Systematic Review Article

The Effect of Particulate Matter on the Development of Dementia

Chelsea Li

Amador Valley High School, 1155 Santa Rita Road, Pleasanton, CA 94566, United States

ABSTRACT

Air pollution has been associated with risk to human health. Recently, an increasing amount of studies have found a connection between air pollution and a decline in cognitive function, specifically dementia. The purpose of this review is to evaluate existing literature to investigate if air pollution is truly a risk factor of dementia. The analysis explores the classification of particulate matter and its entryways into the brain, specifically detailing transport across the blood-brain barrier and translocation via the olfactory bulb. A systematic review of 20 relevant studies was conducted using MEDLINE, ScienceDirect, and Google Scholar to evaluate the link between particulate matter and cognitive decline. Studies were selected based on their focus on PM-induced neuroinflammation (cytokine levels), impaired neurogenesis (neuronal density), and altered neural activity (EEG data). While the majority of data stems from rodent models, human epidemiological findings are also analyzed to investigate the correlation between exposure levels with clinical diagnoses. The consolidated evidence suggests that PM heightens dementia risk by increasing pro-inflammatory cytokines and reducing neuronal density, though results regarding EEG oscillations remain varied, and epidemiological studies supported this conclusion. Future research should clarify the mechanistic thresholds of PM exposure and the impact of varying particle compositions, and more human studies should be conducted to verify the results of the rodent experiments.

Keywords: particulate matter; air pollution; dementia; cognitive function; neuroinflammation; neurogenesis; electroencephalography

INTRODUCTION

Dementia is defined as a cognitive disorder involving cognitive decline and deterioration of cognitive abilities. Dementia is most common among elderly individuals with a 7% prevalence among those 65 years globally, proving that it is quite a common syndrome. It is becoming increasingly relevant as publications on dementia on sites like Medline have increased by 5.6 times from the late 1900s to now (1-2). Therefore, it is necessary to characterize risk factors for developing dementia to help prevent it.

A recently discovered potential risk factor is exposure to air pollution, specifically particulate matter. Coming in a range of sizes, common kinds of particulate matter are PM 0.1, PM 2.5, and PM 10, *PM* representing *particulate matter* and the numbers following representing the diameters of the particles in micrometers (3, 4). Of the different sizes of particulate matter are subtypes such as diesel exhaust particles (DEPs). The California Air Resources Board states that 90% of DEPs are less than 1 μm in diameter, meaning it is part of the PM 2.5 category (5).

In recent years, an increasing number of studies

Corresponding author: Chelsea Li, E-mail: chelseali4567@gmail.com.

Accepted June 23, 2026
https://doi.org/10.70251/HYJR2348.43687695

are finding a connection between air pollution and dementia (6). However, the mechanisms involved are not completely known and still under investigation. Also, there is a lack of research that investigates different results across different variables such as different exposure levels or kinds of particulate matter. The purpose of this review is to address these discrepancies by showing how these pollutants enter the human body and analyzing important data regarding the pollutants' effect on cognitive function.

LITERATURE REVIEW

Air Pollution

First, small enough particulate matter is able to pass through the alveolar-capillary barrier. For instance, coarser particles such as PM 10 are usually deposited in large airways, whereas particles that are PM 2.5 or smaller are able to penetrate through the alveolar-capillary barrier and enter the bloodstream, reaching other internal organs such as the brain (7). However, not all particles are able to enter the brain as it is protected by the blood-brain barrier (BBB). After entering the blood, the particles 0.2 μm or smaller are also able to cross the BBB, passing into the brain. When there is an abundance of particulate matter circulating in the bloodstream, the BBB increases in permeability allowing larger particles (PM 2.5) to enter the brain as well (8). These two pathways are illustrated in Figure 1.

