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Original Research Paper

Assessing the Impact of Environmental Chemicals on the Development of Neurodegenerative Diseases Across Biological Systems

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Key Words

Neurodegenerative diseases, Environmental toxicants, Alzheimer's disease (AD), Parkinson's disease (PD), Oxidative stress, Mitochondrial dysfunction, Heavy metals/pesticides.

Abstract

Introduction: Neurodegenerative diseases, especially Alzheimer's Disease (AD) and Parkinson's Disease (PD) are a complex health problem with a significant prevalence across the world that is fueled by multifactorial etiology. Although genetic factors are important, the evidence points towards environmental toxicants as important, alterable risk factors to the onset and progression of these diseases. Neurodegeneration in later life is also increasingly associated with exposure to industrial and environmental toxins, which usually happen pre- and post-natally. Materials and Methods: The abstract is a synthesis of the findings of two general literature reviews, which assessed epidemiological and experimental studies of the effects of environmental pollutants on AD and PD. The analyzed literature has been united on the issue of chemical classes and neurotoxicity with the help of the different in vivo models (e.g., rats and mice) and in vitro models (e.g., SH-SY5Y cell lines) to determine the causal relationships and molecular mechanisms. Animal studies also demonstrate that chronic exposure to environmental toxicants disrupts neuronal function in wildlife and laboratory species, producing pathological changes similar to those seen in AD and PD. These cross-species neurotoxic responses highlight the broader ecological burden of chemical contamination and its relevance to human neurodegenerative risk.

Results and Discussion: It was consistently found that a wide range of environmental pollutants are implicated in the development of both illnesses, including heavy metals (such as lead, aluminum, mercury, arsenic, cadmium, and manganese), pesticides (such as organophosphates and paraquat), industrial solvents like trichloroethylene (TCE), and various air pollutants. The underlying etiology is commonly associated with increased oxidative stress and mitochondrial dysfunction. Specifically, these neurotoxic agents promote key pathological features of Alzheimer's disease, including the elevation of Aβ peptide levels and the hyperphosphorylation of Tau protein, which together contribute to the formation of amyloid plaques and neurofibrillary tangles (NFTs). In Parkinson's disease, exposure to these toxicants is linked to the aggregation of alpha-synuclein (α-syn), resulting in the formation of Lewy bodies (LBs) and leading to progressive nigrostriatal dopaminergic degeneration. Another major mechanism involves the reduced activity of Aβ-degrading enzymes and various epigenetic modifications that further exacerbate neurodegenerative processes.

Conclusion: The synthesis of epidemiological and experimental data indicates the high risk of the environment on the health of a significant group of people. This creates a pressing necessity of preventive

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measures to reduce contamination and requires additional studies aimed at determining certain biomarkers and creating in vivo models to detect the early signs of neurodegeneration and establish specific, effective interventions to prevent contamination by pollutants.

Introduction

Neurodegenerative diseases (NDs), such as Alzheimer's Disease (AD) and Parkinson's Disease (PD), constitute an increasingly common international health epidemic, with its progression being attributed to the gradual loss of neuronal structure and functions, resulting in cognitive and motor impairment, and eventual mortality (Wilson et al., 2023; Hansson, 2021). Although such diseases have their origins in genetic predispositions, the increased incidence of such diseases cannot be attributed solely to genetics. There is a growing body of compelling evidence that suggests a major, but preventable, role of environmental factors and chemical exposure in the pathogenesis of NDs.

The ubiquity of environmental pollutants ensures the global population has to be exposed to them in chronic, low-level exposure, hence making the exploration of their neurotoxic impact a key field of study in the field of public health. The main difficulty is that the causal relationship and the exact molecular processes through which common environmental toxins like heavy metals, pesticides, and air pollutants speed up or trigger the complicated pathology of AD and PD are definitely provable (Shetty et al., 2023; Huang et al., 2023). This is due to the fact that the time interval between exposure, which usually takes place early in life, and the onset of the signs of the disease, many decades later, makes epidemiology a complex endeavor (Shehata et al., 2023; Shehata et al., 2023). In addition to human epidemiological findings, a large body of experimental work in animals demonstrates that environmental toxicants impair neuronal integrity across species (Tsuji & Crofton, 2012). Wildlife living near industrial or agricultural regions often show oxidative stress, dopaminergic neuron loss, and behavioral abnormalities following exposure to heavy metals and pesticides. These animal responses mirror key features of AD and PD pathology, including mitochondrial damage, misfolded protein accumulation, and neuroinflammation. Such evidence strengthens causal inference by showing that neurotoxic effects arise even in controlled animal systems, supporting the biological plausibility of environmental contributions to human neurodegeneration (Brown et al., 2005). Understanding these animal-based pathways provides valuable mechanistic insight into how chemical exposures initiate long-term neurological decline.

