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Analyzing Impact of Climate Parameters and Air Pollution on Mortality and Hospital Admissions: A Systematic Review

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Abstract
The Detrimental impact of air pollution and adverse weather conditions on global public health is a significant concern, resulting in increased mortality rates and hospital admissions. This paper presents a systematic review investigating the intricate association between air pollution, weather conditions, and health outcomes, particularly emphasizing mortality rates and hospital admissions. The primary goal of this research endeavor is to discern uncharted territories within the realm of scientific inquiry and underscore the significance of further investigations of effect modifiers and the intricate interplay between weather and air pollution variables. While the review predominantly examines countries within the UK, EU, and Asia single markets, it also includes comparative analysis with other nations. The outcomes of this study will provide valuable insights for developing effective environmental health measures, resource allocation, system interventions, and enhancements in the healthcare system. A distinctive aspect of this review lies in examining the combined effects of various exposures, such as air pollution and weather, on critical health outcomes such as hospitalization and mortality rates.

Keywords: Climate parameters, Air pollution, Mortality, Hospital admissions, public health, Environmental health, Climate change, Health risks.

1. Introduction

Exposure to weather conditions exceeding certain thresholds in specific regions and air pollution can have significant and diverse implications for public health [1-3]. The rapid increase in urbanization, population growth, industrialization, economic development, energy consumption, and use of vehicles worldwide have contributed to an exponential rise in air pollution emissions and fluctuations in surrounding temperature and additional climatic factors [4][5]. It is responsible for the release of various pollutants into the air, including particles that are under $10^{\mu m}$ and $2.5^{\mu m}$ (PM_{10}, $PM_{2.5}$) in size, NO_2, along with NO_x, SO_2, O_3, and CO, which researchers have linked to various short- and long-term health issues [6][7]. Exposure to weather conditions, such as temperature variations, precipitation, relative humidity, and additional climate parameters, may lead to severe illnesses and deaths, particularly among susceptible populations without sufficient hospitalization and physiological resources. When climatic exposure exceeds certain thresholds, mortality rates can increase, with a time delay of 20 days or less [8][9]. The existing research indicates a significant correlation between exposure to weather fluctuations and air pollution surpassing thresholds specific to a particular region and the subsequent rise in hospitalization rates and overall mortality. Extensive research in scientific literature consistently reveals compelling evidence of positive correlations between air pollution and exposure to weather variables surpassing region-specific thresholds. These associations are observed with cause-specific and all-cause mortality and hospital admissions, with a pronounced focus on respiratory and cardiovascular diseases. Such findings underscore the significant impact of air pollution and weather conditions on public health outcomes, particularly in the context of respiratory and cardiovascular-related health issues [9-16]. However, there is a need for more research to explore other parameters that may influence the effect of air pollution, such as ethnicity, as well as the communication between meteorological and air pollution conditions. Research has indicated that ethnic minority individuals typically reside in urban communities that are more crowded and impoverished, with low living conditions and elevated exposure to air pollution, which increases their risk of chronic health problems [17-19]. Despite highlighting the impact of the interaction between health outcomes and air pollution, the literature frequently emphasizes the importance of weather conditions in creating and disseminating air pollutants. For instance, sunlight catalyzes the formation of ozone pollutants, resulting in increased ozone densities during summer seasons [20]. To improve our understanding of how meteorological and air pollution conditions affect hospitalization and mortality rates, it is essential to examine existing literature to identify any research deficiencies thoroughly. This knowledge is crucial for improving the planning, organization, and allocation of resources for environmental and system interventions and the medical system. This review offers a detailed and comprehensive summary of the literature on the relationship between weather, air pollution, and mortality rates in hospital settings. This review stands out for investigating several exposures, particularly the effect of air pollution and weather on consequences such as hospitalization and mortality rates, which previous research has explored to a limited extent.

Our literature review aims to concentrate on countries that belong to the world's various regions, as they share similar environmental and health policies and socioeconomic characteristics that may influence the effect of air pollution and weather on mortality and hospitalization rates. This approach will help minimize contextual differences and facilitate comparing results across different studies. Nonetheless, it also provides comprehensive

analysis for comparative purposes; we will also review pertinent articles published in countries beyond the UK, EU, and Asia. This review study highlights the detrimental consequences of air pollution and its exposure to extreme meteorological conditions on human health. It emphasizes the upsurge in pollutant emissions and variations in weather variables worldwide. It notes the release of pollutants like SO_2, PM_{10}, O_3, $PM_{2.5}$, CO, and NO_x into the atmosphere, linked with various chronic and acute medical conditions. It highlights the implication of exploring the influence of weather and air pollution on susceptible populations, including ethnic minorities, and examining the interplay between meteorological and air pollution conditions. In addition, it also highlights the importance of a literature review to identify gaps in research on the correlation among weather, air pollution conditions, and mortality/ hospitalization rates. The research will examine how weather conditions and air pollution affect the rates of mortality and hospitalizations. Specifically, it will concentrate on countries all over the world's single market to reduce variations in context.

1.1 Motivation

The fundamental motivation for this systematic review is to comprehend the complex relationship between climate parameters, air quality, and human health. Climate change, exacerbated by human activity, has increased extreme weather and biological changes. The above changes affect air pollution, which is vital to public health. Hospital admissions and mortality rates are highly responsive to environmental changes; therefore, they can be used to measure their health effects. This paper aims to analyze and appraise current knowledge on this topic through a systematic literature review. This study examines the links between climate factors, air pollution, hospitalization and mortality rates. Additionally, this research aims to identify the primary pollutants and weather parameters that significantly impact human health. The findings from this analysis can inform targeted interventions, policy development, and public health protection in response to changing environmental issues.

1.2 Our contribution

This systematic study advances environmental health research in several ways. We synthesize the research to present a current understanding of the complicated relationship between climate factors, air pollution, and human health. This synthesis combines global research and health outcomes, enhancing our grasp of this complex problem. Second, we improve the systematic review method in this field. We carefully refine our search, inclusion, and data extraction procedures. These methodological enhancements reinforce our findings by making our review studies reliable and relevant.

Our comprehensive review identifies research gaps and limitations. We identify pollutants, locations, sensitive populations, and emerging health consequences for further study. It also identifies emerging trends and patterns that prior studies may have missed. We study the impact of changing climatic conditions, air pollution interactions, and their health effects.

