

Review

Combined Effect of Hot Weather and Outdoor Air Pollution on Respiratory Health: Literature Review

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Abstract: Association between short-term exposure to ambient air pollution and respiratory health is well documented. At the same time, it is widely known that extreme weather events intrinsically exacerbate air pollution impact. Particularly, hot weather and extreme temperatures during heat waves (HW) significantly affect human health, increasing risks of respiratory mortality and morbidity. Concurrently, a synergistic effect of air pollution and high temperatures can be combined with weather–air pollution interaction during wildfires. The purpose of the current review is to summarize literature on interplay of hot weather, air pollution, and respiratory health consequences worldwide, with the ultimate goal of identifying the most dangerous pollution agents and vulnerable population groups. A literature search was conducted using electronic databases Web of Science, Pubmed, Science Direct, and Scopus, focusing only on peer-reviewed journal articles published in English from 2000 to 2021. The main findings demonstrate that the increased level of PM₁₀ and O₃ results in significantly higher rates of respiratory and cardiopulmonary mortality. Increments in PM_{2.5} and PM₁₀, O₃, CO, and NO₂ concentrations during high temperature episodes are dramatically associated with higher admissions to hospital in patients with chronic obstructive pulmonary disease, daily hospital emergency transports for asthma, acute and chronic bronchitis, and premature mortality caused by respiratory disease. Excessive respiratory health risk is more pronounced in elderly cohorts and small children. Both heat waves and outdoor air pollution are synergistically linked and are expected to be more serious in the future due to greater climate instability, being a crucial threat to global public health that requires the responsible involvement of researchers at all levels. Sustainable urban planning and smart city design could significantly reduce both urban heat islands effect and air pollution.

Keywords: air pollution; extremely hot weather; heat waves; combined effect; respiratory health; wildfires; urban heat island



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1. Introduction

The increase in respiratory health indicators, including allergic disease, is well-documented globally and, most harmfully, is more pronounced in those regions where it was previously low [1]. Studies conducted around the world show that both air pollution, on the one hand, and weather and climate conditions, on the other, have a negative impact on human respiratory health [1,2]. Endeavors to raise awareness and mitigate the health implications of air contamination are long-standing and successful. Extensive scientific assessments make a plausible argument for the theory of negative influence of air pollution on cardiac and pulmonary health, notwithstanding some whitespaces in scientific knowledge and, consequently, some lingering doubts [3]. Among them, less is known about the impact of the dangerously polluted air on health outcomes in various weather and climatic conditions, with the greatest interest being their potential synergy on respiratory tract during hot seasons and episodes with extremely high temperature [1,4–9]. Of special

interest are extreme events such as prolonged heat waves and wildfires, which cause air pollutants increase to dangerous levels, which affect respiratory health [10].

Outdoor air pollution is a major environmental threat to global public health that requires the liable involvement of researchers at all levels responsible for a number of health hazards and diseases. Air pollution and its health consequences reached severe levels, causing up to 7 million premature deaths each year with even more hospitalizations and sick leave days [11,12]. Epidemiological and toxicological studies show links between the main atmospheric pollutants such as sulfur dioxide (SO₂), carbon monoxide (CO), photochemical oxidants (such as ozone, O₃), nitrogen oxides (NO_x), and airborne particulate matter (PM) and human health. PM is all solid and liquid materials that, due to its small size, become suspended in the air [13–17]. Total suspended particles, inhalable particles (PM₁₀), fine inhalable particles (PM_{2.5} and PM₁), and smoke are some types of PM, with PM₁₀ and PM_{2.5} being the two major pollutants currently controlled [18–20].

Evidence obtained from experimental and epidemiologic research supports the association between air pollution, its short- and long-term influence, and respiratory health [1,2,12,14,17,21–25]. Different indoor and outdoor exposures are associated with various acute and chronic respiratory health outcomes that range in severity, from allergies and general respiratory complaints to lung cancer and mortality, as air pollution is genotoxic and contributes to tumor development, causing persistent inflammation [24]. The high burden of pollution on respiratory diseases includes exacerbation of respiratory problems in individuals already suffering from chronic respiratory diseases, such as asthma, rhinosinusitis, bronchiolitis, decreased lung function, chronic obstructive pulmonary disease (COPD), and respiratory tract infections, leading to repeated hospitalizations and emergency room visits for the underlying disease, premature mortality, as well as the occurrence of new respiratory problems, such as new-onset asthma [1,17,26–28].

Environmental toxicants cause a wide range of effects on the respiratory system, from reversible local reactions to long-term chronic impact: local irritation, direct epithelium cells of the respiratory airways damage, allergic reactions (such as asthma), and cancer [1]. Although most recent epidemiological research focused on short-term (such as 24 h average) exposures to PM, O₃, NO_x, SO₂, CO, black smoke, and other pollutants [1,17,29–32], several studies suggest the long-term influence is more common in people with a predisposing disease—lung and cardiopulmonary diseases include COPD and asthma exacerbations, lung cancer mortality, etc. [19,33–39]—and may be more important from a general public health perspective [33,40–42].

Over the past few years, considerable advances were made in detecting the main biological mechanisms regarding how air contaminants affect human respiratory health [43,44]. The physiopathology of respiratory problems associated with atmospheric pollution can be explained by a cytotoxicity, which is said to be responsible for morphological and functional changes to the respiratory epithelium, as well as the induction of an inflammatory reaction, which may provoke a bronchial hyperresponsiveness and a possible interference with the immune system [45,46]. Epidemiological, clinical, and toxicological studies confirm the main pathway associated with oxidative systems. Particulate matter affects respiratory system depending on their size and penetrating activity: PM₁₀ can be deposited in the respiratory tract, PM_{2.5}, in the alveoli [9]. PM interacts with the alveolar-capillary cells, induces oxidative stress reactions that can disrupt the integrity of lung endothelial cell barrier and inflammation, and leads to airway damage, lung dysfunction, and negative cardiopulmonary outcomes [14,25,47–49]. Ground-level ozone is a known lung irritant that affects the airway mucosa, other lung tissues, and respiratory function, causing oxidative injury to the respiratory epithelium and leading to lung inflammation, decreased lung function, worsening of airway symptoms, and excessive respiratory reactivity [9,50–52].

The higher temperatures and the increased heat waves (HW) frequency amplify exacerbation rate, morbidity, and mortality from weather-related respiratory diseases, especially in vulnerable populations [1,30,53–63]. Health impacts are determined by the level of exposure in terms of HW duration, severity, and frequency, as well as the susceptible population

and its sensitivity [64–66]. Studies illustrate that the implications of high temperature are generally intense on the same day or with a delay of one to four days [53,67,68].

The majority of surplus hospital admissions were shown to be caused by dehydration, heat stroke, and heat exhaustion among people with comorbidities. In addition, an excessive number of patients with secondary diagnoses of pneumonia and influenza-related respiratory problems, chronic obstructive pulmonary disease, chronic bronchitis, emphysema, and asthma were hospitalized [69]. A literature review conducted by Schlegel [68] highlighted the problems of individuals with chronic lower respiratory diseases on hot days as well as in heat waves. In the summer months, acute exacerbations in patients with chronic obstructive pulmonary disease (COPD), which is the main subtype of respiratory diseases, increase sharply during episodes of HW [70–73]. It was found that chronic respiratory diseases can be an important indicator of increased mortality risk among patients in hospitals on hot days [74,75]. The potential explanation may be that the health status of people with chronic respiratory diseases deteriorates rapidly during hot periods [1,54]. In these patients, it can be hypothesized that thermoregulatory responses to heat stress, especially those affecting the respiratory system, may be futile in dissipating excess heat, increasing risk of developing heat stress outcomes such as dehydration and heatstroke [1,76], which in turn leads to a risk of developing pulmonary vascular resistance secondary to peripheral blood accumulation or hypovolemia [30]. The physiologic effects of excess heat on the thermoregulatory system leads to an increase in cardiac output, elevating skin blood flow and respiratory rate [6]. High temperature raises heat loss through the cutaneous surface blood circulation, which may be associated with increased mortality [48,77].

It is well known that generation and dispersion of air contaminants depend on weather characteristics. High temperatures and sunshine cause the elevation in concentration of particulate matter, ozone, sulfate aerosols, and others, especially in hot summers and during heat waves episodes [1,8,9,20,78]. At the same time, the temperature rise is amplified by high levels of pollution, particularly PM and ozone [7,62,79]. Accumulated data show that pollutants, primarily soot (black carbon) and PM, can absorb heat, thereby enhancing local temperatures and affecting the climate [11,80]. Sunlight associated with high air temperatures increases local air pollution, even without additional emissions [79,81,82]. Excessive heat raises energy consumption and consequently affects air pollutants, their distributions, accumulation, changes in chemical composition of particulate matter, and even generation of more toxic pollutants [21,51,79,82–84].

Although many studies on the relationship between temperature, mortality, and morbidity took into account concentrations of air pollutant, only a few examined the potential synergistic effect of high temperature and air contaminants. Understanding the combined effects of both elevated temperature and air pollution, particularly PM and ozone, would allow us to better characterize the climate-related health outcomes under the changing climate [85]. The conclusions drawn from these findings would have compelling medical and social implication for public health, being important for the development of long-term prevention measures. When an episode of hot weather or even HW is predicted, additional measures may be required to reduce the concentrations of pollutant in the air at the beginning of emergency response [82].

