



The Impact of Climate Change on the Pollen Allergy and Sporulation of Allergic Fungi

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Abstract

Purpose of review The purpose of this chapter is to review allergic disease and how it is potentially impacted by climate change. It is difficult to measure the direct impact climate change has on allergic disease. This is difficult because there are many variables impacting human health as well as what capacity humans have to adapt to these changes. Asthma is tightly associated with allergies and environmental factors, especially in children. In this review, we will explore evidence of environmental changes associated with climate change and the potential impacts on allergy and associated respiratory disease. Furthermore, this paper is to review the impact of climate change on allergy to atmospheric fungi which are known to cause a common allergic response. In this review, we will explore evidence of environmental changes associated with climate change and the potential impacts on allergy.

Recent findings The climate has been measurably changing for the past 100 years and has been described as the most significant health threat of the twenty-first century. How climate change impacts human health is varied and coming more into focus. While direct

effects, such as heatwaves, severe weather, drought, and flooding, are well reported, effects that are indirect or secondary impacts involving changes in ecosystems are less obvious, though the body of data is growing and becoming more robust. It is these changes in ecosystems that may have the greatest impact on allergic and respiratory diseases. Otherwise, the airborne pollens and spores have also been linked with upper and lower respiratory conditions. Atmospheric pollen and spore concentrations are influenced by a wide array of environmental, meteorological, and biological factors and various interspecies interactions. Pollen and spores underlie seasonal variations. Especially climatic factors and circadian patterns influence the spectrum of their species and their concentrations in the environment. It may have the greatest impact on respiratory allergic diseases.

Summary This review will explore some of the impacts our changing climate, current and predicted, has which influences upper and lower respiratory allergic diseases. The discussion will focus on changing pollination with altered pollen patterns, as well as alteration of the composition and transformation of atmospheric allergic fungi with increased CO₂ air pollution and heat stress. The sporulation of fungi is likely to be amplified as CO₂ concentration increases with climate change, potentially contributing to the increasing prevalence and severity of asthma and other respiratory allergies.

The impact of climate change on pollen allergy

Introduction

Climate change may indeed be the most significant health threat of the twenty-first century [1, 2]. In 1988, the Intergovernmental Panel on Climate Change was created in cooperation with the United Nations Environmental Program and the World Meteorological Organization and presents comprehensive reports based on scientific, technical, and socio-economic knowledge on climate change, its impacts, and future risks [3]. The science is compelling that our climate is warming at an unprecedented rate; consequently, surface air temperatures have increased globally over this last century, reflected by warming oceans, rising sea levels, melting glaciers, and shrinking sea ice [3]. Global annually averaged surface air temperature has increased 1.8 °F (1.0 °C) over the last 115 years (1901–2016) with annual average near-surface air temperatures across Alaska and subarctic regions increasing more than double that [4]. Globally, our planet is the warmest in the history of modern civilization. This past decade has experienced record-breaking, climate-related weather extremes, and this trend is expected to continue into the foreseeable future [4]. Indicators of climate change are varied, including physical responses such as changes in surface temperature, atmospheric water vapor, precipitation, severe weather events,

retracting glaciers, rising sea level, and changes in sea and land ice [3].

Changing climate variables and both the potential and realized impacts on health have become more clearly focused over the past 20 years. Health outcomes associated with heat stress, flooding, crop failure, and vector-borne and waterborne diseases have been well reported [4, 5]. Allergies and associated respiratory disease are heterogenous, though tightly linked with environmental and consequently immunologic interaction [6•]. Some of these environmental impacts include changing pollen patterns, altered composition and transformation of atmospheric allergic fungi, and the influence of climate on air pollution. Heat stress, especially in combination with pollution, promotes inflammation and lowers the airway hyperreactivity threshold, thereby increasing the mortality rate [7].

Changing patterns of pollination

Seasonal pollen levels have long been directly linked with the development and worsening of upper and lower respiratory allergic disease. Carbon dioxide (CO₂) is the leading greenhouse gas implicated in global warming and has been shown to impact the growth rate, biomass, and allergenicity of some wind-pollinated

plants. Atmospheric CO₂ levels have topped 400 ppm, up from the pre-industrial level of 280 ppm [3, 4].