Our findings influence public health policy and practice beyond academic research. We summarize the latest climate and air pollution health research to inform decisions. This can make environmental public health policies and initiatives more effective. In addition, we suggest future research directions. These include in-depth studies on climate-related health issues, adaptation approaches, and a thorough evaluation of air pollution's health effects in a changing climate. Finally, our review could enhance awareness of climate-related health risks and air pollution beyond science. Our findings are widely shared to increase scientific and public knowledge of these vital issues. We enrich environmental health research, direct policy and practice, and improve climate-public health awareness with our systematic review.

1.3 Paper organization

This paper's organization is intelligently crafted to guide readers through research investigation. Section I introduces the research problem and its significance. It also briefly summarizes our unique contributions to the subject, setting the stage for the following parts. In Section II ('Materials and Methods'), we painstakingly explain our study materials and methods. This transparency helps readers comprehend the study's data gathering, processing, and statistical or analytical methodologies. Section III ('Literature Survey') overviews our research's knowledge base. This section places our study in the context of academic discourse, emphasizing its significance and uniqueness. Section IV (In Appendix) and V (Results (In Appendix) and Discussion) present our research's empirical findings and outcomes. We demonstrate our contributions via statistics and analysis. The paper's empirical foundation supports our claims and findings. Section VI ('Conclusion') concludes the paper. Here, we summarize the vital findings, their ramifications, and their relevance to our research. We also emphasize our study's significance and suggest future research, giving readers a lasting feeling of its importance.

2. Materials and Methods

2.1 Search Strategy and Sources

To ensure the comprehensiveness and reliability of our scoping review, we followed the recommendations outlined by PRISMA ScR (Preferred Reporting Items for Systematic Reviews and Meta-Analyses for Scoping Reviews) (Checklist S1) [21]. The literature search was performed on May 23, 2023, utilizing the "Web of Science" and "PubMed" sources, explicitly focusing on medical, health, and environmental literature. Our study aimed to evaluate the influence of weather patterns and air pollution levels on hospitalization and mortality rates in many European nations. The investigation spanned ten years, specifically from January 6, 1990, to June 22,

2023, focusing of ten years, specifically from January 6, 1990, to June 22, 2023, with a focus on the implementation of the "Integrated Strategy on Climate Change" by the European Union (EU) in March 2007. The objective of this approach was to achieve a 30% reduction in the impact of climate change, variability in weather patterns, and air pollution compared to the levels recorded in 1990 by the year 2012 [22]. To ensure a comprehensive search, we categorized our search strategy into three groups: "Health Outcomes," "Geographical Areas," and "Environmental Factors." The "Geographical Areas" category included the EU, UK, China, India, and other countries, while the "Health Outcomes" category focused on hospitalizations and deaths. The "Environmental Factors" category encompassed specific pollutants and weather variables. Our search combined MeSH and All Fields terms for each type, connected with the OR Boolean operator, and combined using the AND Boolean operator. To eliminate confounding variables, our search strategy excluded studies related to "influenza infections." To ensure the accuracy of the search results, we limited the search criteria in the Web of Science database to specific categories relevant to our research, such as environmental science, public health environmental work, and biomedical research. We excluded non-English articles, grey literature, books, conference abstracts, theses, reports, and unpublished studies from our search criteria.Explicit criteria were established for screening titles, keywords, and abstracts to ensure the articles' relevance in our scoping review. Our criteria focused on initial quantitative articles published in the UK, EU, and various regions of the globe, analyzing mortality and/or hospitalization outcomes and examining exposures related to ambient air pollutants, weather exposure, and extreme weather events. Our study encompassed cohort studies, time series studies, and quantitative self-control studies that employed statistical measures such as odds ratio (OR), hazard ratio (HR), percentage increase, or relative risk (RR) to quantify the variables associated with hospitalization and death. These study designs facilitated the assessment of the impact of long-term exposure on mortality and hospitalization outcomes. We have excluded research studies that investigated the impacts of weather and air pollution on flora and fauna, indoor air pollution, workplace-related consequences, and studies conducted in languages other than English. We also excluded prediction studies and qualitative research. Furthermore, we excluded reviews that were not based on original research. Following the PRISMA-ScR guideline, comprehensive search strategy, and strict inclusion criteria, we identified 108 articles that met our requirements. The chosen publications were utilized as significant sources in our investigation, offering essential perspectives on the correlation between weather occurrences, air quality, mortality, and hospitalization rates in different countries.

2.2 Data Abstraction and Screening

The search technique examined the “Web of Science” and "PubMed" libraries and additional sources. It identified 990 studies. After eliminating 187 identical articles, the remaining 951 articles' titles, keywords, and abstracts were examined using specified inclusion and exclusion criteria. To validate the veracity of the criteria, we analyzed a sample of 28% of the abstracts to identify the titles of the 990 profiles.

Following the initial screening, 153 articles fulfilled the inclusion criteria and were subjected to full-text review. After excluding 45 articles, we included 108 in the final narrative compilation. Figures 1 and 2 visually represent the screening process; Table 1 highlights the percentage-wise distribution of included studies, and Table 2 offers detailed descriptions of added studies.

The narrative collection required data extraction from 108 articles. This data set contains various topics, including the study population's location, exposure variables, research design, outcomes, sample size, exposure assessment method, statistical methods/modeling, correction for confounding factors, exposure duration, and metric use. In the analysis, metrics such as Incident Relative Risk (IRR), Odds Ratio (OR), Relative Risk (RR), and Hazard Ratio (HR)were employed. In addition, pertinent research studies included confidence intervals or percentage increases to evaluate the associations between outcome variables such as hospitalization and/or mortality and factors such as weather, air pollution, and events. The abovementioned elements were considered essential in assessing the study's overall quality and the robustness of the identified associations.

