

Review

New Developments in Climate Change, Air Pollution, Pollen Allergy, and Interaction with SARS-CoV-2

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Abstract: In recent years, the environmental impacts of climate change have become increasingly evident. Extreme meteorological events are influenced by climate change, which also alter the magnitude and pattern of precipitations and winds. Climate change can have a particularly negative impact on respiratory health, which can lead to the emergence of asthma and allergic respiratory illnesses. Pollen is one of the main components of the atmospheric bioaerosol and is able to induce allergic symptoms in certain subjects. Climate change affects the onset, length, and severity of the pollen season, with effects on pollen allergy. Higher levels of carbon dioxide (CO₂) can lead to enhanced photosynthesis and a higher pollen production in plants. Pollen grains can also interact with air pollutants and be affected by thunderstorms and other extreme events, exacerbating the insurgence of respiratory diseases such as allergic rhinitis and asthma. The consequences of climate change might also favor the spreading of pandemics, such as the COVID-19 one.

Keywords: respiratory allergy; climate change and allergy; biodiversity and allergy; pollen allergy; thunderstorm asthma

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1. Introduction

Climate change is a physic meteorological fact and, among its effects, is an impact on human health. Heat waves, an increase in precipitation, floods, droughts, hurricanes, thunderstorms, and sandstorms are just some of the environmental consequences of climate change. Its other effects include impacts on respiratory health and allergies due to pollen exposure and modifications in its chemical composition, concentration, and allergenic potential, also causing the growth of new, allergenic plant species.

In addition to global health, allergies are among the diseases most influenced by climate change [1]. Evidence is accumulating that, besides the climate, climate change affects food supplies, water, and soil and air quality. Several experimental and epidemiological studies have tackled the topic of how respiratory diseases, such as asthma and allergy, are

linked to air pollutants, meteorology, aeroallergens, and other environmental factors [2–6]. Around the world, climate change has negative effects on health, increasing the cases of respiratory diseases, acute cardio-respiratory cases, and allergies due to pollen and fungal spores.

Respiratory diseases, such as bronchial asthma and allergies, have become more prevalent over the past few decades in most industrialized countries due to westernized lifestyles and urbanization, with its high levels of automobile pollution [3–5]. Biological particles and chemical elements in the air can be affected by climate and meteorological factors [3–5]. Increases in temperature, humidity, and extreme events such as thunderstorms can have effects on different biopollutants, worsening their potentially dangerous health effects.

2. Climate Change, Why and How?

Nowadays, millions of tons of carbon dioxide (CO₂), one of the gases that contributes the most to the greenhouse effect, are produced each year by burning thousands of hectares of forests worldwide and as a result of several human activities [7–9].

Higher CO₂ concentrations in the atmosphere modify the growth and phenology of plants in many ways, for example by enhancing photosynthesis or increasing their pollen production and the duration of pollination periods [10,11].

Regional differences in the trend of climate change are caused by factors related to geography, meteorology, land use, and energy output. This results in different degrees of increase in cases of allergic diseases, which can be also influenced by the application of mitigation measures regarding the limitation of greenhouse gas emissions [2]. The main greenhouse gases are nitrous oxide (NO₂), methane (CH₄), and fluorinated gases, but especially CO₂, produced by the combustion of fossil fuels [12].

Since the beginning of the industrial revolution, the CO₂ concentration in the atmosphere has notably increased. Starting from values of 280 parts per million (ppm) in 1870, carbon dioxide levels reached a peak of 421 ppm in May 2022 at the National Oceanic and Atmospheric Administration's (NOAA) atmospheric observatory at Mauna Loa. With an average of 420.99 ppm, the increase in CO₂ levels was 1.8 ppm over 2021 [13].

The need for a reduction in anthropogenic CO₂ emissions has been clearly stated by the Intergovernmental Panel on Climate Change (IPCC) [6] and could have several benefits for human health. CO₂ emissions not only cause an increase in global temperatures in the short term as the gas is released into the atmosphere, but they also determine a long-term effect, as this temperature rise can still carry on when the concentration of carbon dioxide in the atmosphere stabilizes, continuing for a period of a century. This phenomenon can lead to higher concentration of other harmful gases, such as ground level ozone.

3. Pollen Allergy

Pollen allergy is an important public health problem considering the upward trend of pollinosis cases in this historical period over the world. In Europe, up to 35% of young adults are allergic to grass pollen, one of the most dispersed pollen types in the world [14]. Pollen allergy is a relevant issue also because of its related costs, with a subsequent need for medical medications and consultations [3]. It can also have relevant impacts on quality of life and cause difficulties in the workplace for allergic subjects.

In nature, pollen grains develop in specialized structures of the plant, namely the microsporangia of the male cones in gymnosperms and in the anthers of the flowers in angiosperms. When mature pollen grains are discharged by the mature anthers, usually on warmer and drier days, they also tend to dehydrate. Then, if they come into contact with a wet surface, the pollen grains are modified by the absorption of water, changing both in their shape and metabolic activities. This is the case when pollen enters the conjunctival, nasal, or oral mucosa, an occurrence that causes osmotic shock in grains. When this happens, the pollen gets hydrated, swells, and can quickly release the allergens contained in the cytoplasm, or discharges its water soluble content, e.g., its allergenic proteins,

through micropores. The expulsion of these substances subsequently causes allergic symptoms in the affected mucosae. As described by Taylor et al. [15,16], roughly 65% of pollen grains developed in pollen tubes of up to 300 μm long before rupturing, and they released cytoplasmic material under conditions of extreme humidity.

An aerosol containing allergenic material is formed by the released particles, such as the pollen cytoplasm of the broken up grain. Exposure to allergens, the inflammation of the respiratory tract (both lower and upper), and the manifestation of clinical symptoms are all linked in a number of ways. There are several factors that can influence the severity of allergic symptoms in predisposed individuals. One of these factors is certainly the quantity of the inhaled pollen, but the type of allergenic pollen is also important. Pollen grains can easily enter the upper respiratory system, but it is very hard for them to advance as far as the bronchi, considering that an integer pollen grain has a diameter greater than 10 μm [3]. However, among allergic subjects, symptoms related to bronchial asthma are not rare.

Despite the widespread belief that rain clears the air of pollen, it has been shown that, when pollen comes into contact with water, its allergens can be liberated from the grain in just a matter of seconds [17–19]. The effect of extreme weather events such as heavy rains and thunderstorms could cause the release of very small particles from pollen grains, which are known as paucimicronic particles. These paucimicronic particles are represented by granules with a diameter lower than 5 μm , deriving from the tissues of the anthers, which can carry an important amount of allergens with negative effects on allergic and asthmatic subjects [3].

4. Impact of Climate Change on Allergenic Plants

Modifications in pollen allergens are affected by climate change as a result of the rising CO_2 levels in the atmosphere. In fact, CO_2 can cause plants to grow more quickly and vigorously, as well as increase their pollen allergen potency and flowering intensity and duration. Climate change also increases their exposure and sensitivity to subtropical grasses. Plants that bloom at the beginning of spring and those that react favorably to a warmer climate tend to exhibit an earlier onset of the pollen season and its peak. Similarly, the blooming of urban plants tends to happen 2–4 days before that of plants living in rural areas. In addition, an increase in temperature linked to climate change can worsen its effects on pollen, both alone or in combination with other factors such as CO_2 levels. For this purpose, Ziska et al. [20] recorded that, during the day, the mean CO_2 concentration went up by 21% due to urbanization, while the daytime maximum temperatures increased by 1.6 $^\circ\text{C}$ in more urbanized areas compared to rural areas and the minimum temperatures differed by 3.3 $^\circ\text{C}$. The modifications observed in urban environments were coherent with most of the short-term (~50 year) predictions regarding air temperature and CO_2 concentrations. Rising temperatures and higher CO_2 concentrations have been shown to positively affect maximum plant height and productivity, to values of up to 60% in suburban sites and 115% in urban areas, relative to rural sites. Ragweed pollen allergenicity has been demonstrated to be directly related to CO_2 increases, with a consequently higher prevalence and/or severity of allergic disease cases [10]. Ragweed pollen production can also be increased by 61% as a consequence of the doubling of the CO_2 concentration in the atmosphere and its allergenicity can get much higher along heavily anthropized areas such as high traffic roads, as observed by Wayne et al. [21].