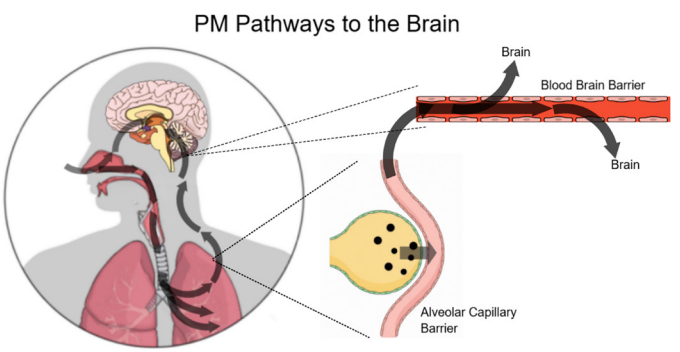

Figure 1. *The figure displays the pathway of inhaled PM into the brain through translocation through the olfactory bulb. It then illustrates a second pathway where the PM travels through the airways into the lungs, where it passes through the alveolar capillary barrier into the bloodstream, and the PM subsequently migrates through to the brain where it passes through the blood brain barrier.*

Furthermore, PM 0.1, also known as ultra-fine particles (UFP for short) may be able to bypass the BBB. Inhaled PM 0.1 deposits in the olfactory mucous membrane and is then able to travel along the olfactory nerve, reaching the olfactory bulb in the brain, highlighting a newly recognized and concerning pathway for air pollutant exposure to affect the central nervous system. In one study, rats were exposed to 160 μg/m³ of PM 0.1, with count median diameter being 36 nm, for 6 hours per day. Results showed measurable accumulation in the olfactory bulb, with concentrations increasing from 0.35 μg/g on Day 1 to 0.43 μg/g by Day 7, indicating persistent buildup. This study also concluded that in the rats, over 50% of inhaled PM 0.1 is accumulated in the nasopharynx and 20% of PM 0.1 deposited in the mucous membrane in that region can be translocated to the olfactory bulb (9). While it may appear minor initially, once particulate matter enters the brain, it significantly impacts key dementia risk factors including neuroinflammation, neurogenesis, and EEG wave patterns.

Neuroinflammation

Cytokines play a key role in regulating immune activity in the brain by facilitating communication between cells and controlling inflammatory processes. In Alzheimer's disease, disturbances in these signaling pathways lead to persistent inflammation, which accelerates neuronal injury and drives disease progression (10). Depending on their type and concentration, cytokines may either amplify or suppress inflammation. Inhaled PM 2.5 has been associated with increasing secretion of pro-inflammatory cytokines by inducing higher levels of oxidative stress, thus causing neuroinflammation (9). Therefore, to determine whether particulate matter leads to neuroinflammation or not, researchers measure the change in the amount of pro-inflammatory cytokines in the hippocampus after exposure to particulate.

Neurogenesis

Neurogenesis, or the formation of new neurons, occurs mainly in the sub-granular zone and sub-ventricular zone in the dentate gyrus, a structure in the hippocampus that processes information, as well as the olfactory bulb. Therefore, studies measuring the change in production of neurons in these regions of the brain will provide an accurate representation of neurogenesis in general. Neurogenesis is important to cognitive function in terms of memory because neurons, especially the signaling

between them, are greatly responsible for the ability to learn and form memories (11). Therefore, hindrances to neurogenesis can lead to cognitive impairment and a range of neuronal disorders.

Electrophalenceography (EEG)

Finally, by analyzing EEG data, the trend that particulate matter decreases neural activity in the hippocampus can be observed. EEG, also known as electroencephalography, is a non-invasive technique that records the brain's spontaneous electrical activity from the scalp, providing a direct measure of cortical neural oscillations. These oscillations are categorized, based on frequency, into four groups, delta, theta, alpha, beta, which reflect different states of brain arousal and processing (12).

METHODS AND MATERIALS

Search Strategy

To evaluate the relationship between air pollution and cognitive function, a systematic literature search was conducted across three primary electronic databases: MEDLINE via PubMed, Google Scholar, and ScienceDirect. MEDLINE and ScienceDirect were selected because they contain a wide range of reputable journals regarding neurological, biomedical, and environmental research, whereas Google Scholar was selected to obtain information, such as statistics involving particulate matter, published by organizations rather than academic journals. The search was limited to sources between January 2010 and May 2026 to ensure the usage of the most recent data. The search was executed using structured Boolean strings related to the exposure ("particulate matter", "PM2.5", or "PM") which were then combined using the AND operator with the following secondary terms: ("dementia"), ("neuroinflammation"), ("neurogenesis"), ("EEG"), and ("epidemiological" OR"human").

