Systematic Review of Environmental Risk Factors for Parkinson's Disease: Insights from Recent Studies

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Introduction: The etiology of Parkinson's disease (PD) is unknown, however, several environmental risk factors may increase risk for developing PD. The aim of this review was to search recently published literature and provide a summary of environmental risk factors for PD.
Methods: This paper searched PubMed from January 1, 2024 to April 21, 2024 to find systematic reviews and meta-analyses of studies examining environmental risk factors and their associations with PD. The eligibility criteria required papers to be in English and to specifically investigate environmental risk factors for PD. It excluded those focused on gene-environment interactions, other neurodegenerative diseases, demographic factors, and molecular mechanisms. A total of 145 reviews were identified, with 18 meeting the inclusion criteria after applying the eligibility requirements.
Results: The search identified 145 articles and included 18 in this umbrella review. In reviewing these articles, several environmental risk factors in association with PD were identified. Particulate matter, NO2, NOx, O3, CO, metals such as manganese, copper, and lead, pesticide exposure, n-6 polyunsaturated fatty acids, dairy consumption, and trichloroethylene, increase the risk of developing PD. Other environmental risk factors such as vitamin D exposure, smoking and nicotine usage, caffeine, diets rich in antioxidants, flavonoids, and polyunsaturated fatty acids, decrease the risk of developing PD.
Conclusion: While PD does not have a cure, taking precautions in avoiding certain environmental risk factors will reduce one's risk of developing PD.

Keywords: Parkinson's disease, risk factors, environment, oxidative stress, heavy metals, occupational exposure, neuroinflammation

Introduction

Parkinson's disease (PD) is the second most widespread neurodegenerative disease, following Alzheimer's disease[1]. Over one million Americans suffer from PD and the number is only expected to increase over the next decades with the aging global population[2,3]. PD is caused by the degeneration of neurons and accumulation of Lewy bodies in the nervous system, leading to motor dysfunction, slow movement, postural instability and rigidity, resting tremors, anxiety, depression, and progressive cognitive decline[4]. These symptoms significantly lower the quality of life for individuals with PD, emphasizing the urgent need to better understand the disease's etiology and risk factors.

While genetic factors for PD have been extensively studied and are known to contribute to PD risk, they make up only a small portion of cases. The exact cause of PD is unknown, suggesting that environmental risk factors may play a role in disease development. However, compared to genetic research, environmental risk factors are comparatively understudied, and their effects on PD are not yet fully understood.

Understanding the relationship between environmental factors and the development of PD is vital to illuminate the risk factors and prevent PD. Recent reviews and meta-analyses have primarily focused on genetic risk factors, leaving a gap in the synthesis of research on environmental risk factors for PD. Previous reviews, such as Bellou et al. (2016), have identified and synthesized environmental risk factors for PD up until 2016. However, since 2016 there have been new insights into the role of environmental factors in PD. The aim of this review was to search the recently published literature and provide an overview of recent literature on the environmental risk factors of PD.

Methods

Umbrella Review

This review followed an umbrella review, which involved collecting and evaluating systematic reviews and meta-analyses focused on a specific research topic. Umbrella review methodology enabled a synthesis of a large number of existing reviews.

Search strategy

This paper searched PubMed from January 1, 2024 to April 21, 2024 to find systematic reviews and meta-analyses of studies examining environmental risk factors and their associations with PD. To refine the search strategy, several scoping reviews were conducted to assess the volume of research and identify gaps in the literature. Additionally, the scoping reviews clarified the definition of environmental risk factors and refined the inclusion and exclusion criteria. A similar review on environmental risk factors for PD was published in 2016 by Bellou et al. but only pertained to things prior to 2016, which is why this review specifically covers literature published since 2016. This enabled the review to make a novel contribution to the published literature and ensured only the most recent environmental risk factors were included. Additionally, tools such as AMSTAR-2 and Cochrane guidelines were not used to evaluate the quality of the studies.

Search Terms

The search for this paper included the terms "Parkinson disease" or "Idiopathic Parkinson disease", "risk factor" and "environment". It also included all the relevant MeSH terms associated with Parkinson's Disease.