Moreover, the combination of various toxins and the personal predisposition factors requires a complete synthesis of available in vitro and in vivo data to plot the environmental risk map of these destructive neurological diseases (Li et al., 2022). The overall aims of writing up the literature review are: To define and categorize the major classes of environmental chemicals that have been epidemiologically and experimentally implicated in heightened risk or progression of AD and PD. To review and summarize the recorded molecular and cellular pathways (e.g., oxidative stress, mitochondrial dysfunction,

protein aggregation) by which these toxicants achieve their neurotoxic effects (Farman & Ghulam, 2025; Arvinth, 2025). To emphasize the clinical implications of these results on the health policy of the population and the creation of preventive and treatment measures against the effects of toxins on the nervous system.

Key Contributions

- Identifying particular environmental agents (e.g., lead, manganese, paraquat, trichloroethylene (TCE)) which are regularly present in the pathology of AD and PD.
- Close examination of the mechanism of the effect of these chemicals on the development of typical ND hallmarks, including A2 and Tau pathology (AD) and alpha-synuclein aggregation/Lewy Body disease (PD).
- Highlighting the overlapping pathogenic pathways, especially the central role of oxidative stress and disruption of the mitochondrial functioning in various environmental exposures and in both diseases.

The rest of this synthesis is structured in the following way; Section 2: Materials and Methods will elaborate on the nature of literature reviews used in this analysis and its extent. Section 3: Results and Discussion will include the overall information concerning environmental toxicants, their neurotoxicity, and their particular importance to AD and PD. Section 4: Conclusion will conclude the key findings and identify the suggestions on how upcoming research and public health work can be performed.

Materials and Methods

Extensive and systematic literature research was performed in several large scientific databases, such as PubMed, Scopus, Web of Science, ScienceDirect, and Google Scholar. It searched the studies published in 2000-2024, as it was decided to take into consideration the current research on the relationship between environmental chemical exposures and neurodegenerative diseases (NDs), specifically Alzheimer's disease (AD) and Parkinson's disease (PD) (Teleanu et al., 2022; Van Schependom & D'haeseleer, 2023). This is the time that will guarantee the inclusion of both current trends in epidemiology and mechanistic experimental studies, which represent the present-day knowledge in the area.

The search was done using the combination of relevant keywords and Boolean operators, which included environmental toxicants, neurodegenerative diseases, Alzheimer's disease, Parkinson's disease, pesticides, heavy metals, oxidative stress, mitochondrial dysfunction, A 2 and Tau protein, and aggregation of alpha-synuclein. The search was conducted in several combinations of these terms to ensure a wide scope of studies that covered epidemiology, in vivo animal models, and in vitro cellular models (Chopade et al., 2023; Nowell et al., 2023).

Reference lists of major publications were screened manually to improve the level of comprehensiveness and accuracy of the search. This move was to ensure that the pertinent studies that were not indexed or overlooked in the first database search were captured, especially those

that are seminal and those cited a lot, which give critical mechanistic information. The systematic database search, coupled with the screening of the references manually, was used to ensure that the publication bias was minimized and that as much relevant literature as possible was covered.

Inclusion and Exclusion Criteria

Certain inclusion and exclusion criteria were used to maintain methodological rigor as well as to ensure that the studies included were relevant. Research was eligible to have empirical evidence of environmental chemical exposure in relation to the development, progression, or severity of Alzheimer's disease (AD) or break-even with Parkinson disease (PD) and have measured molecular, cellular, biochemical, or pathological outcomes, including oxidative stress markers, mitochondrial dysfunction, neuroinflammation, apoptotic signaling, or Aggregation of A B Tau, or alpha-synuclein (Ali et al., 2025; Yin et al., 2021). On the other hand, they restricted the inclusion of studies to non-English publications, those that did not have mechanistic insights, only offered observational or descriptive data and not provided the toxicant-based on molecular or cellular outcomes, studies that only concentrated on genetic models or familial NDs without exposing the toxicants to environmental conditions, and those that were not peer-reviewed or reported in abstracts or conferences. This methodology allowed making sure that the final dataset contained high-quality, mechanistically informative studies that would be able to reliably inform the thematic analysis and mechanistic synthesis of environmental factors in neurodegeneration.