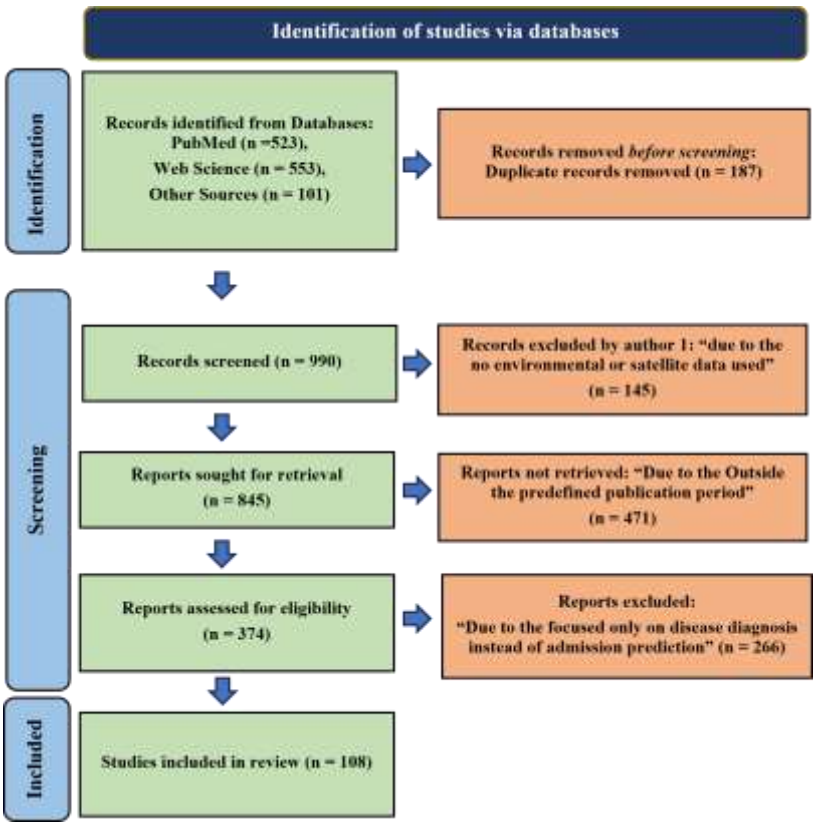


Figure 1. PRISMA Flow Diagram for Literature Search and Screening Process.

Table 1. The proportion of Included Studies (N=108).

Characteristics	Studies Included	Percentage
Hospital Admission	34	31.48%
Mortality	61	56.48%
Hospital Admission & Mortality	13	12.03%
Total	108	100%

Table 2. Description of Included Studies.

Health Outcome	No studies Included	References
Mortality And Hospital Admission	13	[23], [24], [25], [26], [27], [28], [29], [30], [31], [32], [33], [34], [35]
Mortality	61	[36], [37], [38], [16], [39], [40], [41], [42], [43], [20], [44], [45], [46], [47], [48], [49], [50], [51], [52], [53], [54], [55], [56], [57], [58], [59], [60], [61], [62], [63], [64], [65], [66], [67], [68], [69], [70], [71], [72], [73], [74], [75], [76], [77], [78], [79], [80], [81], [82], [83], [84], [85], [86], [87], [88], [89], [90], [91], [92], [92], [93]
Hospital Admission	34	[94], [95], [96], [97], [98], [99], [100], [101], [102], [103], [104], [105], [106], [107], [108], [109], [110], [111], [112], [113], [114], [15], [115], [116], [117], [118], [119], [120], [121], [122], [123], [124], [125], [126]



Figure 2. Categorization of included research studies.

3. Results

The final narrative review consisted of 108 studies, of which 61 focused on mortality outcomes, 34 examined hospitalization outcomes, and 13 investigated hospital admissions and mortality, as presented in Tables 1 and 2. Most research articles employed a time-series study design, utilizing Poisson modelling for data analysis. However, a few studies used a cross-sectional design with conditional logistic regression. We conducted cohort design and data analysis using Cox risk regression. The study investigated multiple outcomes, encompassing cardiovascular and respiratory diseases, all-cause mortality, hospital admissions, and particular diseases such as lung cancer, mental disorders, stroke, atrial fibrillation, cerebrovascular disease, arrhythmia, diabetes, COPD (chronic obstructive pulmonary disease), ischemic heart disease, and heart failure.

3.1 Air Pollution V/s Hospital admission rate and mortality

This review found a notable association between hospitalization and mortality rates with six significant pollutants: NO_x/NO_2, CO, SO_2, O_3, $PM_{2.5}$, and PM_{10}. Respiratory, cardiovascular, and cerebrovascular diseases were the most commonly reported diseases linked to these pollutants. While some health effects were observed immediately after exposure, others took several days to become apparent.

Particulate matter (PM) pertains to the existence of solid particles and water droplets within the atmosphere, creating a colloidal suspension. Particulate matter can originate from natural sources, such as dust, volcanic ashes, and pollen, and anthropogenic activities, including industrial processes, traffic emissions, fossil fuel burning, and mining operations [127]. The structure and size of these elements determine where they are deposited in our respiratory system. Larger particles (PM_{10}) tend to settle in the upper airways and nasal cavities. In contrast, tiny particles ($PM_{2.5}$) can permeate more profoundly into the respiratory system and even spread into the bloodstream and alveoli. Unfortunately, these particles carry harmful substances that can lead to various health issues in humans, such as coughing, Difficulty breathing, Irregular heartbeat, decreased lung function, and Asthma [20] [128].

The analysis of the research findings, as illustrated in Table 6, demonstrated a statistically significant correlation between PM_{10} concentrations and heightened rates of death and hospitalization associated with respiratory and cardiovascular ailments. The research results in the Netherlands revealed substantial findings, indicating a hazard ratio of 1.06% with a 95% confidence range. These findings suggest an association between a 10 g/m^3 increase in PM_{10} levels and death caused by cardiovascular conditions [41]. In the same way, the presence of PM_{10} pollution was found to be associated with a 1.11% rise in the likelihood of mortality due to respiratory illnesses, as well as a 1.056% increase in relative risks, with a 95% confidence interval [16][129], as well as a 0.69 % rise in hazard risk for hospital admission (CI: 95%) [15]. $PM_{2.5}$ pollution has the same impact on human health as PM_{10}. However, $PM_{2.5}$ is more hazardous than PM_{10} since its tiny particles can enter our lungs deeper and disrupt the functionality of the human respiratory system [129]. Research conducted in France highlighted an elevated likelihood of hospitalization due to any cause (relative risk of 1.11) and increased death (relative risk of 1.02) associated with each incremental increase of 10 $\mu g/m^3$ in $PM_{2.5}$ pollutant concentration [130].

