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Association between air pollutants particulate matter ($PM_{2.5}$, PM_{10}), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), volatile organic compounds (VOCs), ground-level ozone (O_3) and hypertension

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ABSTRACT

Background: Air pollution causes numerous debilitating diseases and premature deaths. This study explores the relationship between air pollutants particulate matter ($PM_{2.5}$, PM_{10}), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), volatile organic compounds (VOC), ground-level ozone (O_3) and hypertension (HTN).
Methods: The air pollutants and hypertension data were recorded from the "Pub Med, Web of Science, Scopus, and Google Scholar." We searched the data using the keywords on exposure (air pollutants) and outcome (hypertension). Primarily, 108 documents were selected, and after studying the summaries and complete articles, 28 studies were selected for analysis and discussion. The impact of air pollutants on hypertension was investigated through a compilation of 28 studies, from multiple countries, encompassing a total sample size of 2,540,441.
Results: Increased exposure to environmental pollutants $PM_{2.5}$ (OR = 1.05; 95 % CI:1.02, 1.08; $p < 0.01$); PM_{10} (OR = 1.25; 95 % CI: 1.04, 1.49; $p = 0.02$); NO_2 (OR = 1.12; 95 % CI: 1.01, 1.25; $p = 0.04$); SO_2 (OR = 1.17; 95 % CI:1.04, 1.31; $p = 0.02$); and VOCs (OR = 2.45;95 % CI:1.36, 4,41; $p = 0.01$) were significantly associated with increased incidence of HTN. However, O_3 exposure was positive but not significantly linked with an elevated risk of HTN (OR = 1.35; 95 % CI: 0.67, 2,72; $p = 0.27$).
Conclusions: Air pollutants $PM_{2.5}$, PM_{10}, NO_2, SO_2, and VOC positively and significantly enhanced the risk of hypertension. Environmental pollutants-reducing policies could be a dynamic planned approach to lessen cardiovascular risks in global populations. The strategies such as emission controls, promotion of clean energy sources, and transportation policies, that directly impact air pollution levels will have direct implications on cardiovascular health and decrease the overall occurrence of hypertension in the global population. Additionally, public health campaigns to promote cardiovascular health should incorporate education about the risk of air pollution exposure.

1. Introduction

Environmental pollution creates chaos on the planet, spreads diseases, develops various human health hazards, and can contribute to premature deaths (Meo et al., 2021). Worldwide, about 99 % of the residents reside in regions where air pollution exceeds the required air quality guidelines levels. The major pollutants in the atmosphere are "particulate matter (PM), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), and carbon monoxide (CO)", often emitted from motor vehicles, industrial processes, and the burning of fossil fuels. Environmental pollution poses risks to human health, affecting the respiratory, cardiovascular, nervous, and other body systems. Worldwide, air pollution caused premature deaths of 6.7 million people per annum (World Health Organization (WHO), 2024).

Worldwide about 1.28 billion people have hypertension. The majority of the people are from low-middle-income nations, and about 46 % of people with high blood pressure are not aware that they are suffering from this chronic condition (World Health Organization, 2024). The literature highlights the role of genetics and lifestyle on hypertension (Takase et al., 2024). However, recently emerging evidence has provided additional insight into the link between environmental pollution and hypertension. Air pollution is made up of an

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intricate combination of particulate matter, gases, and chemical compounds from both man-made and natural sources that have been found to harm human health (Niu et al., 2023).

The possible mechanisms through which air pollution might increase blood pressure have been proposed with a role for "oxidative stress, inflammation, Autonomic Nervous System (ANS), imbalance, and endothelial dysfunction" (Brook et al., 2010; Krittanawong et al., 2023). While there has been a substantial and rapidly expanding body of evidence regarding air pollution and hypertension. Air pollutant levels continue to rise, so any evidence involving air pollution to health outcomes like hypertension is imperative for strengthened air quality regulations and public health policies, especially for susceptible inhabitants, pregnant females, children, the aged population, and those already suffering from chronic health conditions. This study aimed to investigate the association between "air pollutants particulate matter ($PM_{2.5}$, PM_{10}), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), volatile organic compounds (VOC), ground-level ozone (O_3)" and hypertension".

2. Materials and Methods

This study was conducted in the "Department of Physiology, College of Medicine, King Saud University, Riyadh, Saudi Arabia.".

2.1. Search Strategy

The PRISMA's "Preferred Reporting Items for Systematic Reviews and Meta-Analyses" protocol was used to select our study's documents. "PubMed, Web of Science, Scopus, and Google Scholar" were used to find literature exploring the relationship between the risk of hypertension and exposure to environmental pollutants. For the exposure, the keywords used were "air pollutants, particulate matter, $PM_{2.5}$, PM_{10}, nitrogen dioxide (NO_2), carbon monoxide (CO), ozone (O_3), sulfur dioxide (SO_2) and volatile organic compounds (VOC)". For outcome, key terms used were hypertension, high blood pressure (BP), and hypertensive diseases. A total of 108 articles were identified initially. After that, screening and applying the eligibility criteria, 38 studies were finally chosen for the analysis and to support the discussion (Fig. 1).

2.2. Inclusion and exclusion criteria

The selection criteria for the studies were set as follows: The studies investigating exposure to air pollutants "$PM_{2.5}$, PM_{10}, NO_2, SO_2, O_3, CO, VOC" and the risk of hypertension; Effect size measured was OR/RR; article language was in English; original studies. The exclusion criteria were established as follows: Air pollutants such as airborne metals; literature such as letters, editorials, review articles, conference abstracts, or systematic reviews; and studies for which full articles could not be found.

