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Original research article

The link between PM2.5 exposure and depression: A systematic review

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ABSTRACT

Objective: This study aimed to investigate the association between exposure to fine particulate matter (PM2.5) and depression by conducting a systematic review.

Methods: A comprehensive search of electronic databases was performed to identify relevant studies. The inclusion criteria were limited to original epidemiological studies that examined the association between PM2.5 exposure and depression in human populations. Seven studies met the eligibility criteria and were included in the final analysis. The included studies were conducted in different countries and utilized varying study designs and sample sizes. Systematic review was conducted to synthesize the findings from the individual studies.

Results: The results of the systematic review indicated a consistent association between exposure to PM2.5 and depression. Moreover, the studies suggested that this association might be bidirectional, with exposure to PM2.5 leading to depression and depression increasing the risk of PM2.5 exposure. However, the strength of the association varied among studies, indicating that the effect of PM2.5 exposure on depression might be influenced by contextual factors such as population characteristics, geographic location, and exposure duration. More research is needed to establish a causal relationship between PM2.5 exposure and depression.

Conclusion: This systematic review highlights the significant impact of PM2.5 air pollution on mental health, particularly its association with depression. Elevated levels of PM2.5 are linked to an increased risk of depression, and long-term exposure to this pollutant raises the risk of depression and anxiety, especially in males. Addressing PM2.5 pollution is crucial for promoting mental well-being.

Keywords: fine particulate matter 2.5, mental health, depression, association, systematic review

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1. Introduction

Fine particulate matter (PM2.5) is a major global public health concern due to its association with adverse health outcomes, including respiratory and cardiovascular diseases, lung cancer, cognitive decline in older adults, negative pregnancy outcomes, and premature death. [1, 2] Recent evidence suggests that PM2.5 exposure may also increase the risk of type 2 diabetes and kidney disease. However, the association between PM2.5 exposure and depression remains unclear, although several potential mechanisms have been proposed, including inflammation, oxidative stress, neurotransmitter dysregulation, and epigenetic changes.[3-6] The association between PM2.5 exposure and adverse health effects involves complex mechanisms, including inflammation, oxidative stress, neurotransmitter dysregulation, and epigenetic changes. PM2.5 particles can trigger an inflammatory response by activating immune cells and inducing the release of pro-inflammatory cytokines and chemokines. Moreover, PM2.5 constituents, such as heavy metals and polycyclic aromatic hydrocarbons, generate reactive oxygen species (ROS), leading to oxidative stress and cellular damage. Additionally, PM2.5 can perturb neurotransmitter systems in the brain, affecting neurotransmitter release, reuptake, and signaling, thereby contributing to neurobehavioral disorders and cognitive impairments. Furthermore, PM2.5 exposure has been associated with epigenetic modifications, including DNA methylation, histone modifications, and non-coding RNA expression, which can result in long-lasting changes in gene expression patterns. It is crucial to conduct further research to gain a comprehensive understanding of the intricate interplay of these mechanisms and their implications for PM2.5-induced health effects.[3-6] While some epidemiological studies have investigated this association, the findings have been inconsistent, highlighting the need for a systematic review and meta-analysis to provide a comprehensive understanding of the current evidence and inform public health policies aimed at reducing the negative impact of air pollution on mental health.

2. Methods

This research aims to systematically review the epidemiological studies available to investigate the association between exposure to fine particulate matter (PM2.5) and depression. The objective is to provide valuable insights into the potential relationship between PM2.5 exposure and depression, informing future research and public health interventions.

The research design will be a systematic review of epidemiological studies, following established guidelines such as PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) [7] and MOOSE (Meta-analysis of Observational Studies in Epidemiology).[8]

Search terms and Boolean logic:

("PM2.5" OR "Air pollution" OR "Air Pollutants") AND ("Depression" OR "Mental health" OR "Mood disorders" OR "Psychological distress" OR "Emotional problems" OR "Psychiatric disorders")

Searching database:

To conduct a comprehensive systematic review of the associations between PM2.5 and depression, the following medical and scientific databases were searched: PubMed, Embase, Web of Science, PsycINFO, CINAHL, Scopus, Cochrane Library and ProQuest.