The current review summarizes the scientific data on combined effect of air pollution and climate-related hazards, such as hot weather and heat waves, on respiratory health outcomes obtained from both climatological and epidemiological studies. In particular, we considered those climate events that could be most affected by climate change and for which climate conditions are related to air quality: temperature extremes, hot weather, or episodes of heat wave. Wildfires and urban heat island (UHI) effects, pathogenic mechanisms, vulnerable population subgroups, confounder of multiple environmental stressors, climate change impact, and policy implications are discussed.

2. Materials and Methods

A systematical literature search was conducted in November 2020–May 2021 using electronic databases Pubmed, Web of Science, Science Direct, and Scopus for papers published before 20 May 2021. Hand searching of the applicable literature was also performed in relevant journals and bibliographies of included studies. Three conceptual categories were used: “ambient/outdoor air pollution”, “extremely hot weather”, and “respiratory health outcomes”, revealing a total of 253 records. We were seeking for key words “heat wave”, “hot weather”, “high temperature”, “warm season”, “air pollution”, “particulate matters”, “fine particles”, “ozone”, “respiratory mortality”, “respiratory disease”, “asthma”, or “COPD” and also looked for studies cited in the recognized articles. The search identified 40 studies that were selected for the review. The narrative synthesis included all original studies with time-series or case cross-over design, ambient air-pollution and weather exposure, and mortality and/or hospital admission/ambulance call outcomes. All included studies examined respiratory (8th or 9th revision of the International Classification of Diseases (ICD8) or (ICD9)): 460–519; or 10th revision of the International Classification of Diseases (ICD10): J00–J99) mortality or morbidity in all age groups. Study duplicates were removed, and abstracts were screened independently by two authors (E.G. and A.L.). Final selected studies were chosen based on the inclusion criteria that they attempted to feature recorded respiratory/pulmonary health outcomes (deaths and hospital admissions/emergency room visits), had air pollution, warm/hot weather, heat waves, and health outcomes recorded quantitatively, and controlled for the main confounding variables. We placed no restrictions on study design, however, only studies published in the English language were included.

The conceptual framework for these interactions is summarized in Figure 1.

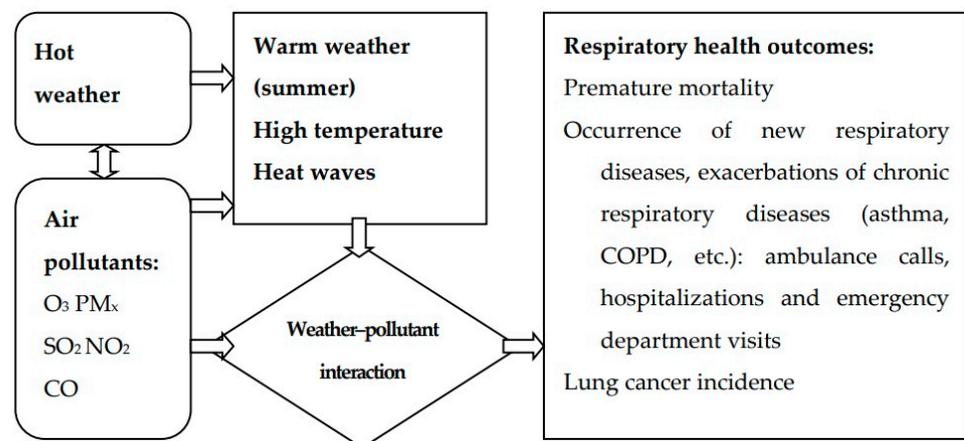


Figure 1. The conceptual framework for combining effect of hot temperatures and air pollution on respiratory health outcomes.

Warm and hot temperatures and heat waves combined with air-pollution (O_3 , PM_x , SO_2 , NO_2 , CO) were shown to act consistently as risk factors for respiratory mortality, hospital admissions, and daily emergency transports, where respiratory health outcomes were taken from the main categories: premature mortality; general respiratory complaints, ambulance calls, hospitalizations, and emergency department visits for respiratory reasons; chronic respiratory diseases such as COPD, asthma, etc.; and lung cancer, as shown in Figure 1.

3. Results

We reviewed the titles and the abstracts of 153 studies, of which 40 met our pre-specified inclusion criteria. Most (23) were from East Asia [37,41,73,86–105], 10 were from Europe [5,30,53,74,75,79,106–109], 5 were from USA and Canada [110–114], and far fewer were included from the Pacific region [115], Latin America, and the Caribbean [116].

The following characteristics of studies involved in the review were extracted: study; study area; study period; weather parameters such as air temperature and humidity, dew point temperature, wind speed and direction, air pressure, precipitation, hours of sunshine; hot weather description, including definition of HW or high temperature used in the study; air pollutants such as PM, O₃, NO₂, SO₂, CO; respiratory health indicators; exposure and confounders; statistical methods and study design; results with outcome and associated lag time. The role of effect modification was investigated in terms of gender and age. Respiratory health outcomes included daily mortality from respiratory causes, respiratory emergency department visits, hospital admissions, ambulance calls, and lung cancer incidence.

Hot weather descriptions were different among the studies. Almost all of them checked for health burden of high temperatures—those levels which exceed the specified percentile of the temperature distribution: 75th, 85th, 95th, 97th, 97.5th, or even 99th percentile for extremely hot weather. In a few papers, high temperature threshold was determined as temperature of minimum mortality [53,116] or temperature with the lowest frequency of ambulance calls [101]. Four studies focused on HW effect in European countries using different definitions of HW and counting for their intensity and duration [5,79,108,110]. Some papers looked at seasonal changes and warm air modifications during warm seasons [41,103,111,113] or in summer [102,104]. In some papers, hot weather influence was corrected by relative humidity expressed in apparent temperature (AT) [117,118] to analyze the perceived rather than the actual thermal load [5,30,41,53,74,79,91,109,110]. Two studies demonstrated the impact of polluted air on respiratory mortality and morbidity for different types of weather, where those with high temperature effects were referred to as dry tropical (DT) and moist tropical (MT) air masses [112,114].

Some studies examined effects of temperature changed by contaminants, and some observed the impact of pollutants modified by hot weather. For instance, a study by Breitner et al. [75] in Germany demonstrated modification in the temperature–mortality association by ozone. Park et al. [96] for South Korea notified the impact changed by temperature on the O₃ exposure, illustrating a pattern consistent with some modification of the effect. The potential interaction between air pollution health outcomes and high temperatures was tested mostly by means of a stratified analysis in time-series; few were tested by case-crossover analyses or through interaction terms. Statistical models used were mainly generalized additive models (GAM), generalized estimating equations (GEE), and generalized linear models (GLM). Almost all models were adjusted for time (seasonal and long-term trends), day of the week, public holidays, and influenza epidemics. Most of them had additional weather confounders such as relative humidity and barometric pressure.

Almost all studies provided consistent evidence of a synergy between the two exposures, with only two showing no evidence of interaction [5,106] and few of them reflecting that low temperature polluted air had a greater impact on respiratory health outcomes than hot air [73,87,101]. In addition, Anderson and Bell [110], for research in urban communities of the USA, showed heat effects on daily mortality from respiratory causes were slightly lower when models included ambient pollution (O₃ and PM₁₀) [110].

Most papers discussed all-cause and specific-cause mortality or morbidity, including respiratory outcomes. Of the identified papers, 24 discussed mortality from respiratory causes [5,53,73–75,79,86,88,90,92,93,95–98,100,106–110,114–116], 14 incorporated respiratory emergency department visits and hospital admissions [30,41,87,89,91,94,99,102–105,111–113], 1 was for ambulance calls for respiratory distress [101], and 1 was for lung cancer incidence [37]. The details of those studies are reported below, separately for respiratory morbidity and mortality and for heat wave episodes.

3.1. Heat, Air Pollution and Morbidity

Strong evidence was obtained for the synergistic impact of ambient air pollution with PM, CO, O₃, SO₂, and NO₂ and hot weather on triggering of health problems [78,119,120].

The magnitude of these effects on respiratory morbidity varies by diseases and individual characteristics; estimates seem to be higher among children and the elderly population. Illustrations of increasing number of daily emergency transports, hospital admissions, and ambulance calls for respiratory problems are summarized in this section. The effect was shown in European cities, especially for elderly aged ≥ 75 [30], in places with high levels of pollution in China, including Beijing and Nanjing [41,102,103,105], in Tokyo, Japan [104], etc. Increased asthma hospitalization was demonstrated to be extremely related with high levels of ozone in connection with hot weather in Indianapolis in the USA [111], for emergency department visits among children in St. Louis, USA [113] during warm seasons, as well as for cities in the USA such as North Carolina on hot, dry days [112]. Episodes of high $PM_{2.5}$ and O_3 and extremely hot temperatures ($T > 27.9$ °C) were demonstrated for hospital admissions with COPD exacerbations in Taiwan. Especially significant impact was detected in the elderly with odds ratio (OR) = 1.037 and greater lagged effect [89]. An increased rate for lung cancer incidence in association with high temperatures and PM_1 exposure was discussed by Guo with coauthors [37], who showed stronger incident rate for males. High daily ambulance calls for respiratory distress on extremely hot days (with temperature $T > 99$ th percentile of distribution) with high concentrations of $PM_{2.5}$ and constituents were demonstrated for Kaohsiung in Taiwan [94]. Respiratory emergency transports during summer for episodes with high exposure of PM_{10} and NO_2 were shown to be greater for males than for females in Tokyo, Japan [104]. High temperatures in connection with particulate matter pollution were associated with increases in hospital emergency room visits for respiratory system diseases in Beijing, China, indicating the elderly (age ≥ 65) and women were the more vulnerable groups [105].