2.3. Data analysis

The analyses were performed using "RStudio version 4.3.2 and package 'meta.' Odds Ratio (OR) with 95 % confidence intervals (CIs)" were obtained from encompassed studies and pooled to evaluate the association concerning air pollutants and the risk of hypertension. The most adjusted model was used if multiple ORs were given within the same study. Different study designs were not mixed, and documents with the most repeated study design (cross-sectional) were used in the final analysis. The analysis was done for the pollutants with three or more studies, and a random effect model was used.

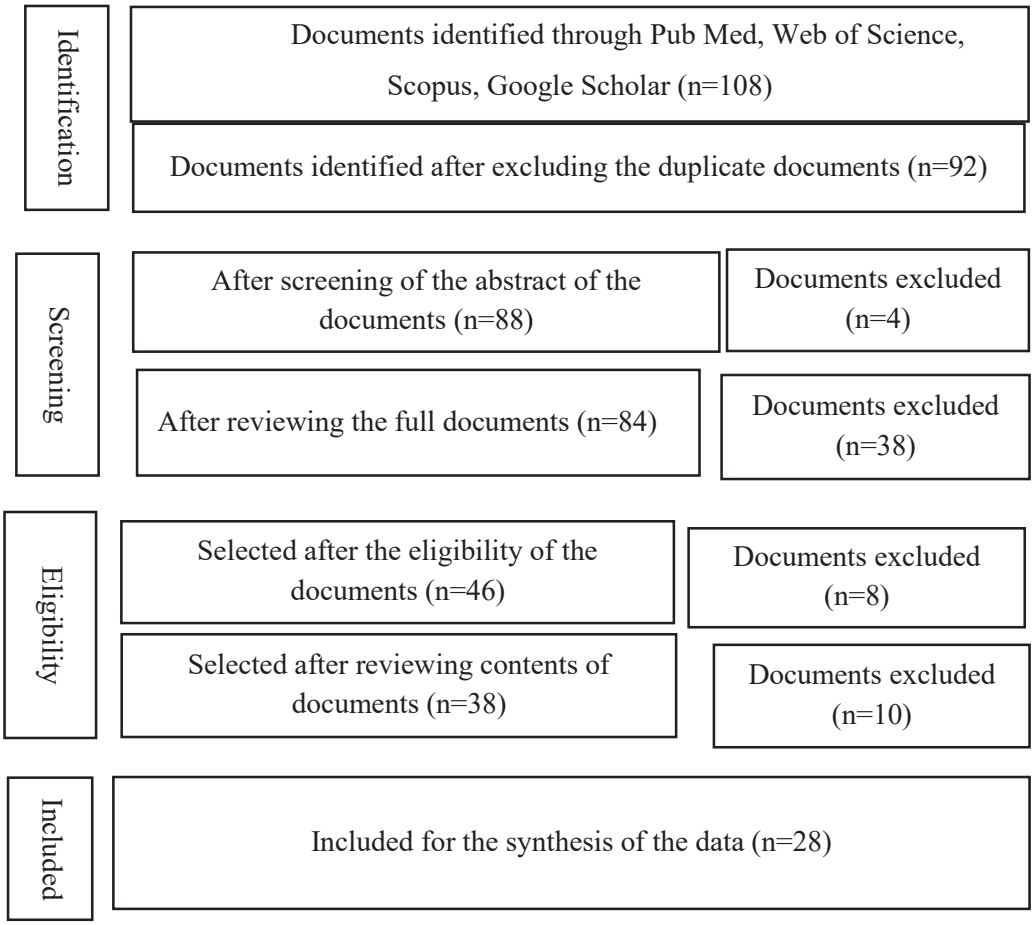

Fig. 1. PRISMA Flow Diagram for the selection of documents.

Table 1
Impact of Air Pollutants on Blood Pressure.