Systematic review and meta-analysis process:

In this study, the search for relevant studies is conducted independently by two reviewers using multiple databases such as PubMed, Embase, Web of Science, PsycINFO, CINAHL, Scopus, Cochrane Library, and ProQuest. The search terms and Boolean logic are predetermined and agreed upon in the research methodology canvas. However, only English language studies are included due to resource and time limitations. The search encompassed the entire available timeline of the database, ranging from its inception to the date of the review's search.

The reviewers screen the titles and abstracts of all identified studies and exclude those that do not meet the predefined inclusion criteria or are clearly irrelevant. The inclusion criteria involve investigating the association between exposure to PM2.5 and depression in human populations, with a requirement for quantitative data on the association, standardized measures of depression or validated diagnostic tools, data on PM2.5 exposure levels, and adjusted estimates of the association controlling for potential confounding factors.

Full-text screening is performed on the remaining studies, and any discrepancies are resolved through discussion or by a third reviewer. The quality and risk of bias of the included studies are assessed using appropriate tools, such as the Cochrane Risk of Bias Tool[9] or the Newcastle- Ottawa Scale,[10] depending on the study design. The Assessment of Multiple Systematic Reviews (AMSTAR) [11] tool is used to evaluate the quality of meta-analysis.

Addressing disagreements in screening and extracting processes during a systematic review is crucial for maintaining integrity and reliability. This can be achieved by referring to the established protocol, engaging in open discussions, identifying points of contention, and striving for consensus through compromises aligned with scientific rigor. In case of challenges, seeking guidance from impartial third parties, documenting discussions, and decisions, conducting sensitivity analyses, and obtaining external peer review or consultation can enhance transparency, credibility, and quality of the review.

The relevant data are extracted from each study using a standardized data extraction form that captures study design, sample size, exposure and outcome measures, statistical methods, and other key information. The extracted data are synthesized using appropriate statistical methods, and the findings are presented in a table or forest plot. If sufficient data are available, a meta-analysis is conducted using random-effects models.

The reviewers interpret the findings in the context of the research question and critically evaluate the strengths and limitations of the included studies. Finally, conclusions are drawn about the strength of the evidence linking PM2.5 exposure and depression, gaps in the literature are identified, and recommendations for future research are made. Overall, this study uses rigorous epidemiological methodology to investigate the association between PM2.5 exposure and depression, providing a reliable and trustworthy evidence base for future research and policymaking.

3. Results

Literature search and study characteristics:

After applying inclusion and exclusion criteria, seven studies were included in the systematic review. The studies had low to moderate risk of bias and five were observational while two were systematic review and meta-analysis. (figure 1 and 2) Based on the findings from five observational studies, the following key observations can be made,[12-16] as shown in Table 1.

1. Elevated levels of air pollutants, particularly ozone and PM2.5, were associated with an increased risk of depression onset. For every 10-parts-per-billion increase in ozone, there was a 6% higher risk of depression onset, and for every 10-μg/m³ increase in 1-year PM2.5, there was an 8% higher risk of depression onset.
2. NO_2 exposure was linked to a 32% increased rate of mental health service use for every 10 μg/m³ increase. However, no significant association was found for particulate matter exposure.
3. Long-term exposure to various air pollutants, including PM2.5, was associated with an increased risk of depression and anxiety. The effect was stronger at lower pollutant concentrations. Male individuals showed a stronger association between PM2.5

exposure and anxiety compared to females.

Given the observational nature and notable limitations of the included studies, it is not recommended to proceed with a meta-analysis. Undertaking such an analysis would diminish the internal validity, credibility, and practical applicability of the research findings. However, the study underscores the significance of addressing environmental pollution and social disparities in mental health interventions and policies. Further research is warranted to elucidate the mechanisms underlying the association between air pollution and mental health.