Study Selection

35 relevant sources were first identified by collecting articles that followed the pathway of particulate matter into the brain or measured air pollution's effect on neuroinflammation, neurogenesis, or neural activity. Finally, epidemiological studies of dementia cases after exposure to air pollution were collected. Afterwards, the sources used in the paper were selected by first excluding research involving pollutants other than particulate matter. Then, the rodent studies were further narrowed down based on the following criteria: whether they specifically measured neuroinflammation through the change in cytokines, neurogenesis through change in neuronal density in the sub-granular and sub-ventricular zones, and neural activity through EEG data. Ultimately, 27 sources were included in the paper with 25 peer reviewed studies and 20 of those studies analyzed in depth.

Data Extraction and Synthesis

Data was then sorted to ensure consistency on human and animal models. For rodent-based studies, information was collected on the specific type of pollutant, the concentration of exposure, and the daily and total duration of exposure. As for human studies, information was collected on sample size, the duration of follow-up, and the calculated hazard ratios or risk ratios associated with long-term exposure to particulate matter.

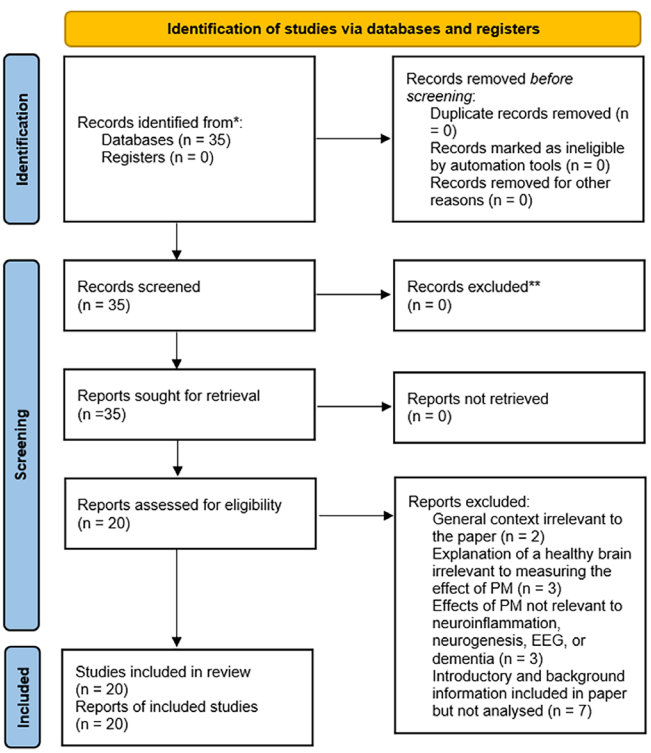

Figure 2. *PRISMA flow diagram illustrating the study selection process. Records were identified through database searches, screened for eligibility, and evaluated according to predefined inclusion and exclusion criteria, resulting in 20 studies included in the final review.*

RESULTS

A total of 20 peer-reviewed articles were examined in this review after applying selection criteria focused on particulate matter and cognitive outcomes. Table 1 indicates the specific pathways through which various pollutants exacerbate these neurological markers, synthesizing these findings linking environmental exposure to clinical indicators of cognitive decline.

Table 1. *Summary of experimental and epidemiological studies evaluating the neurological and cognitive effects of particulate matter exposure. Abbreviations: DEP, diesel exhaust particles; EEG, electroencephalography; HR, hazard ratio; IL, interleukin; nPM, nanoscale particulate matter; PM, particulate matter; TNF-α, tumor necrosis factor alpha.*