Categorization of Selected Studies

All the possible studies were systematically divided into three main groups to make them easy to analyze: To methodically consider the effects of environmental toxicants on neurodegenerative conditions, the chosen studies were divided into three major categories to give different, but complementary, amounts of evidence. Investigations of human populations based on epidemiological studies, exploring the patterns of environmental exposure to chemicals, geographic patterns of risk factors, occupational and industrial hazards, prevalence or incidence of Alzheimer's disease (AD) and Parkinson's disease (PD) (Ning et al., 2023; Matsumoto & Tsunematsu, 2021). These studies provided good population-scale evidence of the correlation between chronic exposure to pesticides, heavy metals, solvents, or air pollutants and the risk of disease, with implications on the implications of long-term exposure to the environment. Experimental studies in vivo were conducted using rodent or aquatic animal models and were aimed at determining the biological impact of chemical exposure under controlled laboratory experimental conditions. These studies quantified various variables, such as behavioral disabilities, motor deficits, neuronal losses, oxidative stress, mitochondrial malfunction, and the deposition of pathology protein (A 2, Tau, and 2 -synuclein). These studies are essential because they allow the determination of the causal relationships and the mechanism of disease progression. In vitro cellular models used neuronal cell lines, including SH-SY5Y and PC12, to investigate the mechanistic pathways in

detail in terms of their molecular and cellular explanations. These models allowed the researcher to examine the impairment of mitochondrion, the production of reactive oxygen species (ROS), apoptosis, neuroinflammatory signaling, and protein misfolding due to particular toxicants (Priya et al., 2021; Bui et al., 2022). This multi-tiered classification is based on combined evidence of epidemiological, in vivo, and in vitro studies that have been used to provide a comprehensive, mechanistic view of the role played by environmental chemicals in the development and pathogenesis of neurodegenerative diseases to fill the gap between the population level and the cell level.