Ozone pollution's impact on mortality and hospitalization did not follow the same pattern as particulate matter pollution. In some studies, ozone was a preventative measure against hospitalization and mortality; in others, it raised the risk or had no effect. This is due to ozone formation, a highly reactive pollutant when sunshine is present [20]. A research study in England found that O_3 protects against cardiovascular, all-cause, lung cancer, and respiratory mortality [16]. Other studies, however, have indicated that ozone can be a risk factor and contribute to an increase of up to 2% in all-cause mortality with a quarterly rise in ozone levels [49][56][70][131].

This review shows that nitrogen oxide pollution is equally hazardous as other air pollutants. Inhaling nitrogen dioxide and nitrogen oxide can make you sick and increase the risk of mortality or hospitalization. [23] [41] [79] [129]. According to a study conducted in Belgium, there was an observed increase of 3.5% in hospitalization for cardiovascular issues with each 10 $\mu g/m^3$ rise in NO_2 pollution [104]. Furthermore, there was a notable rise of 4.5% and 4.9% in hospital admissions for ischemic and hemorrhagic strokes,[94] respectively [104].

Sulfur dioxide (SO_2) pollution, created mainly by industrial operations and power plants that use fossil fuels, has been linked to various health problems. These health issues range from mild eye, nose, and throat irritations to more severe consequences like bronchial spasms and respiratory insufficiency leading to death [133]. Only 12 articles assessing the impact of SO_2 on hospitalization and mortality identified that a higher level of SO_2 upsurges the chances of all-cause and respiratory/cardiovascular mortality [134] [79] [85].
Carbon monoxide (CO) is a poisonous gas generated through the partial combustion of petroleum and other fossil fuels, which causes various respiratory and other diseases. It can attach to hemoglobin, limiting red blood cells' ability to transport oxygen adequately [133]. Only ten studies examined the link between carbon monoxide exposure and mortality or hospitalization. Most of these research studies have discovered that carbon monoxide exposure can initiate numerous health issues, including the respiratory and cardiovascular systems. One study found that exposure to carbon monoxide pollution doubled the possibility of pulmonary embolism-related hospitalization [31]. A study conducted by Renzi et al. (2017) discovered that each 1 mg/m^3 rise in CO correlates with a rise of 0.12% in all-cause mortality [79]. However, one of the analyzed studies found that carbon monoxide was a preventive factor against hospitalization for chest disease in people with sickle cell anaemia. This discovery was ascribed to carbon monoxide's ability to bond with hemoglobin, which increases oxygen-binding sites while decreasing vasoconstriction and inflammation. This observation suggests that it may exert a beneficial influence rather than solely serving as a risk factor for sickle cell disease individuals [123].

3.2 Temperature V/s Hospital admission rate and mortality

Exposure to temperatures above or below region-specific thresholds can indirectly and directly impact public health. The short-term impacts may entail heat stress or hyperthermia resulting from high temperatures and ischemic stroke or hypothermia caused by cold temperatures. Even minor fluctuations in temperature over an extended period can indirectly impact the human body's cardiovascular and respiratory systems [135].
The reviewed articles examined the association between mortality, hospitalization rates, and weather conditions, particularly air temperature during specific lag periods. The studies examined cold temperatures with a lag of 0 days to 5 weeks, and they investigated hot temperatures within 0 days to 25 days of exposure. These studies analysed the effects of these temperatures on various diseases, including respiratory, cardiovascular, and psychiatric disorders.
A study by researchers [136] has demonstrated a heightened risk of hospitalization and mortality in different health conditions due to low temperatures. Nevertheless, one of the studies examined in the literature review documented a mitigating impact of lower temperatures on total mortality, with no discernible time delay, as indicated by a relative risk of 0.99 (CI: 95%). Despite the previous study suggesting a protective effect of cold temperatures, it also revealed that cold temperatures with a 14-day lag can still pose a risk for mortality, as indicated by an RR of 1,003, demonstrating a delayed impact of cold temperatures on casualties.
Most of the research in the analysis indicates a positive correlation between severe temperatures, increased mortality rates, and hospital admissions related to various health outcomes. In contrast, [101] found that warm temperatures were associated with a reduced hospitalization rate for ischemic heart disease. Additionally, Monteiro et al. (2013) observed reduced rates of all-cause hospitalizations and cardiovascular hospitalizations concerning warm temperatures [26][101]. Numerous research articles have analyzed the impact of rising year-round temperatures on mortality and hospitalization, and more than 50% of these articles have demonstrated a notable positive correlation between increased temperature and hospitalization/mortality rates.

3.3 Other Weather Parameters V/s Hospital admission rate and mortality

Numerous studies have demonstrated that exposure to various weather conditions, including humidity, precipitation, sun, air pressure, snow cover, wind speed, daylight, and direction within 0 to 7 days, can impact various diseases. Psychiatric, respiratory, and cardiovascular diseases appear most commonly affected [119]. Certain meteorological factors, such as the impact of rain on psychiatric hospitalizations, sunlight and daylight on psychiatric and trauma hospitalizations, are positively associated with hospital admissions [109], wind speed on lung disease hospitalization [96], and barometric pressure on hospitalization for mania and depression [109]. However, there is inconsistency in the literature regarding the effects of sun exposure on psychiatric hospitalization, with some studies reporting it as a risk factor and others as a protective factor [109].
The analysis revealed that most of the studies considered in the review incorporated adjustments in their statistical analyses, considering factors such as age and gender; some articles identified socioeconomic factors such as occupational class, education level, income level, and marital status as confounding modifiers or factors [20][104][111][137]. One study found that ethnicity moderated the relationship between "summer smog" days and all-cause death concerning high temperatures and PM_{10} pollution [138]
A significant amount of academic research has been dedicated to exploring the impacts of air pollution on hospitalization and mortality rates while also considering the contemporary influence of air temperature. Moreover, numerous research studies have examined the potential correlation between the degree of exposure to diverse weather conditions and the incidence of death and hospitalizations. The primary objective of these inquiries has been to determine the influence of air pollution on various results [49][59][57][69]. The study discovered that several additional factors could influence the correlation between mortality and/or hospitalization and weather exposure and/or air pollution. These factors included weekends and holidays, tobacco smoke, flu waves, seasons, weekdays, and population decline during summer [136] [65][30].