To conduct the search, first, this paper located articles using each of the individual search terms. Second, the individual search terms were grouped (separated by the Boolean operators "OR" or "AND") and then used to conduct a search of the grouped terms. Third, this paper used a filter to include only meta-analyses, reviews, and systematic reviews. Finally, another filter was applied to include only studies published after January 1, 2016.

The last search was conducted on April 21, 2024. The details for the final PubMed search strategy was:

((Parkinson Disease, Idiopathic) OR (Parkinson disease)) OR (Idiopathic Parkinson's Disease)) OR (Idiopathic Parkinson Disease)) OR (("Parkinson Disease/classification"[Mesh] OR "Parkinson Disease/complications"[Mesh] OR "Parkinson Disease/diagnosis"[Mesh] OR "Parkinson Disease/epidemiology"[Mesh] OR "Parkinson Disease/ethnology"[Mesh] OR "Parkinson Disease/etiology"[Mesh] OR "Parkinson Disease/history"[Mesh] OR "Parkinson Disease/prevention and control"[Mesh] OR "Parkinson Disease/psychology"[Mesh]))) AND (risk factor)) AND (environment) Filters: Meta-Analysis, Review, Systematic Review, from 2016 - 2024

Eligibility Criteria

The found reviews were then assessed with the following inclusion and exclusion criteria:

Inclusion Criteria

The inclusion criteria required the paper to be in English and investigate an environmental risk factor of PD.

Exclusion Criteria

The exclusion criteria excluded articles that (1) focused on epigenetics or gene-environment interactions, (2) did not focus on PD specifically, covered neurodegenerative diseases as a whole, (3) focused on other risk factors such as demographic, genetic, lifestyle, or infections and viruses, (4) focused on the mechanisms or how environmental risk factors affected the body on a molecular level.

Study selection

Overall, 145 reviews were found and 18 were included in the final review. Using the search terms, there were 145 records found, of which 120 were excluded after applying the eligibility criteria to their abstracts. After reading the full-text versions of the remaining reviews, an additional 7 articles were excluded because they did not follow the eligibility criteria, leaving the 18 reviews included in this paper. The final number of reviews can be explained by how understudied the environmental risk factors for PD are and that many studies did not meet eligibility criteria by focusing on genetic factors, molecular mechanisms, or other neurodegenerative diseases

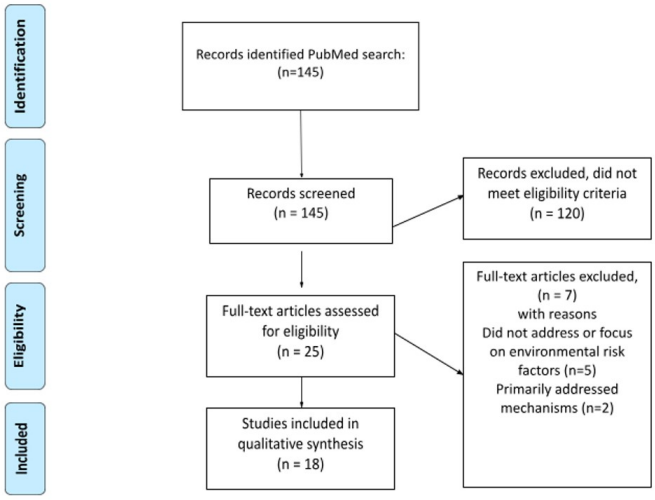

Fig. 1 Prisma diagram outlining study selection.

The diagram outlines the process of identifying, screening, and selecting studies for inclusion in the review. A total of 145 records were identified through database searches, of which 120 were excluded based on title and abstract screening. After full-text review, an additional 7 articles were excluded, leaving

18 studies that met the eligibility criteria and were included in the final review.

Results

This review identified several environmental risk factors associated with PD, which were categorized into those with positive and negative associations.