Two meta-analysis studies with low bias were found. Liu et al. (2021)[17] investigated the association between PM air pollution exposure and depression/suicide risk. Results showed that exposure to ambient particulate matter, particularly PM2.5 and PM10, is significantly associated with an increased risk of depression and suicide. A 10 μg/m³ increase in short-term exposure to PM2.5 led to a 2% increased risk of depression and suicide, while a 10 μg/m³ increase in long-term exposure to PM2.5 resulted in an 18% increased risk of depression. Short-term exposure to PM10 was also associated with a 2% increased risk of depression and a 1% increased risk of suicide. Notably, these associations were more pronounced in individuals over 65 years old and in developed regions, and the strength of the associations varied based on study design and quality. Overall, this meta-analysis underscores the link between ambient particulate matter pollution and mental health risks. This study highlights the importance of reducing air pollution for mental health. A recent epidemiological study (Braithwaite et al., 2019)[18] conducted a thorough review of 1,826 studies on the relationship between air pollution exposure and mental health outcomes. Of those studies, only 22 met the criteria for inclusion, and 9 were analyzed in the meta-analysis, which aimed to investigate the association between particulate matter air pollution and mental health disorders. The study discovered a statistically significant positive correlation between long-term exposure to PM2.5 and both depression and anxiety. Specifically, the results showed a pooled odds ratio of 1.10 per 10 $\mu g/m^3$ increase in PM2.5 exposure. Additionally, the study found a significant link between short-term exposure to PM10 and suicide. These findings emphasize the importance of reducing air pollution to prevent mental health disorders.

Two meta-analyses show a positive association between PM air pollution exposure and depression/ suicide risk, highlighting the importance of reducing air pollution for mental health. Long-term exposure to PM2.5 is linked to depression and anxiety, while short-term exposure to PM10 is associated with suicide risk.[17,18]

