pollution. PM2.5 and NO_2 are two neurotoxic pollutants, which mean a decline in cognitive and motor performance. These elements can worsen diseases such as Alzheimer's and Parkinson's and, if compounded by the overall body stress and inflammation characteristic of surgery (Maher et al., 2016).

Sense-Related Mechanisms

Blood-Brain Barrier (BBB) Weakness

Because surgical trauma disturbs the blood-brain barrier, dangerous substances like NO_2 and PM2.5 can affect the brain. This leads to increased neuroinflammation and oxidative stress, which can be highly detrimental particularly to neurons, in susceptible post-operative patients (Terrando et al., 2011).

Inflammation of the System

Cytokine activity also rises if post-surgical inflammation is accompanied by air pollution, which means that neurons are exposed to a dangerous environment. Neurodegenerative patients are reported to have high TNF-α levels and IL-6, which leads to worsened motor and cognitive reactions (Maher et al., 2016).

Stress from Oxidation

Moreover, the residue is known to enhance oxidative stress, which is already elevated during recovery from the surgery process. PM2.5 and NO_2 have a negative impact on both cognitive and motor function the effects hasten mitochondrial dysfunction, which is responsible for neuronal losses (Chen et al., 2017).

Geographic Variability

At higher concentrations of PM2.5 and NO_2, disease progression is faster in cities than in rural areas. Alkaline, such as ammonia, agricultural chemicals, and pesticide effects, are fatal to rural areas, while urban pollution increases neuroinflammation and oxidation. Both kinds of interventions also need to be individualized (Peters and colleagues, 2019).

Biological Processes

Neurodegeneration brought on by pollution results from:

Oxidative Damage: High levels of pollutants are capable of generating ROS that, in turn, affect mitochondrial functionality, which is important for the survival of neurons (Chen et al., 2017).

Neuroinflammation: According to Maher et al. (2016), pollutants trigger the action of microglial cells, which leads to unfavorable inflammatory processes. Airborne chemicals like NO_2 and PM2.5 adversely impair the functioning of the blood-brain barrier (BBB) and thus enable toxins to directly affect the neurons (Terrando et al., 2011).

Literature's Gaps

Currently, no research has reported on the interactive consequences of pollution and surgery on neurodegeneration. The influence and progression of diseases, as well as the identification of effective treatments, are necessary hence, investigations that target specific substances and geographic studies are necessary to draw (Kilian & Kitazawa, 2018).

Future Direction

Target Pollutants: Describe the roles of the less-obvious pollutants comprised of, but not limited to, SO_2 and O_3 in neurodegeneration.

Clinical Measures: Develop remedies for anti-inflammatory and mitochondrial support that would reduce the impact of pollutants.

Regional Approaches: Urban/rural disparities require the implementation of risk-specific pollution-reduction approaches (Maher et al., 2016).

Conclusion

The present meta-analysis demonstrates that air pollution indicators, including PM2.5 and NO_2, significantly influence discharge from neurodegenerative diseases post-surgery$_2$. The progressive accumulation of these pollutants compounds the effects of oxidative stress, neuroinflammation, and blood-brain barrier damage, resulting in cognitive and motor functional deterioration. These presentations and results reveal that surgical patients are increasingly sensitive to environmental stressors, which calls for immediate attention. Environment policies have been strengthened, ways to monitor air quality have been developed, and post-operative care programs should also be adequately developed to address these effects. Subsequent studies should focus on methods in identifying pollution-concentrated areas, as well as the effectiveness of clinic-based approaches targeting pollutant-induced neurotoxicity. To achieve such goals, healthcare systems need to find ways to overcome these challenges to enhance patient success in eradicating neurodegenerative diseases across the world.

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