4. Discussion

The included studies employed three primary epidemiological designs to investigate the relationship between ambient environmental exposures and adverse health outcomes: time-series analyses, case-crossover studies, and cohort studies. As summarized in Table 3 (In Appendix), time-series studies [23][24][26][28][33][34] consistently examined both cardiovascular and respiratory hospital admissions and mortality, offering temporally granular data on the associations between short-term pollutant fluctuations and health outcomes. These designs are well-suited to detect immediate physiological responses to environmental exposures, particularly for cardiorespiratory endpoints. Similarly, as presented in Table 4 (In Appendix), case-crossover studies [25][27][30][31][32][35] provided self-matched comparisons of health events across a diverse spectrum of conditions ranging from pulmonary embolism and stroke to psychiatric disorders and all-cause hospitalization, effectively controlling for time-invariant individual confounders. Table 5 (In Appendix) presents the sole cohort study [29] examining both hospitalization and mortality outcomes, underscoring the relatively limited use of prospective cohort designs for simultaneously capturing both endpoints. Table 6 (In Appendix) catalogues a considerably larger body of cohort evidence [16][20][36]–[46] dedicated exclusively to mortality outcomes, covering a broad range of diagnoses including all-cause mortality, cardiovascular disease, respiratory illness, lung cancer, and cerebrovascular events. The hospital admission-related cohort studies in Table 10 (In Appendix) ([95][106][121]) further expand the scope to psychiatric morbidity and emergency presentations. Collectively, the time-series and case-crossover studies presented in Tables 7, 8, and 9 (In Appendix) ([47]–[93]) form the largest component of the evidence base, demonstrating the widespread epidemiological interest in short-term environmental exposures and a broad array of hospital admission-related health outcomes. The diversity of study designs across Tables 3–11 (In Appendix) strengthens the overall evidence by providing convergent findings from methodologically distinct approaches, although it also introduces heterogeneity that warrants cautious interpretation when synthesizing quantitative estimates.

Particulate matter with an aerodynamic diameter of *≤10 μm (PM_{10})* has been consistently implicated in cardiorespiratory morbidity and mortality across multiple study designs and geographic settings. As reported in Table 12 (In Appendix) , exposure to PM_{10} was associated with a statistically significant elevation in pulmonary embolism hospitalizations (OR 1.04, 95% CI: 1.02–1.05) [31], while the corresponding mortality risk (OR 1.02, 95% CI: 0.96–1.07) did not achieve significance, suggesting that PM_{10} may exert a more pronounced effect on thromboembolic morbidity than on fatal outcomes. Concurrently, [32] reported a modest but statistically significant increase in acute myocardial infarction hospitalization risk (RR 1.02, 95% CI: 1.00–1.04), with stroke mortality risks (RR 1.02, 95% CI: 0.99–1.05) approaching but not crossing the threshold of statistical significance. In the context of mortality-related cohort evidence detailed in Table 20 (In Appendix), a consistent pattern of elevated hazard ratios was observed across multiple causes of death. Study [36] reported significant increases in all-cause mortality (HR 1.03, 95% CI: 1.01–1.04), cardiovascular mortality (HR 1.04, 95% CI: 1.01–1.07), and ischemic heart disease mortality (HR 1.05, 95% CI: 1.00–1.09) per unit increment in PM_{10}. Similar findings were demonstrated by [42], which reported significant elevations in all-cause (HR 1.08), cardiovascular (HR 1.06), respiratory (HR 1.13), and lung cancer mortality (HR 1.26), with the latter representing the largest relative risk across all PM10 mortality outcomes. Study [20] reported particularly elevated cardiovascular mortality risks (HR 1.34, 95% CI: 1.14–1.57), highlighting marked inter-study variability potentially attributable to differences in baseline PM_{10} concentrations, population demographics, and healthcare system contexts. Regarding hospital admissions recorded in Table 36 (In Appendix) , PM_{10} was significantly associated with increased admissions for multiple sclerosis (RR 1.33, 95% CI: 1.23–1.43) [100], COPD exacerbations (OR 1.35, 95% CI: 1.04–1.76) [103], cardiovascular conditions including heart failure (1.37% rise, 95% CI: 0.74–2.00) [104], and respiratory diseases across a range of magnitudes [15], [116], [124], [125], [126]. Collectively, the weight of evidence across Tables 12, 20, and 36 (In Appendix) strongly supports a causal association between PM10 exposure and elevated risks of cardiovascular, respiratory, and all-cause morbidity and mortality, consistent with established biological mechanisms involving systemic inflammation, oxidative stress, and autonomic dysfunction.

Fine particulate matter ($PM_{2.5}$), owing to its smaller aerodynamic diameter and deeper pulmonary penetration, has been associated with a broader and more pronounced spectrum of health effects compared to PM_{10}. Evidence from Table 13 (In Appendix) regarding dual hospitalization and mortality outcomes reveals that $PM_{2.5}$ exposure was associated with a significant increase in all-cause mortality across age groups (OR 0.31 for all ages, rising to OR 0.42 among seniors), along with significant circulatory mortality risks [35]. The consistently higher estimates in older age groups underscore the particular vulnerability of elderly populations to fine particulate exposure. In the extensive cohort evidence collated in Table 21 (In Appendix), $PM_{2.5}$ demonstrated significant associations with all-cause mortality across multiple independent studies. Study [36] reported HR 1.03 for all-cause and HR 1.05 for cardiovascular mortality; [37] documented an all-cause HR of 1.07 (95% CI: 1.02–1.13); and [39] reported significant elevation across all-cause (HR 1.02), cardiovascular (HR 1.04), ischemic heart disease (HR 1.06), lung cancer (HR 1.03), and respiratory mortality (HR 1.05). Study [20] reported among the highest estimates, with cardiovascular HR of 1.32 (95% CI: 1.16–1.50), reinforcing the primacy of cardiovascular disease as a $PM_{2.5}$-sensitive endpoint. Notably, [93] introduced a unique ecological perspective, reporting that $PM_{2.5}$ from heavy-duty diesel trucks disproportionately affects younger adults aged 20–44 and carries higher mortality burden among males, introducing important considerations of source-specific exposure and demographic vulnerability. The hospital admission evidence in Table 37 (In Appendix) corroborates these findings, with $PM_{2.5}$ significantly associated with increased respiratory hospitalization risk (7.5% rise, 95% CI: 4.9–10.2) [107], cardiovascular admissions [15], COPD exacerbations (RR 1.32) [119], and asthma hospitalizations (RR 1.016, 95% CI: 1.008–

1.025) [126]. The convergent evidence across Tables 13, 21, and 37 (In Appendix) unequivocally supports $PM_{2.5}$ as a major independent risk factor for cardiorespiratory morbidity and mortality, with effect magnitudes consistently exceeding those reported for PM_{10}, reflecting the greater biological toxicity of fine particles at equivalent mass concentrations.