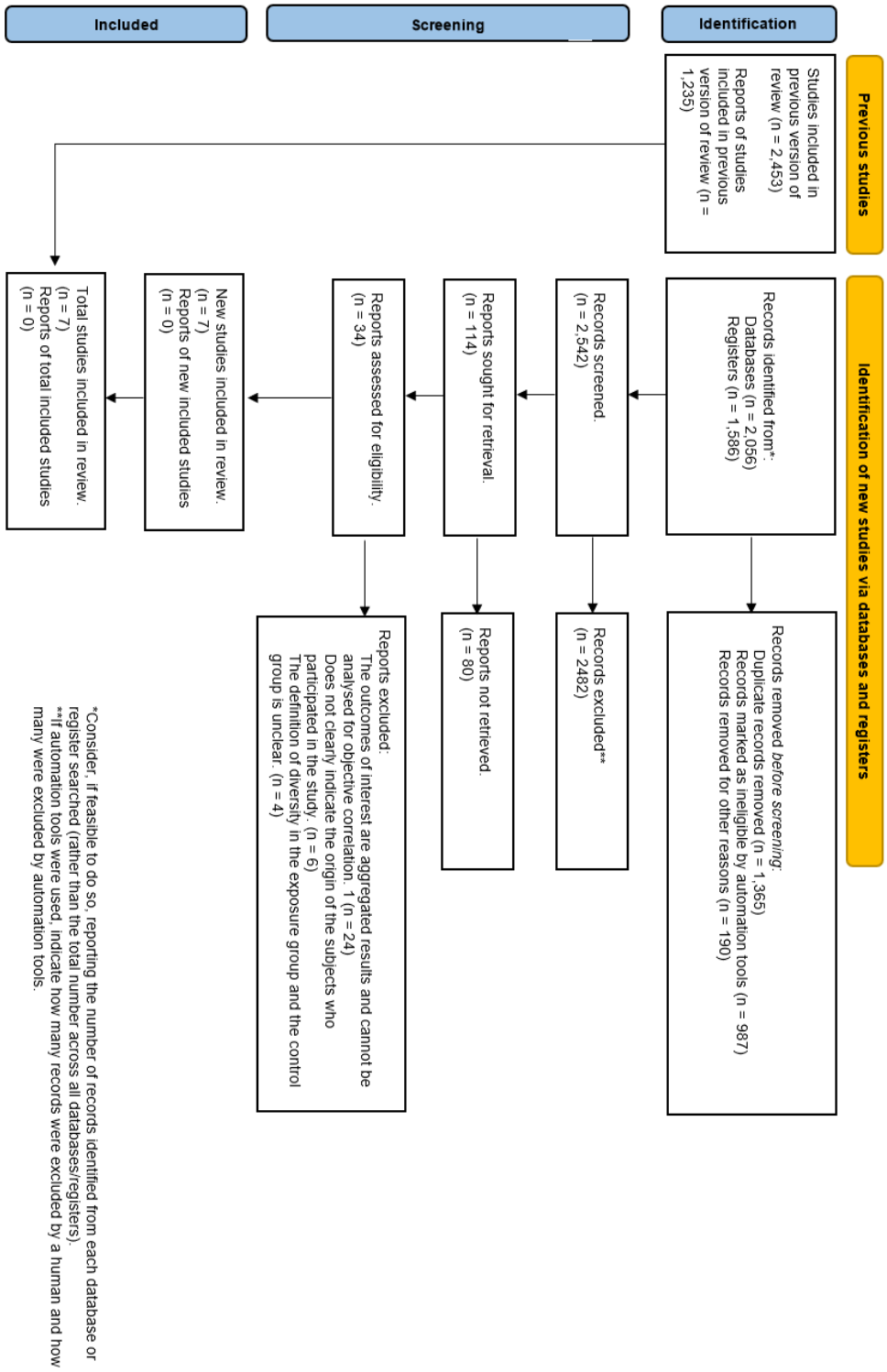

Fig. 1. PRISMA flow.

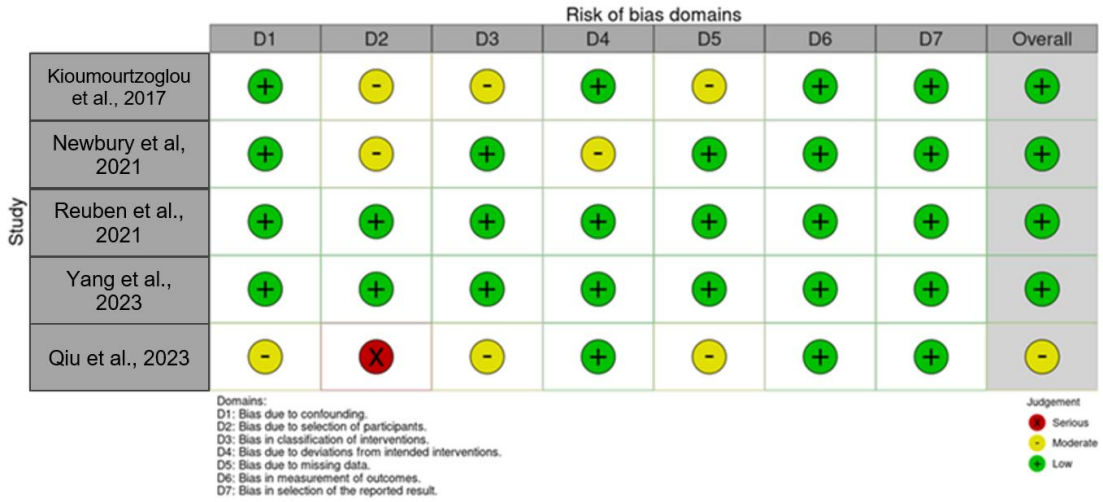

Fig. 2. Risk of bias in observational studies**.**

Table 1 The characteristics of the observational studies[12-16]

Details	Kioumourtzoglou et al., 2017[12]	Newbury et al, 2021[13]	Reuben et al., 2021[14]	Yang et al., 2023[16]	Qiu et al., 2023[15]
Study design	A prospective cohort study	A retrospective cohort study	A population-based cohort study	A prospective, population-based cohort study	A population-based longitudinal cohort study
Participants	Married registered nurses aged 30 to 55, enrolled in the Nurses' Health Study in 1976. They were followed over time to collect health-related data.	Mental health service-seeking individuals with first episode of psychotic or mood disorder.	Part of the Environmental-Risk (E-Risk) Longitudinal Twin Study, involving 2,232 twins born in England and Wales in 1994-1995. They were followed from age 5 to 18, representing diverse socioeconomic conditions in the UK. Ethical approval and consent were obtained.	Participants without prior depression or anxiety diagnoses at baseline, with complete exposure and covariate data, were analyzed from May 1 to October 10, 2022.	This study involved over 8.9 million US Medicare enrollees aged 65+ from 2005 to 2016.
Number of participants	121,701	61,270	2,232	389,185	8,907,422