Author, Year of Study, Country	Study Type	Sample Size	Pollutants	Outcomes
Li J et al., 2022, China (Li et al., 2022)	Cross-sectional Study	Total: 37,610, Female: 18762, Male: 18848 7–18 years, mean age: 11 ys	$PM_{2.5}$	High $PM_{2.5}$ 19.6 μg/m^3 was significantly related to HTN where average exposure was 61.3 μg/m^3 OR 1.56 (95 % CI: 1.08, 2.25).
Song et al., 2021, China (Song et al., 2021)	Cross-sectional Study	Total: 883827, Female: 533832, Male 349995Mean age: 55.5	$PM_{2.5}$	$PM_{2.5}$, 10 μg/m^3 increase in 1-year exposure linked with increased hypertension with average exposure was 49.2 μg/m^3 OR 1.04 (95 % CI, 1.02 to 1.05).
Arku et al., 2020, 21 countries (Arku et al., 2020)	Cross-sectional Study	Total: 137809, Feamle: 79929, Male: 57880Mean age 50.6	$PM_{2.5}$	OR 1.04 (95 % CI: 1.01, 1.07). $PM_{2.5}$ was related to increased odds of HTN per 10 μg/m^3 rise in $PM_{2.5}$
Yang et al., 2019a, China (Yang et al., 2019)	Cross-sectional Study	Total: 24,845, Female: 12184, Male:12661Mean age 45.6	$PM^{2.5}$	$PM_{2.5}$ exposure was linked with higher odds of hypertension. OR 1.03, 95 % CI (1.00, 1.07). A 10-μg/m^3.
Lin H et al., 2017, China (Lin et al., 2017)	Cross-sectional study	Total: 12,665, Female: 6770; Male: 5895Mean age: 63	$PM_{2.5}$	Each 10 μg/m^3 increase in $PM_{2.5}$ exposure was correlated with high hypertension OR: 1.14 (95 % CI: 1.07, 1.22).
Yang et al, 2019b, China (Yang et al., 2019)	Cross-sectional Study	Total:15477, Female:7321; Male:8156; Mean age 45 ys	$PM_{2.5}$	OR 1.07, 95 % CI (1.02–1.13), $PM_{2.5}$ was linked with hypertension per 10-μg/m^3 increase with a mean concentration of 82.0 μg/m^3.
			PM_{10}	PM_{10} was associated with hypertension, with a mean level of 123 μg/m^3. OR 1.09, 95 % CI (1.05–1.12).
			NO_2	NO_2 was linked to hypertension, with a mean level of 35.3 μg/m^3 OR 1.19, 95 % CI (1.10–1.29).
			SO_2	The mean levels of SO_2 54.4 μg/m^3 were related to hypertension, OR 1.05, 95 % CI (1.00–1.10).
			O_3	O_3 mean concentration of 49.4 μg/m^3 established a link with hypertension OR 1.05, 95 % CI (1.00–1.12.
Honda et al. 2018, USA (Honda et al., 2018)	Cross-sectional Study	Total: 4,121; Female: 2212; Male: 1909Mean age: 70	$PM_{2.5}$	3.91 μg/m^3 one-year exposure to $PM_{2.5}$ was linked with hypertension. OR 1.24, 95 % CI: (1.11, 1.38).
Li H et al., 2023, China (Li et al., 2023)	Cross-sectional study	Total: 165075 Female: 79889 Male: 85186	$PM_{2.5}$	$PM_{2.5}$ was allied to an elevated risk of hypertension prevalence. OR = 1.081, 95 % CI: 1.034–1.129.
Zhang B et al., 2023, USA (Zhang et al., 2023)	Cross-sectional Study	Total: 27857; Female: 15747; Male:12110Mean age 61 ys	$PM_{2.5}$	No link between long-term $PM_{2.5}$ exposure per 10.9 to 14.9 μg/m^3 with hypertension. OR 0.99, 95 % CI (0.92,1.07.
Xu et al., 2021, China (Xu et al., 2021)	Cross-sectional	Total: 31462; Female: 16224; Male: 15238Mean age: 55 ys	$PM_{2.5}$	Long-term $PM_{2.5}$ was significantly allied with an increasing risk of hypertension. OR 1.08; 95 % CI, 1.04–1.12;
Babisch et al., 2014, Germany (Babisch et al., 2014)	Cross-sectional study	Total: 4166; Female: 2120; Male: 2046Mean age: 49.2	$PM_{2.5}$	OR 1.08 (95 % CI: 0.99, 1.17). There was a relationship between $PM_{2.5}$ and hypertension.
Chan et al., 2008 Taiwan (Chan et al., 2024)	Cross-sectional	Total: 18,867	PM_{10}	OR: 1.10, (1.06–1.15), There was a significant association of PM_{10} on hypertension with concentration ranging from 47.7-84.0 μg/m^3.
			NO_2	OR: 1.16, (1.08–1.25). There was a significant association of NO_2 with hypertension with the concentration ranging from 4.5-27.4 ppb.
			SO_2	OR:1.21, (1.12–1.29). There was a significant association of SO_2 with hypertension, with concentration ranging from 2.3-12.7 ppb.
Chen et al., 2015, Taiwan (Chen et al., 2015)	Cross-sectional Study	Total: 27,752; Female: 13338; Male:14414Mean age 74.8 ys	$PM_{2.5}$	OR 0.991, 95 % CI (0.959, 1.023), No association between one-year exposure to air pollution and HTN.
			PM_{10}	OR 1.000, 95 % CI (0.939, 1.066), No association was found between hypertension and 1-year exposures to air pollution.
			NO_2	OR 0.999, 955 CI (0.956, 1.043). No relationship was found between one year of exposure to air pollution and HTN.
Dong et al., 2013, China (Dong et al., 2013)	Cross-sectional Study	Total: 24845; Female: 12184; Male: 12661 Mean age 45.59 ys	PM_{10}	OR 1.12 (95 % CI, 1.08–1.16), Per 19 μg/m^3 High PM_{10} increased odds of hypertension.
			SO_2	OR 1.11 (95 % CI, 1.04–1.18). Per 20 μg/m^3 rise in SO_2, there was increased odds of hypertension
			O_3	OR 1.13 (95 % CI, 1.06–1.20). 22 μg/m^3 increase in O_3, there was increased odds of hypertension
Johnson et al., 2009, USA (Johnson and Parker, 2009)	Cross-sectional Study	Total: 134,224. Men:57864.Women: 76380; age: 30	$PM_{2.5}$	OR 1.05, 95 % CI (1.00–1.10), A 10 μg/m^3 rise in $PM_{2.5}$ was linked with an elevated risk of HTN.
Sørensen et al., 2012, Denmark (Sørensen et al., 2012)	Cross-sectional Study	Total: 44,436 Men: 21344 Women: 23029Median age: 55.9	NO_2	OR 0.97, 95 % CI (0.87–1.08). No clear associations were found between NO_2 > 19.6 (μg/m3) and risk for hypertension.
Fuks et al., 2016, Germany (Fuks et al., 2016)	Cross-sectional Study	Total: 4359; Female: 2179; Male:2180Mean age: 59.5	$PM_{2.5}$	OR 0.99, 95 % CI (0.94, 1.03), There was no association found between $PM_{2.5}$ and hypertension.
Zeng et al., 2017, China (Zeng et al., 2017)	Cross-sectional Study	Total: 9354 Male: 4583 Female: 4771Mean age: 11.4	PM_{10}	Positive but not significantly linked with an increase in PM_{10} and an increase in blood pressure. OR 2.17 (95 % CI, 1.61, 2.93).
			NO_2	OR 1.26 95 % CI (0.92-). A positive and significant association between an increase in NO_2 5-day mean exposure and an increase in blood pressure.
			O_3	OR 2.77 95 % CI (1.94, 3.95); Positive but not significant association between an increase in O_3 5-day mean exposure and increase in blood pressure