Nitrogen dioxide (NO_2), a key marker of traffic-related air pollution, emerged as one of the pollutants with the most consistent evidence across both mortality and hospitalization endpoints. As presented in Table 15 (In Appendix) , NO_2 was associated with significant increases in pulmonary embolism hospitalization (OR 1.11, 95% CI: 1.09–1.13) and mortality (OR 1.05, 95% CI: 1.00–1.11) [31], while [32] reported a 1.7% rise in overall cardiovascular hospitalization, rising to 2.8% for arrhythmias (0.6–5.2%) and 4.4% for heart failure (2.0–6.8%), highlighting a differential susceptibility of specific cardiac conditions to NO_2 exposure. Study [35] documented significant increases in all-cause hospitalization across all age groups, with particular elevation in young individuals (RR 0.22 per 10 $\mu g/m^3$). The mortality evidence assembled in Table 22 (In Appendix) reveals a broader and more consistent pattern. Cohort study [16] reported significant increases in all-cause mortality (HR 1.06, 95% CI: 1.03–1.08), lung cancer mortality (HR 1.11, 95% CI: 1.05–1.17), and respiratory mortality (HR 1.15, 95% CI: 1.10–1.20). Study [39] reported significant elevations across all-cause, cardiovascular, ischemic heart disease, lung cancer, and ischemic stroke mortality. Notably, [20] reported the highest HR for cardiovascular mortality (HR 1.13, 95% CI: 1.07–1.20), while [45] documented a significant increase in overall cardiovascular mortality (HR 1.26, 95% CI: 1.06–1.51) and all-cause mortality (HR 1.13, 95% CI: 1.04–1.23). The hospital admission evidence in Table 38 (In Appendix) further confirms NO_2's pathophysiological relevance, with [105] reporting significant increases in cardiovascular (3.5% rise), stroke (4.5% rise), arrhythmia (3.7% rise), and acute myocardial infarction admissions (2.8% rise), and [106] documenting significant cerebrovascular disease risk (RR 1.02, 95% CI: 1.01–1.03). Across all three tables, the evidence collectively identifies NO_2 as a robust predictor of cardiovascular, cerebrovascular, respiratory, and oncological morbidity and mortality, with effect sizes of particular magnitude for cardiac arrhythmias and lung cancer endpoints.

Ozone (O_3), a secondary air pollutant with strong oxidative properties, demonstrated a more complex and sometimes heterogeneous pattern of associations with health outcomes compared to particulate matter. As indicated in Table 14 (In Appendix), O_3 was associated with a statistically significant reduction in pulmonary embolism hospitalization (OR 0.93, 95% CI: 0.91–0.95) [31], a counterintuitive finding that may reflect confounding by season or temperature, as O_3 concentrations peak during warm periods when thromboembolic risk may be differentially modified. Ischemic stroke risks (RR 1.01, 95% CI: 0.99–1.02) for hospitalizations and (RR 0.99, 95% CI: 0.96–1.02) for mortality [33] were not statistically significant. The mortality evidence in Table 23 (In Appendix), however, reveals a more consistent positive association for short-term exposure metrics. Time-series studies [50] and [71] reported significant increases in all-cause mortality (2.20% rise and 2.0% rise respectively) per O_3 increment, while [57] documented a modest but significant 0.04% rise in all-cause mortality. In contrast, cohort studies [16] and [20] reported significant inverse associations for several causes of death, with [16] reporting HRs of 0.96 for all-cause, 0.96 for cardiovascular, 0.94 for lung cancer, and 0.93 for respiratory mortality. These inverse associations in cohort studies likely reflect chronic exposure adaptation effects and the complex interplay between ozone and other co-pollutants over extended follow-up periods. Hospital admission evidence in Table 39 (In Appendix) was relatively sparse, with non-significant associations for ischemic stroke [113] and a significant positive association for respiratory admissions [122]. The overall pattern across Tables 14, 23, and 39 (In Appendix) suggests that acute O_3 exposure confers significant short-term mortality risk, while the direction of long-term associations remains more complex and context-dependent, underscoring the need to account for temporal exposure windows when interpreting ozone epidemiology.

Sulfur dioxide (SO_2), a pollutant predominantly emitted from fossil fuel combustion, demonstrated variable associations with health outcomes across the included studies, with effect magnitudes generally smaller than those observed for $PM_{2.5}$ or NO_2. Evidence from Table 16 (In Appendix) indicates that SO_2 exposure was associated with non-significant trends in pulmonary embolism hospitalization (OR 1.01, 95% CI: 1.00–1.03) and mortality (OR 1.02, 95% CI: 0.97–1.06) [31], and a non-significant positive trend for acute myocardial infarction hospitalization (RR 1.06, 95% CI: 0.94–1.18), with ischemic stroke hospitalization showing an inverse trend (RR 0.93, 95% CI: 0.85–1.01) [33]. The mortality evidence in Table 24 (In Appendix) presents more consistent positive signals. Cohort study [16] reported significant increases in all-cause mortality (HR 1.03, 95% CI: 1.01–1.05), cardiovascular mortality (HR 1.03, 95% CI: 1.01–1.05), and respiratory mortality (HR 1.07, 95% CI: 1.04–1.10), suggesting that chronic SO_2 exposure carries meaningful long-term cardiovascular and respiratory mortality risk even at relatively low concentrations. Of particular note, study [94] reported a significant hypertension-attributable mortality risk of 4.344% per 10 $\mu g/m^3$ increase in SO_2, the largest pollutant-specific hypertensive effect estimates in the reviewed evidence base. Study [80] reported a large but statistically imprecise rise in all-cause mortality (16.13%, 95% CI: −2.00 to 34.58), reflecting significant heterogeneity in SO_2 attributable risk across geographic settings. Hospital admission findings from Table 40 showed significant associations between SO_2 and respiratory disease admissions (RR 1.229, 95% CI: 1.073–1.409) [125] and non-significant trends for ischemic stroke and acute myocardial infarction [113]. Overall, the evidence from Tables 16, 24, and 40 (In Appendix) supports a modest but real contribution of SO2 to cardiovascular, respiratory, and hypertensive health burden, with effects that are most apparent in cohort studies examining long-term chronic exposures.