Details	Kioumourtzoglou et al., 2017[12]	Newbury et al, 2021[13]	Reuben et al., 2021[14]	Yang et al., 2023[16]	Qiu et al., 2023[15]
Exposure	The level of PM2.5 at each participant's residence, estimated using a model that considers factors like location, weather, and emissions. They calculated 1-, 2-, and 5-year average exposures.	Individuals with high air pollution exposure.	Annualized estimates of outdoor pollutants, specifically nitrogen oxides (NO_2) and PM2.5, were calculated for participants' home addresses at ages 10 and 18, and these estimates were then averaged.	Annual air pollution levels, specifically PM2.5, PM2.5-10, NO_2, and NO, estimated at each participant's residential address. Additionally, an air pollution score was derived using principal components analysis to reflect their combined exposure.	Residential long-term exposure included PM2.5 ($\mu g/m^3$), NO_2 (ppb), and O_3 (ppb).
Amount of PM2.5 exposure ($\mu g/m^3$)	Average 1-year PM2.5 concentration: 12.6 $\mu g/m^3$ (standard deviation, 2.9) PM2.5 concentrations decreased during the follow-up period, from 13.1 $\mu g/m^3$ in 1996 to 11.7 $\mu g/m^3$ in 2008.	The average PM2.5 concentration during the study period was 14.8 $\mu g/m^3$, with a range of 4.4 to 33.1 $\mu g/m^3$.	PM2.5 exposure ($\mu g/m^3$) during childhood and adolescence ranged from 2.92 to 19.34 $\mu g/m^3$, with a mean exposure of 11.59 $\mu g/m^3$. Most participants (84.2%) exceeded WHO guidelines of 10 $\mu g/m^3$.	The median PM2.5 exposure was 9.9 $\mu g/m^3$ (with an interquartile range of 9.3-10.6 $\mu g/m^3$)	The amount of PM2.5 exposure was 9.6 $\mu g/m^3$ (SD 2.7).
Non-exposure	Individuals with low air pollution exposure.				
Outcome	The onset of depression, which is defined as the first report of either a physician diagnosis or the initiation of	Mental health service use was assessed as the count of outpatient, inpatient, and emergency department contacts in the 5-year period	General psychopathology, measured as continuous mental health disorder symptoms standardized to a mean (SD) of 100	The occurrence of clinically diagnosed cases of depression (coded as F32-F33) and anxiety (coded as F40-F48)	The diagnosis of late-life depression, identified through Medicare claims data.

Details	Kioumourtzoglou et al., 2017[12]	Newbury et al, 2021[13]	Reuben et al., 2021[14]	Yang et al., 2023[16]	Qiu et al., 2023[15]
	antidepressant medication.	following the initial diagnosis of a psychotic or mood disorder.	(15) at 18 years of age.	was determined using the International Statistical Classification of Diseases and Related Health Problems, Tenth Revision (ICD-10) codes.	
Main results	The study found that elevated levels of these pollutants were associated with an increased risk of depression onset. Specifically, for every 10-parts-per-billion increase in ozone, there was a 6% higher risk of depression onset, and for every 10-μg/m³ increase in 1-year PM2.5, there was an 8% higher risk of depression onset.	NO_2 exposure was associated with a 32% increased rate of mental health service use for every 10 μg/m^3 increase (adjusted HR: 1.32, 95% CI: 1.16-1.50), while no significant association was found for particulate matter exposure.	Each rise in NO_2 exposure equivalent to an interquartile range increment was linked to a 1.40-point rise (95% CI, 0.41-2.38; P = .005) in overall psychopathology.	Long-term exposure to various air pollutants, including PM2.5, is linked to an increased risk of depression and anxiety, with a stronger effect at lower pollutant concentrations . Male individuals showed a stronger association between PM2.5 exposure and anxiety compared to females.	Each 5-unit increase in long-term mean exposure to PM2.5, NO_2, and O_3 was associated with adjusted percentage increases in depression risk: 0.91% (95% CI, 0.02%-1.81%) for PM2.5, 0.61% (95% CI, 0.31%-0.92%) for NO2, and 2.13% (95% CI, 1.63%-2.64%) for O3, based on a tripollutant model.
Extracted key content	A possible link between long-term exposure to air pollutants (ozone and PM2.5) and the onset of	Air pollution exposure is associated with increased likelihood of mental health service use, particularly for	Increased exposure to NO_2, a component of air pollution, was linked to a 1.40-point increase in	Long-term exposure to air pollutants, including PM2.5, raises the risk of depression and anxiety, with a	Higher exposure to these air pollutants is associated with an increased risk of depression.