Model	Type of PM Used	Dosage	Results	Reference
Rodent	PM 0.1	160 μg/m³ (7 days)	Persistent buildup in the olfactory bulb (up to 0.43 μg/g).	[9]
Rodent	DEPs	300-350 μg/m³ (5 or 7 hrs/day)	Increased TNF-α, IL1β, and IL1α mRNA, decreased hippocampal neural density.	[13]
Rodent	PM 1	16.38.2 μg/m³ (3 months)	Increased hippocampal IL-6 production; brain impairment.	[9]
Rodent	Dusty PM	4 Groups: <150, 200–500, 500–2000, and 2000–8000 μg/m³ (30 min, 2x daily, 4 weeks)	Increased inflammatory cytokines and oxidative stress.	[14]
Rodent	nPM	340 μg/m³, 5 hrs/day, 3 days/week (Birth to 25 weeks)	20% decrease in anti-inflammatory cytokines (IL-4, 10, 13); 70% decrease in neurogenesis.	[15]
Rodent	DEPs	250-300 μg/m³	Decreased BrDU+ cells in sub-granular zone (males) and olfactory bulb.	[16]
Rodent	DEPs	25050 μg/m³	Significant reduction in Tbr2+ neurons by postnatal day 60.	[17]
Rodent	nPM	342 μg/m³	25% reduction in neurite density in CA1 (young mice); no effect in dentate gyrus or aged mice.	[18]
Rodent	PM 2.5	8 hrs/day (8 weeks)	Significant decrease in alpha and beta EEG wave activity.	[19]
Rodent	Dusty PM	2000-8000 μg/m³ (60 min/day for 10 days)	Decline in electrical activity in the hippocampus.	[20]
Rodent	PM 1	16.38.2 μg/m³ (3 months and 6 months)	No significant changes in EEG activity.	[9]
Human	DEPs	300 μg/m³	Increased beta EEG waves (stress response).	[21]
Human	DEPs	N/A	Cognitive impairment and memory loss in 16 adult men.	[22]
Human	PM 2.5	N/A	Positive association with dementia and Alzheimer's Disease.	[23]
Human	PM 2.5 / NO_2	N/A	Associated with 6.1% of dementia cases (HR 1.04 for PM 2.5).	[24]
Human	PM 2.5	N/A	Hazard ratio of 1.049 for dementia per μg/m³ annual exposure.	[25]
Human	PM 2.5	N/A	19.3% faster immediate recall decline; 14.8% faster learning decline.	[26]
Human	PM 2.5	N/A	Correlation between PM 2.5 exposure and physical AD brain pathology.	[27]

Neuroinflammation

A study exposed mice to 300-350 μg/m³ of DEPs for 12 days, with different groups experiencing two hours, five hours, and seven hours per day. A significant increase in TNF-α, IL1β, and IL1α mRNA levels was observed after the five- and seven-hour group, while the two-hour group stayed relatively close to unexposed levels. Also, the five- and seven-hour groups had significantly higher IL1α mRNA levels compared to the two-hour groups, and TNF-α and IL1β mRNA levels were significantly higher in the seven-hour groups and two-hour groups (13). Thus, the results suggest that this inflammatory response is both dependent on dose and duration. This trend is corroborated by longitudinal studies where even lower concentrations of PM1 (16.38.2 μg/m³ for 3 months) produced measurable increases in hippocampal IL-6 over a three-month period, notably sparing the olfactory bulb from similar inflammatory markers (9). Furthermore, four weeks of exposure to dusty PM led to an increase in inflammatory cytokines in adult mice proving consistency among findings of various kinds of PM (14).

Not only do pro-inflammatory cytokines increase in number, a study finds the secretion of anti-inflammatory cytokines decrease too. It found that after exposing mice to 340 μg/m³ of nano-particulate matter (nPM), also known as PM 0.2, from birth until 25 weeks of age for five hours a day three days per week, their levels of anti-inflammatory cytokines, specifically IL-4, IL-10, and IL-13, decreased by 20% (15). Therefore, this suggests that PM exposure not only triggers an inflammatory response but shifts the brain into a chronic pro-inflammatory state by reducing the ability to resolve inflammation. Consequently, this imbalance likely accelerates the progression of neurodegeneration.

Decreased neurogenesis

DEPs, as well as traffic related air pollution, have been found to decrease neurogenesis in the hippocampus. For example, a study found that after exposing adult mice to DEPs with density 250–300 μg/m³ decreased BrDU+ cells in the sub-granular zone and olfactory bulb for male mice, while female mice experienced a decrease in double-labeled NeuN+ and BrdU+ cells only in the olfactory bulb (16). This suggests that males may possess a higher hippocampal sensitivity to PM-induced damage.