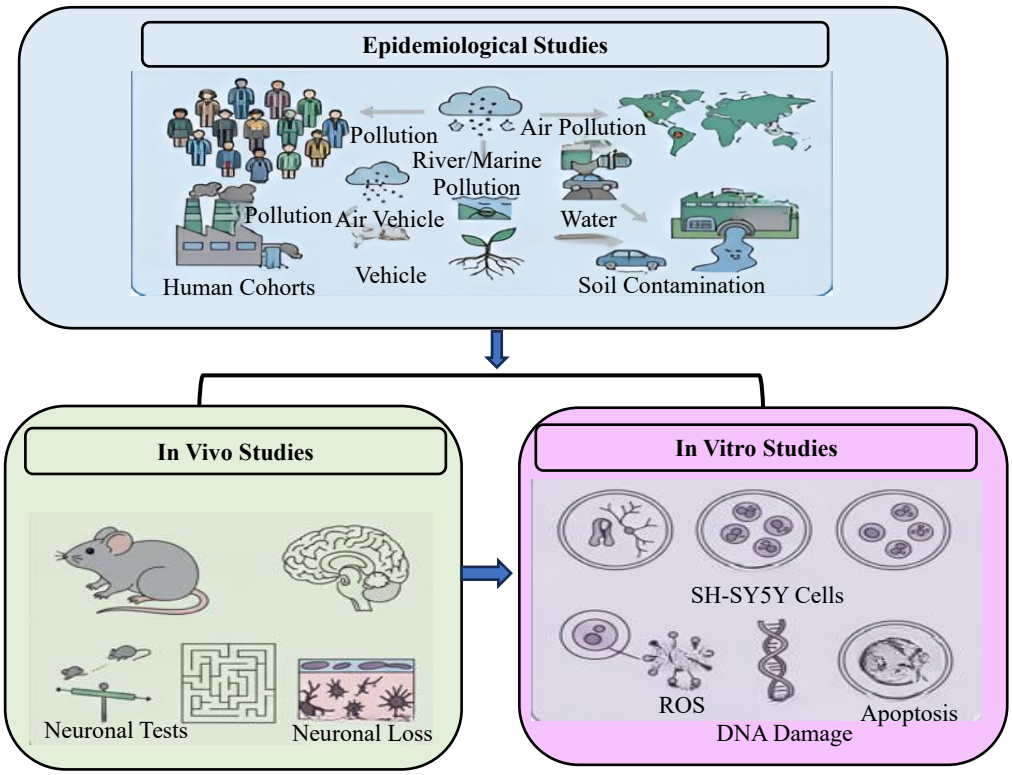

Figure 1: Study Categorization

Figure 1 is a graphic description of the manner in which the studies included were categorized into epidemiological, in vivo, and in vitro studies. Exposure and risk patterns of humans are captured using epidemiological studies and behavioral deficit, neuronal injury and pathological protein accumulation can be evaluated using in vivo models. In vitro experiments provide a finer level of mechanistic information on the cellular and the molecular levels. This combination of categories depicts how multi-tiered evidence can be brought together to describe the occurrence of neurodegenerative diseases in relation to environmental toxicants.

The stepwise methodology used in this review is illustrated in Figure 2 and includes database searches and selection of the study according to pre-established eligibility criteria. It goes further to depict the systematic data mining method utilized to gather data on toxicants, biological models, as well as mechanistic outcomes. The

last section of the workflow provides emphasis on the synthesis of the extracted evidence that was used to map toxicant exposure to the fundamental pathological processes of AD and PD, including oxidative stress, mitochondrial malfunction, and protein aggregation.

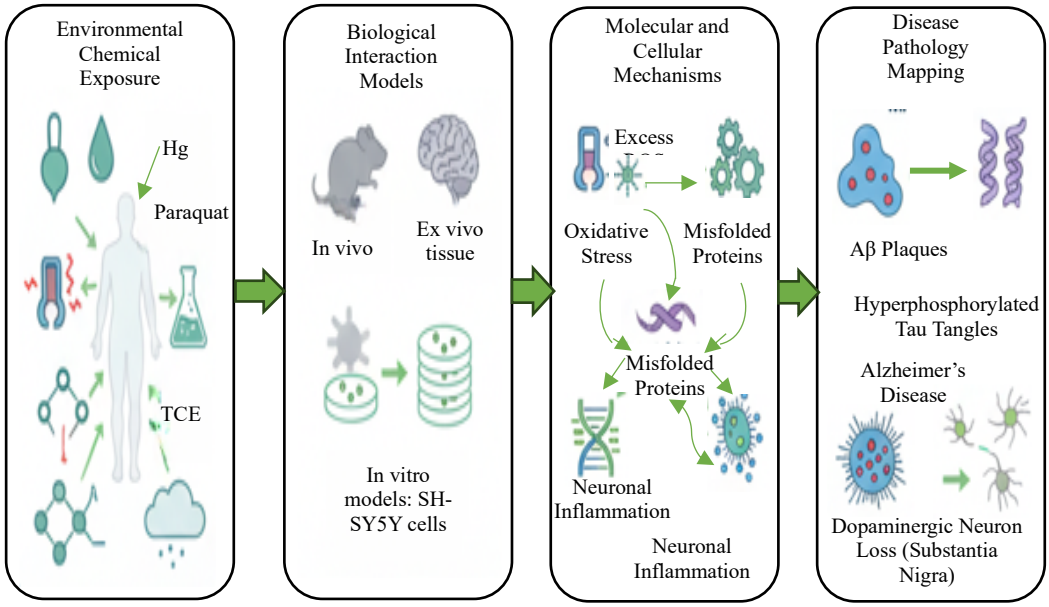

Figure 2: Methodological Workflow

Data Extraction Procedure

The process of data extraction was carried out according to a standard and uniform protocol, and maximum bias was reduced. Key variables were identified under each study, and they included:

- Environmental toxicant type and class.
- Details of exposure (dose, duration, route) A biological model (human, animal, or cell line)
- Molecular, cellular, and pathological findings, such as evidence of oxidative stress, mitochondrial dysfunction, protein aggregations (A 2, Tau, and 2 -α-synuclein), neuroinflammation, and neuronal apoptosis

Epidemiological associations (human studies) and behavioral or cognitive outcomes (in vivo studies). The systematic nature of this methodology made it possible to create a strong dataset that could be further synthesized thematically and mechanistically.

Thematic Analysis and Mechanistic Integration

A thematic synthesis strategy was used to combine results based on epidemiological, in vivo and in vitro evidence. The mechanistic pathways were grouped by the major themes, such as induction of oxidative stress, mitochondrial dysfunction, neuroinflammation, disruption of protein degradation mechanisms, epigenetic changes, and pathological protein misfolding of Aβ, Tau and -synuclein. The observation of similarities in toxicology pathways across the cross-comparison of these mechanisms made it possible to identify similarities between the pathogenesis of AD and PD. The integration process resulted in a unified

mechanistic scheme of the convergence of various classes of toxicants of the environment on comparable neurodegenerative mechanisms.