There is compelling evidence that higher ambient CO₂ concentrations and warmer temperatures result in increased pollen production, especially in the urban communities, where temperature and CO₂ are higher than in surrounding rural areas [5, 7–9]. Heightened pollen levels have been associated with increased CO₂ levels as demonstrated in environmental chambers with controlled variables, as well as in open-field studies [8–11]. Furthermore, when CO₂ levels doubled, individual ragweed plant pollen production increases 30 to 90% [8–10, 12]. In urban areas, where CO₂ levels and temperatures are elevated, ragweed flowers earlier and produces significantly more pollen [9].

Lengthening pollen seasons in North America have been correlated with increased temperatures, and lengthening frost-free periods, with increases greater in higher latitudes [13]. Elevated CO₂ levels have also been demonstrated to increase the major allergenic peptide of ragweed, Amb a 1, making each pollen grain and pollen particle more allergenic [11]. The major allergen of birch pollen, Bet v 1, has also been similarly demonstrated to be increased when trees are exposed to higher daily mean temperatures [14].

Correlating these data clinically, in an Italian study [15•], Ariano et al. monitored pollen counts and meteorological data, along with changes in allergen sensitization patterns of their patients. Over that 27-year period, there was a progressive 25% increase in measured tree and weed pollen levels as well as prolonged spring and fall pollen seasons were observed. These measured pollen cycle changes corresponded with rising daily temperature and more frequent days > 30 °C. An increase incidence of pollen sensitivity was observed during this same period, while there was no significant change to dust mite, a perennial allergen. The authors concluded that progressive climate changes, with increased temperatures, correlated with extended pollen seasons and increased pollen load, thus may have influenced the observed increased incidence of seasonal allergic sensitization [15•].

In summary, warmer temperatures, earlier frost off in the spring, and later onset of frost in the fall promote a prolonged allergy season for millions of people with seasonal allergy and asthma [13, 16]. Increasing CO₂ levels have been shown to significantly increase pollen load and intensify allergenicity in studied weed and tree pollens. Moreover, as the climate changes, plant species will likely adapt and undergo an expansion of

demographic distribution, expanding the zone of many allergenic plants.

Air pollution and health impacts

Pollution has long been associated with chronic lung disease, cancer, and cardiac disease. More recently, data indicate that pollen grains and air pollution mutually act, altering metabolic and physiologic features of pollen, resulting in potentiation immune reactivity [17]. Pollutants in conjunction with meteorological changes, especially in urban environments, provoke significant stress response in plants, further increasing biomass and allergenicity [18]. Additionally, air pollutants intensify aeroallergen sensitization by a direct impact on the respiratory mucosal environment. Two proposed principal mechanisms are an increased epithelial permeability associated with oxidative injury and a direct adjuvant effect [19•]. Ozone is known to induce an increase in NADPH in ragweed pollen, generating reactive oxygen species [18, 19]. A pollutant gas, ground-level ozone (O₃), has a direct impact on the human respiratory system, increasing frequency of asthma exacerbations, increasing susceptibility to infection and possible promotion of chronic obstructive pulmonary disease (COPD) [20]. At ground level, O₃ is generated by a photochemical reaction involving nitrogen dioxide (NO₂), hydrocarbons, heat, and ultraviolet radiation. Ground-level O₃, a major pollutant, has increased since pre-industrial times. Though air quality in the USA has improved over recent decades, ground ozone remains a major pollutant principally related to increasing emissions of methane, carbon monoxide, and nitrogen oxides, chiefly produced by transportation-related activities [6, 21]. Ground-level O₃ levels are highest during summers having the highest number of hot days (> 32 °C) [5]. Exposure to O₃ induces increased airway permeability, promotes airway inflammation, decreases lung function, and elevates the risk of asthma exacerbations [21, 22]. Beck et al. [23] demonstrated that exposure to elevated levels of O₃ enhances the allergic response to birch pollens in asthmatics. Prolonged exposure to O₃ promotes inflammation, impairs pulmonary function, particularly those predisposed to respiratory disease, and is associated with a higher risk of fatality from respiratory causes [24]. Furthermore, in the Children's Health Study [25], higher levels of O₃, NO₂, and fine particulate matter PM_{2.5} were linked with worsening pulmonary function as demonstrated by decreases in FEV₁.