Among the factors increasing PD risk, air pollution and particulate matter (NO2, NOx, O3, CO,) were consistently linked to PD, as they are shown to cause oxidative stress and neuroinflammation[3]. Metal exposure, particularly to manganese, copper, and lead, was another factor linked to PD[4,5]. However, the evidence for some metals remains inconclusive and requires more research. Pesticide exposure emerged as a major risk factory, with various studies highlighting the neurotoxic effects of compounds such as paraquat, rotenone, and organochlorines[6]. Additionally, trichloroethylene, a common industry solvent, was linked directly to PD, with some studies reporting a 500% increased risk following exposure[7]. Dietary factors such as n-6 polyunsaturated fatty acids and dairy consumption were also linked to higher PD risk, likely due to contaminants or inflammatory properties[8].

Factors found to have protective effects against PD include Vitamin D exposure, attributed to its anti-inflammatory and neuroprotective properties[9]. Smoking and nicotine usage also showed an inverse relationship with PD, although the harmful effects of smoking counterbalance its use as a preventive measure[10,11]. Caffeine intake and diets rich in antioxidants, flavonoids, and polyunsaturated fatty acids were also linked to lower PD risk, suggesting that dietary changes may play a role in disease prevention[2].

While these findings provide valuable insights, there are still inconsistencies, specifically with particulate matter and metals. These gaps of information underscore the need for further research.

Summary of Common Patterns, Limitations, and Inconsistencies

Across the studies, several consistent patterns, or common findings, emerged. Air pollution and pesticide exposure were repeatedly associated with increased PD risk, likely due to their roles in inducing oxidative stress and neuroinflammation. Similarly, metal exposures and trichloroethylene showed strong positive associations with PD. On the other hand, vitamin D, caffeine, and diets rich in antioxidants and flavonoids were consistently linked to reduced PD risk, suggesting potential protective effects.

However, there were contradictory findings with the effects of particulate matter. For example, some studies reported strong associations between PM exposure and PD risk, while others found no relationship. Several studies also exhibited heterogeneity among its results, specifically in relation to air pollution, occupational metal exposure, and pesticide exposure. These inconsistencies could also stem from differences in study design, exposure assessment methods, and geographic differences.

Additionally, as this paper is compiled of systematic reviews and meta-analyses, it is important to consider publication bias and study quality. Publication bias, where studies with significant results are more likely to be published, may have influenced the findings. Variations in study quality too, may affect the reliability of results. For example, differences in sample sizes, exposure assessment methods, and study designs may all affect the reliability. Studies using self-reported data or estimates may introduce bias, whereas those using precise measurements provide more reliable results. Standardizing methods in future research could work to prevent these inconsistencies. Despite these limitations however, the overall body of evidence suggests that air pollution and particulate matter remain important areas of investigation in PD research.

The main findings of these studies that are concluded in this review are described in Table 1.

Umbrella Review

Air pollution and particulate matter

Air pollution and particulate matter (PM) exposure have been linked to numerous adverse effects on human health, such as brain inflammation and oxidative stress, both of which are processes known for developing and progressing PD[3]. A literature review done by Palacios found that ozone, NOx, NO2, CO, and traffic air pollution had positive associations with developing PD, while PM2.5-10 had no association[3]. However, Palacios found contradicting studies between PD and PM10, PM2.5, NO2 or traffic air pollution. Dhiman et al. researched the effects of ambient air pollution in relation to PD and found that NO2, O3, PM2.5, and CO increased the risk of developing PD[12]. They were unable to determine the association between PD and pollutants like SO2, PM10, and NOx because of the lack of existing literature on them[12]. A review done by Hu et al. (2019) found a positive association between PD and long-term exposure to NO2, NOx, CO, and O3, and no statistically significant associations between long-term PM exposure and PD. It was acknowledged that there is high bias in these results as much of the information was derived from observational studies, which have ingrained biases such as selection and information bias[14]. Wang et al. focused on the effects of long-term particulate matter exposure on PD risk and found no statistically substantial association between PD and long-term PM2.5 and PM10 exposure. However, the possibility of risk between PM2.5/PM10 and PD risk should not be ruled out because of their variation and large effect size[11]. The increased PD risk is likely caused through mechanisms such as neuroinflammation and oxidative

Table 1 Summary of Included Studies: Author(s), Main Topic, Study Design, Number of Studies Included, and Relationship to PD Risk. Information on author(s), main topic, methodology summary, and main finding of review papers used in this paper.