(*continued on next page*)

Table 1 (*continued*)

Author, Year of Study, Country	Study Type	Sample Size	Pollutants	Outcomes
			SO_2	OR 1.21 95 % CI (0.93–1.56). Positive but not significant association between an increase in SO_2 5-day mean exposure & increase in blood pressure
Dong et al., 2014, China (Dong et al., 2014)	Cross-sectional study	Total: 9354; Female: 4583; Male: 4771Mean age 10.9 ys	PM_{10}	OR = 1.68; 95 % CI: 1.53–1.86 A significant relationship between M_{10} and the prevalence of hypertension
			NO_2	(OR = 1.33; 95 % CI: 1.22–1.44). Significant link between prevalence of NO_2 and hypertension
			SO_2	(OR = 1.33; 95 % CI: 1.21–1.47). A significant link between SO_2 and hypertension
			O_3	(OR = 1.12; 95 %CI:1.10–1.13). Significant link between the prevalence of O_3 & HTN
Zhang Z et al., 2019, China (Zhang et al., 2019)	Cross-sectional	Total: 43745; Men: 22037 Women: 21708.Mean age: 11.3 ys	$PM_{2.5}$	OR 1.31, 95 % CI (0.86,1.98). No significant relationship between $PM_{2.5}$ and hypertension.
			PM_{10}	OR 1.45, 95 % CI (1.07,1.95). PM_{10} was associated with a high occurrence of hypertension
Hassanvand et al., 2018, Iran (Hassanvand et al., 2018)	Cross-sectional study	Total: 2847; Men: 1181 Women: 1666.Mena age: 52 ys	PM_{10}	PM_{10} was related to the occurrence of hypertension, OR 1.55, 95 % CI (1.21,1.99).
Lee et al., 2016, South Korea (Lee et al., 2016)	Cross-sectional study	Total: 680,202	PM_{10}	OR 1.042 (95 % CI: 1.009, 1.077). PM_{10} exposure 10 μg/m3 was linked to hypertension
			NO_2	OR 1.077 (95 % CI: 1.044, 1.112). NO_2 exposure (10 ppb) was associated with hypertension
Yang et al., 2019c, China (Yang et al., 2019)	Cross-sectional	Total: 194104; Men:102785 Women: 91319.Mean age 10.2 ys	$PM_{2.5}$	A significant association was found between $PM_{2.5}$ and hypertension. OR 1.03, 95 % CI (1.02–1.05).
			PM_{10}	OR 1.03, 95 % CI (1.02–1.04). Positive significant associations between PM_{10} and high blood pressure
Chang et al., 2009a, Taiwan (Chang et al., 2009)	Cross-sectional study	Total: 59 Men:47 Women: 12Mean age: 39	VOCs	OR 7.0, 95 % CI (1.0–48.4), organic solvents and toluene mixture were associated with high HTN although it was not significant.
Mohammadi et al., 2012, Iran (Mohammadi et al., 2012)	cross-sectional study	Total: 433 Female: 433Mena age: 31.72	VOCs	High exposure: aOR:3.00, 95 % CI: 1.30–6.91, significantly increased the risk for hypertension.
Chang et al., 2020b, Taiwan (Chang et al., 2020)	cross-sectional study	Total: 155 Men: 105 Women: 50Mean age 27.6	VOCs	(OR = 1.25; 95 % CI: 0.39–3.98;). Subjects co-exposed to ethanol, cyclohexanone and toluene ≥ 2500 ppb had an increased but not significant risk of HTN.
Attarchi et al., 2013, Iran (Attarchi et al., 2013)	Cross-sectional	Total: 101 All menMen age: 33.1	VOCs	aOR:4.38, 95 % CI 1.27–10.53. Organic solvent exposure was significantly associated with hypertension in group 2.
Kotseva et al., 1998, Bulgaria (Kotseva and Popov, 1998)	Cross-sectional study	Total: 345 cases and 345 controls	VOCs	OR = 2.00; 95 % CI 1.11–3.61. Workers exposed to high concentrations of benzene and xylene (group 2) have a significantly increased prevalence of arterial hypertension

The inclusive outcome was found to be significant at p-value < 0.05. The pooled studies evaluated heterogenicity using the Cochrane chi-square test (Q) and I^2. "Moderate to a high degree of heterogenicity was indicated when the p-value of the chi-square test was < 0.05 and the I^2 value was ≥ 50 %" (5). RStudio version 4.3.2 was used to evaluate the existence of publication bias using Egger's regression test and funnel plot, and sensitivity analysis was performed to evaluate the reliability of the analysis.