Children and adolescents were shown to be a sensitive age cohort. Increased hospitalization of acute bronchiolitis-related disease among children accompanied with high temperature and exposure to NO_2 and PM_{10} at different lag times was shown for Hong Kong [91]. Air pollution during hot weather raised asthma ED visits in St. Louis, USA by 9.45% (95% CI = 1.02, 1.17) among patients 11–17 years old [113].

Geography and type of climate were shown to be important. Michelozzi et al. [30], in a study for Europe, emphasized the raise in apparent temperature was more effective for Mediterranean cities with a +4.5% (95% CI: 1.9–7.3) increase in respiratory emergency visits compared with northern continental cities, where a +3.1% (95% CI: 0.8–5.5) increase in respiratory visits was demonstrated.

On the other hand, there was some polemical evidence for no statistically significant effect of air pollution on respiratory emergency department visits in moderately hot and hot days, for example, in Beijing [99]. Likewise, for ambulance calls with respiratory distress in Taiwan, exposure to the extreme level of $PM_{2.5}$ was more likely to occur at low temperatures [101].

3.2. Heat, Air Pollution and Mortality

The mean daily concentrations of some pollutants can be very high in East Asia; for instance, PM_{10} concentration in eight Chinese cities was found to range from $65 \mu\text{g}/\text{m}^3$ to $124 \mu\text{g}/\text{m}^3$, which is significantly higher than in Western countries [95]. Combined impacts of hot weather and ambient pollution on respiratory mortality are presented in many papers around the world, with more recent research being undertaken in East Asian countries, especially in China, than in Europe or other countries.

Hot weather was found to substantially strengthen the impact of air pollution on respiratory mortality, with PM_{10} and O_3 being the main pollutants, such as that shown by research in Japan [88]. Examples can be shown for heat effect during the excess PM_{10} exposure in Beijing, Tianjin, Guangzhou, Hefei, Wuhan, and other cities in China [92,93,95,97,98,100]. The higher mortality risk was shown for places in China with 0–1 days lag of PM_{10} exposure: 0.45% (95% CI: 0.13–0.78) for respiratory mortality in Beijing [100]; 0.74% (95% CI: –0.33, 1.82) for respiratory and cardiopulmonary mortality in Tianjin [92]; 6.09% (95% CI: 2.42, 9.89) in Guangzhou [93]; 4.6% (95% CI: 2.83–5.85%) for COPD excess mortality in

11 cities [73]; 1.79% (95% CI: 0.75–2.83) for 8 cities, with impact being higher in hotter locales [95]. High temperatures jeopardize the effect of PM₁₀ pollution in South Korea [90]; in Christchurch, New Zealand [115]; in Sao-Paulo, Brazil, with rate ratio (RR) = 1.60% (95% CI: 0.74–2.46) [116]; in Italian cities, with RR = 2.54% (95% CI: 1.31–3.78) for 35+ age cohort [109]. High temperatures also impact exposure to O₃ pollution in Seoul, South Korea [96], Germany [75], England, and Wales [107]. Besides, some studies discussed additional significant effects for SO₂ and NO₂, e.g., research in Hefei, China [98]. The combined effect of polluted air and weather was shown to be high during weather with tropical air masses, both dry and moist, for Canadian cities in summer [114]. The results highlighted that the synergistic effect varies across regions and countries depending on local climate peculiarities, activity patterns, and physical adjustments [1].

The studies demonstrated strong mortality growth on days with increases in concentration of air pollutants, with the effect being higher in older individuals ≥ 65 years [92], ≥ 75 years [53], and ≥ 85 years [75,96] old, in men [90] and women [97,98], in those who are illiterate [97,98], and in subjects in southern cities compared with northern ones [95]. It can be assumed the differences may arise due to personal behavioral and physiological dissimilarities to high temperature and pollution exposure [97].

Predictably, temperature extremes were found to create the greatest mortality burden, with studies in Wuhan, China, a highly polluted “oven” city [97,98], or cities in South Korea [96] as examples. Additionally, examples are presented for extremely high thermal exposure and pollution that resulted in excess mortality in subjects with chronic pulmonary disease in Italian cities (OR = 2.48 (95% CI: 1.50–4.09)) [74] and COPD for cities of Jiangsu province, China (OR = 4.6% (95% CI: 2.83–5.85%)) [73]. However, in contrast, no significant interaction between polluted air and extremely high temperatures was shown for Shanghai [86] and France [106], and a higher effect for low temperature exposure was demonstrated for locales in Jiangsu province, China [73].

3.3. Heat Wave Episodes

As frequency, intensity, and duration of heat waves are expected to increase with climate change, HW episodes are of special interest for public health [37,121]. While most of the studies cited above addressed joint effects of pollutants with high temperature, only four focused on heat wave episodes [5,70,108,110].

Heat waves in urban places of the USA were explored in detail by Anderson and Bell [110]. The joint effect of air pollution caused by elevated levels of PM₁₀ and O₃ during the HW showed the higher estimates of exposure for respiratory disease compared with the total mortality [110].

European studies by Analitis et al. [5,79] focused on specific heat wave episodes, analyzing total and cause-specific mortality, including respiratory disease, where they provided evidence of a significant interaction between high temperature and pollutants, mainly ozone, especially for the elderly [79].

A synergistic effect of heat and air pollution on mortality was shown for extremely long and intensive heat waves in European Russia accompanied with a disastrous wildfire in summer 2010, with Moscow as an example [108]. Main pollutants estimated were PM₁₀ and O₃. The major HW lasted for 44 days, with 24 h average temperatures ranging from 24 °C to 31 °C and PM₁₀ levels exceeding 300 $\mu\text{g}/\text{m}^3$ on several days [108]. An increase of 339 deaths in Moscow from respiratory diseases was found compared to the same period in summer 2009, with RR = 2.05 (95% CI: 1.80–2.39) [108].

Although there was evidence of a synergistic effect of air pollution and high temperatures during HW episodes, a slight decrease in thermal effects was found when models included O₃ and PM₁₀ [5,110], which should be analyzed in the future.

All results from 40 papers mentioned above are summarized in Table 1, where the papers are arranged alphabetically by the first author in the included studies.

Table 1. Characteristics of studies included in the review.

Study	Study Area	Study Period	Weather Parameter ¹	Hot Weather Description ²	Air Pollutant ³	Health Indicator ⁴	Exposure and Confounders ⁵	Statistical Methods and Study Design ⁶	Results
Analitis et al. [79]	Europe: Athens, Barcelona, Budapest, London, Milan, Munich, Paris, Rome, Valencia	Summer months, 1990–2004	3-h T, Td, V, P	HW: period of at least 2 days with AT _{max} > 90th percentile of the monthly city-specific distribution, or a period of at least 2 days with T _{min} > 90th percentile of the minimum monthly distribution and AT _{max} > the median monthly value; intensity and duration of HW	PM ₁₀ , O ₃ , NO ₂ , SO ₂ , CO High pollution >75th percentile of the overall pollutant distribution	MRC (ICD9: 460–519)	HW—main exposure, adjusted for AP, confounders: P, V, calendar month, DOW, holiday, time trend	An interaction term between HW and each single pollutant; random effects meta-analysis for summarizing the city-specific results; time-series analysis	MRC increase during HW 54% higher on high O ₃ days compared with low, among people age 75–84 years; significant positive interaction of HW and PM ₁₀ for MRC in Mediterranean cities (65–74 years); HW effect on MRC is larger than on other causes of death, although the effect modification was less evident for MRC
Analitis et al. [5]	Europe: Athens, Barcelona, Budapest, Helsinki, London, Paris, Rome, Stockholm, Valencia	2004–2010	1-h T, Td, RH, V, D, P	HW: (1) period of at least 2 days with AT > the 90th percentile of the monthly distribution; or (2) period of at least 2 days with T _{min} > the 90th percentile of its respective distribution and AT > median monthly value	PM ₁₀ , O ₃ , NO ₂ High pollution >75th percentile of the city-specific pollutant distribution	MRC (ICD9: 460–519; ICD10: group J), age groups: 15–64, 65–74, >75 years and all ages	HW—main exposure, adjusted for pollutants, P, V, calendar month, day of the week, holiday, time trend	GEE modeling approach; Poisson regression for city-specific analysis; random effects meta-analysis to combine city-specific results; time-series of HW days	Synergistic effect between hot temperature and O ₃ , PM ₁₀ , and NO ₂ on MRC <u>No evidence for synergy for any of the pollutants and health endpoints analyzed during heat wave days ⁷</u>
Anderson and Bell [110]	USA: 107 urban communities	1987–2000	Daily T _{mean} , T _{max} , T _{min} , Td	HW, 6 types: periods of 2 or more or 4 or more days of continuous AT more than 98.5th, 99th, or 99.5th percentile of the community's temperature distribution	PM ₁₀ (lag 0–1), O ₃	MRC (ICD9 codes 480–486, 490–497, or 507), age (<65, 65–74, ≥75 years)	T—main exposure, adjusted for pollutants; confounders: day of the week, Td; income, unemployment, education, public transportation, race, urbanicity, population	Bayesian hierarchical model used for community-specific estimates of absolute and relative heat and HW effects	Association between HW and MRC, although estimates are uncertain; somewhat higher estimates for respiratory, compared with total deaths. Heat effects slightly lowered when models included O ₃ and PM ₁₀
Baccini et al. [53]	Europe: Athens, Barcelona, Ljubljana, Milan, Rome, Turin, Valencia (Mediterranean), Budapest, Dublin, Helsinki, London, Paris, Prague, Stockholm, Zurich (north-continental)	1990–2000	3-h T, Td, V, P	High temperature: percent change in mortality associated with a 1 °C increase in AT _{max} above the city-specific threshold, defined as AT with the minimum mortality rate (lag 0–3)	NO ₂ (lag 0–1)	MRC (ICD9: 460–519), age (15–64, 65–74, 75+ years)	AT—main exposure, adjusted for AP; confounders: holidays, DOW, day of calendar month, long-term time trend, P (lag 0–1), V	GEE, combined in a Bayesian random effects meta-analysis; distributed lag models for studying the delayed effect of exposure; time-varying coefficient models are used to check the assumption of a constant heat effect	Higher associations between heat and MRC, with estimated percent changes equal to 6.7 (2.4 to 11.3) and 6.1 (2.6 to 11.1) for Mediterranean and north continental cities, respectively; heat effect particularly large in the elderly (75+): 8.1% for the Mediterranean region and of 6.6% for the north-continental region; adjustment for AP changed the MRC increase with 1 °C rise in AT _{max} from 6.2% to 5.5% in Athens to a negligible effect in Stockholm

Table 1. Cont.