Additionally, the timing of exposure is a critical determinant of long-term neurological outcomes. A study demonstrated that mice exposed to 250±50 μg/m³ of diesel exhaust particles during the first 21 days of life (6 hours/day, 5 days/week) exhibited a significant reduction in Tbr2+ neurons by postnatal day 60, regardless of sex (17). This is supported by another study that finds exposure to PM 0.2 traffic related air pollution, 340 μg/m³ for five hours per day three days a week from the second day of gestation until five years old, decreased neurogenesis for mice by 70% in the dentate gyrus (15). Thus, these studies imply that exposure to particulate matter during early, developmental years can lead to permanent damages in the hippocampus.

Furthermore, a study discovered that DEP (PM0.1) exposure for 300-350 μg/m3 for 5 and 7 hours per day led to a decline in neural density in the CA1 and CA3 regions in the hippocampus as well as dentate gyrus in male mice but not for those only exposed for 2 hours per day (13). This indicates that an increased length of exposure is associated with increased risk of the reduction of neurogenesis. Collectively, these findings imply that particulate matter hinders cognitive function through compromising the brain's long-term ability to learn and repair itself.

Conversely, mice exposed to demonstrated that young mice exposed to nanoscale particulate matter (PM 0.2) for ten weeks at 342 μg/m³ exhibited a 25% reduction in neurite density specifically in the hippocampal CA1 region, but the same levels were not present in the dentate gyrus and older mice (18 months) showed minimal further structural decline compared to aged controls. This highlights that while PM accelerates aging processes in younger brains, the same response is not present across all stages in life or hippocampal regions. (18).

Electroencephalography (EEG)

Adult mice who were exposed to PM 2.5 for eight hours per day for four, six, or eight weeks. The eight-week group showed a significant decrease in alpha and beta waves compared to the four- and six-week group as well as the control group. Also, the study noticed that the decrease in these waves occurred relatively later, thus why the eight-week group showed such notable changes (19). Therefore, these findings suggest higher length of exposure correlates to greater reductions in EEG activity and that there is a potential threshold length of exposure for observable alterations in EEG activity. Similarly, hippocampal-specific EEG recordings following dusty PM exposure (2000 - 8000 μg/m³ for 60 minutes each day, 10 consecutive days) and ischemia-reperfusion models (4VO I/R) demonstrated a marked decline in electrical activity in the hippocampus, a memory critical

region (20). Thus, the functionality of this region could decline, leading to cognitive impairment.

However, other studies reveal inconsistencies in results. When adult mice were exposed to 16.3 ± 8.2 (4.7 ~ 68.8) μg/m³ of PM1, there was no major difference after three and six months of exposure. The negligible change could be caused by the distance between the pathological location and the electrons placed on the animal's scalp (9). Moreover, a study conducted on ten human, male volunteers from 18 to 39 years old found that beta waves increased after exposure to DEPs (300 μg/m³), which indicates a stress response in the brain (21). Therefore, although the effect of PM on EEG varies, most results prove PM exposure disrupts EEG brainwaves in ways which may impact cognitive function.

Epidemiological studies

Aside from conclusions that can be drawn using rodent models, epidemiological studies have established the connection between air pollution and cognitive decline as well. For instance, a study on 16 adult men who were exposed to DEPs through their work environment displayed cognitive impairment, memory loss, and other health issues, proving the correlation between particulate matter and cognitive impairment on the small-scale (22).

However, large-scale studies yield similar results. Researchers observed 18.5 million people across the US for dementia and 19.2 million people for Alzheimer's disease and found that the presence of PM2.5 had a positive association with dementia and Alzheimer's, notably finding no association with dust (23). Additionally, a study on 2.1 million residents of Ontario who were initially dementia-free found that 257,816 individuals were diagnosed with dementia over a span of 13 years, with air pollution (PM2.5 and NO_2) being associated with 6.1% of those cases. PM 2.5 and NO2 had hazard ratios for dementia of 1.04 and 1.10 per interquartile range increase respectively, which are significant numbers in a high population (24). A study that analyzed 94 million follow-up records for Medicare patients found that per each μg/m³ of annual PM 2.5 exposure, the hazard ratio was 1.049 (25). The massive-scale studies not only prove particulate matter's effect on the risk for dementia but also establish PM2.5 specifically as a main driver of neurodegeneration.