The geographical distribution of plant species can change as a consequence of climate change too. Following modifications in temperatures, rainfall, and other factors, the distribution range of many plants could shift toward the poles, i.e., northward in the Boreal hemisphere and southward in the Austral hemisphere. Disseminated species, such as grasses, can also be influenced by changes in land use, and, in general, human activities [22].

According to their carbon fixation metabolism, plants can be classified either as C3 plants, which include Pooideae (temperate grasses), or C4 plants, which include Chloridoideae and Panicoideae (subtropical grasses). C3 grasses tend to increase in winter and flower in spring, while C4 ones tend to grow and bloom in summer and at the start of autumn, at least in seasonal climates. Peaks of airborne grass pollen have been recorded at the end of summer, corresponding to the blooming of subtropical grasses [23,24]. However, in the temperate areas of the northern hemisphere, species from the subfamily Poaceae are the main culprits behind grass pollen allergy [25,26].

The above mentioned grass subfamilies are all represented in southern hemisphere countries such as Argentina, Australia, Brazil, and Uruguay, with Pooideae being the most abundant. Studies have revealed geographic differences in sensitivities to subtropical plant pollen, mainly in the southern hemisphere [27–31].

Nowadays, agriculture is having a positive impact on subtropical grass expansion in addition to climate change, which favors the growth of plant populations and their expansion to previously uncommon locations. For example, Australia and Argentina are among the countries with increasing areas being dedicated to agriculture, a factor that may certainly have consequences regarding allergies [32–34].

A bigger incidence of pollen-related respiratory allergies has been recorded in individuals living in urban areas, in contrast with a lower incidence in rural areas, a trend that can be linked to phenomena such as high levels of vehicle emissions, urbanization, and having a western lifestyle [3,35]. Biodiversity loss, global warming, pollution, and the microbiome are all interconnected and this increase in allergy in urban environments can be also due to a reduction in the microbiome, mainly during the first years of life [36,37].

Allergenic particles, such as airborne pollen grains, can be altered in the atmosphere and release allergens, resulting in allergen-containing aerosols in the ambient air, due to the impact of pollutants present in the environment, which, in addition to their direct effects on human health (e.g., as irritants of skin and mucosal membranes), can also have an indirect effect. In addition to serving as a carrier of allergens, it has been shown that pollen also releases highly active lipid mediators (pollen-associated lipid mediators), which have pro-inflammatory and immunomodulating effects in allergic illnesses [1]. Between them, linolenic-acid-derived hydroxy fatty acid derivatives, namely 13-HODE and 13-HOTE, are able to induce the activation and migration of polymorphonuclear granulocytes [38,39].

5. Effect of Climate Change on Chemical Air Pollution

Severe episodes of asthma exacerbation have been linked to the consequences of climate change and the presence of high levels of chemical pollutants in the air.

An important chemical pollutant in the atmosphere is ozone, which can have negative effects on the human respiratory system, resulting in inflammation, decreased lung function, systemic oxidative stress, and an increased responsiveness to injury [8,40,41].

In particular, Gent et al. [8] analyzed the respiratory symptoms caused by the conjunct effects of ozone concentrations below the standard values of the U.S. Environmental Protection Agency and fine particulate matter (PM 2.5) on children in need of crisis medications. The results of the study showed a significant association between ozone levels and the insurgence of respiratory symptoms needing rescue medications in asthmatic children. An increase of 50 parts per billion of ozone for one hour has been associated with an insurgence of chest tightness (47%) and wheezing (35%), while higher ozone levels were related to increased dyspnea and a requirement for emergency medication.

In hypersensitive individuals, asthma can be induced by allergens carried by pollen or other plant particles that enter the peripheral airways by air inhalation. The permeability of airways can be increased by factors such as ozone, particulate matter (PM), nitrogen dioxide, sulfur dioxide, and diesel exhaust particles [42–46]. This increased permeability can cause the enhanced interaction of the immune system cells with allergens due to the penetration of mucosal membranes. Consequently, air pollutants play a determinant role in the inflammation of airways in susceptible individuals.

Air pollutants are able to stick to the external walls of pollen grains and paucimicronic particles derived from plants, increasing their allergenicity and affecting, in various ways, their morphology [47]. In addition, pollutants adhering to the walls of pollen grains can surpass the mucosal barrier as a consequence of the inflammation and increased permeability of the airways, causing enhanced responses to pollinosis in atopic patients [42–44].

The increased effect of aeroallergens on sensitive individuals and the augmented severity of respiratory symptoms is clearly shown in the literature [48–50].

6. Respiratory Allergies, Urban Environment, and Climate Change

The frequency and severity of air pollution events can be affected by the impact of climate change, which can have an effect on variations in wind speed and direction, the timing and quantity of rains, and temperature increases. Manmade emissions can also change as a response to climate change, with consequences such as an increase in energy demand for home heating or air conditioning. Levels of ozone and other air pollutants can be increased by the urban heat island effect, which also has an indirect effect on the natural phenomena that cause the emission of particles, such as forest fires, soil erosion, and vegetation breakdown [51,52]. The reaction between nitrogen oxides and volatile organic compounds is a source of tropospheric ozone (O_3) in the presence of bright sunlight. Observations in outdoor smog chambers and evaluations in ambient air have demonstrated a relationship between temperature and tropospheric ozone levels [53,54]. In comparison to pollen exposed to lower amounts of ozone, birch pollen exposed to high levels of ozone causes larger wheals and erythema in skin prick tests, suggesting a possible role of ozone in the insurgence of allergic reactions [55].

The intensity of forest fires, which can cause respiratory ailments, can also rise in response to changes in temperature and rainfall. In addition to extending the growth time of ozone concentrations, rising temperatures can exacerbate peak ozone levels.

Pollutants and pollen grains can travel farther when wind patterns are altered, making this transport mechanism just as significant as the local one.

7. Thunderstorm Asthma

Thunderstorms occurring during the pollen season, especially in late spring and summer, can induce severe asthma outbreaks in allergic patients living in a circumscribed area, a phenomenon known as “thunderstorm asthma”. Asthma exacerbations caused by thunderstorms usually begin with a sudden increase in visits of asthmatic patients to general practitioners and emergency services in hospitals. In these cases, asthmatic symptoms can manifest even in patients that normally only suffer from seasonal rhinitis. This phenomenon is strongly associated with the altitude of the dispersal of allergenic pollen grains such as grasses. This enhanced allergenicity during thunderstorms could be caused by the hydration of pollen grains caused by rainwater, with a release of inhalable allergenic particles. In the first half an hour of a thunderstorm, individuals who suffer from pollen allergies may breathe in large amounts of the allergens that are dispersed in the air [56]. Thunderstorms have been linked to asthma outbreaks and exacerbations in a number of places, mainly in European cities (Naples in Italy and London and Birmingham in the United Kingdom) and Australia (Wagga Wagga and Melbourne) [56–58]. At least some of the mentioned cases have been related specifically to grass pollen, suggesting that the main sources of this pollen could be located outside cities, in nearby pastures [58]. After being transported high in the atmosphere due to the movement of hot air, the pollen gets concentrated by converging turbulences and ruptures with increasing humidity, later affecting the people living in the area with the release of small, allergenic particles, which are brought down by the storm.

8. Pollen Allergy and Occupational Health

Numerous categories of workers may be exposed to several biological, chemical, and physical agents that may induce and/or exacerbate allergic diseases in sensitized individuals [59–67].

In addition to this, synergic and/or additive effects due to environmental exposure to allergens, chemical pollutants, and individual sensitization may also play critical roles. Climate change adds complexity to allergies [68–72]. Occupational exposure both in outdoor and indoor workplaces should be taken into account while dealing with these diseases [73–76].

Previous studies conducted on indoor workplaces have evidenced the importance of the presence and actions of occupants as triggers for the increase in the concentrations of pollen and other bioaerosol particles, highlighting the role of working days and working-hours as co-factors of the increases in and diffusion of pollen [77–79].

