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Meta Analysis Study of Environmental Pollution and Its effect on Human Brain Behaviour

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Abstract

Background: The effects of ambient air pollution on brain behaviours are poorly supported by epidemiological research. The aim of this study was to ascertain if changes in brain behaviours and exposure to air pollution are related. **Methods:** Between duration of March 2022 and April 2022, a thorough search of the literature was done using databases, including Google scholar. The PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analysis) statement served as the foundation for the current study's approach. **Results:** The results of the publication bias of the study indicated the absence of bias in the examined results (p = 0.431). There was a relationship between NOx index and brain behaviour, but this correlation was not significant (RR = 1.36, 95% CI 0.93–1.48, P = 0.163; Q-value = 42.33, df = 7; I2 = 59.69 %, P = 0.108). **Conclusion:** In summary, our findings offer more, albeit conflicting, proof of the harmful effects of air pollution on cognitive brain development, which is largely in line with mechanistic data and other published studies. Nevertheless, no correlation was found between the brain behaviour and other air contaminants. However, further research is required to more precisely define the hazards, particularly with regard to particular contaminants and the crucial exposure times.

Keywords: Environmental, Brain Disorder, Behaviours Science, Meta Analysis

Introduction

These days, air pollution is one of the most dangerous environmental issues and a major problem in many parts of the world [1]. There are several negative health effects of exposure to air pollution, and the health of people is

at risk due to the pollutants' increasing trend in a number of global locations [2]. Human DNA damage, oxidative stress, and inflammation can all be frequently caused by air pollution [3]. Among the systems impacted by air pollution is the central nervous system [4]. The complex combination of gases and particulate matter that makes up the atmosphere caused by air pollution includes nitrogen oxides, ozone (O3), volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons (PAHs), and different sizes of particle matter (PM) [5]. Both mobile (like motor vehicles) and stationary (like industries, power plants) sources are examples of anthropogenic causes of air pollution. Significant geographical variations exist in the amounts of air pollution, with about 78% of emissions affecting more than 50% of the global population coming from metropolitan [7,8]. Furthermore, there is a significant amount of spatiotemporal fluctuation in the concentrations of air pollution, and climatic factors (such as temperature, humidity, precipitation, etc.) are responsible for up to half of this variance [9]. A growing body of research suggests that air pollution exposure may also have negative effects on the central nervous system (CNS), including impaired attention, diminished IQ, memory, and academic performance, as well as negative impacts on cognitive and behavioral functioning [10]. Air pollution has also been found in recent research to be a significant risk factor for internalizing psychopathology. For instance, a recent meta-analysis discovered a substantial correlation between elevated ambient PM (PM2.5 and PM10) concentration and an increased risk of both depression and suicide [11].

It is yet unknown how exactly pollutants like PM impact the central nervous system and raise the possibility of internalizing psychopathology [12]. Over 3.2 million premature deaths worldwide were attributed to outdoor air pollution [13]. An estimated one million fatalities worldwide (or 27.3% of all deaths) are attributed to the combustion of fossil fuels, such as coal, oil, and natural gas [14,15]. The central nervous system may be impacted by environmental contaminants [16]. through a number of mechanisms, including the passage of gaseous pollutants and particles through the blood-brain barrier, the inner wall of the lung tissue into the circulatory system, the transmission of particles and pollutants directly from the sinuses to the brain tissue through the olfactory nerves, the passage of pollutants through the alveoli, and the creation of a systemic inflammatory response in the lungs that may result in the release of oxygen free radicals in distant tissues like the central nervous system [17 - 19]. In this study, we reviewed previous research using a systematic review and meta-analysis in order to investigate the effects of air pollution criteria exposure in urban areas on the incidence of central nervous system (CNS) cancer using scientific evidence [20]. According to the US EPA, neurotoxicity is defined as a detrimental alteration in the central or peripheral nervous system's structure and/or function that is assessed at the neurochemical, behavioral,