Details	Kioumourtzoglou et al., 2017[12]	Newbury et al, 2021[13]	Reuben et al., 2021[14]	Yang et al., 2023[16]	Qiu et al., 2023[15]
	depression in a cohort of 41,844 women. Stronger associations were observed when defining cases based on antidepressant use.	psychotic disorders, with a 27% increase in odds for every 10 $\mu g/m^3$ increase in NO2 exposure. These findings highlight air pollution's potential role as a risk factor for mental health disorders and the need for further research.	general mental health problems, even after considering other factors. Those with high PM2.5 exposure also had more mental health issues. When both pollutants were considered, NO_2 had a stronger impact. NO_2 exposure was associated with different mental health problems, with thought disorders showing the strongest connection. Importantly, these associations remained even in neighborhoods with challenging living conditions.	stronger effect at lower pollution levels, especially in males.	
Major limitations of the research	Key limitations include potential outcome misclassification, exposure measurement errors, and a lack of precise timing of depression onset. The	Unmeasured confounding factors, like socioeconomic status, may have influenced observed associations. The study was limited to London, UK, and	Limitations include reliance on modeled exposure estimates, lack of prenatal and preschool exposure data, focus on NO_2 and PM2.5 only, potential influence of	The crucial limitation of the study is that it cannot establish causation; it only shows associations between air pollution and mental health outcomes. Other factors	Potential misclassification of depression cases due to the use of Medicare data. Unmeasured individual-level risk factors for depression.

Details	Kioumourtzoglou et al., 2017[12]	Newbury et al, 2021[13]	Reuben et al., 2021[14]	Yang et al., 2023[16]	Qiu et al., 2023[15]
	study's cohort of middle-aged and older women may limit generalizability. Despite these limitations, the findings suggest a link between air pollution and depression onset, calling for further research and cautious interpretation.	generalizability to populations with different air pollution levels and sources may be limited. The study design identifies associations but not causal relationships.	unmeasured traffic-related pollutants and noise, and uncertainty about generalizability to highly polluted regions. This observational study cannot establish causation but considered various factors to address alternative explanations at different levels.	not considered may influence the results. More research is needed to confirm causation and understand the mechanisms involved.	Exposure measurement error from using residential zip codes instead of specific addresses.
Assessable bias level	Moderate	Moderate	Low	Low	Moderate

4. Discussion

Evidence suggests that exposure to PM2.5 may be linked to depression through mechanisms involving inflammation, oxidative stress, and disruption of the hypothalamic- pituitary- adrenal (HPA) axis.[19] PM2.5 particles can enter the brain through the olfactory bulb and activate microglia, leading to the release of pro-inflammatory cytokines and oxidative stress. This can impact neurotransmitter systems such as serotonin and dopamine, which are involved in regulating mood and emotions.[20] Additionally, PM2. 5 exposure has been shown to increase cortisol levels and reduce the sensitivity of the HPA axis, leading to dysregulation of the stress response and potentially contributing to the development of depression.[21]