3. Results

In this study, 28 studies were selected that explored the link between different air pollutants and the risk of hypertension. These studies spanned multiple countries. A total of 2,540,441 individuals were included in these studies, of which 56 % of the demographic was women, and the remaining 44 % were men. In all the studies, the definition of hypertension was included in the required diagnostic criteria. The summary table for all the studies is present in the respective pollutant section, and it includes the author's name, year of publication, sample characteristics (sample size, mean age, and number of men and women), and primary outcome (Table 1).

3.1. Particulate matter ($PM_{2.5}$ μm) and hypertension

A total of 16 studies were found for PM2.5. The Cochrane chi-squared test (Q) and I^2 statistic revealed a significant heterogeneity (Q = 49.77, p < 0.01, I^2 = 70. Our forest plot (Fig. 2) showed a significantly elevated risk between increased exposure to PM 2.5 and incidence of HTN (OR = 1.05; 95 % CI: 1.02, 1.08; p < 0.01). Egger's Regression Test shows no publication bias, though the funnel plot shows slight asymmetry.

3.2. Particulate matter (PM_{10} μm) and hypertension

A total of 10 studies were found. The Cochrane chi-squared test (Q) and I^2 showed significant heterogeneity (Q = 164, p < 0.01, I^2 = 95 %). The forest plot analysis (Fig. 3) suggested that PM 10 was positively and significantly associated with an increased risk of HTN (OR = 1.25; 95 % CI: 1.04, 1.49; p = 0.02). Egger's Regression and the funnel plot showed asymmetry indicating publication bias.

3.3. Nitrogen dioxide (NO_2) and hypertension

A total of 7 studies were found. The Cochrane chi-squared test (Q) and I^2 statistic revealed a significant heterogeneity (Q = 51, p < 0.01, I^2 = 88 %). The forest plot (Fig. 4) analysis suggested that increased NO2 exposure was found to be a positive and significant risk factor for HTN (OR = 1.12; 95 % CI: 1.01, 1.25; p = 0.04). Egger's Regression Test shows no publication bias, though the funnel plot shows slight asymmetry.

3.4. Sulfur dioxide (SO_2) and hypertension

A total of 5 studies were found. The Cochrane chi-squared test (Q) and I^2 statistic revealed a significant heterogeneity (Q = 24, p < 0.01, I^2 = 83 %). The forest plot (Fig. 5) analysis suggested that SO2 was

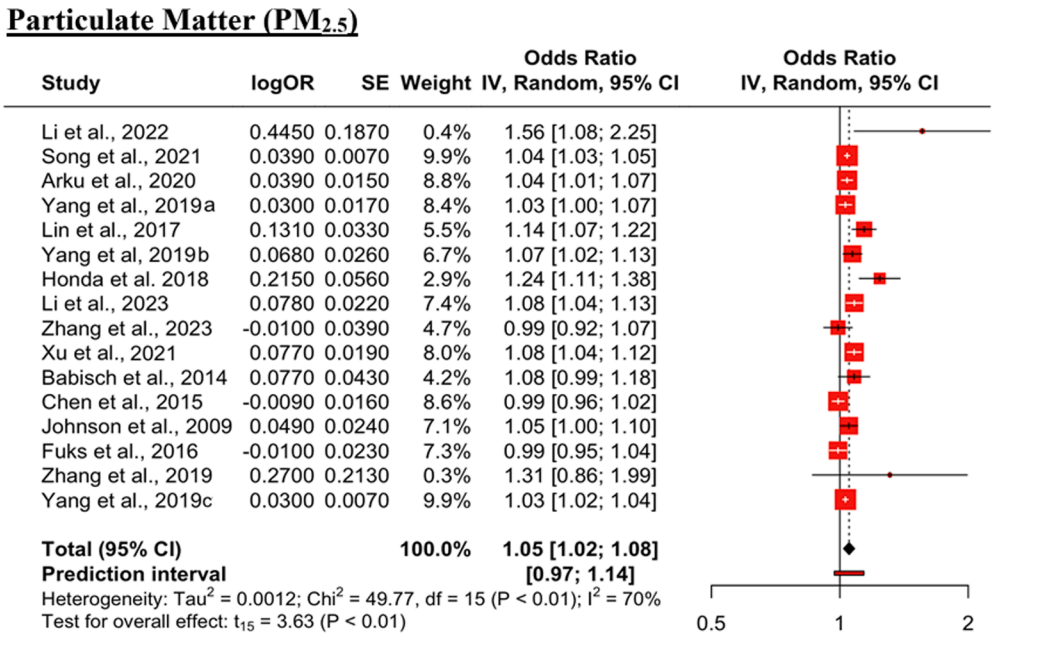

Fig. 2. Effect of $PM_{2.5}$ on hypertension. The black diamond represents the combined OR for all the studies. The red squares represent the individual OR for each study. The solid vertical line represents OR = 1. The dashed line represents the point estimate of the overall OR for all studies.

significantly associated with an increased incidence of HTN (OR = 1.17; 95 % CI: 1.04, 1.31; p = 0.02). Egger's Regression Test shows no publication bias, though the funnel plot shows slight asymmetry.