Environmental exposure to particulate matter is mainly seen to affect memory and learning. A study found that in 998 older women (ages 73 – 87), each 2.81 μg/m³ increase in long-term PM 2.5 exposure was linked to 19.3% faster decline in immediate recall and 14.8% faster decline in new learning. Higher PM 2.5 was also associated with brain changes indicating greater Alzheimer's risk, mediating 22.6% of effects on immediate recall and 10.7% of effects on learning impairment observed. The results show specifically the areas of cognitive function possibly impacted by exposure. The associations persisted after adjusting for dementia, stroke, and vascular disease, suggesting PM 2.5 accelerates early Alzheimer's-related decline, so PM 2.5 acts independently as a catalyst for Alzheimer's rather than only exacerbating existing conditions (26).

A critical validation of the particulate-dementia link is found in post-mortem data, which provides direct anatomical evidence of environmental exposure causing neurodegeneration. In a study of 602 autopsy cases, higher exposure to PM 2.5 before death was linked to more severe Alzheimer's disease–related brain changes. This finding is significant because it moves beyond behavioral observation to physical evidence. Furthermore, in this study, among 287 individuals with cognitive assessments, greater PM 2.5 exposure was also associated with worse cognitive and functional impairment, and about 63% of this relationship was explained by Alzheimer's disease–relating brain pathology (27). Therefore, since this study finds the same cognitive assessment results as other studies, the physical damage caused by exposure to particulate matter is likely the cause of the common observations in cognitive decline.

DISCUSSION

This review aimed to evaluate whether air pollution, specifically particulate matter, is a risk factor for cognitive decline and dementia. Overall, the findings support this hypothesis, although results vary across study types and methodologies.

The changes in neuroinflammation and neurogenesis can increase risk for neurodegenerative diseases such as dementia. Increased levels of pro-inflammatory cytokines along with decreased levels of anti-inflammatory cytokines suggests that particulate matter may not only cause the brain to experience inflammation associated with neurodegenerative diseases but also make it difficult for the brain to leave that state (9, 13, 14). On the other hand, the decline in neurogenesis tracked through reductions in neuronal markers could indicate another possible method of progression for dementia (15-18). However, the differences in results displayed among the different brain regions and sexes

indicates the need for further research on the specific vulnerabilities. Additionally, variability in EEG results and differences in experimental conditions indicate that the neurological effects of particulate matter are complex (9, 19-21). These factors highlight the need to investigate effects of methodological differences such as exposure duration and measurement techniques.

Epidemiological studies further support the association between particulate matter and cognitive decline, applying results from rodent models into large-scale societal observations (22-27). The epidemiological evidence suggests that reductions in annual PM 2.5 levels could significantly lower the global burden of dementia, which showcases the importance of maintaining air quality to improve public health.

A primary strength of this review is its multi-dimensional approach, including from molecular to population-level data. However, several limitations must be noted. Most mechanistic evidence relies on rodent models, which may not perfectly replicate human longitudinal exposure. However, conducting more mechanistic studies would help clarify and confirm the effect of air pollution on the biological markers causing dementia. Additionally, further investigation into the effects of different particle sizes and compositions may help explain inconsistencies observed across current studies. Furthermore, there is a need for studies to investigate at what specific concentration and duration PM exposure becomes irreversible. Finally, more human studies aside from epidemiological studies are necessary to confirm observations made with rodent studies.

CONCLUSION

This review aimed to examine the correlation between air pollution and dementia. Through analyzing pollutants' effects on cytokine production, neuronal markers, and EEG waves, studies suggest that particulate matter such as PM 2.5 is associated with neuroinflammation, disrupted neurogenesis, and changes in electrical activity. Consequently, these factors can contribute towards the development of dementia, which is corroborated by studies on various groups of people in environments with differing amounts of pollution. Notably, the field still lacks detailed mechanistic insight into how particulate matter exerts these effects and specifics about the effects of different kinds of particulate matter among different variables. Additionally, future research should be conducted to establish the specific exposure concentrations at which brain damage becomes permanent. Addressing these ambiguities is essential for designing effective clinical interventions and creating targeted environmental policies to protect populations worldwide.

ACKNOWLEDGEMENTS

I would like to acknowledge the professors and mentors at Cambridge Centre for International Research who have helped me learn about neuroscience and review my paper.

CONFLICT OF INTEREST

The author declares that there are no conflicts of interest related to this work.