Study	Study Area	Study Period	Weather Parameter ¹	Hot Weather Description ²	Air Pollutant ³	Health Indicator ⁴	Exposure and Confounders ⁵	Statistical Methods and Study Design ⁶	Results
Breitner et al. [75]	Germany: Munich, Nuremberg, Augsburg	1990–2006	Daily T, RH, P	High temperature: increase from the 90th (20.0 °C) to the 99th percentile (24.8 °C) of 2-day T _{mean} (lag 0–3)	PM ₁₀ , O ₃	MRC (ICD9: 460–519; ICD10: J00–J99)	T—main exposure, adjusted for AP; confounders: long-term trend/seasonality, calendar effects, DOW, influenza, RH, P	Poisson regression models combined with distributed lag non-linear models; time-series analysis	High 2 day and 15 day T _{mean} and consistent MRC increases; 85+ most susceptible to heat effects; some effect modification by O ₃ but not for PM ₁₀
Byers et al. [111]	USA: Indianapolis	2007–2011	T, RH, P	Warm season (April–September)	PM _{2.5} , O ₃ , SO ₂	Daily asthma-related EDV; ≥5 years old	AP—main exposure, adjusted for T; confounders: year, month, DOW, holidays; smoothing function of time	Poisson GLM to estimate the association between AP and asthma ED; RR for risk estimation, using interquartile range in single pollutant models, accounting for age group and season	O ₃ and SO ₂ increases associated with increased asthma morbidity
Chen and Kan [86]	China: Shanghai	January 2001–December 2004	T, RH	High temperature: T > 85th percentile of distribution	PM ₁₀ , O ₃ , NO ₂ , SO ₂	MRC (ICD9: 460–519; ICD10: J00–J99)	AP—main exposure	GAM; time-series analysis	<u>No significant interaction between air pollution and extremely high temperature for MRC</u>
Chen et al. [87]	China: Guangzhou	January 2014–December 2017	T, RH	High temperature: T > 75% quartile, or 4th DTL defined as 27.4–31.1 °C	CO, PM _{2.5} , SO ₂ , NO ₂ , O ₃	REDV	DTL—main exposure, adjusted for AP; confounders: DOW, holiday, RH	Quasi-Poisson varying coefficient regression models	Significant adverse effect on REDV of interactions between SO ₂ and the 4th DTL (27.4–31.1 °C)
Chung et al. [88]	Taiwan: Taipei, Taichung, Kaohsiung; South Korea: Seoul, Incheon, Daejeon, Daegu, Gwangju, Busan; Japan: Sapporo, Sendai, Tokyo, Nagoya, Osaka, Kitakyushu	Taiwan (1994–2007), South Korea (1992–2010), Japan (1972–2009)	Daily T _{mean} , T _{max} , T _{min} , RH, P	High temperature: T > 99th percentile; comparing the 90th and 99th percentiles of T _{mean} percentiles specific to each city	O ₃ , PM ₁₀	MRC (ICD8 460–519; ICD9 460–519; ICD10 J00–J99), age (<65, 65–75, >75 years)	T—main exposure, adjusted for AP; confounders: DOW, influenza, RH, P	Generalized Poisson semiparametric regression model; city-specific effect estimates combined to generate an overall estimate for each country using Bayesian hierarchical modeling	Heat effects greater for cities in Korea and Japan; in all countries, heat effect increases with age and is higher for cardiorespiratory mortality than for non-cardiorespiratory mortality

Table 1. Cont.

Study	Study Area	Study Period	Weather Parameter ¹	Hot Weather Description ²	Air Pollutant ³	Health Indicator ⁴	Exposure and Confounders ⁵	Statistical Methods and Study Design ⁶	Results
Ding et al. [89]	Taiwan: Taipei, New Taipei	2000–2013	Daily T, RH, P	High temperature: T > 27.9 °C	PM _{2.5} , O ₃ , SO ₂ , CO, NO ₂	REDV COPD-associated, including: chronic bronchitis (ICD9 491), emphysema (492), chronic, airway obstruction (496); >40 years old (40–64, 65–79, >80)	AP—main exposure, adjusted for T, RH, P	Conditional logistic regression models with ORs and 95% CIs; case-crossover study design	PM _{2.5} and O ₃ have significantly greater impact on elderly COPD-associated EDV on hot days; greatest effect in the days with T > 27.9 °C (OR = 1.037 (95% CI 1.001–1.074))
Guo et al. [37]	China: 345 counties (districts)	2014–2015	Annual and monthly T, RH	High temperature: T > 75% percentile of distribution	PM ₁	Lung cancer incidence (ICD10 C33–34)	T and RH—main exposure, adjusted for AP, confounders (smoking, education, economic status, occupation, employment, urban, population size)	Multivariable linear regression model for stratified and combined datasets	Strong association between PM ₁ and the incidence rate of male lung cancer with high T
Hales et al. [115]	New Zealand: Christchurch	June 1988–December 1993	T _{max}	High temperature: T _{max} > 3rd quartile (20.5 °C)	PM ₁₀	MRC (ICD9 460–519)	AP—main exposure, adjusted for T _{max} , controlling for season	GLM; interaction terms; time-series analysis	Increase of 1 °C on the day of death associated with a 3% MRC increase; increase in PM ₁₀ (lag 0–1)—4% MRC increase
Hanna et al. [112]	USA: 5 cities in North Carolina (Asheville, Charlotte, Greensboro, Raleigh, Wilmington)	1996–2004	Daily T _{max} , Td, V, D, P, CC, R	Weather types: DT (hot and dry), MT (hot and humid)	O ₃ (lag 0–5)	HA for asthma (ICD9 493.x)	AP—main exposure, adjusted for DOW, seasonality, long-term trend, Td	GLM; time-series analysis	Increased asthma hospitalizations during episodes with high O ₃ levels associated with DT and MT air masses
Kim et al. [90]	South Korea: seven cities	2000–2009	Daily T, RH, P	High temperature: T > 95–99th percentile of distribution	PM ₁₀	MRC (ICD10 J00–J99)	AP—main exposure, adjusted for T; confounding factors: seasonal variation, DOW, RH, P	GAM; stratification; time-series analysis	Strong harmful effects from PM ₁₀ with the highest temperature range (>99th percentile) in men, with a very high temperature range (95–99th percentile) in women
Leung et al. [91]	Hong Kong	January 2008–December 2017	Daily T, RH, R, V	High temperature: high T and AT	PM ₁₀ , NO ₂ , SO ₂ , O ₃	29,688 acute bronchiolitis-related HA (ICD9: 466.1 487.0, 487.1, 487.8), children ≤2 years old	T and AP—main exposure, adjusted for DOW, holiday	Quasi-Poisson GAM in conjunction with DLNMs; time-series analysis	Increased adjusted RR of acute bronchiolitis-related hospitalisation among children is associated with high T and exposure to NO ₂ and PM ₁₀ at different lag times
Li et al. [92]	China: Tianjin	2007–2009	Daily T, RH	High temperature: T > 20 °C	PM ₁₀ , NO ₂ , SO ₂	MRC: respiratory (ICD8 J00–99), cardiopulmonary (J00–99), age groups <65, ≥65	T—main exposure, adjusted for AP; confounders: days of calendar time, DOW, RH, holiday	Poisson GAM model natural logarithm of the expected daily death counts; time-series analysis	10 µg/m ³ increment of PM ₁₀ on T high for MRC (0.74% (95% CI: −0.33, 1.82), lag 0–1); PM ₁₀ effects on T high stronger on older (≥65 years)

Table 1. Cont.