3.5. Volatile organic compounds (VOC) and hypertension

A total of 5 studies were found for volatile organic compounds associated with HTN. The Cochrane chi-squared test (Q) and I^2 statistic revealed a non-significant heterogeneity (Q = 4.25, p = 0.37, I^2 = 6 %). The forest plot (Fig. 6) analysis suggested that high exposure to VOCs was positively and significantly related to increased HTN incidence (OR = 2.45; 95 % CI: 1.36, 4,41; p = 0.01). Egger's Regression Test and funnel plot both indicate no publication bias.

3.6. Ground level ozone (O_3) and hypertension

A total of 4 studies were found. The Cochrane chi-squared test (Q) and I^2 statistic revealed a significant heterogeneity (Q = 29.94p < 0.01, I^2 = 90 %). The forest plot (Fig. 7) showed that increased exposures to O3 were positively but not significantly linked with an increased risk of HTN (OR = 1.35; 95 % CI: 0.67, 2,72; p = 0.27). Egger's regression test shows no publication bias, though the funnel plot shows slight asymmetry.

4. Discussion

Worldwide, air pollution has become a highly threatening and challenging concern. The present study findings showed that $PM_{2.5}$, PM_{10}, NO_2, SO_2, and VOCs were positively and significantly allied with hypertension. The present study findings are based on global literature that established the rapport between air pollutants and hypertension. The worldwide scientific literature conducted by Li et al., 2022 China (Li et al., 2022), Song et al., 2021 China (Song et al., 2021), Arku et al., 2020, 21 countries (Arku et al., 2020), Yang et al., 2019 China (Yang et al., 2019), Lin et al., 2017 China (Lin et al., 2017), Yang et al, 2019 China (Yang et al., 2019), Honda et al. 2018 USA (Honda et al., 2018), Li et al., 2023 China (Li et al., 2023), Zhang et al., 2023 USA (Zhang et al., 2023), Xu et al., 2021 China (Xu et al., 2021), Babisch et al., 2014 Germany (Babisch et al., 2014), Chan et al., 2008 Taiwan (Chan et al., 2024), Dong et al., 2013 China (Dong et al., 2013), Johnson et al., 2009 USA (Johnson and Parker, 2009), Zeng et al., 2017 China (Zeng et al., 2017), Dong et al., 2014 China (Dong et al., 2014), Zhang et al., 2019 China (Zhang et al., 2019), Hassanvand et al., 2018 Iran (Hassanvand et al., 2018), Lee et al., 2016 South Korea (Lee et al., 2016), Yang et al., 2019 China (Yang et al., 2019), Chang et al., 2009 Taiwan (Chang et al., 2009), Mohammadi et al., 2012 Iran (Mohammadi et al., 2012), Chang et al., 2020 Taiwan (Chang et al., 2020), Attarchi et al., 2013 Iran (Attarchi et al., 2013), and Kotseva et al., 1998 Bulgaria (Kotseva and Popov, 1998), These studies reported that exposure to various environmental pollutants have an association with hypertension. However, Chen et al., 2015 Taiwan (Chen et al., 2015); Sørensen et al., 2012 Denmark (Sørensen et al., 2012), and Fuks et al., 2016 Germany (Fuks et al., 2016) did not find a significant link between air pollution and hypertension (Table 1).

The mechanisms behind air pollution associated with hypertension are integral to designing public health involvements to condense the cardiovascular impacts of air pollution. The known mechanisms via air pollutants could exacerbate or cause hypertension, which includes "systemic inflammation, oxidative stress responses, endothelial dysfunction, and autonomic nervous system imbalance" (Brook et al., 2010; Krittanawong et al., 2023). The inhalation of particulate initiates systemic proinflammatory and oxidative responses and the release of proinflammatory cytokines and ROS into the bloodstream, leading to inflammation in various systems, including the cardiovascular system. Oxidative stress can reduce the availability of nitric oxide, which is a key vasodilator molecule. This leads to vasoconstriction, increased vascular resistance, and high blood pressure (Brook et al., 2010). Vasoconstriction affects blood pressure and flow, making it harder for the heart to pump blood into the circulatory system through the already constricted blood vessels. This forces the heart to work harder, increasing the cardiac workload, this may, therefore, increase the chances of having other heart diseases. Systemic inflammation and oxidative stress can cause increased sympathetic activity, which then results in arterial remodelling (Krzemińska et al., 2022; Thangavel et al., 2022). Arterial remodelling is a change in blood vessel structure and or function. This causes the arteries to be more rigid and not be able to regulate the blood