A more integrated analysis should be conducted on occupational environments, considering both pollen exposure and its health effects on workers, deepening studies on the sources of exposure and also distinguishing between urban, semi-urban, and rural workplaces [78,80–83].

These studies on occupational settings allow for higher control of the environmental exposure and health conditions of the individuals exposed. The latter information can be obtained by the specific clinical–anamnestic questionnaire and the use of innovative methodologies enabling the evaluation of multiple sensitizations against numerous allergens derived by plants, animals, and food [84]. Moreover, specific studies may be useful for deepening the interactions between pollen and chemical pollutants [85], promoting synergic studies and including pollen and other allergens, as reported by the air quality guidelines recently published by WHO [86].

Future research on climate change, pollen, air pollution, extreme events, and allergy should include occupational health and workers' roles in numerous indoor and outdoor workplaces.

Strategies for control and prevention could be “tested” in occupational settings involving all “actors” of prevention, in a collaborative perspective between public health, environmental health, and occupational health.

9. Climate Change and Its Impact on Infectious Respiratory Disease (SARS-CoV-2)

An extensive body of literature shows climate change's impact on the incidence and severity of infectious respiratory diseases through modifications in a host's immune response, exposure to fungal and mycobacterial species, vector vitality, and the spread of novel viruses. Recently, studies on climate change have considered its influence on the outbreak of pandemics of novel pathogenic species, such as COVID-19, caused by the emergence of the new coronavirus SARS-CoV-2 [87,88].

Dramatic temperature shifts can lead to an increased exposure to environments where vector-borne pathogens thrive. Rises in temperatures are able to increase these vectors' vitality and therefore the risk of disease spread. This has been shown, for example, in rodents that are reservoirs for Hantaviruses, a virus known for regional outbreaks manifesting as pneumonia and diffuse systemic disease [89,90].

Furthermore, desertification, the expansion of drylands, and dust storms have contributed to the release and diffusion of fungal dust-borne spores commonly found on soil that can cause respiratory infections, as observed in the southwestern USA with *Coccidiomycosis* [91,92].

Another example is the geographic spread of *Cryptococcus gattii*, a causal agent of Cryptococcosis, a disease that most commonly affects immunocompromised human hosts. This respiratory disease, originally only present in subtropical areas, is expanding in the Mediterranean regions of Europe and Pacific northwest regions of the USA, and it

has been hypothesized that trees and livestock trading, flocks of migratory birds, anomalous atmospheric events (e.g., tsunamis), and human interactions have substantially contributed to the diffusion of this pathogen [93].

A similar case is observed with *Histoplasma capsulatum*, an endemic fungus transmitted through inhalation in areas with bird or bat droppings in northern parts of the USA. It is known to cause severe pneumonia in immunocompromised hosts. Changes in animal behavior and geographic distribution due to global warming have likely had an impact on the diffusion of this disease [94].

Not only the spread of fungal respiratory infections, but also that of mycobacteriosis is intertwined with climate change. It has been demonstrated that hurricanes, whose number has lately increased due climate change, contribute to an increase in non-tuberculous mycobacteria (NTM) disease [95]. Different from TB, NTM lung diseases are typically conveyed through environmental sources, such as municipal water and soil, and environmental cross-contamination by NTM is greatly favored by hurricanes [95,96].

It is evident to all how public health and safety are threatened and damaged by emerging viral diseases, e.g., the avian flu, severe acute respiratory syndrome (SARS), Ebola, and novel viruses in the Coronavirus family.

Climate change must be considered a co-factor in their outbreak and spread. Notably, both biodiversity decreases and air pollution increases caused by climate change might favor the onset and diffusion of the COVID-19 pandemic [97]. A rise in air pollution not only modifies the respiratory tract's permeability through oxidative stress and the over-expression of Angiotensin-converting enzyme 2 (ACE-2), but also triggers a chronic inflammatory status and promotes respiratory co-morbidities that greatly increase the risk of a severe course and the mortality of COVID-19 [98]. A large study conducted in 2021 [99] on 130 stations, across 31 countries and five continents, found that pollen, also in synergy with temperature and humidity, can explain about 44% of the infection rate variability. Moreover, it is indicated that pollen exposure itself may modulate the antiviral defense of the respiratory epithelium, suggesting that some individuals should avoid exposure in outdoor activities during the coincidence of pollen and respiratory virus seasons [100].

Finally, it is known that exposure to high temperatures and pollution, as direct effects of climate change, can affect a host's immune system [101]. Therefore, the fight against fossil fuel emissions and air pollutant release can prevent the outbreak of new viral diseases and therefore new epidemics, but also limit the damage to societies and health systems caused by these diseases.

10. Conclusions

Climate change has several effects on human health, in particular on respiratory health. A rise in temperature causes direct health effects due to the higher risks of specific pathologies such as chronic obstructive pulmonary diseases (COPD) and hospital admissions and deaths due to respiratory diseases. Extreme environmental events such as thunderstorms and a higher humidity and temperature can cause an increase in the frequency of hospital admissions for thunderstorm asthma [102,103].

Increases in air pollutants, including higher levels of ozone and (bio)pollutants, may be responsible for indirect respiratory health effects [104,105]. Air pollution is a key component linked to the climate-change-driven worsening of respiratory health effects, since pollen and fungal spores are able to interact with these pollutants. The exposure to several (bio)contaminants in urban settings is linked to severe episodes of asthma attacks and/or exacerbations, mostly regarding individuals that are IgE sensitized [4,106,107].

Climate change has important effects on the origin of hypersensitivity and pollen allergy. Climate change can determine an increased pollen production in plants and amp up the allergenic properties of pollen grains. An alteration in plant growth could worsen the negative effects on human health even more in the future. Similarly, although the data are sparse, climate change impacts mold proliferation through precipitation increases and

floods. As a further consideration, the microbiomes of different forest ecosystems and geographic areas can be differently affected by climate change [108].

As a consequence of this, in the medium and long term, an increase in the prevalence of allergic diseases brought on by pollen and mold is expected [109–111].

The study of pollen allergy needs to be deepened with regard to several aspects, starting with world changes and analyzing the numerous aspects linked to them. The promotion of studies aimed at deepening the interactions between pollen and chemical pollutants reported by the WHO guidelines [86] is an important tool that provides numerous research opportunities.

New developments should address the management of the integrated aspects of environment and human health, with specific attention to the general and occupational population. At the same time, an improvement in the methodologies aimed at evaluating the sources of exposure, as well as the responses of individuals to numerous (bio)contaminants, should be considered in the management of health effects [112,113].

Around the world, there is an urgent need to address public education and the formation of governmental initiatives for reducing pollution and mitigating the impacts of climate change. To deal with the effects of climate change on pollen, molds, and air pollution, several strategies are accessible, consisting either of mitigation measures (measures dealing with the causes of climate change, i.e., the accumulation of greenhouse gases in the atmosphere) or adaptation measures (dealing with the impacts of climate change). Adaptation is certainly important, since the negative effects of climate change are already in action and impacting the world. However, the effectiveness of these adaptation measures is inevitably linked to the limitation of greenhouse gas emissions, making mitigation more essential than ever and the most important action available. Extreme weather events such as thunderstorms cause severe asthma attacks and asthma exacerbations, with relevant socio-economic consequences, and also have to be prevented by meteorological broadcasting. Finally, the general population, and in particular patients with asthma and pollen allergies, should be educated about the health risks related to climate change. Education programs should address different categories for both adults and children.

The information should include several aspects, starting with the phenomena responsible for environmental variations, as well as the adverse health effects and mitigation measures, explained with effective messages and tools.

Health professionals should be, in turn, educated on the appropriate methods for transferring key messages in their clinical practices. Physicians should be able to develop basic knowledge on climate change in relation to environmental variations and health effects.

New perspectives on synergizing the different topics of climate change and education with health effects should take into account the evolution of (bio)medical sciences and promote sustainable actions and key messages as being able to extend the *Curricula* of different professionals [114–118]. Specific training programs need to be developed.