Carbon monoxide (CO) was among the less extensively studied pollutants in the reviewed literature, yet available evidence indicates biologically plausible associations with specific morbidity endpoints. As shown in Table 17 (In Appendix), CO exposure was significantly associated with pulmonary embolism hospitalization (OR 1.03, 95%

CI: 1.02–1.05) [31], consistent with CO's established role in promoting a pro-coagulant physiological state through carboxyhemoglobin formation and endothelial dysfunction. Corresponding mortality risks for pulmonary embolism (OR 1.01, 95% CI: 0.96–1.06) [31] and for acute myocardial infarction hospitalization (RR 1.00, 95% CI: 0.87–1.15) [33] did not achieve statistical significance, potentially reflecting insufficient statistical power rather than a true absence of effect. Mortality data from Table 25 (In Appendix) present divergent findings: study [80] reported a statistically significant modest increase in all-cause mortality (0.12% rise, 95% CI: 0.07–0.17), while [54] observed non-significant inverse trends for all-cause (−0.79%), cardiovascular (−1.47%), and a modest non-significant increase for respiratory mortality (0.41%). These contrasting findings likely reflect differences in baseline CO concentrations, climate contexts, and confounding by co-pollutants. Although CO's contribution to ambient outdoor pollution mortality appears smaller than for $PM_{2.5}$ or NO_2, its significant association with thromboembolic hospitalizations documented in Table 17 (In Appendix) warrants continued investigation, particularly in contexts of wildfire-related CO exposure and vehicular emissions.

Temperature extremes both heat and cold represent among the most extensively documented environmental determinants of morbidity and mortality in the reviewed literature. Evidence from Table 18 (In Appendix) demonstrates that cold temperature significantly increased myocardial infarction hospitalization risk (RR 1.10, 95% CI: 1.03–1.17) [33], while cold-associated stroke mortality was markedly elevated (RR 1.38, 95% CI: 1.13–1.68) and hot temperature significantly increased stroke mortality risk (RR 1.48, 95% CI: 1.11–1.98) [33]. These findings confirm a bidirectional thermal risk for cerebrovascular and cardiovascular events. The mortality evidence in Table 26 (In Appendix), the most extensive temperature-related table in this review, documents striking heat-related mortality elevations. Study [48] reported that heat waves increased all-cause mortality by 10.65% and respiratory mortality by 17.60%, while [51] reported hot temperature-associated increases of 14.6% for all-cause, 19.9% for cardiovascular, and 33.1% for respiratory mortality. Study [53] confirmed significant associations with both cold (all-cause RR 1.10) and hot (all-cause RR 1.06) temperature mortality. Study [55] reported a very large cold-associated cardiovascular mortality risk (RR 1.83, 95% CI: 1.31–2.55), among the largest cardiovascular estimates across all pollutants and climate parameters reviewed. Study [67] revealed significant gender- and age-differential effects, with older women (≥85 years) experiencing the largest proportional mortality increase (5.2% per temperature unit). Study [73] confirmed that extreme heat elevated all-cause mortality by RR 1.22 in Rome and RR 1.08 in Stockholm, with significant city-specific variation in psychiatric, diabetic, and COPD-related mortality. Hospital admission evidence from Table 29 (In Appendix) demonstrated that cold temperature significantly elevated admissions for ischemic heart disease (RR 1.51, 95% CI: 1.09–2.08) and myocardial infarction (RR 1.39, 95% CI: 1.01–1.91) [102], while cold-associated cardiovascular admissions were significantly elevated (IRR 1.20, 95% CI: 1.10–1.30), heart failure admissions (IRR 1.27), and stroke admissions (IRR 1.22) [111]. Trauma admissions increased significantly with temperature (IRR 1.008, 95% CI: 1.002–1.014) [112]. The convergent evidence across Tables 18, 26, and 29 establishes temperature extremes as among the most potent environmental determinants of both mortality and hospitalization, with cold-related cardiovascular effects and heat-related respiratory and psychiatric effects representing key vulnerable endpoints.

Rainfall, as a meteorological parameter, demonstrated more limited but potentially clinically relevant associations with specific health endpoints. Mortality evidence from Table 27 (In Appendix) was sparse, with [84] reporting a non-significant association between rainfall and all-cause mortality (RR 1.004, 95% CI: 0.997–1.010), suggesting that rainfall's direct contribution to all-cause mortality independent of temperature and other meteorological variables remains uncertain. Hospital admission findings in Table 30 present a more nuanced picture: study [110] documented a significant positive association between rainfall and psychiatric admissions (coefficient 0.003, p=0.001), while [123] reported a significant association with sickle cell disease admissions (RR 1.06, 95% CI: 1.01–1.12), potentially reflecting rainfall-related changes in barometric pressure and temperature that precipitate vaso-occlusive crises. In contrast, [96] found a non-significant association with psychiatric admissions. The evidence on humidity—an atmospheric parameter closely related to rainfall—from Table 34 reveals that low humidity significantly increased both angina pectoris (RR 1.002) and ischemic heart disease (RR 1.38, 95% CI: 1.05–1.79) hospitalization, while high humidity had a paradoxically protective association with these endpoints [98], [102]. These findings collectively suggest that moisture-related meteorological parameters affect specific disease-susceptible subpopulations and health conditions, though the mechanisms underlying rainfall–health associations warrant further investigation to disentangle direct effects from those of temperature and pollutant co-exposures.