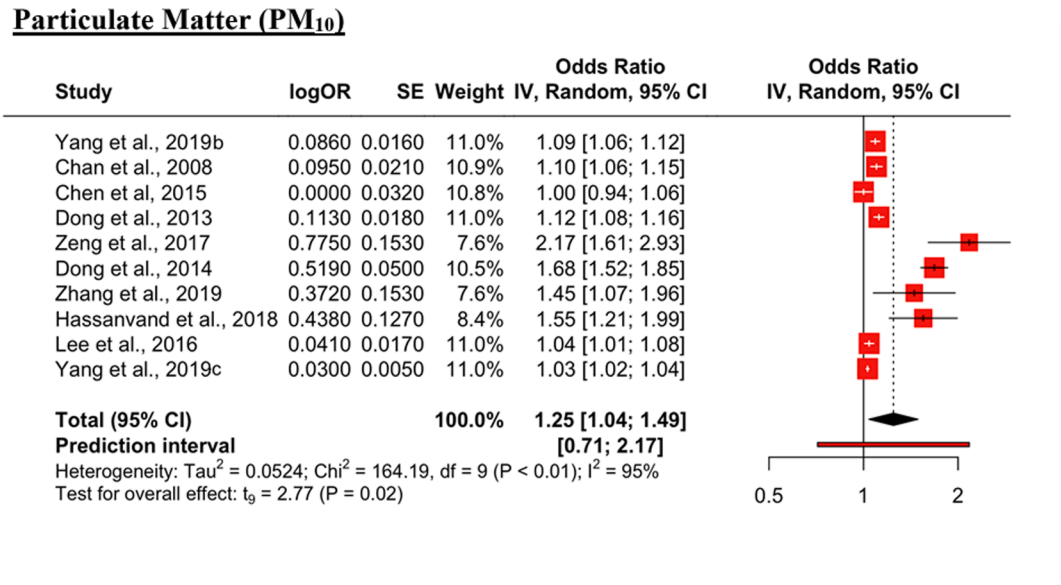

Fig. 3. Forest Plot for PM_{10} impact on HTN. The black diamond represents the combined OR for all the studies. The red squares represent the individual OR for each study. The solid vertical line represents OR = 1. The dashed line represents the point estimate of the overall OR for all studies.

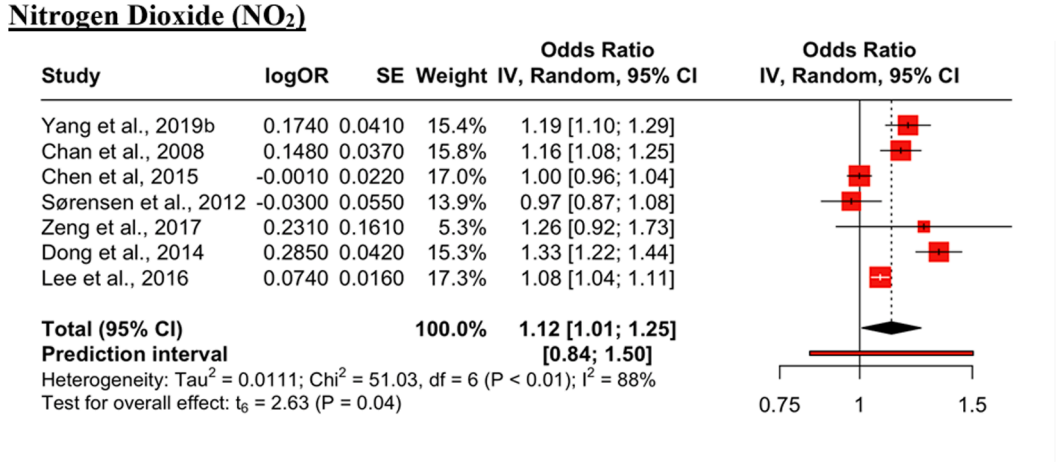

Fig. 4. Forest Plot for impact of NO_2 on HTN. The black diamond represents the combined OR for all the studies. The red squares represent the individual OR for each study. The solid vertical line represents OR = 1. The dashed line represents the point estimate of the overall OR for all studies.

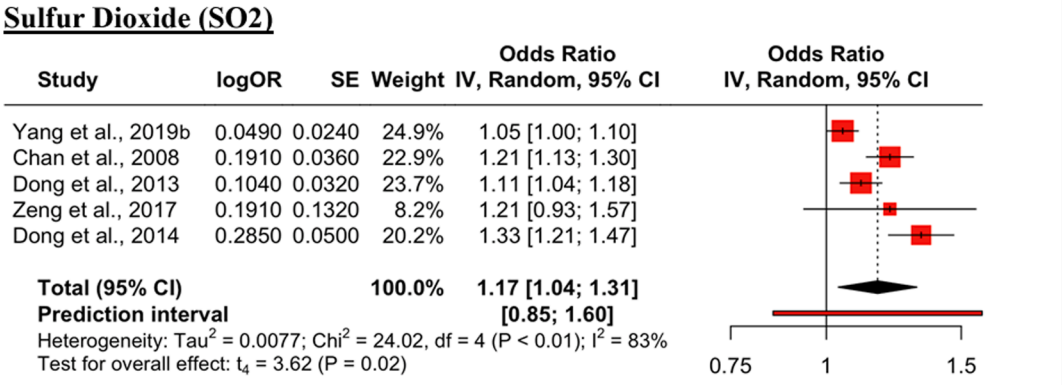

Fig. 5. Forest Plot for SO_2 Effect on Risk of HTN. The black diamond represents the combined OR for all the studies. The red squares represent the individual OR for each study. The solid vertical line represents OR = 1. The dashed line represents the point estimate of the overall OR for all studies.