Anatomical or neurophysiological level [21]. Conventionally, exposure to well-known neurotoxicants—like lead, mercury, and pesticides with organic phosphates—leads to the loss of neurons and other measurable brain disorders. We acknowledge, however, that certain pollutants—such as endocrine disrupting substances—do not meet the traditional criteria for being considered neurotoxicants, despite the fact that they have significant and physiologically significant effects on behaviours and neurophysiology [22]. Studies on the neurotoxicity of polyfluoroalkyl substances (PFAS) are few in the literature, despite the fact that the toxicity of some PFAS to several organ systems has been investigated [23]. As Since the brain is the organ most prone to lipophilic compound penetration, there is reason to be concerned that some PFAS may have significant neurotoxic consequences. Furthermore, because PFAS attach to serum albumin with very little affinity, one of the main compartments in which PFAS partitions is the blood [24]. Because of this, there is a higher chance that toxicants in the blood, such as PFAS, will be exposed to the brain due to the highly vascularized architecture of the brain [25].

Methods

The following search phrases were used to find titles and abstracts in the google scholar database. Between duration of March 2022 and April 2022, 90 abstracts were obtained from google scholar and 26 publications were chosen for this analysis by a researcher based on the inclusion criteria listed below: The main research question in all epidemiologic studies on the detrimental health effects on the Saudi population was (i) exposure to ambient levels rather than indoor, occupational, or accidental exposures; (ii) health outcomes were related to hospital utilization; (iii) subjects were not designed to be members of specific age subgroups or high-risk groups (e.g., smokers or patients); (v) health risk estimates were expressed in terms of unit change in pollutant mass

concentration; and (vi) the studies that were conducted in only English. The PRISMA standards were followed in this investigation. Exclusion criteria includes: (1) studies looking at how these pollutants were exposed to animals; (2) studies looking at tobacco smoke exposure, such as cigarette smoke; (3) studies looking at occupational exposure; and (4) papers for which the entire text was not available even after the study Researchers' follow-up (email correspondence with authors).

The overall average of these numbers, weighted by the inverse variance, was used to summarize the risk estimations from the chosen research. Every risk assessment. There were just findings from the single-pollutant model provided. To prevent overrepresentation, the most recent study's publication on the same population was chosen where there were multiple ones.

Heterogeneity and publication bias.

The total relative risk (RR) for brain behavior was combined, and funnel plots with the Egger test were used to diagnose overall heterogeneity resulting from individual studies. This helped to mitigate publication bias.

Statistical Analysis

Using I2 statistics, the heterogeneity among the included studies was evaluated. Statistical analysis was performed using the Jasp program.The risk ratio (RR) served as the study's indicator. When the relative risk (RR) is 1 or nearly 1, it indicates that there is little to no variation in risk (the incidence in each group is equal). A larger than one RR indicates that the exposed group has a higher chance of the outcome. A lower risk in the exposed group is indicated by an RR of less than one.

Results

Study Selection

90 articles were discovered when the databases were searched. The papers went through the title and abstract review stage after duplicates were removed. Following a review of the study titles and abstracts, 63 publications were moved on to the next stage, where the articles' entire texts were examined and 26 publications made it into the final examination. Studies may have been rejected at the screening stage for a variety of reasons, such as irrelevant research populations or unrelated topic matter. Figure 1 displays a flowchart of the studies that are covered.

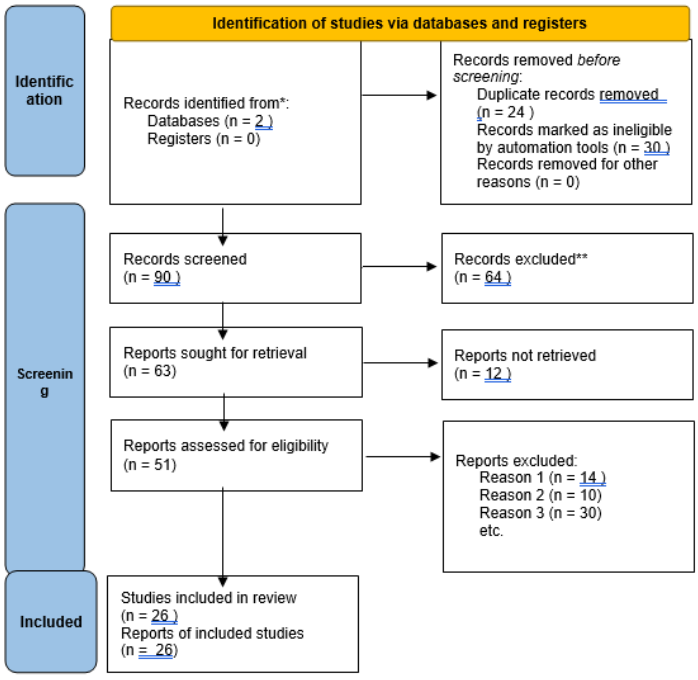

Figure 1: PRISMA 2020 flow diagram for new systemic review which includes searches of database, representation of literature review article as inclusion and exclusion.