STUDY	MAIN TOPIC	DESIGN	METHODOLOGY SUMMARY	NO. OF STUDIES	RELATIONSHIP TO PD
Boulos et al. (2019)[8]	Nutritional Risk Factors	Review	Predefined search terms	68	Dairy increases; uric acid, PUFAs, coffee protective
Chambers-Richards et al. (2023)[4]	Pesticides, Metals, EM Fields	Systematic Review	Predefined search terms	24	All increase PD risk
Delamarre and Meissner (2017)[2]	Environmental Risk Factors	Review	Literature synthesis	N/A*	Smoking, coffee, tea decrease; pesticides increase
Dhiman et al. (2023)[12]	Ambient Air Pollution	Meta-analysis	Statistical pooling	11	NO2, O3, PM2.5, CO increase risk
Dorsey et al. (2023)[7]	Trichloroethylene	Review	Predefined search terms	8	Increases PD risk
Erro et al. (2023)[7]	Nutrition	Literature Review	Literature synthesis	N/A*	No relationship
Gonzalez-Alvarez et al. (2022)[5]	Metals	Narrative Review	Literature synthesis	14	Increased risk
Gunnarson and Bodin (2017)[13]	Occupational Exposures	Systematic Review	Predefined search terms	47	Pesticides increase; others no relationship
Hu et al. (2019)[14]	Air Pollution (PM2.5, NO2, etc.)	Systematic Review	Predefined search terms	10	NO2, NOx, CO, O3 increase risk
Ma et al. (2017)	Nicotine	Review	Literature synthesis	N/A*	Decreased risk
Maggio et al. (2019)[15]	Light	Review	Literature synthesis	N/A*	Artificial light increases; infrared decreases
Marras et al. (2019)[16]	Environmental Risk Factors	Review	Literature synthesis	N/A*	Pesticides, dairy increase; vit D, smoking decrease
Nandipati and Litvan (2016)[13]	Environmental Risk Factors	Review	Literature synthesis	N/A*	Pesticides increase; metals no relationship
Palacios (2017)[3]	Air Pollutants	Literature Review	Literature synthesis	14	Ozone, PM, NOx, CO, traffic pollution increase
Tranchant et al. (2019)[10]	Environmental Risk Factors	Review	Literature synthesis	N/A*	Paraquat, maneb, rotenone increase risk
Wang et al. (2020)[11]	Particulate Matter	Review	Literature synthesis	6	No relationship
Yan et al. (2018)[6]	Pesticide Exposure	Meta-analysis	Statistical pooling	10	Increased risk
Zhou et al. (2019)[9]	Vitamin D	Lit Review/Meta-analysis	Statistical pooling	8	Vitamin D deficiency increases risk

Note: *"n/a" indicates that the number of studies included was not explicitly stated in the original review or meta-analysis.*

stress[3].

Many of the reviews found positive associations between PD and NO2, NOx, O3, and CO while findings indicating the association between PM2.5 and PM10 were contradictory. Hu et al. and Wang et al. found that exposure to PM2.5/PM10 had no statistically substantial risk associated with PD, but the possibility should not be ruled out. All four studies recommended further investigation into air pollution and particulates and their associations with PD for a clearer picture of the effects.