Study	Study Area	Study Period	Weather Parameter ¹	Hot Weather Description ²	Air Pollutant ³	Health Indicator ⁴	Exposure and Confounders ⁵	Statistical Methods and Study Design ⁶	Results
Li et al. [93]	China: Guangzhou	2005–2009	Daily T, RH	High temperature: T > 95th percentile of distribution	PM ₁₀ , NO ₂ , SO ₂	MRC (ICD9: J00–99)	T—main exposure, adjusted for AP; confounders: days of calendar time, DOW, RH, holiday	GAM; time-series analysis	10 µg/m ³ increase in PM ₁₀ concentrations (lag 0–1) on high temperature days for MRC (6.09% (95% CI: 2.42, 9.89))
Lin et al. [94]	Taiwan: Kaohsiung	2006–2010	Daily T, RH, V	High temperature: T > 99th percentile of distribution	PM _{2.5} , EC, NO _x , SO _x	Daily AC of cases diagnosed with respiratory distress	T—main exposure, adjusted for AP, and vice-versa; confounders: time trend, seasonality, DOW, influenza, holiday, RH, V	Quasi-Poisson function and DLNM; time-series analysis	Significant association with T, PM _{2.5} , and concentrations of constituents
Ma et al. [73]	China: Jiangsu, 11 cities	2014–2017	Daily T, RH, R, P, S	High temperature: T > 97.5th percentile of distribution	PM ₁₀ , PM _{2.5} , SO ₂ , NO ₂ , CO, O ₃	MRC (ICD10 J00–J99), including COPD (J41–J44)	T—main exposure, adjusted for AP	1: DLNM combined with generalized linear model (GLM); 2: univariate random-effect meta-analysis for construction the overall cumulative exposure-response curves for all cities	Excess mortality for COPD (4.6% (95% CI: 2.83%–5.85%)) 20.58% (95% CI: 13.97%–24.96%) at <u>cold exposure</u>
Meng et al. [95]	China: Guangzhou, Hangzhou, Shanghai, Shenyang, Suzhou, Taiyuan, Tianjin, Wuhan	2001–2008	Daily T, RH	High temperature: T > 95th percentile of the distribution	PM ₁₀ (lag 0–1)	MRC (ICD10 J00–J98)	PM ₁₀ —main exposure, adjusted for T; confounders: DOW, RH	Poisson GAM; time-series analysis	10 µg/m ³ increment in PM ₁₀ during T high: increase for MRC (1.79% (95% CI: 0.75–2.83)); higher effect for southern cities
Michelozzi et al. [30]	Europe: Barcelona, Budapest, Dublin, Ljubljana, London, Milan, Paris, Rome, Stockholm, Turin, Valencia, Zurich	1990–2001	T, Td	High temperature: AT _{max} > 90th percentile of the distribution	NO ₂ as an indicator of traffic-related pollution	REDV (ICD9 460–519); age groups: all ages, 65–74, 75+	T—main exposure, adjusted for NO ₂ (lag 0–1); confounders: holidays, DOW, calendar month, P (lag 0–3), V	Semiparametric approach, which includes penalized cubic regression splines; time-series analysis	1 °C increase in AT _{max} gives increase in REDV by +4.5% (95% CI: 1.9–7.3) and +3.1% (95% CI: 0.8–5.5) in the 75+ age group in Mediterranean and North-Continental cities
Mohr et al. [113]	USA: St. Louis	1 June 2001–31 May 2003	T _{max}	Warm season: median seasonal T (86.5 °F = 30.3 °C)	EC, O ₃ , SO ₂ , NO _x	EDV for asthma (ICD9 J493), children aged 2–17 years	AP—main exposure, controlling for season (T), weekend, DOW, exposure, allergens	Poisson generalized estimating equations using a 1-day lag between exposure and ED visit; time-series analysis	0.10 µg/m ³ increase in EC gives 9.45% increase in asthma ED visits among 11–17-year-olds (95% CI = 1.02, 1.17); risk increased with increasing temperatur

Table 1. Cont.

Study	Study Area	Study Period	Weather Parameter ¹	Hot Weather Description ²	Air Pollutant ³	Health Indicator ⁴	Exposure and Confounders ⁵	Statistical Methods and Study Design ⁶	Results
Park et al. [96]	South Korea: Seoul	June 1999–December 2007	1-hourly T, RH	High temperature: T ≥ 90th, 95th percentile of distribution	PM ₁₀ , CO, NO ₂ , O ₃ , SO ₂ (lag 0–1)	MRC (ICD10 J00–J99)	AP—main exposure, adjusted for T; confounders: long-term and seasonal trends, DOW, holiday, RH, influenza	GLM with natural cubic splines; temperature-stratified model; time-series analysis	O ₃ effect stronger in summer; 85+ especially vulnerable to AP during extremely high T
Pascal et al. [106]	France: Bordeaux, Le Havre, Lille, Lyon, Marseille, Paris, Rouen, Strasbourg, Toulouse	2000–2006 (August 2003 excluded due to extreme HW)	T	High temperature: T > 97.5th percentile	PM ₁₀ , PM _{10–2.5} , PM _{2.5} , O ₃	MRC (ICD10 J00–J99)	AP—main exposure, adjusted for T; confounders: long-term and seasonal trends, DOW, influenza, holiday	GAM; stratification; time-series analysis	Statistically non-significant results for MRC
Pattenden et al. [107]	England and Wales: 15 conurbations	May–September, 1993–2003	Daily T, RH	High temperature: ‘Hot days’—2-day T > 95th percentile whole-year distribution	O ₃ , PM ₁₀	MRC (ICD9 4600–5199, ICD-10 ‘J’)	AP—main exposure, adjusted for T; confounders: DOW, holiday, seasonal and long-term time trends; time series analysis	Poisson regression GLM; time-series analysis	Independent association of heat with MRC, mean rate ratio 1.139 (95% CI: 1.079–1.202); adjusted for O ₃ mean interaction rate ratio 1.008 (95% CI: 0.992–1.023); high heat and O ₃ effect for aged <75
Pinheiro et al. [116]	Brazil: Sao Paulo	1998–2008	Daily T, T _{max} , RH _{min}	High temperature: T > temperature of the minimum mortality	PM ₁₀	MRC (ICD10-X), >60 years old	T—main exposure, adjusted for AP; AP—main exposure, adjusted for T; controlled for seasonality, DOW, holidays	(1) Case-crossover approach with different types of case-control matching for isolated effects; (2) GAM, bidirectional case-crossover analysis matched by period, time-series analysis; (3) conditional logistic regression models to compare results (1) and (2)	10 µg/m ³ increase in PM ₁₀ for MRC (RR = 1.60% (95% CI: 0.74–2.46)); higher RR for MRC at high T and PM ₁₀ = 60 µg/m ³
Qian et al. [97]	China: Wuhan	1 July 2000–30 June 2004	T, RH	High temperature: T > 95th percentile distribution (31.7 °C)	PM ₁₀ , SO ₂ , NO ₂ , O ₃	MRC (ICD9 460–519; ICD10 J00–J98)	AP—main exposure, adjusted for T; confounders: DOW, time trend, RH	GAM; stratification; time series analysis	Synergistic effects of PM ₁₀ and T high: 10 µg/m ³ increase in PM ₁₀ (lag 0–1) during T high gives increase in MRC 1.15% (CI: 3.54% to 6.07%)

Table 1. Cont.

Study	Study Area	Study Period	Weather Parameter ¹	Hot Weather Description ²	Air Pollutant ³	Health Indicator ⁴	Exposure and Confounders ⁵	Statistical Methods and Study Design ⁶	Results
Qin et al. [98]	China: Hefei	January 2008–December 2014	Daily T, RH, P	High temperature: T > 95th percentile distribution (30.25 °C)	PM ₁₀ , SO ₂ , NO ₂	MRC (ICD10 J00–J98) stratified by gender, age (<75, 75–84, 85+)	AP—main exposure, adjusted for T; confounders: holiday, RH, P, age, gender, and educational levels	Quasi-Poisson regression GAM model with natural cubic splines; time series analysis	10 µg/m ³ at high temperatures was 7.18 (95% CI: 2.44 to 12.13) for PM ₁₀ , 28.23 (95% CI: 8.25 to 48.61) for SO ₂ , and 25.58 (95% CI: 3.66 to 47.99) for NO ₂
Shaposhnikov et al. [108]	Russia: Moscow	6 June–18 August 2010	Daily T, RH	HW: T > 97th percentile of the year-round distribution, duration >5 days	PM ₁₀ , O ₃	MRC (ICD10 J00–J98)	T—main exposure, adjusted for AP; confounders: day number, DOW, RH	GLM; identity link and Gaussian errors; data for 2006–2010 used as a corresponding period; time series analysis	Added deaths due to interaction between HW and AP, elevated risks for MRC RR = 2.05 (95% CI: 1.80–2.39)
Song et al. [99]	China: Beijing	January 2009–December 2012	Daily T, RH, P, V, S	High temperature: moderately hot (50th–75th), hot > 75th percentile of distribution	PM ₁₀ , NO ₂ , and SO ₂ , API	REDV (ICD10 J00–J99)	AP—main exposure, adjusted for T	Quasi-Poisson GAM; bivariate response surface model and stratification model; time series analysis	No statistical significance of API effect on REDV on moderately hot days and hot days
Staffoglia et al. [74]	Italian cities	1997–2004	Daily T, RH, P	High temperature: city-specific AT = 20–30 °C	PM ₁₀ (lag 0–1), O ₃ (lag 0)	MRC, in-hospital chronic pulmonary diseases (ICD9 490–505), aged 65+	T—main exposure, adjusted for AP; confounders: DOW, RH, P, decrease in population during summer, holidays, influenza epidemics	Case-crossover approach; random-effects meta-analysis; pooled OR of dying on a day with AT = 30 °C compared to a day with AT = 20 °C	High mortality for patients with chronic pulmonary diseases OR = 2.48 (95% CI: 1.50–4.09)
Staffoglia et al. [109]	Italian cities	1997–2004	Daily T, Td, P	High temperature: city-specific AT = 50th–75th percentiles and >75th percentile distributions	PM ₁₀ (lag 0–1)	MRC (ICD9 460–519), aged 35+	AP—main exposure, adjusted for AT(lag 0–1); confounders: DOW, RH, P, decrease in population during summer, holidays, influenza	Case-crossover approach to study the association between PM ₁₀ /AT and MRC; time-stratified approach to select control days	Higher PM ₁₀ effects on mortality during T high (lag 0–1 for PM ₁₀ and T): 10 µg/m ³ variation in PM ₁₀ : MRC, RR = 2.54% (95% CI: 1.31–3.78)
Tian et al. [100]	China: Beijing	January 2006–December 2009	Daily T, RH	High temperature: T > 15.9 °C	PM ₁₀ , NO ₂ , SO ₂	MRC (ICD10: J00–J99), gender, age (<65 years and ≥65 years)	AP—main exposure, adjusted for T; confounders: RH, long-term trend, DOW, holidays, influenza epidemics	GAM; T-stratified parametric model	Strong adverse effects of PM ₁₀ on MRC at T high: 0.45% (95% CI: 0.13–0.78); stronger effect for elderly

Table 1. Cont.