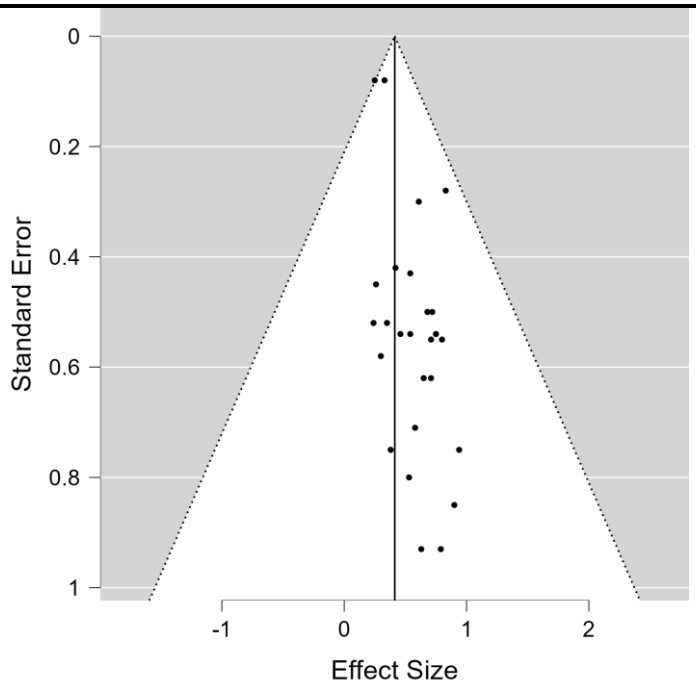

Figure 2: Funnel plots were used to investigate the publication bias in the results. The results of the publication bias of the study indicated the absence of bias in the examined results (p = 0.431)