Occupational exposures

Metals

Some occupations face harmful environmental exposures that may increase an individual's risk of developing PD. Oftentimes, welding is used as an example of an occupation that exposes individuals to, sometimes, harmful metals. Chambers-Richards et al. (2023) found that out of the fourteen articles used to examine the relationship between metal exposures and PD in their study, nine out of fourteen studies showed an association be-

tween occupational metal exposures and PD, with one reporting a significant increase in its association with PD. Palacios (2017) found that airborne metals, such as manganese, copper, and lead had positive associations with PD. A review by Nandipati and Litvan (2016) found that studies relating to metals such as manganese and iron were inconclusive and did not support associations with PD. Gunnarson and Bodin (2017) investigated the link between welding and PD risk and found a slightly reduced risk estimate regarding exposure to welding. A reason for this may be the healthy worker effect, the selection of healthier people into highly skilled physical work[13]. In a review on exposure to manganese, mercury, and iron, Gonzalez-Alvarez et al. (2022) found that a fraction of the studies used suggested an association between the metals and PD and conclude that further research is required to confirm whether or not there is a potential risk of metal exposure and its relationship to PD.

Overall, the reviews suggest that metal exposure is a risk factor for PD, specifically manganese, copper, and lead. However, because of the lack of research on many of these metals and their associations with PD, further research would greatly benefit and verify the risk associations between the metals and PD.

Pesticides

Pesticide families such as carbamates, organochlorines, and organophosphates have adverse effects on the nervous system and recent studies have been made investigating the association between pesticides and PD[6]. Chambers-Richards et al. (2022) found that out of the twelve studies used to examine the relationship between metal exposures and PD, eleven of them reported an association, with half of the studies showing a significantly increased association.

Nandipati and Litvan (2016) investigated pesticides such as rotenone, paraquat, maneb, organochlorines, organophosphates, and insecticide, pyrethroids, and found that all of them had associations with PD. Tranchant (2019) also found that exposure to paraquat, rotenone, and maneb all showed signs of increased PD risk. Gunnarson and Bodin (2017) concluded similar results and that exposure to any pesticide exerts an increased risk for PD. These chemicals are known to induce oxidative stress and neuroinflammation, which are key mechanisms in the pathology of PD[3]. Higher and longer-term exposures correlating with greater PD risk have also been observed, as Yan et al. (2018) found that pesticide exposure was a risk factor for PD, and 5 to 10 years of exposure was associated with a 5% and 11% increase in PD risk, respectively. Marras et al. (2019) found that pesticide exposure is associated with PD risk and that well water consumption was also associated with PD due to pesticide contamination. Delamarre and Meissner (2017) concluded similar results with pesticides showing an increased risk of PD.

This concludes that pesticide exposure is associated with an increased risk for PD. However, there are still limitations as many of the studies used examined entire families of pesticides, with little to no data done on specific agents.

Electromagnetic exposures

Electromagnetic exposures are often encountered in occupations such as being an electrician. Chambers-Richards et al. (2023) found that out of nine articles used to examine the relationship between electromagnetic exposures and PD, six studies reported an association with PD. Gunnarsson and Bodin (2017) found no associations between electromagnetic exposures and PD. Due to the lack of research on electromagnetic exposures, it is difficult to reach a conclusion on the relationship between electromagnetic exposures and PD.

Light and Vitamin D

Vitamin D, or 25-hydroxyvitamin D, is often derived from diet and sunlight and its deficiency is associated with a variety of pathological changes in many internal systems as well as an increased risk for several chronic diseases, including PD[9]. Zhou et al. (2019) found that together, meta-data analyses showed that individuals deficient in vitamin D were significantly more likely to develop PD. Maggio et al. (2019) saw similar results, finding that a deficiency in vitamin D is associated with Parkinson's disease. Some evidence for a link between PD and excess exposure to artificial or natural light was found, but more focused studies are needed to further verify this association[15]. Infrared lights have been found to be beneficial in the treatment of PD and have been considered for therapeutic purposes[15]. In terms of diet, Marras et al. (2019) found that higher levels of vitamin D have been shown in multiple studies to decrease PD risk.

Overall, making sure to get adequate amounts of vitamin D a day through both sunlight and diet is equally important in the prevention of PD. Furthering research in the effects of overexposure to light would help cement the relationship between light and PD even more.