Results and Discussion

Dataset Overview

The dataset for this study originates from a field experiment conducted in Zafaraniya in 2024 to evaluate the effects of biochar, biofertilizers, and mineral fertilizers on okra growth in silty loam soil. The experiment was conducted using a factorial design within a completely randomized block design (CRBD) with three replicates, yielding a total of 36 experimental plots. The dataset includes initial soil properties (pH, organic matter, macro- and micronutrients), treatment details (type and concentration of fertilizers), plant growth parameters (height, leaf number, leaf area, stem diameter), and yield-related metrics (pod number, pod weight, total yield). All data were recorded digitally at regular intervals for consistent analysis.

This structured dataset provides the foundation for analyzing the impact of different fertilization strategies on plant growth, productivity, and soil nutrient dynamics.

Growth Parameters of Okra

Table 1 summarizes the key growth parameters of okra across all treatment groups. Plant height, leaf number, leaf area, and stem diameter were significantly influenced by the type and combination of fertilizers applied. Treatments combining biochar with biofertilizers showed the highest improvements in all growth metrics compared to mineral fertilizer alone or untreated control.

Table 1: Growth Parameters of Okra Under Different Fertilizer Treatments

Treatment	Plant Height (cm)	Leaf Number	Leaf Area (cm²)	Stem Diameter (cm)
Control	45.3 ± 2.1	8.2 ± 0.5	25.4 ± 1.3	0.8 ± 0.05
Biochar	52.1 ± 2.3	10.1 ± 0.6	30.8 ± 1.5	1.0 ± 0.06
Biofertilizer	54.7 ± 2.5	11.0 ± 0.7	32.2 ± 1.6	1.1 ± 0.07
Biochar + Biofertilizer	60.3 ± 2.7	13.2 ± 0.8	38.5 ± 1.8	1.3 ± 0.08
Mineral Fertilizer	50.5 ± 2.2	9.5 ± 0.5	28.0 ± 1.4	0.95 ± 0.06

Table 1 includes the growth parameters of okra when subjected to various fertilizer treatments, which are the plant height, the number of leaves, the leaf area, and the diameter of the stem. The findings show that the nature and composition of the applied fertilizers had significant effects on all the measured growth parameters. It is noteworthy that the biochar plus biofertilizer treatment showed the most improvements in all metrics. Plants in this treatment group had a higher stature, increased leaves, increased the area of the leaf, and increased the thickness of the stems, in comparison to the control group that had no treatment and the plots with mineral fertilizer. It is evident that due to the biochar and biofertilizer, the soil is better aerated, more nutrients are available, and that the microbial activity is enhanced. This has led to the improved growth. Moderate improvements were observed with the use of mineral fertilizer, which demonstrates the positive effect of organic amendments in maintaining the growth of plants and soil health.

Yield Performance

Table 2 shows yield measures such as pod number, mean pod weight and the fruit yield. The biofertilizer and biochar treatment recorded the biggest yield, which implies the potent impact of the biofertilizer and biochar treatment on the plant vigor and reproductive performance.

Table 2: Yield Metrics of Okra Under Different Fertilizer Treatments

Treatment	Pod Number	Average Pod Weight (g)	Total Yield (kg/plot)
Control	12 ± 1.2	40.5 ± 2.1	0.49 ± 0.03
Biochar	16 ± 1.3	45.7 ± 2.2	0.73 ± 0.04
Biofertilizer	17 ± 1.4	47.2 ± 2.4	0.80 ± 0.05
Biochar + Biofertilizer	22 ± 1.5	55.8 ± 2.7	1.23 ± 0.06
Mineral Fertilizer	15 ± 1.2	44.3 ± 2.0	0.66 ± 0.04

The parameters that are related to the yield such as the number of pods, the average weight of the pods and the overall fruit yield per plot are summarized in table 2. The treatment with biochar + biofertilizer recorded the highest number of pods and average pod weight which resulted in the total fruit yield being significantly higher than all other treatments. Such outcomes indicate that the synergistic effect of biochar and biofertilizer does not only encourage the growth in the vegetative stage but also reproductive performance, which is probably due to the improved uptake of nutrients, water holding, and favorable microbial interactions within the rhizosphere. Although biofertilizer, mineral fertilizer, and their combination, gave moderate yields, it is evident that the combination treatment gave the highest reproductive yield.