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References

1. D'Amato, G.; Akdis, C. Global warming, climate change, air pollution and allergies. *Allergy* **2020**, *75*, 2158–2160. <https://doi.org/10.1111/all.14527>.
2. Beggs, P.G.; Bambrick, H.J. Is the global rise of asthma an early impact of anthropogenic climate change? *Environ. Health Perspect.* **2005**, *113*, 915–919. <https://doi.org/10.1289/ehp.7724>.
3. D'Amato, G.; Holgate, S.T.; Pawankar, R.; Ledford, D.K.; Cecchi, L.; Al-Ahmad, M.; Al-Enezi, F.; Al-Muhsen, S.; Ansotegui, I.; Baena-Cagnani, C.E.; et al. Meteorological conditions, climate change, new emerging factors, and asthma and related allergic disorders. A statement of the World Allergy Organization. *World Allergy Organ. J.* **2015**, *8*, 25. <https://doi.org/10.1186/s40413-015-0073-0>.
4. D'Amato, G.; Pawankar, R.; Vitale, C.; Lanza, M.; Molino, A.; Stanziola, A.; Sanduzzi, A.; Vatrella, A.; D'Amato, M. Climate change and air pollution: Effects on respiratory allergy. *Allergy Asthma Immunol. Res.* **2016**, *8*, 391–395. <https://doi.org/10.4168/aaair.2016.8.5.391>.
5. D'Amato, G.; Vitale, C.; Lanza, M.; Molino, A.; D'Amato, M. Climate change, air pollution, and allergic respiratory diseases: An update. *Curr. Opin. Allergy Clin. Immunol.* **2016**, *16*, 434–440. <http://doi.org/10.1097/ACI.0000000000000301>.
6. Hegerl, G.C.; Zwiers, F.W.; Braconnot, P.; Gillett, N.P.; Luo, Y.; Marengo Orsini, J.A.; Nicholls, N.; Penner, J.E.; Stott, P.A. Understanding and attributing climate change. In *Climate Change 2007: The Physical Science Basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*; Solomon, S., Qin, D., Manning, M., Chen, Z., Marquis, M., Averyt, K.B., Tignor, M., Miller, H.L., Eds.; Cambridge University Press: Cambridge, UK; New York, NY, USA, 2007.
7. Californian Department of Forestry and Fire Protection. Available online: <http://www.fire.ca.gov/index.php> (accessed on 17 April 2023).
8. Gent, J.F.; Triche, E.W.; Holford, T.R.; Belanger, K.; Bracken, M.B.; Beckett, W.S.; Leaderer, B.P. Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. *JAMA* **2003**, *290*, 1859–1867. <https://doi.org/10.1001/jama.290.14.1859>.
9. McDonnell, W.F.; Abbey, D.E.; Nishino, N.; Lebowitz, M.D. Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: The AHSMOG Study. *Environ. Res.* **1999**, *80*, 110–121. <https://doi.org/10.1006/enrs.1998.3894>.
10. Singer, B.D.; Ziska, L.H.; Frenz, D.A.; Gebhard, D.E.; Straka, J.G. Increasing Amb a 1 content in common ragweed (*Ambrosia artemisiifolia*) pollen as a function of rising atmospheric CO₂ concentration. *Func. Plant. Biol.* **2005**, *32*, 667–670. <https://doi.org/10.1071/FP05039>.
11. Rogers, H.H.; Runion, G. B.; Krupa, S. V. Plant responses to atmospheric CO₂ enrichment with emphasis on roots and the rhizosphere. *Environ. Pollut.* **1994**, *83*, 155–189. [https://doi.org/10.1016/0269-7491\(94\)90034-5](https://doi.org/10.1016/0269-7491(94)90034-5).
12. United States Environmental Protection Agency (EPA). Overview of Greenhouse Gases. Available online: <http://learning-cleanairasia.org/lms/library/ga3/99-Overview-of-Greenhouse-Gases.pdf> (accessed on 17 August 2019).
13. National Oceanic and Atmospheric Administration. Carbon Dioxide Now More than 50% Higher than Pre-Industrial Levels. Available online: <https://www.noaa.gov/news-release/carbon-dioxide-now-more-than-50-higher-than-pre-industrial-levels> (accessed on 17 April 2023).
14. D'Amato, G.; Cecchi, L.; Bonini, S.; Nunes, C.; Annesi-Maesano, I.; Behrendt, H.; Liccardi, G.; Popov, T.; Van Cauwenberge, P. Allergenic pollen and pollen allergy in Europe. *Allergy* **2007**, *62*, 976–990. <https://doi.org/10.1111/j.1398-9995.2007.01393>.
15. Taylor, P.E.; Flagan, R.; Valenta, R.; Glovsky, M.M. Release of allergens in respirable aerosols: A link between grass pollen and asthma. *J. Allergy Clin. Immunol.* **2002**, *109*, 51–56. <https://doi.org/10.1067/mai.2002.120759>.
16. Taylor, P.E.; Flagan, R.C.; Miguel, A.G.; Valenta, R.; Glovsky, M.M. Birch pollen rupture and the release of aerosols of respirable allergens. *Clin. Exp. Allergy* **2004**, *34*, 1591–1596. <https://doi.org/10.1111/j.1365-2222.2004.02078.x>.
17. Siriwanakul, U.; Piboonpocanun, S.; Traiperm, P.; Pichakam, A.; Songnuan, W. *Amaranthus* species around Bangkok, Thailand and the release of allergenic proteins from their pollens. *Asian Pac. J. Allergy Immunol.* **2015**, *33*, 203–210. <https://doi.org/10.12932/AP0547.33.3.2015>.
18. Zaidi, M.A.; O'Leary, S.; Wu, S.; Gleddie, S.; Eudes, F.; Laroche, A.; Robert, L.S. A molecular and proteomic investigation of proteins rapidly released from triticale pollen upon hydration. *Plant. Mol. Biol.* **2012**, *79*, 101–121. <https://doi.org/10.1007/s11103-012-9897-y>.
19. Spieksma, M.; Nikkels, A.H. Similarity in seasonal appearance between atmospheric birch-pollen grains and allergen in paucimicronic, size-fractionated ambient aerosol. *Allergy* **1999**, *54*, 235–241. <https://doi.org/10.1034/j.1398-9995.1999.00817.x>.
20. Ziska, L.H.; Bunce, J.A.; Goins, E.W. Characterization of an urban-rural CO₂/temperature gradient and associated changes in initial plant productivity during secondary succession. *Oecologia* **2004**, *139*, 454–458. <https://doi.org/10.1007/s00442-004-1526-2>.
21. Wayne, P.; Foster, S.; Connolly, J.; Bazzaz, F.; Epstein, P. Production of allergenic pollen by ragweed (*Ambrosia artemisiifolia* L.) is increased in CO₂-enriched atmospheres. *Ann. Allergy Asthma Immunol.* **2002**, *88*, 279–282. [https://doi.org/10.1016/S1081-1206\(10\)62009-1](https://doi.org/10.1016/S1081-1206(10)62009-1).
22. Cecchi, L.; Morabito, M.; Domeneghetti, M.P.; Crisci, A.; Onorari, M.; Orlandini, S. Long distance transport of ragweed pollen as a potential cause of allergy in central Italy. *Ann. Allergy Asthma Immunol.* **2006**, *96*, 86–91. [https://doi.org/10.1016/s1081-1206\(10\)61045-9](https://doi.org/10.1016/s1081-1206(10)61045-9).
23. Davies, J.M. Grass pollen allergens globally: The contribution of subtropical grasses to burden of allergic respiratory diseases. *Clin. Exp. Allergy* **2014**, *44*, 790–801. <https://doi.org/10.1111/cea.12317>.