Exposure to a high level of particulates poses a significant risk of worsening cardiorespiratory health. PM_{2.5} is a mixture of organic and inorganic solid particles and liquid particles of varying sizes and composition, which are formed from a variety of sources, including, but not limited to, diesel exhaust, forest fire smoke, airborne dusts, volcanic eruptions, glacial silt, and industrial emissions [2, 5]. The correlation between increased severity of asthma and allergic disease with living near high-traffic areas and exposure to diesel exhaust has been well studied and reported [24–28]. Forest fires are becoming more frequent and severe in northern latitudes at least in part related to warmer and drier conditions in these regions. Western United States and Alaska have experienced a significant increased incidence in large forest fires since the early 1980s with further increases projected, risking a profound change in the ecosystem [4]. Higher temperatures spur more thunderstorms and lightning, the most common trigger of forest fires. Drier conditions compromise containment and resultant extensive spread of fire, resulting in a further increase in airborne particulates [29]. Exposure to these elevated levels of particulates and ozone has been correlated with worsening respiratory disease as well as increased mortality [24].

Weather change and associated health impacts

As predicted by climate scientists, extreme weather events have increased and consequently have adversely impacted respiratory morbidity and mortality. Increased frequency and intensity of hurricanes and thunderstorms bring more rainwater and potential flooding [19]. Flooding promotes damp buildings and resultant indoor fungi growth [30•]. In the presence of rainwater from storms, pollen grains rupture releasing respirable aeroallergens [22, 31]. Thunderstorms have been shown to concentrate pollen grains at ground level, resulting in the release of allergenic particles of respirable size in the atmosphere as a result of pollen grain rupture by osmotic shock [32]. During the first 20–30 min of a thunderstorm, pollen-allergic individuals are at risk of exposure to high concentrations of the allergenic peptides and can potentially induce severe asthmatic exacerbations [32, 33].

Globally, we have experienced an average sea level rise of 7 to 8 in. since 1900, with almost half of that rise occurring since 1993 [4]. Although there is widespread variability in these data, recent estimates are that sea level rose to 1.8 mm per year for the past century and has increased between 2.8 and 3.1 mm annually over

the past decade [34]. Sea level rises are expected to result in an increase in indoor moisture in seaside communities, contributing to damp buildings, ultimately promoting mold growth and resultant increase in associated respiratory events [35]. Low-lying coastal areas and along the ocean fronts have seen population expansion over the past 50 years. Worldwide, it is estimated that up to 3.3 billion people live near coastal regions and are vulnerable to the potential increase risk of flooding [36]. A sizable percentage of the US population living in coastal and wetland areas are also at risk of damp buildings, posing an increased risk of indoor mold contamination and resultant exposure. The evidence that damp buildings and consequential mold growth are associated with respiratory disease is compelling [35, 37]. A quantitative meta-analysis of 33 epidemiologic studies revealed an increase of 30 to 50% in adverse respiratory health outcomes in occupants because of dampness and resultant mold exposure [38].