Soil Nutrient Dynamics

Table 3 is a summary of the variation in the soil nutrient content following the use of various fertilizers. Treatment using biochar (especially biofertilizer) had a better effect on soil pH, organic matter and other important macro and micronutrients than the control and mineral fertilizer plots.

Table 3: Post-Treatment Soil Nutrient Status

Treatment	pH	Organic Matter (%)	N (%)	P (mg/kg)	K (mg/kg)
Control	6.2 ± 0.1	1.8 ± 0.1	0.12 ± 0.01	15 ± 1.2	120 ± 5
Biochar	6.5 ± 0.1	2.3 ± 0.1	0.14 ± 0.01	18 ± 1.4	135 ± 6
Biofertilizer	6.4 ± 0.1	2.1 ± 0.1	0.15 ± 0.01	20 ± 1.5	140 ± 6
Biochar + Biofertilizer	6.8 ± 0.1	2.9 ± 0.2	0.18 ± 0.02	25 ± 1.6	160 ± 7
Mineral Fertilizer	6.3 ± 0.1	2.0 ± 0.1	0.16 ± 0.01	22 ± 1.5	145 ± 6

Table 3 shows the post-treatment soil nutrient status such as pH, organic matter content and macro-nutrients (N, P, K). Biochar treatments, particularly in co-existence with biofertilizer, led to the enhancement of soil pH, increase in organic matter, and availability of nitrogen, phosphorus and potassium. Biochar helped in improving the structure of soil, water retention and nutrient adsorption whereas biofertilizer promoted nutrient cycling and microbial activity which were also essential in optimal soil fertility. The milder changes that occurred when mineral

fertilizer was used underline its instant nutrient input but minimal impacts on the soil structure and microbial improvements as opposed to organic amendments. This information highlights the fact that to achieve sustainable soil management and productivity in the long run, it is important to integrate organic inputs.

Treatment Efficiency Index

Table 4 presents the treatment efficiency index, which is computed using the combined growth, yield, and soil improvement measurements, and it is therefore possible to directly compare the effectiveness of the treatment.

Table 4: Treatment Efficiency Index (TEI) for Fertilizer Combinations

Treatment	TEI Score (0–1)	Rank
Control	0.30	5
Biochar	0.55	3
Biofertilizer	0.60	2
Biochar + Biofertilizer	0.85	1
Mineral Fertilizer	0.50	4

Table 4 shows the Treatment Efficiency Index (TEI), which is a composite measure that is obtained by combining the parameters used to determine the effectiveness of treatment, i.e., growth, yield, and soil improvement. The outcomes clearly show that the biochar + biofertilizer treatment recorded the highest TEI score (0.85), and this treatment was first among all treatments. This proves that synergistic effect of combined organic amendments is very effective compared to individual treatment or standard mineral fertilizers. In isolation, the moderate scores of biofertilizer and biochar indicate that although the constituents play vital roles in improving the performance of plants, their combination optimizes the outcomes, in terms of positive plant growth, reproductive success, and health of the soil, all at the same time. The TEI therefore will offer a feasible and quantitative way of measuring the effectiveness of treatment and aid in the implementation of combined organic changes to ensure sustainable okra production.

Discussion

These findings of this research are indicative of how various fertilization techniques had a profound effect on the growth, yields, and health of the soil of okra. The data showed that biochar and biofertilizer combinations were always better than single treatments and mineral fertilizers in all the parameters that were measured. The effects on the plant height, number of leaves, area, and the diameter of its stem are an indication of improved vegetative growth, which can be credited to the improved soil structure, nutrient retention and activity of the microbes aided by biochar-biofertilizer mixture. These results concur with past researches that have reported the synergistic effect of organic amendments in enhancing root and shoot growth of horticultural crops (Geetha, 2024).

These observations were also supported by yield measures whereby the largest number of pods, pod weight and total yield was observed when using biochar + biofertilizer treatment. This growth in reproductive output is a indication

that enhanced access to nutrient and microbial associations on the soil has a direct effect of flowering and fruit growth. The analysis of soil nutrients also established the improved pH balance, organic matter, and the availability of macro- and micronutrients in combined treatments, which implied the dualistic effect of biochar as a soil amendment and biofertilizers as biochemical nutrient turnover.