Study	Study Area	Study Period	Weather Parameter ¹	Hot Weather Description ²	Air Pollutant ³	Health Indicator ⁴	Exposure and Confounders ⁵	Statistical Methods and Study Design ⁶	Results
Vanos et al. [114]	Canada: 10 cities	1981–1999	4 hourly T, Td, V, R	Weather types: DT (hot and dry), MT (hot and humid)	CO, NO ₂ , SO ₂ , O ₃	MRC (ICD9 460–519)	AP—main exposure	GLM stratified by season and each of six distinctive synoptic weather types; time-series analysis	Combined effect of weather and AP is greatest during DT and MT air masses in summer
Wang et al. [41]	China: Nanjing	2013–2016	Daily T, RH	Warm season, AT	PM _{2.5} , PM ₁₀ , SO ₂ , NO ₂ , CO, O ₃	REDV	AT—main exposure, adjusted for AP (lag 0–7); confounders: RH, time trends	Quasi-Poisson regression, GAM; DLNM to evaluate the cumulative and delayed effects of T on health; ER = percent increase with 95% CI in REDV per 10 µg/m ³ increase in AP concentration; time-series analysis	Warm season: 10 µg/m ³ increase in PM _{2.5} and PM ₁₀ concentration on the current day of exposure (lag 0), increase in REDV ER = 0.35% (95% CI: 0.20–0.50); more serious impact than in cold season
Wang et al. [101]	Taiwan: 15 cities and counties (without Taipei)	2006–2014	Hourly T, T _{max} , T _{min} , RH, V, P	High temperature: T > 99th percentile of distribution relative to 25 °C (T with the lowest AC frequency)	PM ₁₀ , PM _{2.5}	AC for respiratory distress	T—main exposure, adjusted for AP; confounders: time trend, V, RH, DOW, holiday, pneumonia and influenza	DLNM with a quasi-Poisson function	Exposure to 99th percentile of PM _{2.5} and T control: significant for respiratory distress events; more likely to occur in low temperatures
Wang et al. [102]	China: 18 sites	2014–2017	Daily T, RH, P	High temperature: T during summer (June to August)	PM _{2.5} (lag 0–2), O ₃ , (lag 0–2), PM ₁₀ , CO, NO ₂ , SO ₂	REDV (ICD10: J00–J9), divided by gender, age-groups (<17, 18–44, 45–59, 60–74, ≥75)	T—main exposure, adjusted for AP; confounders: RH, P, DOW, long-term trend	Quasi-Poisson GAM for each study site; time series analysis	High T exposure increased REDVs risks; patients >18 more vulnerable to T high
Wang et al. [103]	China: Beijing	January 2016–December 2017	Daily T, RH	Warm season: May to October	PM ₁ , PM _{2.5} , PM ₁₀ , NO ₂ , SO ₂ , CO, O ₃	REDV (ICD10: J00–J9), divided by gender, age-groups (<25, 25–65, ≥65)	AP—main exposure, adjusted for T; confounders: RH, DOW, long-term trend	GLMs; time series analysis	Stronger associations between PM exposure and EDVs during warm season
Ye et al. [104]	Japan: Tokyo	1980–1995, July and August	T _{max} , RH	High temperature: T _{max} during summer	NO ₂ , O ₃ , SO ₂ , CO, PM ₁₀ (lag 1–4)	REDV (ICD9: acute bronchitis 466, pneumonia 486, chronic bronchitis 491, asthma 493), gender, 65+	AP; T _{max} ; confounders: annual trends, gender	GLMs; time series analysis	PM ₁₀ and NO ₂ are associated with daily REDV; greater for males than for females, except for angina and acute bronchitis

Table 1. Cont.

Study	Study Area	Study Period	Weather Parameter ¹	Hot Weather Description ²	Air Pollutant ³	Health Indicator ⁴	Exposure and Confounders ⁵	Statistical Methods and Study Design ⁶	Results
Zhang et al. [105]	China: Beijing	January 2009–December 2011	T, RH	High temperature: T > 28 °C	PM ₁₀ , PM _{2.5} (lag 0–1)	REDV (ICD10: J00–J99)	AP—main exposure, adjusted for T; confounders: calendar day, DOW, holidays, RH, influenza	Three GAMs, including a bivariate response surface model, a non-stratification parametric model, and a stratification parametric model	Increase in REDVs per 10 µg/m ³ increase in PM _{2.5} and PM ₁₀ at T high (>28 °C): 0.35% for PM _{2.5} , 0.08% for PM ₁₀ ; elderly (age ≥ 65) and women are more vulnerable to PM at high temperatures

¹ T—air temperature (°C); Td—dew point temperature (°C); RH—relative humidity (%); V—wind speed (m/s); D—wind direction (degrees); P—barometric pressure (hPa); S—hours of sunshine (h); R—precipitation (mm); CC—cloud cover (points). ² AT—apparent temperature calculated as a combination of T and Td [117,118]; DTL—daily temperature level; HW—heat wave; DT—dry tropical air mass; MT—moist tropical air mass. ³ API—air pollution index; PM₁₀—particulate matter with aerodynamic diameter <10 µm (µg/m³); PM_{2.5}—particulate matter with aerodynamic diameter <2.5 µm (µg/m³); PM₁—particulate matter with aerodynamic diameter <1 µm (µg/m³); O₃—ozone (µg/m³ or ppb); NO₂—nitrogen dioxide (µg/m³); NO_x—total oxides of nitrogen (µg/m³); SO₂—sulfur dioxide (µg/m³); SO_x—total oxides of sulfur (µg/m³); CO—carbon monoxide (µg/m³); EC—elemental carbon particulate matter less than or equal to 2.5 µm in diameter (µg/m³). ⁴ AC—ambulance call; ICD8, 9, 10—Primary International Classification of Disease, Eighth, Ninth, or Tenth Revision; COPD—chronic obstructive pulmonary disease; EDV—emergency department visits; MRC—daily mortality from respiratory causes; PEF—peak expiratory flow, used for lung function test (l/min); REDV—respiratory emergency department visits; HA—hospital admissions. ⁵ AP—air pollution; DOW—day of the week. ⁶ CI—confidence interval; DLNM—distributed lag non-linear model; DNN—deep neural network; ER—excess risk; GAM—generalized additive model; GEE—generalized estimating equations; GLM—generalized linear model; OR—odds ratio; RR—rate ratio. ⁷ Controversial results are underlined.

4. Discussion

Episodes of hot weather and poor air quality pose significant consequences for public health. Numerous researchers revealed that both ambient temperature and air pollution are associated with human health. Although many studies on the association of temperature and mortality/morbidity adjusted for concentration of air pollutants, few of them considered the potential synergistic effects of temperature and air contaminants. In this review, we looked for relevant studies and summarized the findings to get a general understanding of this problem. We focused on the main air pollutants: sulphur dioxide, oxides of nitrogen, including nitrogen dioxide; carbon monoxide; particulate matter (PM) with PM₁₀ and PM_{2.5} being the main pollutants; and ozone. Most studies suggested that there were interactive effects between temperature and air pollution on human health, and the results varied among different geographic regions. The health consequences varied from premature death to decreases in lung function, new onset of diseases, exacerbation of chronic respiratory diseases, and lung cancer.

Much is known about the relation between high ambient temperature, air pollution, all-cause and cardiovascular mortality [122], independent associations of hot weather, polluted air environment, and respiratory health [1,67,123], but the combined effect on disease-specific events, such as respiratory, is less clear. The current review shows the role of pollutants on respiratory health impacts may vary with hot weather conditions. The modifying effect of the high temperature on the air pollution–respiratory health indicators relationship was scarcely reported in recent years, although it is well known that high temperatures have an important action on the transport and the dispersion of pollutants in the air and vice versa [78], causing the plausible effect on respiration. Increased mortality and morbidity are associated with heat wave episodes; an interesting aspect is the role of air pollution in this relationship [1,67].

In the reviewed studies on effects of air pollution and temperature on human health, temperature was generally corrected to efficiently analyze the health impacts of ambient particles; otherwise, air pollutants were also adjusted when investigating the influence of temperature on mortality. Each identified a significantly enhanced mortality and morbidity effect for increasing values of both air contaminants and high temperature. However, a relatively small number of papers were found to confirm our hypothesis, which considerably limits the scientific significance of the current paper and is a great potential for further research. The large differences in the results obtained do not allow us to collect them in a meta-analysis and provide reliable, mathematically confirmed evidence with statistical weights about the jeopardized effect of exposure to both high temperature and air pollution on respiratory outcomes. Another problem identified is that the interaction between the temperature and the pollution continues to be vague, as some studies showed non-statistically significant coefficients for the interaction between air pollution, high temperature, and respiratory outcomes [57,86,99,106].