Heatwaves represent another health risk and are likely to increase in frequency, intensity, and duration as a consequence of climate change and world temperature rise [3, 39]. Mortality related to heatwaves is typically greatest in urban areas, largely due to the “heat island effect” [40]. The heat island effect is largely created by large, treeless swaths of asphalt and large heat-retaining structures that block cooling breezes. In a meta-analysis, heatwaves associated with increased mortality were observed, especially among more vulnerable populations, such as the elderly and those with health issues, such as ischemic heart disease, stroke, heart failure, and chronic obstructive pulmonary disease [41]. In a review of 33 relevant articles, Witt et al. [7] reported that death rates related to chronic lung disease during a heatwave were 1.8 to 8.2% higher, compared to average summer temperature. Heat stress causes fluid loss, disrupting pulmonary perfusion and in conjunction with increased concentrations of pollutants, and promotes bronchial inflammation and decreased threshold for bronchoconstriction [42, 43]. McCormack et al. [43] studied patients with moderate to severe COPD and found that increases in maximal indoor temperature correlated with worsening dyspnea, cough, and sputum production. Lin et al. [44] reported that the hospitalization rate for patients in New York with COPD increased 7.6% for every 1 °C increase in temperatures above 29 °C. Indoor temperature and indoor air pollution have also been associated with worsening respiratory symptoms [43]. With projections for worsening heatwaves in the future, it is likely that we will experience more heat-related worsening cardiorespiratory disease and death.

The impact of climate change on the sporulation of atmospheric fungi

Introduction

Fungi are ubiquitous microorganisms which have been estimated that around 1 to 1.5 million fungal species exist worldwide, but so far, only 80,000 have been described [45]. Their main mode of dispersal is to liberate sexually or asexually produced spores into the atmosphere. These spores can travel great distances, so they are important vectors by which fungi colonize new substrates. From these, 112 genera are thought to be a source of allergens. The 4 genera most commonly associated with the development of allergy are *Alternaria*, *Cladosporium*, *Penicillium*, and *Aspergillus* [46]. These genera belong to the phylum Ascomycota, but allergens have also been described from Basidiomycota and Zygomycota. Overall, 107 allergens from 28 fungal genera have been approved by the International Allergen Nomenclature Sub-committee (<http://www.allergen.org>).

These airborne spores have also been linked with human respiratory allergies [46, 47]. Atmospheric spore concentrations are influenced by a wide array of environmental, meteorological, and biological factors and various interspecies interactions. The number of spores in the atmosphere underlies seasonal variations. Especially climatic factors and circadian patterns influence the spectrum of fungal species and their concentrations in the environment [48, 49]. The number of spores in the air rises in particular in late summer and early autumn, while they diminish in winter. Considering daily fluctuations, the highest concentration of spore can be found in the afternoon and early evening [49].

A few spores of fungi are known to cause an allergic disease. *Alternaria alternata* and *Cladosporium herbarium* can cause allergenic asthma and rhinitis [48, 50]. As *Alternaria* is a saprophyte living on most vegetation, the volumes of dust generated by combine harvesters will include many *Alternaria* spores which will be dispersed over a wide area. The threshold level of spore necessary to elicit allergic symptoms in sensitized patients is not known and varies between different species. Spore concentrations of *Alternaria* equal or greater than 100 spores/m³ are believed to evoke allergic symptoms [48, 51], whereas the reference value for *Cladosporium* is estimated to be 3000 spores/m³ [52].

Sensitization to allergic fungi

The exact sensitization rate to fungi is not known but is estimated to range from 3 to 10% in the general population. The prevalence of sensitization in atopic individuals varies depending on many factors as it was exemplified in several studies [53–55]. A multicenter study in 7 European countries investigated the prevalence of *Alternaria* and *Cladosporium* sensitization in 877 children and adults with rhinitis and/or asthma. From these, approximately 9.5% were skin prick positive to at least one or both fungal species [56]. In another very recent multinational study, specific IgE antibodies to food and aeroallergens were determined in 2184 with atopic eczema. The overall sensitization rate to

Alternaria in this population was 3.7%, but variations were observed between the different countries [57]. The third National Health and Nutrition Examination Survey in the USA revealed that approximately 12.9% of the population showed positive skin prick results in *Alternaria alternata* [58]. A survey performed by Global Allergy and Asthma European Network in 16 European countries showed general sensitization rates of 11.9% for *Alternaria alternata* and 5.8% for *Cladosporium herbarum* with the highest prevalence in the UK, Ireland, and Northern Europe. Based on studies that consider the age of patients in the evaluation of prevalence data, it was assumed that sensitization to fungi is an age-dependent process with a higher prevalence in children. *Alternaria*-specific IgE antibodies increase significantly in early childhood until they reach a maximum level and then seem to decline again with increasing age [57–60]. Fewer people are allergic to fungi but spores have been implicated as a major risk factor for fatal asthma attacks in Chicago. These people react immediately on exposure in the upper airways with symptoms like allergic rhinitis or asthma [61]. The risk of asthma-related deaths is more than doubled when there is a high incidence of fungi spores [61] and *Alternaria* is a known risk factor of asthma in childhood and young adults [62]. Atmospheric *Alternaria* was the commonest fungal spore to cause seasonal allergy in the late summer and early autumn; only very small numbers of this spore penetrate from the outside to the inside [63].