24. Osborne, N.J.; Alcock, I.; Wheeler, B.W.; Hajat, S.; Sarran, C.; Clewlow, Y.; McInnes, R.N.; Hemming, D.; White, M.; Vardoulakis, S.; et al. Pollen exposure and hospitalization due to asthma exacerbations: Daily time series in a European city. *Int. J. Biometeorol.* **2017**, *61*, 1837–1848. <https://doi.org/10.1007/s00484-017-1369-2>.
25. Jaeger, S. Exposure to grass pollen in Europe. *Clin. Exp. Allergy Rev.* **2008**, *8*, 2–6. <https://doi.org/10.1111/j.1472-9733.2008.00125.x>.
26. Kleine-Tebbe, J.; Davies, J. Grass pollen allergens. In *Global Atlas of Allergy*; Akdis, C.A., Agache, I., Eds.; European Academy of Allergy and Clinical Immunology: Zürich, Switzerland, 2014; pp. 22–26.
27. Davies, J.; Timbrell, V.; Reibelt, L.; Simmonds, C.; Solley, G.; Smith, W.B.; Mclean-Tooke, A.; Nunen, S.; Smith, P.; Upham, J.; et al. Regional variation in allergic sensitivity to subtropical and temperate grass pollen allergens; outcomes of the multicenter cross-sectional Grass Pollen Allergy Survey (GPAS). *Eur. J. Immunol.* **2016**, *46*, 841.
28. Ramon, G.D.; Viego, V.; Arango, N.; Long, M.A.; Kahn, A.; Barrionuevo, L.B. Allergy to *Cynodon dactylon* (Cyn d) pollen in seasonal rhinitis, comparison between skin tests and component-resolved diagnosis (CRD) in Bahía Blanca (Argentina). *J. Allergy Clin. Immunol.* **2018**, *141*, AB129. <https://doi.org/10.1016/j.jaci.2017.12.412>.
29. Rogers, C.A.; Wayne, P.M.; Macklin, E.A.; Muilenberg, M.L.; Wagner, C.J.; Epstein, P.R.; Bazzaz, F.A. Interaction of the onset of spring and elevated atmospheric CO₂ on ragweed (*Ambrosia artemisiifolia* L.) pollen production. *Environ. Health Perspect.* **2006**, *114*, 865–869. <https://doi.org/10.1289/ehp.8549>.
30. Soreng, R.J.; Peterson, P.M.; Romaschenko, K.; Davidse, G.; Zuloaga, F.O.; Judziewicz, E.J.; Filguerias, T.S.; Davis, J.I.; Morrone, O. A worldwide phylogenetic classification of the Poaceae (Gramineae). *J. Syst. Evol.* **2015**, *53*, 117–137. <https://doi.org/10.1111/jse.12150>.
31. Soreng, R.J.; Peterson, P.M.; Romaschenko, K.; Davidse, G.; Teisher, J.K.; Clark, L.G.; Barberá, P.; Gillespie, L.J.; Zuloaga, F.O. A worldwide phylogenetic classification of the Poaceae (Gramineae) II: An update and a comparison of two 2015 classifications. *J. Syst. Evol.* **2017**, *55*, 259–290. <https://doi.org/10.1111/jse.12262>.
32. Gornall, J.; Betts, R.; Burke, E.; Clark, R.; Camp, J.; Willett, K.; Wiltshire, A. Implications of climate change for agricultural productivity in the early twenty-first century. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **2010**, *365*, 2973–2989. <https://doi.org/10.1098/rstb.2010.0158>.
33. García-Mozo, H.; Oteros, J.A.; Galán, C. Impact of land cover changes and climate on the main airborne pollen types in Southern Spain. *Sci. Total. Environ.* **2016**, *548*–*549*, 221–228. <https://doi.org/10.1016/j.scitotenv.2016.01.005>.
34. Raiten, D.J.; Allen, L.H.; Slavin, J.L.; Mitloehner, F.M.; Thoma, G.J.; Haggerty, P.A.; Finley, J.W. Understanding the Intersection of Climate/Environmental Change, Health, Agriculture, and Improved Nutrition: A Case Study on Micronutrient Nutrition and Animal Source Foods. *Curr. Dev. Nutr.* **2020**, *4*, nzaa087. <https://doi.org/10.1093/cdn/nzaa087>.
35. D’Amato, G.; Annesi-Maesano, I.; Urrutia-Pereira, M.; Del Giacco, S.; Rosario Filho, N.A.; Chong-Neto, H.J.; Solé, D.; Ansotegui, I.; Cecchi, L.; Sanduzzi Zamparelli, A.; et al. Thunderstorm allergy and asthma: State of the art. *Multidiscip. Respir. Med.* **2021**, *16*, 806. <https://doi.org/10.4081/mrm.2021.806>.
36. Haahtela, T. A biodiversity hypothesis. *Allergy* **2019**, *74*, 1445–1456. <https://doi.org/10.1111/all.13763>.
37. Haahtela, T.; Holgate, S.; Pawankar, R.; Akdis, C.A.; Benjaponpitak, S.; Caraballo, L.; Demain, J.; Portnoy, J.; von Hertzen, L. The biodiversity hypothesis and allergic disease: World allergy organization position statement. *World Allergy Organ. J.* **2013**, *6*, 3.
38. González Roldán, N.; Engel, R.; Düpow, S.; Jakob, K.; Koops, F.; Orinska, Z.; Vigor, C.; Oger, C.; Galano, J.M.; Durand, T.; et al. Lipid Mediators From Timothy Grass Pollen Contribute to the Effector Phase of Allergy and Prime Dendritic Cells for Glycolipid Presentation. *Front. Immunol.* **2019**, *10*, 974. <https://doi.org/10.3389/fimmu.2019.00974>.
39. Traidl-Hoffmann, C.; Kasche, A.; Thilo, J.; Huger, M.; Plötz, S.; Feussner, I.; Ring, J.; Behrendt, H. Lipid mediators from pollen act as chemoattractants and activators of polymorphonuclear granulocytes. *J. Allergy Clin. Immunol.* **2002**, *109*, 831–838. <https://doi.org/10.1067/mai.2002.124655>.
40. McConnell, R.; Berhane, K.; Gilliland, F.; London, S.J.; Islam, T.; Gauderman, W.J.; Avol, E.; Margolis, H.G.; Peters, J.M. Asthma in exercising children exposed to ozone: A cohort study. *Lancet* **2002**, *359*, 386–391. [https://doi.org/10.1016/S0140-6736\(02\)07597-9](https://doi.org/10.1016/S0140-6736(02)07597-9).
41. Islam, T.; Gauderman, W.J.; Berhane, K.; McConnell, R.; Avol, E.; Peters, J.M.; Gilliland, F.D. Relationship between air pollution, lung function and asthma in adolescents. *Thorax* **2007**, *62*, 957–963. <https://doi.org/10.1136/thx.2007.078964>.
42. Kreit, J.W.; Gross, K.B.; Moore, T.B.; Lorenzen, T.J.; D’Arcy, J.; Eschenbacher, W.L. Ozone-induced changes in pulmonary function and bronchial responsiveness in asthmatics. *J. Appl. Physiol.* **1989**, *66*, 217–222. <https://doi.org/10.1152/jappl.1989.66.1.217>.
43. Scannell, C.; Chen, L.L.; Aris, R.M.; Tager, I.; Christian, D.; Ferrando, R.; Welch, B.; Kelly, T.; Balmes, J.R. Greater ozone-induced inflammatory responses in subjects with asthma. *Am. J. Respir. Crit. Care Med.* **1996**, *154*, 24–29. <https://doi.org/10.1164/ajrccm.154.1.8680687>.
44. Bayram, H.; Sapsford, R.J.; Abdelaziz, M.M.; Khair, O.A. Effect of ozone and nitrogen dioxide on the release of proinflammatory mediators from bronchial epithelial cells on nonatopic, nonasthmatic subjects and atopic asthmatic patients in vitro. *J. Allergy Clin. Immunol.* **2001**, *107*, 287–294. <https://doi.org/10.1067/mai.2001.111141>.
45. Diaz Sanchez, D.; Tsien, A.; Fleming, J.; Saxon, A. Combined diesel exhaust particulate and ragweed allergen challenge markedly enhances human in vivo nasal ragweed specific IgE and skews cytokine production to a T helper cell 2-type pattern. *J. Immunol.* **1997**, *158*, 2406–2413. <https://doi.org/10.4049/jimmunol.158.5.2406>.
46. D’Amato, G.; Liccardi, G.; D’Amato, M.; Holgate, S.T. Environmental risk factors and allergic bronchial asthma. *Clin. Exp. Allergy* **2005**, *35*, 1113–1124. <https://doi.org/10.1111/j.1365-2222.2005.02328.x>.