Many additional factors could shed more light on the confounding effect of interactions in the relationship of hot weather–air pollution–respiratory health and ways to mitigate these implications, which are discussed below.

4.1. Wildfires

A very significant consideration is the occurrence of wildfires caused by hot weather, which are associated with dangerous levels of pollutants and therefore affect respiratory health [9,63,108,124,125]. A vivid manifestation is the several large wildfires of unprecedented scale and duration that occurred recently, including wildfires in European Russia in 2010 [108,126], Australia in 2019 to 2020, the Amazon rainforest in Brazil in 2019 and 2020, the western United States in 2018 and 2020, and British Columbia, Canada in 2017 and 2018, with the huge impact on the population in terms of adverse effects on respiratory health [127].

The mechanisms that explain the respiratory effects of wildfires are mainly related to the air pollution associated with these events. Wildfire smoke is a complex mixture of

thousands of compounds, primarily PM_{2.5}, carbon dioxide, hydrocarbons, and nitrogen oxides, which contribute to increase air pollution locally and regionally. In multiple studies, air pollution from wildfire smoke was associated with more asthma exacerbations, ED visits, and hospitalizations for bronchitis, dyspnea, and COPD symptoms. Wood smoke particles can activate systemic and pulmonary inflammation, even in healthy human subjects [1,125,128–136].

Extreme events such as wildfires and prolonged heat waves and drought are causing increases in pollutants such as particulate matter and ground level ozone, often to dangerous levels. Due to high ambient temperature during wildfire, there is also a potential for interaction as evidenced by the combination of HW and wildfire conditions [1,108,126,137]. This mutual influence can be explained at the point of emissions by changes in smoke components caused by temperature, sunlight, water vapor, and interaction with other pollutants [138].

Next, a few more important points should be discussed. An *in vivo* experiment showed that the responses to wood smoke consist of higher inflammatory and cytotoxic reactions than those caused by urban particulate matter [139], suggesting that wildfire contamination is more dangerous than urban pollution. Another problem is that, although forest fires can only occur in certain regions, their smoke plumes can spread over long distances [130]. More research is needed to assess long-term health effects from wildfires [129]. The understanding of such factors is vital to ensuring that health care services are prepared for these events.

4.2. Urban Heat Island Effect

The problem of outdoor pollution is not new, but rapid urbanization is making the problem of air pollution more visible and its load on human health more noticeable [12,140,141]. Extreme heat events at urban centers in combination with air pollution pose a serious risk to human health. Studies showed the coupled interaction of urban heat island and air pollution increases hospital respiratory admissions and hospitalizations in the warm center of urban areas [141–143]. The prognostic doubling of urban population within the next two decades and the disproportionate growth of megacities make it critical to explore the synergism between urban heat and pollution [143].

Urban populations are likely to be particularly at risk, but the role of urban characteristics in changing the direct health effects of temperature is still debated due to a variety of modulating causes. For example, the impact of heat on mortality is higher in cities characterized by high population density, higher levels air pollution, fewer green spaces, and lower availability of health services [144].

4.3. Possible Biological Mechanisms

Several different explanations are proposed for the synergistic effects between ambient pollution and high temperature on human health [1,7,93,145]. Since the respiratory surface is a primary route by which air pollutants enter the body, prolonged heat exposure may activate three key mechanisms of thermoregulatory responses, which include: secretion of sweat glands, vasodilatation, and increase in ventilation rate and lung volumes, which in turn can directly or indirectly affect toxic substances entering the body and raise the total intake of airborne pollutants [1,7,145–147]. Next, high ambient temperature increases skin permeability, providing an easier way for air pollutants to be absorbed by the skin surface [145,148].

Pathophysiological mechanisms are discussed in detail in [55]. First, a combination of heat load and gaseous polluted environment leads to an inflammation of the bronchial mucosa and lowers the bronchoconstriction threshold; the accompanying fluid loss additionally contributes to subsequent changes in perfusion and ventilation, causing acute and chronic injury to lung tissue by gaseous particles in polluted air, such as greenhouse gases, nitrogen dioxide, sulfur dioxide, and ozone [149]. Secondly, fine PM of different size “destroys the integrity of endothelial cells via the signal transduction pathways that depend

on reactive oxygen derivatives and p38-activated protein kinase and is involved in the pathogenesis of cardiopulmonary disorders”, which is explained in detail in [47,50]. Moreover, the synergistic interaction of heat and concentrations of noxious/toxic substances in the air leads to exacerbations, especially of asthma and COPD [150,151]. Experiments on animals show the importance of hot air as a pathological substrate of bronchoconstriction and penetration of tissue-toxic elements [152,153]. Additionally, some evidence was found to demonstrate the increase in airway resistance during hot and humid days more rapidly than in cold air, triggering asthma symptoms, most likely by stimulating airway C-fiber nerves [130,154,155].

The delayed associations between exposure and outcome—or the “lagged” effect of air pollution, thermal exposure, or both—of different lengths (mostly 0–2 days) on respiratory mortality and morbidity were demonstrated in many studies [68,78,89,97,111,115]. It can be supposed that respiratory diseases are slower in their development and are therefore more lagged [29].

However, excess mortality may be followed by fewer deaths than expected during the next period of time, which is called a harvesting effect or mortality displacement [30,53,55,75,156–160]. A harvesting effect is found to partly or even fully balance the observed excess health outcomes due to heat. One more consideration can be shown for a sharp decrease in deaths to hospital admissions, with some suggestion of displacement or harvesting for respiratory admissions [159].

4.4. Virus and Bacteria Infections and Epidemics

Evidence supports a clear effect of air pollution on respiratory infections interacting to adversely affect the respiratory system, contributing to the recent coronavirus disease 2019 (COVID-19) pandemic. Most research indicated that chronic exposure to air pollutants leads to more severe and lethal forms of COVID-19, delaying and complicating the recovery period after the disease [161,162]. Areas with frequently high levels of air pollution—exceeding safe levels of ozone or particulate matter—had higher numbers of COVID-19 related infected individuals and deaths [163].

The biochemical and the physiological mechanisms behind this effect include a number of functional changes involving endothelial dysfunction, endothelial activation, and injury [44]. These local changes in the lung promote pulmonary responses, affecting airway function and resistance to viruses and bacteria, increasing the risk of infection, for example, upper respiratory tract infections, bronchitis, and pneumonia [44]. Moreover, high concentrations of nitrogen dioxide and particulate air pollutant induce serious damages to the immune system of people, weakening it to cope with infectious diseases of viral agents [164]. Knowing the associations between polluted environment and human respiratory infections in different temperature regimes may decrease turbulence and spread of a new COVID-19 pandemic and any other in the future.

4.5. Indoor Pollution

Indoor pollution obtained from a variety of sources such as ventilation and building materials, use of biomass fuels for heating and cooking, active indoor smoking, pesticides, incense, and biological pollutants such as dust, dandruff, furniture, and mold might be combined with high indoor temperatures [9,21,28,165,166]. In addition to ambient contaminants, exposure to poor indoor air quality is linked to many acute and chronic respiratory health outcomes such as general respiratory complaints, exacerbation of asthma symptoms and COPD, diminished lung function, lung cancer, and mortality, with women and young children being disproportionately affected due to time spent indoors [21,24,28,36,45,167,168].

4.6. Vulnerable or Susceptible Population Groups

Pre-existing respiratory diseases are important vulnerabilities to both thermal effect and air contamination. Since exposure factors are specific to each climate hazard and can vary by populations and over time, more research is needed, particularly for younger age cohorts and children as well as for other parts of the population, such as socioeconomic

groups with low income [46]. Vulnerable population subgroups were identified in regard to the effects of air pollution and/or heat, including: elderly; care homes' residents; young children; patients with cardio-respiratory disease asthma, renal diseases, and diabetes; people with obesity as well as those who takes certain drug therapy; and those who live in densely populated urban neighborhoods [1,5,40,46,75,95,107,169–171]. For example, some evidence was shown in England and Wales that ozone effects were worse on hot days, particularly for those aged <75 [107]. In addition, there were clear differences by sex: specifically, females showed higher mortality risk, and males showed higher hospitalization risk [172].

The elderly age cohort shows an increased sensitivity to exposure of both atmospheric pollution and thermal impact, which is probably explained by an excessive manifestation of concomitant diseases (diabetes, various acute heart disease such as congestive heart failure or myocardial infarction, etc.), by reduced respiratory and antioxidant capabilities, and by rise of inflammatory phenomena, even in healthy people [2,144,173]. The interacting effect between air contaminants and extremely high temperature is biologically plausible for the elderly, whose ability to thermo-regulate body temperatures is reduced, and sweating thresholds are generally elevated in comparison with younger people [95]. Inflammatory and immunological reactions in children are significantly contrasted compared to other age groups, which may possibly determine a higher degree of influence on children [120,174].