Smoking and Nicotine

Although smoking is associated with a variety of adverse health effects, it has been associated with a lower risk of PD. Researchers found a significantly lower risk of PD for smoking 15 to 24 years before symptom onset, but smoking 25+ years before onset did not lower the risks[10]. For tobacco chewing and snus use, the same inverse relationship with PD was seen again[10]. Various flora and vegetables that nicotine can be naturally found in also have an inverse association with PD, albeit with much lower nicotine intake than from tobacco[10]. Delamarre and Meissner (2017) found numerous studies focused on the inverse correlation between smoking and PD risk. Marras et al. (2019) found that PD and smoking has an inverse relationship, as well as passive smoking and PD. Palacios (2017) investigated second-hand smoking in relation to PD and found that with the exception of one study, most evidence suggests a

protective relationship between passive smoking and PD. This protective effect is thought to be caused by nicotine's ability to modulate dopamine release and reduce oxidative stress[10].

Overall, smoking and nicotine usage correlates an inverse association with PD. However, because of the numerous harmful effects caused by smoking and nicotine usage, using it as a protective force against PD is not optimal and may only lead to other health issues.

Diet and Nutrition

Various dietary components have been observed for their both positive and negative associations with PD. Conclusions made on diet in relation to PD have been widely inconsistent, but despite these inconsistencies, greater caffeine intake has proven to have an inverse association with PD[16]. Gunnarson and Bodin (2017) found similar results with a decreased risk for regular tea drinkers and a reported 25% risk reduction for PD for coffee users. Mediterranean diets, antioxidants, flavonoids, and polyunsaturated fatty acids have been seen with protective effects, but n-6 polyunsaturated fatty acids were associated with a higher PD risk[16]. Dairy consumption has also been related to an increased risk of PD, which can be attributed to its greater intake of fat-soluble toxicants[16]. Boulos et al. (2019) found that polyunsaturated fatty acids, uric acids, coffee, and tea had protective associations with PD. They also found that vitamin B intake had no association with PD while dairy did[8].

Based on the current studies, there are still many controversies on diet and its association with PD. It is important to keep in mind the presence of contaminants, such as pesticides, that may affect results.

Trichloroethylene

Trichloroethylene (TCE) is a six-atom molecule that decaffeinates coffee, degreases metal parts, and dry cleans clothes[7]. It was first linked to PD in 1969 and since then, many studies have found even more associations between TCE and PD[7]. An epidemiological study found that occupational or hobby exposure to the colorless chemical led to a 500% increased risk of developing PD[7]. Exposure to TCE is not limited to those working in occupations with the chemical as it can pollute indoor air, outdoor air, and groundwater[7]. Despite being one of the most preventable causes of PD, investigations into TCE have been limited and further research is required.

Conclusion

This review has highlighted that the environmental risk factors, particulate matter, NO2, NOx, O3, CO, metals such as manganese, copper, and lead, pesticide exposure, n-6 polyunsaturated fatty acids, dairy consumption, and trichloroethylene, increase your risk of developing PD. While other environmental risk factors such as vitamin D exposure, smoking and nicotine usage, caffeine, diets rich in antioxidants, flavonoids, and polyunsaturated fatty acids, decrease your risk of developing PD.

PD has many environmental risk factors; however, it is extremely important to highlight the gene-environment interactions that were not addressed in this paper. PD is caused by a variety of aspects such as genes, environmental, demographic, lifestyle, and gene-environment risk factors, not just a single factor. Almost all papers mentioned the uncertainty of their conclusions and emphasized the importance and need of further research to further support their findings.

The most important environmental risk factors are the ones that individuals have control over and can prevent such as diet and pesticide exposure. While PD does not have a cure, taking precautions against certain environmental risk factors, such as reducing pesticide use, improving air quality, and encouraging dietary modifications, may help lower PD risk. To minimize harmful exposures, public health initiatives should prioritize awareness campaigns and policy changes, ultimately preventing PD on a broader scale. While all these strategies offer promising possibilities to reduce PD risk, further research is needed to confirm the associations and to develop more targeted interventions. For this reason, environmental risk factors are extremely important in developing the field of PD and further research is required for many risk factors to verify their associations with PD.

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