REFERENCES

1. Gale SA, Acar D, Daffner KR. Dementia. *The American Journal of Medicine*. 2018 Oct; 131 (10): 1161-9. https://doi.org/10.1016/j.amjmed.2018.01.022
2. Theander SS, Gustafson L. Publications on dementia in Medline 1974-2009: a quantitative bibliometric study. *International Journal of Geriatric Psychiatry*. 2012 Jun 15; 28 (5): 471-8. https://doi.org/10.1002/gps.3848
3. Arias-Pérez RD, Taborda NA, Gómez DM, Narvaez JF, Porras J, Hernandez JC. Inflammatory effects of particulate matter air pollution. *Environmental Science and Pollution Research*. 2020 Sep 1; 27 (34): 42390-404. https://doi.org/10.1007/s11356-020-10574-w
4. Oberdörster G, Sharp Z, Atudorei V, Elder A, Gelein R, Kreyling W, *et al*. Translocation of inhaled ultrafine particles to the brain. *Inhalation Toxicology*. 2004 Jun 1; 16 (6-7): 437-45. https://doi.org/10.1080/08958370490439597
5. Sacuiu SF. Dementias. *Handbook of Clinical Neurology*. 2016; 138: 123-51. https://doi.org/10.1016/B978-0-12-802973-2.00008-2
6. Roy R, Amedeo D'Angiulli. Air pollution and neurological diseases, current state highlights. *Frontiers in neuroscience*. 2024 Mar 6; 18: 1351721. https://doi.org/10.3389/fnins.2024.1351721
7. California Air Resources Board. Overview: Diesel Exhaust & Health (Internet). Ca.gov. Available from: https://ww2.arb.ca.gov/resources/overview-diesel-exhaust-and-health. (accessed on 2026-4-12)
8. Costa LG, Cole TB, Coburn J, Chang YC, Dao K,

Roqué PJ. Neurotoxicity of traffic-related air pollution. *NeuroToxicology*. 2017 Mar 1; 59: 133-9. https://doi.org/10.1016/j.neuro.2015.11.008