47. D'Amato, G.; Cecchi, L. Effects of climate change on environmental factors in respiratory allergic diseases. *Clin. Exp. Allergy* **2008**, *38*, 1264–1274. <https://doi.org/10.1111/j.1365-2222.2008.03033.x>.
48. Burney, P.; Malmberg, E.; Chinn, S.; Jarvis, D.; Luczynska, C.; Lai, E. The distribution of total and specific serum IgE in the European community respiratory health survey. *J. Allergy Clin. Immunol.* **1997**, *99*, 314–322. [https://doi.org/10.1016/s0091-6749\(97\)70048-4](https://doi.org/10.1016/s0091-6749(97)70048-4).
49. D'Amato, G.; Cecchi, L.; Annesi-Maesano, I. A trans-disciplinary overview of case reports of thunderstorm-related asthma outbreaks and relapse. *Eur. Respir. Rev.* **2012**, *21*, 82–87. <https://doi.org/10.1183/09059180.00001712>.
50. Traidl-Hoffmann, C.; Kasche, A.; Menzel, A.; Jakob, T.; Thiel, M.; Ring, J.; Behrendt, H. Impact of pollen on human health: More than allergen carriers? *Int. Arch. Allergy Immunol.* **2003**, *131*, 1–13. <https://doi.org/10.1159/000070428>.
51. Bernard, S.M.; Samet, J.M.; Grambsch, A.; Ebi, K.L.; Romieu, I. The potential impacts of climate variability and change on air pollution-related health effects in the United States. *Environ. Health Perspect.* **2001**, *109*, 199–209. <https://doi.org/10.1289/ehp.109-1240667>.
52. Confalonieri, U.; Menne, B.; Akhtar, R.; Ebi, K.L.; Hauengue, M.; Kovats, R.S.; Revich, B.; Woodward, A. Human health. In *Climate Change 2007: Impacts, Adaptation and Vulnerability. Contribution of Working Group. II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*; Parry, M.L., Canziani, O.F., Palutikof, J.P., van der Linden, P.J., Hanson, C.E., Eds.; Cambridge University Press: Cambridge, UK, 2007; pp. 391–431.
53. USEPA. Motor Vehicles and the 1990 Clean Air Act. Fact Sheet OMS-11. EPA 400-F-92-013. 1994. Available online: <https://nepis.epa.gov/Exe/ZyPDF.cgi/900L1M00.PDF?Dockey=900L1M00.PDF> (accessed on 17 April 2023).
54. USEPA. *Air Quality Criteria for Ozone and Related Photochemical Oxidants*; EPA/600/P-93/004a-cF; Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment: Washington, DC, USA, 1996.
55. Zhu, C.; Farah, J.; Choël, M.; Gosselin, S.; Baroudi, M.; Petitprez, D.; Visez, N. Uptake of ozone and modification of lipids in Betula Pendula pollen. *Environ. Pollut.* **2018**, *242* (Pt A), 880–886. <https://doi.org/10.1016/j.envpol.2018.07.025>.
56. Andrew, E.; Nehme, Z.; Bernard, S.; Abramson, M.J.; Newbiggin, E.; Piper, B.; Dunlop, J.; Smith, K. Stormy weather: A retrospective analysis of demand for emergency medical services during epidemic thunderstorm asthma. *BMJ* **2017**, *359*, j5636. <https://doi.org/10.1136/bmj.j5636>.
57. Davies, J.; Erbas, B.; Simunovic, M.; Al Kouba, J.; Milic, A. *Final Report: Literature Review on Thunderstorm Asthma and Its Implications for Public Health Advice*; Contracted by: Department of Health and Human Services, Victorian State Government. 19 May 2017; Queensland University of Technology: Brisbane, Australia, 2017.
58. Lindstrom, S.J.; Silver, J.D.; Sutherland, M.F. Thunderstorm asthma outbreak of november 2016: A natural disaster requiring planning. *Med. J. Aust.* **2017**, *207*, 235–237. <https://doi.org/10.5694/mja17.00285>.
59. Ganseman, E.; Gouwy, M.; Bullens, D.M.A.; Breynaert, C.; Schrijvers, R.; Proost, P. Reported cases and diagnostics of occupational insect allergy: A systematic review. *Int. J. Mol. Sci.* **2022**, *24*, 86. <https://doi.org/10.3390/ijms24010086>.
60. D'Ovidio, M.C.; Wirz, A.; Zennaro, D.; Massari, S.; Melis, P.; Peri, V.M.; Rafeiani, C.; Riviello, M.C.; Mari, A. Biological occupational allergy: Protein microarray for the study of laboratory animal allergy (LAA). *AIMS Public. Health* **2018**, *5*, 352–365. <https://doi.org/10.3934/publichealth.2018.4.352>.
61. Siegel, J.; Gill, N.; Ramanathan, M., Jr.; Patadia, M. Unified airway disease: Environmental factors. *Otolaryngol. Clin. North. Am.* **2023**, *56*, 39–53. <https://doi.org/10.1016/j.otc.2022.09.003>.
62. Goyal, A.; Ravindra, K.; Mor, S. Occupational exposure to airborne pollen and associated health risks among gardeners: A perception-based survey. *Environ. Sci. Pollut. Res. Int.* **2022**, *29*, 70084–70098. <https://doi.org/10.1007/s11356-022-20595-2>.
63. Tsui, H.C.; Ronsmans, S.; Hoet, P.H.M.; Nemery, B.; Vanoirbeek, J.A.J. Occupational asthma caused by low-molecular-weight chemicals associated with contact dermatitis: A retrospective study. *J. Allergy Clin. Immunol. Pract.* **2022**, *10*, 2346–2354.e4. <https://doi.org/10.1016/j.jaip.2022.05.014>.
64. Kurt, O.K.; Basaran, N. Occupational exposure to metals and solvents: Allergy and airway diseases. *Curr. Allergy Asthma Rep.* **2020**, *20*, 38. <https://doi.org/10.1007/s11882-020-00931-7>.
65. Rocholl, M.; Weinert, P.; Bielfeldt, S.; Laing, S.; Wilhelm, K.P.; Ulrich, C.; John, S.M. New methods for assessing secondary performance attributes of sunscreens suitable for professional outdoor work. *J. Occup. Med. Toxicol.* **2021**, *16*, 25. <https://doi.org/10.1186/s12995-021-00314-2>.
66. World Allergy Organization. *White Book on Allergy: Update 2013*; Pawankar, R., Canonica, G.W., Holgate, S.T., Lockey, R.F., Blaiss, M.S., Eds.; World Allergy Organization: Milwaukee, WI, USA, 2013; ISBN 10:061592915X.
67. European Academy of Allergy and Clinical Immunology. *EAACI White Paper*; Agache, I., Akdis, C.A., Chivato, T., Hellings, P., Hoffman-Sommergruber, K., Jutel, M., Lauerma, A., Papadopoulos, N., Schmid-Grendelmeier, P., Schmidt-Weber, C., Eds.; European Academy of Allergy and Clinical Immunology: Zurich, Switzerland, 2018.
68. D'Amato, G.; Chong-Neto, H.J.; Monge Ortega, O.P.; Vitale, C.; Ansotegui, I.; Rosario, N.; Haahtela, T.; Galan, C.; Pawankar, R.; Murrieta-Aguttes, M.; et al. The effects of climate change on respiratory allergy and asthma induced by pollen and mold allergens. *Allergy* **2020**, *75*, 2219–2228. <https://doi.org/10.1111/all.14476>.
69. Applebaum, K.M.; Graham, J.; Gray, G.M.; LaPuma, P.; McCormick, S.A.; Northcross, A.; Perry, M.J. An overview of occupational risks from climate change. *Curr. Environ. Health Rep.* **2016**, *3*, 13–22. <https://doi.org/10.1007/s40572-016-0081-4>.
70. Rorie, A.; Poole, J.A. The Role of Extreme Weather and Climate-Related Events on Asthma Outcomes. *Immunol. Allergy Clin. North. Am.* **2021**, *41*, 73–84. <https://doi.org/10.1016/j.iac.2020.09.009>.