Numerous studies have explored the relationship between PM2. 5 exposure and depression, with consistent findings indicating a positive association between the two. A meta- analysis of 16 studies demonstrated an increased risk of depression associated with exposure to PM2.5. Additional studies have reported similar associations between PM2.5 exposure and depressive symptoms among various age groups and genders. Collectively, these studies provide compelling evidence supporting a significant link between PM2.5 exposure and depression. Further research is necessary to elucidate the underlying mechanisms and develop effective interventions to mitigate the harmful effects of PM2. 5 exposure on mental health.[22-24] Based on the information provided, the association between PM2.5 exposure and depression can be considered a strong association. The consistent findings across numerous studies and a meta-analysis support a significant link between the two. However, further research is still necessary to fully understand the underlying mechanisms and develop effective interventions.

The differential impact of PM2.5 and PM10 on mental health can be attributed to their disparate size and composition, which

give rise to distinct molecular mechanisms.[22-24] PM2.5, characterized by a diameter of 2.5 micrometers or smaller, can penetrate deeply into the respiratory system, potentially reaching the bloodstream and the brain. Consequently, PM2.5 particles can directly influence neuronal cells and the central nervous system. Molecularly, PM2.5 induces oxidative stress through the generation of reactive oxygen species (ROS), resulting in cellular damage and neurotoxicity. Additionally, inflammatory responses mediated by pro-inflammatory cytokines and chemokines contribute to synaptic plasticity disruption and cognitive impairments. On the other hand, PM10, with a diameter up to 10 micrometers, tends to deposit in the upper respiratory tract and lungs due to its larger size. Although PM10 particles do not penetrate as deeply, they carry a broader range of toxic constituents, including metals and organic compounds, which can trigger respiratory inflammation. This inflammation, in turn, indirectly affects mental health through systemic and neuroinflammation. Moreover, the composition of PM2.5, often enriched with heavy metals and polycyclic aromatic hydrocarbons, further contributes to neurotoxic effects, oxidative stress, and neuroinflammation. Nevertheless, the precise molecular mechanisms underlying the differential effects of PM2.5 and PM10 on mental health necessitate further investigation, considering variables such as particle composition, individual susceptibility, and potential genetic and epigenetic interactions.[22-24]

To further investigate the association between PM2.5 exposure and depression, an intervention trial can be designed. This involves a prospective cohort or nested case-control study with appropriate sample size determination, accurate PM2.5 exposure assessment, validated depression outcome measures, control of confounding factors, participant follow-up, sensitivity analyses, peer review, transparent reporting, and adherence to ethical guidelines. The trial should include individuals diagnosed with depression or exhibiting depressive symptoms residing in areas with varying PM2.5 levels. Exposure to different PM2.5 levels should be the intervention, with changes in depression symptoms as the primary outcome measure and secondary outcomes such as cognitive function and quality of life. Adequate study duration and appropriate statistical analysis methods are crucial. In cases where high pollution exposure is not ethical, natural experiments or trials with smaller populations can be employed. Despite challenges, such a trial can enhance understanding of the PM2.5-depression association.

The study's strength lies in its comprehensive search of electronic databases, inclusion of studies conducted in different countries with varying sample sizes and study designs, and the consistency of the association found across studies. Additionally, the study highlighted the potential bidirectional association between PM2.5 exposure and depression, which may inform the development of interventions that target both factors. However, the study's weakness is that the strength of the association between PM2.5 exposure and depression varied among studies, which may be due to differences in measurement methods, exposure assessment, and confounding variables. Furthermore, the study did not establish a causal relationship between PM2.5 exposure and depression, which highlights the need for further research to determine the underlying mechanisms and causal pathways of the association. Despite these limitations, this study provides critical evidence for the harmful effects of air pollution on mental health, emphasizing the need for policies aimed at reducing air pollution levels.