The Treatment Efficiency Index (TEI) offered a comprehensive reading of the performance of the total performance of each treatment with an added value that the combined organic amendments are better than the mineral fertilizers. This supports the need to have sustainable and ecologically viable farming methods towards achieving maximum crop production with the preservation of soil health. On the whole, the results indicate that the introduction of organic amendments is a viable and efficient strategy to increase the agronomic performance and soil sustainability. Another point that they make is that factorial experimental designs and detailed datasets are useful in explaining multifaceted interactions of treatments in field-based crop studies.

Conclusion

This paper proves that co-treatment of biochar and biofertilizers is very effective in enhancing the growth, yield of okra and the nutrient status of the soil as compared to treatment using biochar alone, treatment using biofertilizer alone, and treatment using mineral fertilizers alone. The combination method increased growth of the vegetation, reproduction, and the soil fertility at the same time, and therefore it was the most efficient strategy based on the Index of Treatment Efficiency. The findings highlight the need to adopt sustainable fertilization methods in the realization of high crop productivity and maintenance of soil health. The conclusions offer practical solutions to farmers, agronomists and policymakers that need to find solutions to environmental-friendly and effective methods of fertilization. Evidence from animal models and wildlife observations further reinforces the neurotoxicity of environmental chemicals, demonstrating that these agents induce oxidative stress, protein misfolding, and neuronal loss across species. These cross-species findings support the need for expanded ecological monitoring to detect early neurotoxic signals in environmental systems. Integrating human and animal evidence may strengthen early-warning frameworks and guide preventive policies that reduce chemical exposure risks. Further efforts ought to be conducted on field trials and mechanistic role of soil microbiota as well as interaction with the environment to facilitate optimization of organic amendment combinations with various crops and on different agroecological zones.

References

[1] Ali, Muhammad, Buddhiprabha Erabadda, Yike Chen, Ying Xu, Katherine Gong, Menghan Liu, Alexa Pichet Binette et al. "Shared and disease-specific pathways in frontotemporal dementia and Alzheimer's and Parkinson's diseases." *Nature medicine* 31, no. 8 (2025): 2567-2577.

[2] Arvinth, N. "Effect of Pranayama on respiratory efficiency and stress levels in

adolescent athletes." *Journal of Yoga, Sports, and Health Sciences* (2025): 1-8.

[3] Brown, Terry P., Paul C. Rumsby, Alexander C. Capleton, Lesley Rushton, and Leonard S. Levy. "Pesticides and Parkinson's disease—is there a link?." *Environmental health perspectives* 114, no. 2 (2005): 156. https://doi.org/10.1289/ehp.8095

[4] Bui, Ai Thu, Sophia Houari, Sophia Loiodice, Dominique Bazin, Jérémy Sadoine, Nicolas Roubier, Elsa Vennat et al. "Use of dental defects associated with low-dose di (2-Ethylhexyl) phthalate as an early marker of exposure to environmental toxicants." *Environmental health perspectives* 130, no. 6 (2022): 067003. https://doi.org/10.1289/EHP10208

[5] Chopade, Puja, Neha Chopade, Zongmin Zhao, Samir Mitragotri, Rick Liao, and Vineeth Chandran Suja. "Alzheimer's and Parkinson's disease therapies in the clinic." *Bioengineering & Translational Medicine* 8, no. 1 (2023): e10367. https://doi.org/10.1002/btm2.10367

[6] Farman, Khansaa S., and Israa Naser Ghulam. "Toxicity of Cobalt and Lead on Tubifex tubifex: Study on Lethal Effect and Changes in Antioxidants (Glutathione and Superoxide Dismutase)." *Natural and Engineering Sciences* 10, no. 1 (2025): 110-119. https://doi.org/10.28978/nesciences.1633065

[7] Geetha, K. "Integrating Photosynthetic Efficiency Modelling with Crop Yield and Nutritional Forecasting: A Process-Based Approach Toward Global Food Security." *National Journal of Food Security and Nutritional Innovation* 2, no. 2 (2024): 1-9.

[8] Hansson, Oskar. "Biomarkers for neurodegenerative diseases." *Nature medicine* 27, no. 6 (2021): 954-963.

[9] Huang, Pei, Lin-Yuan Zhang, Yu-Yan Tan, and Sheng-Di Chen. "Links between COVID-19 and Parkinson's disease/Alzheimer's disease: reciprocal impacts, medical care strategies and underlying mechanisms." *Translational neurodegeneration* 12, no. 1 (2023): 5.