Beyond temperature and rainfall, several additional meteorological parameters demonstrated significant associations with specific hospital admission outcomes. Sunshine and daylight duration data presented in Table 31 (In Appendix) revealed divergent associations: study [96] reported a significant positive association between sunshine and psychiatric admissions (coefficient 0.10, $p<0.01$), while [109] documented a significant inverse association (coefficient −0.012, p=0.002), a discrepancy that may reflect differences in geographic location, population characteristics, and the specific psychiatric diagnoses assessed. Study [120] independently confirmed that hospitalization rates for psychiatric conditions rose significantly with increasing sunshine ($p<0.05$). Trauma admissions increased significantly with both sunshine (IRR 1.016) and daylight duration (IRR 1.014) [112], consistent with behavioral mechanisms linking outdoor activity and trauma risk to ambient light conditions. Snow cover, examined in Table 32 (In Appendix), was significantly associated with a reduction in psychiatric admissions (coefficient −0.06, p=0.04) [96], suggesting a behavioral or biological pathway linking reduced outdoor exposure during snow conditions to lower short-term psychiatric hospitalization burden. Air pressure data

from Table 35 (In Appendix) demonstrated that low atmospheric pressure was significantly associated with reduced acute myocardial infarction admissions (RR 0.67, 95% CI: 0.47–0.96) [102], while maximum air pressure was significantly associated with increased mania and depression admissions [110]. These findings, while based on limited evidence from individual studies, collectively highlight the multidimensional meteorological determinants of hospital admissions for psychiatric, cardiovascular, and trauma-related conditions, supporting the need for integrated multi-parameter meteorological analysis in future environmental epidemiology research.

Ultrafine particles (UFP), defined as particles with an aerodynamic diameter below 0.1 μm, represent an emerging area of environmental health research given their superior capacity to translocate across biological barriers, including the blood-brain barrier and alveolar epithelium. Evidence from Table 28, though limited to two studies, provides preliminary support for UFP-associated mortality risk. Study [88] reported significant increases in all-cause mortality (0.8% rise, 95% CI: 0.2–1.5) and cardiovascular mortality (0.9% rise, 95% CI: 0.2–1.7) per increment in UFP concentration, consistent with the hypothesis that ultrafine particles promote systemic cardiovascular inflammation and autonomic dysfunction. Study [91] documented a significant 4.46% rise in respiratory mortality (95% CI: 1.52–7.48%) per 3,223 particles/cm^3, suggesting that inhalational exposure to UFP at the concentrations typical of urban traffic environments confers a meaningful respiratory mortality burden. These findings, while based on limited published evidence, reinforce the biological plausibility of UFP toxicity and highlight the critical need for expanded epidemiological research on ultrafine particle exposures, particularly in densely populated urban environments where vehicular sources predominate.

The synthesis of evidence across all included tables provides compelling and converging support for the hypothesis that ambient environmental exposures encompassing both air pollutants and meteorological parameters exert substantial, clinically meaningful, and often statistically significant effects on both hospital admissions and mortality across a spectrum of cardiovascular, respiratory, cerebrovascular, psychiatric, and oncological health outcomes. Among air pollutants, $PM_{2.5}$ and NO_2 emerged as the most consistent and potent predictors of mortality and morbidity, demonstrating significant hazard ratios and risk ratios across diverse study designs, geographic settings, and health endpoints. PM_{10} and SO_2 contributed additional and partially independent mortality and hospitalization risks, particularly for cardiovascular and respiratory disease. CO and O_3 demonstrated more heterogeneous evidence, with CO showing notable thromboembolic hospitalization risk and O_3 presenting complex dose-response relationships that differ between acute and chronic exposure paradigms. Among meteorological parameters, extreme temperatures both heat and cold represented the most impactful environmental determinants of mortality and cardiovascular hospitalization, with cold-related cardiovascular and heat-related respiratory mortality dominating the temperature-health literature. Additional meteorological parameters including rainfall, sunshine, air pressure, snow cover, and humidity demonstrated significant associations with specific, often neuropsychiatric and hematological, hospital admission outcomes, broadening the conceptual framework of environmental determinants of health beyond conventional air pollution metrics. The review collectively underscores the urgent need for integrated multi-pollutant, multi-meteorological exposure assessment in future epidemiological research, the adoption of sensitive susceptible population analyses (particularly for the elderly, children, and individuals with pre-existing disease), and the development of coordinated environmental health policies that simultaneously address air quality standards and climate adaptation strategies. The breadth and consistency of findings across Tables 3 through 41 (In Appendix) position ambient environmental quality as a fundamental and modifiable determinant of population health outcomes globally.

5. Conclusion

In summary, our investigation has unveiled that exposure to inclement weather conditions and heightened levels of atmospheric pollution can lead to a diverse range of adverse health consequences, predominantly manifesting as respiratory and cardiovascular ailments. Moreover, evidence links exposure to these elements with a higher likelihood of hospitalizations and fatalities. However, the academic community must research the correlation between weather patterns, air quality, and significant socio-demographic factors such as ethnicity. The results obtained from our research provide empirical evidence in favor of the notion that policymakers should consider the impact of weather fluctuations and air pollution on the overall welfare of communities when formulating and executing initiatives to alleviate these consequences. In addition, it is imperative for research endeavors to proactively consider forthcoming death rates and the alterations in health-related behaviors associated with shifts in air quality and weather patterns. This proactive strategy will yield valuable data that can inform the formulation of efficient prevention programs targeting environmental preservation and public health enhancement.

Our research recognizes that seasonal variations might influence air pollution and individual exposure levels. Nevertheless, this study is subject to several constraints, such as excluding unpublished or non-indexed journal works/articles and restricting publications published exclusively in English. Moreover, it is worth noting that the evaluation focused solely on quantitative research, potentially overlooking other crucial research methodologies, like opinion columns, letters to the editor, and qualitative research. However, our study thoroughly examines the effects of air pollution and weather conditions on hospital admissions and fatality rates. This viewpoint underscores the significance of opinion pieces and editorials in influencing the trajectory of future research investigations, along with the significance of qualitative research in documenting the lived experiences of people. The strong concordance between the two investigators throughout implementing the inclusion and exclusion criteria substantiates the study's conclusions.

6. References

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