4.7. Confounder Effect of Multiple Environmental Stressors

Exposure to poor air quality and thermal load both already affect human health independently, but their combined occurrence poses an intensified threat to human life, especially as synergistic effects lead to a risk beyond the sum of their individual effects [4]. The idea of multiple environmental stressors supports the assumption that it is a scientific apparatus for organizing and evaluating relevant scientific data to identify and evaluate the cumulative effects of various environmental factors that negatively affect human health [175,176]. For instance, the Air Pollution Index assesses the combined effects of main air pollutants to determine whether their impacts on respiratory morbidity and mortality are affected by temperature, age, gender, pre-existing disease, as well as other confounders and effect modifiers, such as socioeconomic conditions, urbanicity, and central air conditioning [99]. Multiple air pollutants may be most responsible for increased impact on cardiopulmonary health, which would need to be taken into account in the future research [41]. Heterogeneity of the results of the joint effect of high temperature and air contamination on respiratory health may reflect the characteristics of the study sites, such as weather patterns, air pollution levels and components of pollution mixture, use of air-conditioning or heating systems, sensitivity of local residents to air pollution (e.g., gender, age, and smoking rate), and possibly other socioeconomic characteristics [79]. An important addition was proposed by Abed Al Ahad with coauthors [177] that the analysis of interaction between air pollution and weather stressors beyond specific limits and their effect on respiratory health should be supplemented by study of the major socio-demographics modifiers, such as ethnicity, occupational/educational/marital status, and others [177].

Other challenges may be specific parameters of the study area. For example, the impact on the population of outdoor temperature and air pollution may vary depending on social-economic status, use of air conditioners, local habits associated with opening windows, traditions in the house design, etc. Results from studies on the interaction of temperature and air pollution suggest that this topic cannot be ignored when detecting the effects of temperature or air pollution on respiratory causes, since the real scale of the relations may be understated. Overall, results differ depending on the pollutant under consideration and the study area, assuming that the original regularities are led by local conditions.

Many studies showed that, to explore the respiratory system status under environmental exposure, not only temperature is used but also air humidity [178,179] as well as special

indices such as apparent temperature, which determines the combined effect of weather parameters together with air pollutants [6,114,117,118]. It should be noted that, although absolute humidity correlates with air temperature, it can itself significantly change the impact of air pollutants on health outcomes [180].

Some results for mortality and hospital respiratory admissions showed the synergistic effects of long-term exposures to air pollution and temperature were associated with larger effects compared to short-term exposures; one possible explanation is the suggested evidence provided in studies on the effects of heat waves and air pollution on mortality that respiratory diseases are slower in their development and are therefore more lagged than, for example, cardiovascular diseases, which can initiate an acute body response [29,59,181].

Although there was evidence of a synergetic effect of air pollution and high temperatures during HW episodes, a slight decrease in thermal effects was found when models included O₃ and PM₁₀ [5,110], which should be analyzed in future research.

4.8. Climate Change

Almost all climate events will worsen under climate change, as will the related disease burden. Climate and climate change are modifying air pollution effects on respiratory health in several ways: climate variations are predicted to increase frequency and intensity of heatwaves and wildfires, cause longer fire and pollen seasons, influence start, duration, and intensity of the pollen season, increase aero-allergenic plant pollen production, and raise long-term transport of air pollutants and allergens [1,9,84,121,182,183].

To better assess the potential impact of current climate change scenarios on human health, it is mandatory to comprehend not only the separate effects of temperature and other weather characteristics (adjusted for confounders, including air contamination) but also to understand any combined effect between meteorology and air pollution. It is proper to assume that the synergistic interactions between high ambient temperature and air pollution will become more important under extreme conditions that could occur in the future due to larger climate volatility [5,37,184]. Global climate models predict not only warmer temperatures on average but more frequent extreme weather events [6,185], which increase the threat of weather-related health outcomes [186].

Mitigation and age-specific adaptation strategies might greatly reduce the temperature-related mortality burden in the future climate change realm. If people cannot adapt to future climate change, heatwave-related excess mortality is expected to increase the most in tropical and subtropical countries/regions, while European countries and the United States will have smaller increases [37]. At the same time, considering future impacts on respiratory diseases, progressive population aging, growing spread of chronic diseases, and socioeconomic transformations that are currently taking place in a number of countries, especially in Europe and Northern America, will probably raise the portion of the population at risk [186,187]. This is crucial for individuals with impaired adaptation to weather variability, such as the elderly.

Another consideration is the increase in both urbanized and wildfire areas due to climate change. The risk of wildfire frequency is increasing in most areas of the world as climate change worsens [119], with projections of the fires to raise excess mortality and morbidity as well as mental health effects from burns and wildfire smoke [124,127,129]. Therefore, there is an urgent need to further understand the health effects and the public awareness of wildfires [108,183]. Due to climate change and other factors, such as the relatively rapid growth of urban population up to 5.2 billion in 2050 [188], the nature of pollution is changing in several urbanized areas worldwide, which has a significant impact on respiratory health, both separately and synergistically with weather state [1,188].

Another key research need for future studies is to compare the health burdens of future air quality under a changing climate with alterations in other risk factors. Warmer temperatures from climate change and increased amounts of carbon dioxide can cause increased growth of aero-allergenic plant pollen production, which leads to extension of pollen seasons with warmer springs and delays in first fall frost, therefore leading to increased production

of pollen [9,182,189]. Climate change may impact the incidence of bacterial, viral, fungal, and tick-borne respiratory infections in terms of incidence, total duration, and severity of these infectious diseases, increasing their overall incidence and geographical spread [9,17]. The only way to solve this problem is to inform the public using the multisectoral approach of the scientific community. National and international organizations must deal with the emergence of this threat and offer sustainable, viable decisions.

4.9. Prevention Policy

At last, decades-long periods of both high air temperature and air pollution, including wildfires, are becoming an important issue of global concern, exacerbating health outcomes and corresponding health-care expenditures. A significant impact of air pollution, including episodes of wildfires, on health-care costs related to respiratory diseases was identified [42,108,159,183,190,191]. Addressing these challenges requires the interdisciplinary cooperation of epidemiologists, climate scientists, respiratory and allergy physicians, policy makers, and public health professionals to jointly guide the world through the climate crisis, making the environment sustainable for future generations. Furthermore, the governments should strengthen environmental management, pay attention to the heterogeneity of the healthcare expenditure burden affected by both high temperatures and environmental pollution, ameliorate the medical insurance system, and improve the health of residents [65,66,137,159,192,193]. Moreover, policy makers should enhance the emission control of air pollution in high temperature days, especially to target pollutants produced by motor vehicles [78,91], and strengthen health education propaganda [89].

Taking into account the interrelationships between current and future climates and pollution challenges, adequate mitigation policy and public health actions are needed to face the two hazardous exposures. Considering the predicted increase in heat waves and stagnation events, it is time to enclose air pollution within public health heat prevention plans.

The prospect of global warming requires a more accurate assessment of how a hotter environment can affect the human response to toxic chemicals [192,193]. In climate change scenarios, the increase in extreme weather events and some air contaminants, particularly ozone, is likely to further exacerbate chronic respiratory disorders. Public health measures should aim to prevent this additional burden of illness in summer periods and especially during HW [30,65,193,194]. Identifying population groups that are more susceptible to weather variability is of paramount importance for the development of public health policies that protect them [5,195,196]. Recognizing the sources and the concentrations of air pollutants helps to establish the new regulations for control measures. This subsequently leads to the diminishing mortality of infants and children under the age of five [25], the elderly [196,197], and the low-income population [194,195]. Short-term public health activities to decrease heat-related morbidity should target sensitive population groups to assure access to air conditioning, evaporative coolers, fans, adequate fluid intake, etc. [194,195,197]. On the other hand, on hot days, emissions of pollutants may be further increased by behavioral changes when inhabitants of cities may choose to use their possibly air-conditioned car more often [79].

Long-term prevention efforts should aim to improve the general health condition of people at risk, implementing special prevention plans for cities [125,137,198,199] and conceptualizing special green and blue infrastructure in the built environment [199–201], with the paramount need to establish joint heat and air pollution warning systems [59,66,186,193,202]. Sustainable urban planning and smart city design developing integrated sustainability performance for innovative energy systems in smart cities could significantly reduce both UHIs and air pollution [203,204], enhance human thermal comfort, and decrease heat- and pollution-related mortality, thus saving lives [200].

It is also important to better comprehend the influence of climate characteristics on the direction and the spatio-temporal spread of wild fires [205]. The use of remote sensors

would help identify the components of pollution during fires and assess the temperature rise associated with fires [125,131,136].

5. Conclusions

Air-pollution and hot weather exposure beyond certain thresholds have serious effects on respiratory health, with the elderly and young children being the most vulnerable groups. However, there is a lack of information on broader perspectives, including the role of some exposure modifiers and the interaction between air pollution and weather characteristics. The purpose of this paper is to provide a holistic overview and descriptive synopsis of the literature on the association of air pollution and weather with mortality and hospitalization and to identify gaps in scientific knowledge that need further research. The final review included 40 articles, from which 24 involved mortality, 15 incorporated respiratory emergency transportations and hospital admissions, and 1 reviewed lung cancer incidence. Air-pollution was shown to act consistently as a risk factor for respiratory mortality and morbidity. Hot temperature was a risk factor for a wide range of respiratory disease. The role of effect modification in the included studies was investigated in terms of gender and age. These findings are important for public health, as the high spread of chronic diseases such as COPD is expected to increase, especially in developed countries as a result of an aging population. Effect alteration of important socio-demographics and the interaction between air pollution and weather is often missed in the literature. Climate and climate change are shifting, combining influences of hot weather and air pollution on respiratory health in several ways. Climate variations are predicted to increase frequency and intensity of heat waves and wildfires, raise long-range transport of air pollutants and allergens, and influence start, duration, and intensity of the pollen season. Moreover, with regard to climate changes, it is generally accepted that global warming increases the health effects of outdoor air pollution, resulting in more heat waves, during which levels of air pollutants raise and high temperatures and air contamination act in synergy, causing more serious health impacts than those estimated from heat or pollution alone.

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