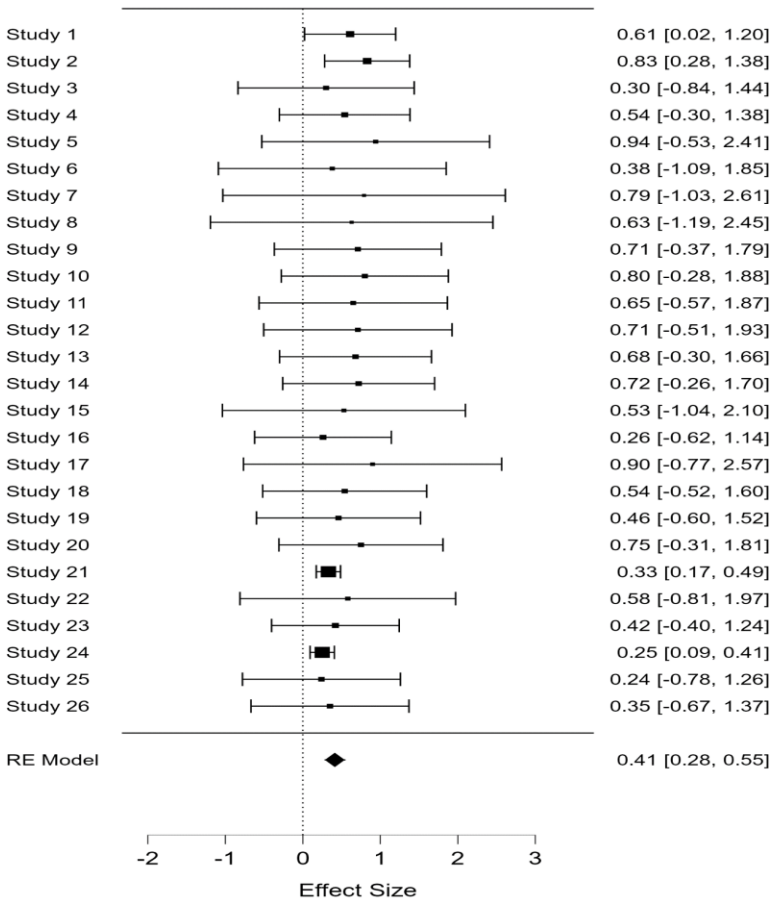

Figure 3: Shows relationship between environmental pollutants and brain behaviour. There was a relationship between NOx index and brain behaviour, but this correlation was not significant (RR = 1.36, 95% CI 0.93–1.48, P = 0.163; Q-value = 42.33, df = 7; I2 = 59.69 %, P = 0.108).

Table 1: Shows Residual Heterogeneity Estimates. *I2 is 98.293 f*or upper limit

Residual Heterogeneity Estimates			
		95% Confidence Interval	
	Estimate	**Lower**	**Upper**
τ^2	77.786	46.587	147.301
τ	8.820	6.825	12.137
I^2 (%)	98.293	97.182	99.091
H^2	58.572	35.481	110.022

Table 2: Eggers Regression test for Funnel plot asymmetry. P value = 0.609.

Regression test for Funnel plot asymmetry ("Egger's test")		
	z	**p**
sei	-0.512	0.609

Discussion

The link between brain behaviours and exposure to the NOx index was shown to exist, albeit it was not statistically significant. Additionally, there was no connection found between brain behaviours and exposure to any of the following pollutants: SO2, CO, NO2, PM10, PM2.5. Recent epidemiological research has looked at how air pollution exposure affects the central nervous system [26]. In particular, studies on the relationships between air pollution and dementia, stroke, Parkinson's disease, and Alzheimer's disease have shown conflicting results [27].Even though there aren't many research in this area, the ones that do exist are mostly cohort studies conducted in wealthy nations. Furthermore, these contaminants have been extensive investigation even though air pollution is mostly regulated in these nations. As a result, there is insufficient knowledge [28-30]. Changes in brain behaviours and NOx exposure were shown to have a modest but positive connection that was not statistically significant, according to Anderson et al.'s data [31].

On the other hand, findings from Denmark (2011) showed a substantial correlation between NOx exposure and alterations in the incidence of brain behaviours. Because in vitro research on animals has demonstrated that inhaled UFPs may enter the brain by either directly passing through the nasal passages and olfactory neurons, or by crossing the blood-brain barrier, there are several worries regarding UFPs' impact on the central nervous system [32-33]. These particles are free of membrane organelles and have a large surface-to-volume ratio. Additionally, they might cause the cell to produce cytokines and inflammatory mediators, which would lead to oxidative stress, inflammation, and DNA damage [34-36].

According to Evans et al exposure to air pollution impairs one's capacity to cope with stress since it can exacerbate feelings of exhaustion, powerlessness, and anxiety, all of which heighten susceptibility [37]. Animal research that demonstrates how long-term stress may make an individual more vulnerable to the negative effects of air pollution supports this. This is consistent with earlier findings that linked fine dust exposure to participant reports of chronic stress, with higher fine dust exposure being linked to higher levels of chronic stress [38]. Over time, this might result in diminished stress resilience or a restricted capacity to cope with stress [39]. Higher levels of personal life satisfaction are correlated with living in less polluted environments. Even when age and wealth are taken into account, this impact still remains. There is no evidence that gender-specific models matter for life happiness [40].

Limitations of the study

Because different studies have different outcome measures and designs, it is challenging to directly compare the evidence across them; nonetheless, our findings are generally in line with previous published research.

Conclusion

In summary, our findings offer more, albeit conflicting, proof of the harmful effects of air pollution on cognitive brain development, which is largely in line with mechanistic data and other published studies. Nevertheless, no correlation was found between the brain behaviours and other air contaminants. Initially, additional studies on the effects of air pollution on the brain and behaviours in children and adolescents are required, and longitudinal analyses would be a nice addition. Subsequently, more investigations using human neuroimaging are required to duplicate or contrast the impacts of air pollution on front limbic brain areas. However, further research is required to more precisely define the hazards, particularly with regard to particular contaminants and the crucial exposure times.

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