9. Shih CH, Chen JK, Kuo LW, Cho KH, Hsiao TC, Lin ZW, *et al*. Chronic pulmonary exposure to traffic-related fine particulate matter causes brain impairment in adult rats. *Particle and Fibre Toxicology*. 2018 Nov 9; 15 (1). https://doi.org/10.1186/s12989-018-0281-1
10. Rather MA, Khan A, Alshahrani S, Rashid H, Qadri M, Rashid S, *et al*. Inflammation and Alzheimer's Disease: Mechanisms and Therapeutic Implications by Natural Products. Oliveira SHP, editor. *Mediators of Inflammation*. 2021 Aug 2; 2021: 1-21. https://doi.org/10.1155/2021/9982954
11. Horgusluoglu E, Nudelman K, Nho K, Saykin AJ. Adult neurogenesis and neurodegenerative diseases: A systems biology perspective. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*. 2016 Feb 16; 174 (1): 93-112. https://doi.org/10.1002/ajmg.b.32429
12. Malver LP, Brokjaer A, Staahl C, Graversen C, Andresen T, Drewes AM. Electroencephalography and analgesics. *British Journal of Clinical Pharmacology*. 2014 Jan 1; 77 (1): 72-95. https://doi.org/10.1111/bcp.12137
13. Ehsanifar M, Jafari AJ, Montazeri Z, Kalantari RR, Gholami M, Ashtarinezhad A. Learning and memory disorders related to hippocampal inflammation following exposure to air pollution. *Journal of Environmental Health Science and Engineering*. 2021 Jan 22; 19 (1): 261-72. https://doi.org/10.1007/s40201-020-00600-x
14. Hajipour S, Farbood Y, Gharib-Naseri MK, Goudarzi G, Rashno M, Maleki H, *et al*. Exposure to ambient dusty particulate matter impairs spatial memory and hippocampal LTP by increasing brain inflammation and oxidative stress in rats. *Life Sciences*. 2019 Dec 23; 242: 117210. https://doi.org/10.1016/j.lfs.2019.117210
15. Woodward NC, Haghani A, Johnson RG, Hsu TM, Saffari A, Sioutas C, *et al*. Prenatal and early life exposure to air pollution induced hippocampal vascular leakage and impaired neurogenesis in association with behavioral deficits. *Translational Psychiatry*. 2018 Nov 29; 8 (1). https://doi.org/10.1038/s41398-018-0317-1
16. Coburn JL, Cole TB, Dao KT, Costa LG. Acute exposure to diesel exhaust impairs adult neurogenesis in mice: prominence in males and protective effect of pioglitazone. *Archives of Toxicology*. 2018 Mar 10; 92 (5): 1815-29. https://doi.org/10.1007/s00204-018-2180-5
17. Cole TB, Chang YC, Dao K, Daza R, Hevner R, Costa LG. Developmental exposure to diesel exhaust upregulates transcription factor expression, decreases hippocampal neurogenesis, and alters cortical lamina organization: relevance to neurodevelopmental disorders. *Journal of Neurodevelopmental Disorders*. 2020 Dec 1; 12 (1). https://doi.org/10.1186/s11689-020-09340-3
18. Woodward NC, Pakbin P, Saffari A, Shirmohammadi F, Haghani A, Sioutas C, *et al*. Traffic-related air pollution impact on mouse brain accelerates myelin and neuritic aging changes with specificity for CA1 neurons. *Neurobiology of Aging*. 2017 May; 53: 48-58. https://doi.org/10.1016/j.neurobiolaging.2017.01.007
19. Li H, Guo X, Li P, Gao X, Song X, Chen X, *et al*. Particulate matter induces depression-like behavior through systemic inflammation and brain-derived neurotrophic factors. *Environment International*. 2024 Jul 20; 194: 108883. https://doi.org/10.1016/j.envint.2024.108883
20. Mirshekari Jahangiri H, Sarkaki A, Farbood Y, Dianat M, Goudarzi G. Gallic acid affects blood-brain barrier permeability, behaviors, hippocampus local EEG, and brain oxidative stress in ischemic rats exposed to dusty particulate matter. *Environmental Science and Pollution Research*. 2019 Dec 17; 27 (5): 5281-92. https://doi.org/10.1007/s11356-019-07076-9
21. Crüts B, van Etten L, Törnqvist H, Blomberg A, Sandström T, Mills NL, *et al*. Exposure to diesel exhaust induces changes in EEG in human volunteers. *Particle and Fibre Toxicology*. 2008 Mar 11; 5 (1). https://doi.org/10.1186/1743-8977-5-4
22. Kilburn KH. Effects of Diesel Exhaust on Neurobehavioral and Pulmonary Functions. *Archives of Environmental Health (Internet)*. 2000 Jan 1; 55 (1): 11-7. https://doi.org/10.1080/00039890009603379
23. Shi L, Zhu Q, Wang Y, Hao H, Zhang H, Schwartz J, *et al*. Incident dementia and long-term exposure to constituents of fine particle air pollution: A national cohort study in the United States. *Proceedings of the National Academy of Sciences*. 2022 Dec 27; 120 (1). https://doi.org/10.1002/alz.067071
24. Chen H, Kwong JC, Copes R, Hystad P, van Donkelaar A, Tu K, *et al*. Exposure to ambient air pollution and the incidence of dementia: A population-based cohort study. *Environment International*. 2017 Nov 1; 108: 271-7. https://doi.org/10.1016/j.envint.2017.08.020
25. Lee M, Schwartz J, Wang Y, Dominici F, Zanobetti A. Long-term effect of fine particulate matter on hospitalization with dementia. *Environmental*

Pollution. 2019 Nov (cited 2020 Oct 8); 254: 112926. https://doi.org/10.1016/j.envpol.2019.07.094

26. Younan D, Petkus AJ, Widaman KF, Wang X, Casanova R, Espeland MA, *et al*. Particulate matter and episodic memory decline mediated by early neuroanatomic biomarkers of Alzheimer's disease. *Brain: A Journal of Neurology*. 2020 Jan 1; 143 (1): 289-302. https://doi.org/10.1093/brain/awz348
27. Kim B, Blam K, Elser H, Xie SX, Van Deerlin VM, Penning TM, *et al*. Ambient Air Pollution and the Severity of Alzheimer Disease Neuropathology. *JAMA Neurology*. 2025 Sep 8; 82 (11). https://doi.org/10.1001/jamaneurol.2025.3316