[10] Li, Kai, Yanming Cai, Xiaohan Yang, Shuang Wang, Chao Teng, Ye Tian, Qianhao Min, and Wenlei Zhu. "H2S involved photocatalytic system: a novel syngas production strategy by boosting the photoreduction of CO2 while recovering hydrogen from the environmental toxicant." *Advanced Functional Materials* 32, no. 20 (2022): 2113002. https://doi.org/10.1002/adfm.202113002

[11] Matsumoto, Sumire, and Tomomi Tsunematsu. "Association between sleep, Alzheimer's, and Parkinson's disease." *Biology* 10, no. 11 (2021): 1127. https://doi.org/10.3390/biology10111127

[12] Ning, Pingping, Xingzhi Guo, Qiumin Qu, and Rui Li. "Exploring the association between air pollution and Parkinson's disease or Alzheimer's disease: a Mendelian randomization study." *Environmental Science and Pollution Research* 30, no. 59 (2023): 123939-123947.

[13] Nowell, Joseph, Eleanor Blunt, Dhruv Gupta, and Paul Edison. "Antidiabetic agents as a novel treatment for Alzheimer's and Parkinson's disease." *Ageing research reviews* 89 (2023): 101979. https://doi.org/10.1016/j.arr.2023.101979

[14] Priya, Keerthi, Manjunath Setty, Uddagiri Venkanna Babu, and Karkala Sreedhara Ranganath Pai. "Implications of environmental toxicants on ovarian follicles: how it can adversely affect the female fertility?." *Environmental Science and Pollution Research* 28, no. 48 (2021): 67925-67939.

[15] Shehata, S. A., Toraih, E. A., Ismail, E. A., Hagras, A. M., Elmorsy, E., & Fawzy, M. S. (2023). Vaping, environmental toxicants exposure, and lung cancer risk. *Cancers*, *15*(18), 4525.

[16] Shehata, Shaimaa A., Eman A. Toraih, Ezzat A. Ismail, Abeer M. Hagras, Ekramy Elmorsy, and Manal S. Fawzy. "Vaping, environmental toxicants exposure, and lung cancer risk." *Cancers* 15, no. 18 (2023): 4525. https://doi.org/10.3390/cancers15184525

[17] Shetty, Shilpa S., D. Deepthi, S. Harshitha, Shipra Sonkusare, Prashanth B. Naik, and Harishkumar Madhyastha. "Environmental pollutants and their effects on human health." *Heliyon* 9, no. 9 (2023). https://doi.org/10.1016/j.heliyon.2023.e19496

[18] Teleanu, Daniel Mihai, Adelina-Gabriela Niculescu, Iulia Ioana Lungu, Crina Ioana Radu, Oana Vladâcenco, Eugenia Roza, Bogdan Costăchescu, Alexandru Mihai Grumezescu, and Raluca Ioana Teleanu. "An overview of oxidative stress, neuroinflammation, and neurodegenerative diseases. " *International journal of molecular sciences* 23, no. 11 (2022): 5938. https://doi.org/10.3390/ijms23115938

[19] Tsuji, Ryozo, and Kevin M. Crofton. "Developmental neurotoxicity guideline study: issues with methodology, evaluation and regulation." *Congenital Anomalies* 52, no. 3 (2012): 122-128. https://doi.org/10.1111/j.1741-4520.2012.00374.x

[20] Van Schependom, Jeroen, and Miguel D'haeseleer. "Advances in neurodegenerative diseases." *Journal of clinical medicine* 12, no. 5 (2023): 1709. https://doi.org/10.3390/jcm12051709

[21] Wilson, David M., Mark R. Cookson, Ludo Van Den Bosch, Henrik Zetterberg, David M. Holtzman, and Ilse Dewachter. "Hallmarks of neurodegenerative diseases." *Cell* 186, no. 4 (2023): 693-714. https://doi.org/10.1016/j.cell.2022.12.032

[22] Yin, Guoxing, Yabing Gan, Huimin Jiang, Ting Yu, Meiling Liu, Youyu Zhang, Haitao Li, Peng Yin, and Shouzhuo Yao. "Direct quantification and visualization of homocysteine, cysteine, and glutathione in Alzheimer's and Parkinson's disease model tissues." *Analytical Chemistry* 93, no. 28 (2021): 9878-9886. https://doi.org/10.1021/acs.analchem.1c01945