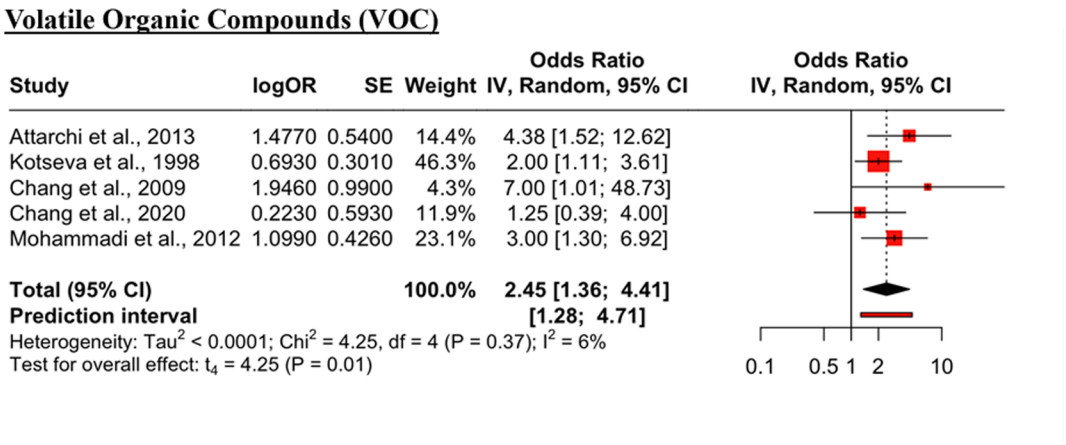

Fig. 6. Forest Plot for VOC and hypertension. The black diamond represents the combined OR for all the studies. The red squares represent the individual OR for each study. The solid vertical line represents OR = 1. The dashed line represents the point estimate of the overall OR for all studies.

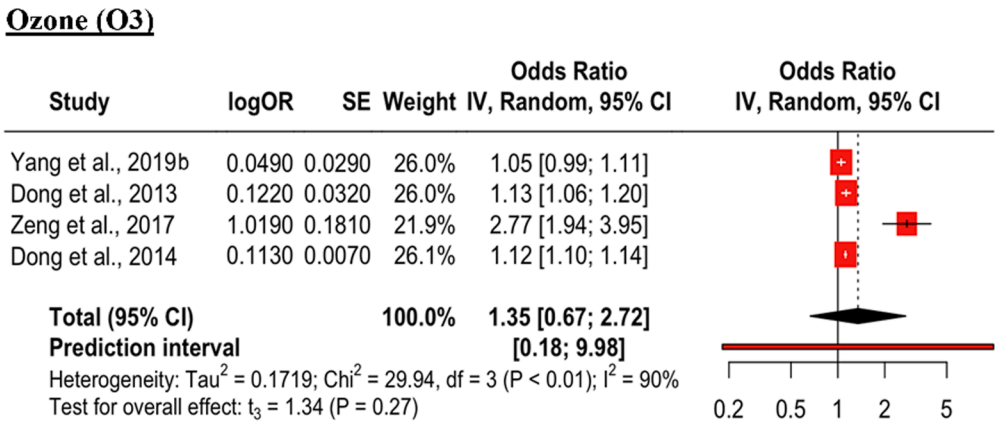

Fig. 7. Forest Plot for O_3 and Risk of HTN. The black diamond represents the combined OR for all the studies. The red squares represent the individual OR for each study. The solid vertical line represents OR = 1. The dashed line represents the point estimate of the overall OR for all studies.

pressure and flow as much. Aside from the indirect influences, particulate matter can also have a direct effect on the cardiovascular system through its ability to cause vasoconstriction. The literature highlights that $PM_{2.5}$ increases the imbalance of the Autonomic Nervous System, favouring the sympathetic over the parasympathetic component, and this imbalance results in vasoconstriction (Krzemińska et al., 2022; Thangavel et al., 2022).

The literature has a consensus on air pollution exposure and hypertension. The three presumed biological mechanisms that are involved in the pathophysiological influence of air pollution on cardiovascular diseases are "systemic inflammation, oxidative stress, Autonomic Nervous System imbalance, endothelial dysfunction, and vasoconstriction", resulting in hypertension (Brook et al., 2010; Krittanawong et al., 2023; Huang et al., 2012).

4.1. Study strengths and limitations

The strength of this study is that the impact of air pollutants on hypertension has been investigated through a compilation of 28 studies from multiple countries, encompassing a total sample size of 2,540,441 individuals. The results based on such large sample-sized studies and populations provide a piece of better evidence. There are a few limitations, some evidence may be missed or overlooked due to human error. We limited our eligibility criteria to an odd ratio to better synthesize the results; however, this could have led to the exclusion of some important evidence. We also did not mix different study designs and used the most reported study design, which was cross-sectional studies, and this also could have led to the exclusion of some evidence. The concentration of the pollutants also varied widely throughout the studies, which may have affected the results.

5. Conclusions

The environmental pollutants $PM_{2.5}$, PM_{10}, NO_2, SO_2, and VOC significantly increased the risk of hypertension. Reducing levels of air pollutants could be a dynamic strategic approach to mitigate cardiovascular health risks in global populations. Public health initiatives aimed at reducing air pollution levels and targeted interventions to protect vulnerable populations and minimize the burden of hypertension associated with environmental exposures. Strategies such as emission controls, promotion of clean energy sources, and transportation policies that directly impact air pollution levels will have direct implications on cardiovascular health and decrease the overall prevalence of hypertension in the general population. Additionally, public health campaigns to promote cardiovascular health should incorporate education about the risk of air pollution exposure. Health officials and policymakers must establish strategies to minimize air pollution both at regional and global levels. Addressing air pollution improves cardiovascular outcomes and also contributes to the overall well-being of populations exposed to environmental stressors.

CRediT authorship contribution statement

Sultan Ayoub Meo: Writing – review & editing, Formal analysis,

Conceptualization. **Narmeen Shaikh:** Methodology, Investigation, Formal analysis, Data curation. **Metib Alotaibi:** Investigation, Formal analysis, Data curation.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Informed Consent: Not required.

Availability of Data: The data may be provided on reasonable request.

Authors' Contribution.

SAM: study concept, manuscript writing and editing, NA, MAO: writing a manuscript, literature review, data collection and data analysis.

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