71. Sheehan, W.J.; Gaffin, J.M.; Peden, D.B.; Bush, R.K.; Phipatanakul, W. Advances in environmental and occupational disorders in 2016. *J. Allergy Clin. Immunol.* **2017**, *140*, 1683–1692. <https://doi.org/10.1016/j.jaci.2017.09.032>.
72. Peden, D.; Reed, C.E. Environmental and occupational allergies. *J. Allergy Clin. Immunol.* **2010**, *125*, S150–S160. <https://doi.org/10.1016/j.jaci.2009.10.073>.
73. D'Ovidio, M.C.; Annesi-Maesano, I.; D'Amato, G.; Cecchi, L. Climate change and occupational allergies: An overview on biological pollution, exposure and prevention. *Ann. Ist. Super. Sanità* **2016**, *52*, 406–414. https://doi.org/10.4415/ANN_16_03_12.
74. Oldenburg, M.; Petersen, A.; Baur, X. Maize pollen is an important allergen in occupationally exposed workers. *J. Occup. Med. Toxicol.* **2011**, *6*, 32. <https://doi.org/10.1186/1745-6673-6-32>.
75. Tizek, L.; Redlinger, E.; Ring, J.; Eyerich, K.; Biedermann, T.; Zink, A. Urban vs. rural—Prevalence of self-reported allergies in various occupational and regional settings. *World Allergy Organ. J.* **2022**, *15*, 100625. <https://doi.org/10.1016/j.waojou.2022.100625>.
76. Han, J.; Kim, Y.; Lee, S.; Lee, S.J. Association between the prevalence of allergic reactions to skin prick tests and workplace types among agricultural workers in South Korea. *Ann. Occup. Environ. Med.* **2020**, *32*, e36. <https://doi.org/10.35371/aoem.2020.32.e36>.
77. D'Ovidio, M.C.; Di Renzi, S.; Capone, P.; Pelliccioni, A. Pollen and fungal spores evaluation in relation to occupants and microclimate in indoor workplaces. *Sustainability* **2021**, *13*, 3154. <https://doi.org/10.3390/su13063154>.
78. Pelliccioni, A.; Ciardini, V.; Lancia, A.; Di Renzi, S.; Brighetti, M.A.; Travaglini, A.; Capone, P.; D'Ovidio, M.C. Intercomparison of indoor and outdoor pollen concentrations in rural and suburban research workplaces. *Sustainability* **2021**, *13*, 8776. <https://doi.org/10.3390/su13168776>.
79. Lancia, A.; Gioffrè, A.; Magri, D.; D'Ovidio, M.C. Aerobiological monitoring in an indoor occupational setting using a real-time bioaerosol sampler. *Atmosphere* **2023**, *14*, 118. <https://doi.org/10.3390/atmos14010118>.
80. Lancia, A.; Capone, P.; Vonesch, N.; Pelliccioni, A.; Grandi, C.; Magri, D.; D'Ovidio, M.C. Research progress on aerobiology in the last 30 years: A focus on methodology and occupational health. *Sustainability* **2021**, *13*, 4337. <https://doi.org/10.3390/su13084337>.
81. Akdis, C.A.; Akdis, M.; Boyd, S.D.; Sampath, V.; Galli, S.J.; Nadeau, K.C. Allergy: Mechanistic insights into new methods of prevention and therapy. *Sci. Transl. Med.* **2023**, *15*, eadd2563. <https://doi.org/10.1126/scitranslmed.add2563>.
82. Dbouk, T.; Visez, N.; Ali, S.; Shahrouh, I.; Drikakis, D. Risk assessment of pollen allergy in urban environments. *Sci. Rep.* **2022**, *12*, 21076. <https://doi.org/10.1038/s41598-022-24819-w>.
83. Sousa-Silva, R.; Smargiassi, A.; Kneeshaw, D.; Dupras, J.; Zinszer, K.; Paquette, A. Strong variations in urban allergenicity riskscapes due to poor knowledge of tree pollen allergenic potential. *Sci. Rep.* **2021**, *11*, 10196. <https://doi.org/10.1038/s41598-021-89353-7>.
84. Diem, L.; Neuherz, B.; Rohrhofer, J.; Koidl, L.; Asero, R.; Brockow, K.; Diaz Perales, A.; Faber, M.; Gebhardt, J.; Torres, M.J.; et al. Real-life evaluation of molecular multiplex IgE test methods in the diagnosis of pollen associated food allergy. *Allergy* **2022**, *77*, 3028–3040. <https://doi.org/10.1111/all.15329>.
85. Sénéchal, H.; Visez, N.; Charpin, D.; Shahali, Y.; Peltre, G.; Biolley, J.P.; Lhuissier, F.; Couderc, R.; Yamada, O.; Malrat-Domenge, A.; et al. A review of the effects of major atmospheric pollutants on pollen grains, pollen content, and allergenicity. *Sci. World J.* **2015**, *2015*, 940243. <https://doi.org/10.1155/2015/940243>.
86. World Health Organization. WHO Global Air Quality Guidelines. In *Particulate Matter (PM_{2.5} and PM₁₀), Ozone, Nitrogen Dioxide, Sulfur Dioxide and Carbon Monoxide*; World Health Organization: Geneva, Switzerland, 2021. Available online: <https://apps.who.int/iris/handle/10665/345329> (accessed on 17 April 2023).
87. Chu, B.; Chen, R.; Liu, Q.; Wang, H. Effects of high temperature on COVID-19 deaths in U.S. counties. *Geohealth* **2023**, *7*, e2022GH000705. <https://doi.org/10.1029/2022GH000705>.
88. Burnham, J.P.; Betz, F.; Lautz, R.; Mousavi, E.; Martinello, R.A.; McGain, F.; Sherman, J.D. Air exchanges, climate change, and severe acute respiratory coronavirus virus 2 (SARS-CoV-2): Results from a survey of the Society of Healthcare Epidemiology of America Research Network (SRN). *Antimicrob. Steward. Healthc. Epidemiol.* **2022**, *2*, e40. <https://doi.org/10.1017/ash.2021.256>.
89. Luo, Y.; Lv, H.; Yan, H.; Zhu, C.; Ai, L.; Li, W.; Yi, J.; Zhang, L.; Tan, W. Meteorological change and hemorrhagic fever with renal syndrome epidemic in China, 2004–2018. *Sci. Rep.* **2022**, *12*, 20037. <https://doi.org/10.1038/s41598-022-23945-9>.
90. Ashique, S.; Sandhu, N.K.; Das, S.; Haque, S.N.; Koley, K. Global comprehensive outlook of hantavirus contagion on humans: A review. *Infect. Disord. Drug. Targets* **2022**, *22*, e050122199975. <https://doi.org/10.2174/1871526522666220105110819>.
91. Lang, R.; Stokes, W.; Lemaire, J.; Johnson, A.; Conly, J. A case report of *Coccidioides posadasii* meningoencephalitis in an immunocompetent host. *BMC Infect. Dis.* **2019**, *19*, 722. <https://doi.org/10.1186/s12879-019-4329-0>.
92. Matlock, M.; Hopfer, S.; Ogunseit, O.A. Communicating risk for a climate-sensitive disease: A case study of valley fever in central California. *Int. J. Environ. Res. Public. Health* **2019**, *16*, 3254. <https://doi.org/10.3390/ijerph16183254>.
93. Maestrale, C.; Masia, M.; Pintus, D.; Lollai, S.; Kozel, T.R.; Gates-Hollingsworth, M.A.; Cancedda, M.G.; Cabras, P.; Pirino, S.; D'Ascenzo, V.; Ligios, C. Genetic and pathological characteristics of *Cryptococcus gattii* and *Cryptococcus neoformans* var. *neoformans* from meningoencephalitis in autochthonous goats and mouflons, Sardinia, Italy. *Vet. Microbiol.* **2015**, *177*, 409–413. <https://doi.org/10.1016/j.vetmic.2015.03.008>.
94. Devnath, P.; Karah, N.; Graham, J.P.; Rose, E.S.; Asaduzzaman, M. Evidence of antimicrobial resistance in bats and its planetary health impact for surveillance of zoonotic spillover events: A scoping review. *Int. J. Environ. Res. Public. Health* **2022**, *20*, 243. <https://doi.org/10.3390/ijerph20010243>.