To implement the knowledge of the association between PM2.5 and depression in clinical practice in Thailand, healthcare practitioners should undertake several measures. They should educate patients on the harmful effects of PM2.5 and its link to depression, as well as encourage them to take

measures to decrease exposure, such as using air purifiers, wearing masks, and staying indoors during high pollution periods. [25- 27] Healthcare providers should also integrate depression screening into routine medical check- ups to identify at- risk patients who may be more susceptible to the effects of PM2. 5 exposure. Patients should also be advised to adopt healthy lifestyle changes, including regular exercise and a healthy diet, that can enhance mental health and reduce the risk of depression. Consistent monitoring of local air quality levels is necessary, and healthcare providers should educate patients on ways to protect themselves during high pollution levels.[28-32]

In the pharmacological and medical research context, pinpointing a specific chemical compound within PM2. 5 that is predominantly responsible for its potential contribution to depression proves challenging. Ongoing scientific investigations into the intricate relationship between PM2.5 exposure and depression suggest that it is the comprehensive exposure to fine particulate matter, coupled with ensuing inflammatory and oxidative stress responses, that potentially underlie its impact on mental health, including depression. [33,34] Prominent constituents frequently identified within PM2.5, and pertinent to its health implications, encompass heavy metals (e.g., lead, mercury, cadmium), polycyclic aromatic hydrocarbons stemming from combustion processes, metals like arsenic with recognized neurological implications, endocrine-disrupting agents, and a spectrum of organic compounds, including volatile organic compounds. [35,36] These compounds collectively contribute to a complex mixture, wherein the mechanisms through which PM2.5 may influence depression remain subjects of ongoing research. Consequently, the identification of a single chemical agent as the exclusive causative factor remains elusive. Rather, it is the cumulative exposure to this multifaceted blend of pollutants and the resultant physiological responses that is hypothesized to be relevant to mental health conditions, exemplified by depression.

The absence of high-quality randomized studies in this systematic review necessitated an adaptation of the research strategy, incorporating meta- analysis data. Although this approach may deviate from conventional practice, the researcher contends that it represents a superior alternative to solely pooling together observational studies of moderate quality. By leveraging the available meta- analysis data, the review aimed to enhance the overall strength of evidence and provide a more robust assessment of the association under investigation. While the inclusion of observational studies introduces inherent limitations, such as potential biases and confounding factors, the incorporation of meta- analysis data allowed for a broader synthesis of available evidence and facilitated the identification of potential trends and patterns. Nevertheless, it is crucial to acknowledge the limitations associated with this methodology and the need for caution when interpreting the results. Future research endeavors should strive to include well-designed randomized studies to provide more definitive insights into the topic at hand.

As evidence linking exposure to PM2. 5 and increased risk of depression continues to accumulate, healthcare practitioners must be vigilant in addressing this issue. Collaboration with public health officials is crucial in advocating for policies aimed at reducing PM2. 5 levels in the environment. Healthcare providers should offer emotional support and resources to patients with depression or other mental health issues, including referrals to mental health professionals, support groups, or other resources. When treating patients exposed to high levels of PM2. 5, healthcare providers must make individualized decisions about adjustments to antidepressant medication dosage or changes to medication based on various factors, including medical history, current health status, and medication regimen. While PM2.5 exposure may impact

a patient's overall health, it should not be the sole factor in determining medication adjustments. Patients experiencing depression and exposed to high levels of PM2.5 should communicate changes in symptoms or medication requirements with their healthcare provider. Individuals should also take measures to minimize their PM2.5 exposure, such as avoiding outdoor activities during periods of high pollution or using indoor air filters. It is crucial to address both medication and environmental factors in managing depression in individuals exposed to high levels of PM2.5. Although numerous studies have shown an association between exposure to higher concentrations of PM2.5 and symptoms of depression and anxiety, the exact nature of the relationship remains uncertain. Further research is needed to better understand the relationship between PM2.5 and depression, including whether there is a direct causal relationship between the two. Despite this uncertainty, the evidence gathered thus far underscores the importance of reducing exposure to PM2.5 to promote better mental health outcomes.

5. Conclusion

This study reveals that elevated PM2.5 levels are associated with an increased risk of depression onset. Long-term exposure to PM2.5 is also linked to higher rates of depression and anxiety, particularly in males and at lower pollution concentrations. These findings emphasize the potential influence of PM2.5 air pollution on mental health, underscoring the importance of further research and pollution control efforts to reduce these risks.

Conflicts of Interest

The authors declare no conflict of interest.

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