95. Turner, N.A.; Sweeney, M.I.; Xet-Mull, A.M.; Storm, J.; Mithani, S.K.; Jones Jr, D.B.; Miles, J.J.; Tobin, D.M.; Stout, J.E. A Cluster of Nontuberculous Mycobacterial Tenosynovitis Following Hurricane Relief Efforts. *Clin. Infect. Dis.* **2021**, *72*, e931–e937. <https://doi.org/10.1093/cid/ciaa1665>.
96. Katsuda, R.; Yoshida, S.; Tsuyuguchi, K.; Kawamura, T. A case report of hot tub lung: Identical strains of *Mycobacterium avium* from the patient and the bathroom air. *Int. J. Tuberc. Lung Dis.* **2018**, *22*, 350–352. <https://doi.org/10.5588/ijtld.17.0642>.
97. Ali, N.; Islam, F. The effects of air pollution on COVID-19 infection and mortality—A review on recent evidence. *Front. Public Health* **2020**, *8*, 580057. <https://doi.org/10.3389/fpubh.2020.580057>.
98. Paital, B.; Agrawal, P.K. Air pollution by NO₂ and PM_{2.5} explains COVID-19 infection severity by overexpression of angiotensin-converting enzyme 2 in respiratory cells: A review. *Environ. Chem. Lett.* **2021**, *19*, 25–42. <https://doi.org/10.1007/s10311-020-01091-w>.
99. Damialis, A.; Gilles, S.; Sofiev, M.; Sofieva, V.; Kolek, F.; Bayr, D.; Plaza, M.P.; Leier-Wirtz, V.; Kaschuba, S.; Ziska, L.H.; et al. Higher airborne pollen concentrations correlated with increased SARS-CoV-2 infection rates, as evidenced from 31 countries across the globe. *Proc. Natl. Acad. Sci. USA* **2021**, *118*, e2019034118. <https://doi.org/10.1073/pnas.2019034118>.
100. Gilles, S.; Blume, C.; Wimmer, M.; Damialis, A.; Meulenbroek, L.; Gökkaya, M.; Bergougnan, C.; Eisenbart, S.; Sundell, N.; Lindh, M.; et al. Pollen exposure weakens innate defense against respiratory viruses. *Allergy* **2020**, *75*, 576–587. <https://doi.org/10.1111/all.14047>.
101. Glencross, D.A.; Ho, T.R.; Camina, N.; Hawrylowicz, C.M.; Pfeffer, P.E. Air pollution and its effects on the immune system. *Free. Radical Bio Med.* **2020**, *151*, 56–68. <https://doi.org/10.1016/j.freeradbiomed.2020.01.179>.
102. D’Amato, G.; Cecchi, L.; D’Amato, M.; Annesi-Maesano, I. Climate change and respiratory diseases. *Eur. Respir. Rev.* **2014**, *23*, 161–169. <https://doi.org/10.1183/09059180.00001714>.
103. De Sario, M.; Katsouyanni, K.; Michelozzi, P. Climate change, extreme weather events, air pollution and respiratory health in Europe. *Eur. Respir. J.* **2013**, *42*, 826–843. <https://doi.org/10.1183/09031936.00074712>.
104. Mendell, M.J.; Mirer, A.G.; Cheung, K.; Tong, M.; Douwes, J. Respiratory and allergic health effects of dampness, mold, and dampness related agents: A review of the epidemiologic evidence. *Environ. Health Perspect.* **2011**, *119*, 748–756. <https://doi.org/10.1289/ehp.1002410>.
105. Ayres, J.G.; Forsberg, B.; Annesi-Maesano, I.; Dey, R.; Ebi, K.L.; Helms, P.J.; Medina-Ramon, M.; Windt, M.; Forastiere, F.; on behalf of the Environment and Health Committee of the European Respiratory Society. Climate change and respiratory disease: European Respiratory Society position statement. *Eur. Respir. J.* **2009**, *34*, 295–302. <https://doi.org/10.1183/09031936.00003409>.
106. Nassikas, N.J.; Spangler, K.; Wellenius, G.A. Asthma exacerbations attributable to ozone air pollution in New England. *R. I Med. J.* **2013**, *104*, 20–23.
107. Santos, U.P.; Arbex, M.A.; Braga, A.L.F.; Futoshi Mizutani, R.; Delfini Cançado, J.E.; Terra-Filho, M.; Chatkin, J.M. Environmental air pollution: Respiratory effects. *J. Bras. Pneumol.* **2021**, *47*, e20200267. <https://doi.org/10.36416/1806-3756/e20200267>.
108. Baldrian, P.; López-Mondéjar, R.; Kohout, P. Forest microbiome and global change. *Nat. Rev. Microbiol.* **2023**. <https://doi.org/10.1038/s41579-023-00876-4>.
109. Singh, A.B.; Kumar, P. Climate change and allergic diseases: An overview. *Front. Allergy* **2022**, *3*, 964987. <https://doi.org/10.3389/falgy.2022.964987>.
110. Hu, Y.; Xu, Z.; Jiang, F.; Li, S.; Liu, S.; Wu, M.; Yan, C.; Tan, J.; Yu, G.; Hu, Y.; et al. Relative impact of meteorological factors and air pollutants on childhood allergic diseases in Shanghai, China. *Sci. Total. Environ.* **2020**, *706*, 135975. <https://doi.org/10.1016/j.scitotenv.2019.135975>.
111. Poole, J.A.; Barnes, C.S.; Demain, J.G.; Bernstein, J.A.; Padukudru, M.A.; Sheehan, W.J.; Fogelbach, G.G.; Wedner, J.; Codina, R.; Levetin, E.; et al. Impact of weather and climate change with indoor and outdoor air quality in asthma: A Work Group Report of the AAAAI Environmental Exposure and Respiratory Health Committee. *J. Allergy Clin. Immunol.* **2019**, *143*, 1702–1710. <https://doi.org/10.1016/j.jaci.2019.02.018>.
112. Rorie, A. Climate change factors and the aerobiology effect. *Immunol. Allergy Clin. North. Am.* **2022**, *42*, 771–786. <https://doi.org/10.1016/j.iac.2022.05.007>.
113. Oh, J.W. Pollen allergy in a changing planetary environment. *Allergy Asthma Immunol. Res.* **2022**, *14*, 168–181. <https://doi.org/10.4168/aaair.2022.14.2.168>.
114. Di Cicco, M.E.; Ferrante, G.; Amato, D.; Capizzi, A.; De Pieri, C.; Ferraro, V.A.; Furno, M.; Tranchino, V.; La Grutta, S. Climate change and childhood respiratory health: A call to action for paediatricians. *Int. J. Environ. Res. Public Health* **2020**, *17*, 5344. <https://doi.org/10.3390/ijerph17155344>.
115. Goshua, A.; Gomez, J.; Erny, B.; Burke, M.; Luby, S.; Sokolow, S.; LaBeaud, A.D.; Auerbach, P.; Gisondi, M.A.; Nadeau, K. Addressing climate change and its effects on human health: A call to action for medical schools. *Acad. Med.* **2021**, *96*, 324–328. <https://doi.org/10.1097/ACM.00000000000003861>.
116. Dupraz, J.; Burnand, B. Role of health professionals regarding the impact of climate change on health-an exploratory review. *Int. J. Environ. Res. Public Health* **2021**, *18*, 3222. <https://doi.org/10.3390/ijerph18063222>.

117. Liu, I.; Rabin, B.; Manivannan, M.; Laney, E.; Philipsborn, R. Evaluating strengths and opportunities for a co-created climate change curriculum: Medical student perspectives. *Front. Public. Health* **2022**, *10*, 1021125. <https://doi.org/10.3389/fpubh.2022.1021125>.
118. McGushin, A.; de Barros, E.F.; Floss, M.; Mohammad, Y.; Ndikum, A.E.; Ngendahayo, C.; Oduor, P.A.; Sultana, S.; Wong, R.; Abelson, A. The World Organization of family doctors air health train the trainer program: Lessons learned and implications for planetary health education. *Lancet Planet. Health* **2023**, *7*, e55–e63. [https://doi.org/10.1016/S2542-5196\(22\)00218-2](https://doi.org/10.1016/S2542-5196(22)00218-2).

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