Increased CO₂ and fungal allergy

A number of studies also indicate that fungi exposure is affected by climate change. Their community-level responses to elevated CO₂, one of the major global climate change factors impacting ecosystems, are not well understood. In terms of pollen, climate change is expected to result in earlier pollination with longer seasonal exposure [16], and higher pollen loads and changes in pollen allergenicity to elevated CO₂ [64, 65]. Such shifts may lead to critical changes in the incidence, severity, and distribution of human health effects, including allergic diseases. Wolf et al. have shown experimentally that *Alternaria*, when grown on leaves of timothy grass plants grown at potential future elevated CO₂ concentrations, produces nearly three times the number of spores and more than twice the total antigenic protein per plant than at lower CO₂ concentrations [66]. As with pollen production, the sporulation of allergenic fungi is likely to be amplified as atmospheric CO₂ concentration increases and is also likely to contribute to the increasing prevalence and severity of asthma and allergies.

Large increases in atmospheric CO₂ concentration have been shown to alter both plant biomass and chemistry in many plant species. These two physiological changes have the potential to alter phloem function [67, 68]. The responses of individual plant species to large increases in atmospheric CO₂ concentration are variable but often include greater total biomass production and greater carbon-to-nitrogen ratios (C:N) of plant tissues [69]. In this study, there were significant positive relationships between leaf C:N and *Alternaria alternata* and *Cladosporium phlei* spore production per gram leaf, although only *Alternaria alternata* sporulation was associated with CO₂ concentration after accounting for changes in leaf C:N. These relationships suggest that considerable increases in sporulation of both species will occur and may already be occurring, if rising

atmospheric CO₂ concentration increases plant C:N in the field. Eventually allergenic fungi that grow on living or dead plant materials are likely to encounter changes in substrate quality or quantity as global CO₂ concentration increases [70].

Climate change and the altered sporulation of allergic fungi

Fungal communities play a major role as decomposers in the earth's ecosystems. Soils in terrestrial systems host biogeochemical transformations of important greenhouse gases. Soils might be drier with global warming and increase evaporative water loss. The harmful impacts of acid rain on natural ecosystems include water contamination, soil acidification, and death to many organisms in the last decades. The major sources of acid rain are sulfur dioxide and nitrogen oxides emitted into the atmosphere [71].

Aerobiological monitoring of atmospheric fungal spores has been taking place in 4 cities (Seoul, Guri, Busan, and Jeju) of South Korea since 1997. This study focused on the evaluation of change of allergenic fungal species and their concentration compared with the allergenic pollens in the same period (Fig. 1). There have been no long-term detailed studies of concentration and seasonal variation of allergic fungi spores in the outdoor. It has become apparent that spore concentration of *Alternaria* and *Cladosporium* has decreased and the other fungi have disappeared for the last 22 years (Figs. 2 and 3). The trend of weather has been changed to a dry moderate weather zone in South Korea during the last 50 years (Fig. 4). It may suggest that the weather change like atmospheric dryness and warming evaporated soils with silent droughts. This condition decreased the concentration of fungi and even disappeared some certain fungi in South Korea (submitted to Allergy, 2020).

Climate change forecast widespread changes in precipitation regimes, including longer, more intense droughts, causing desertification and promoting the expansion of drylands globally [72••]. Drought had a stronger effect on fungal community composition and induced greater functional loss. The magnitude of the fungal community change was directly proportional to the

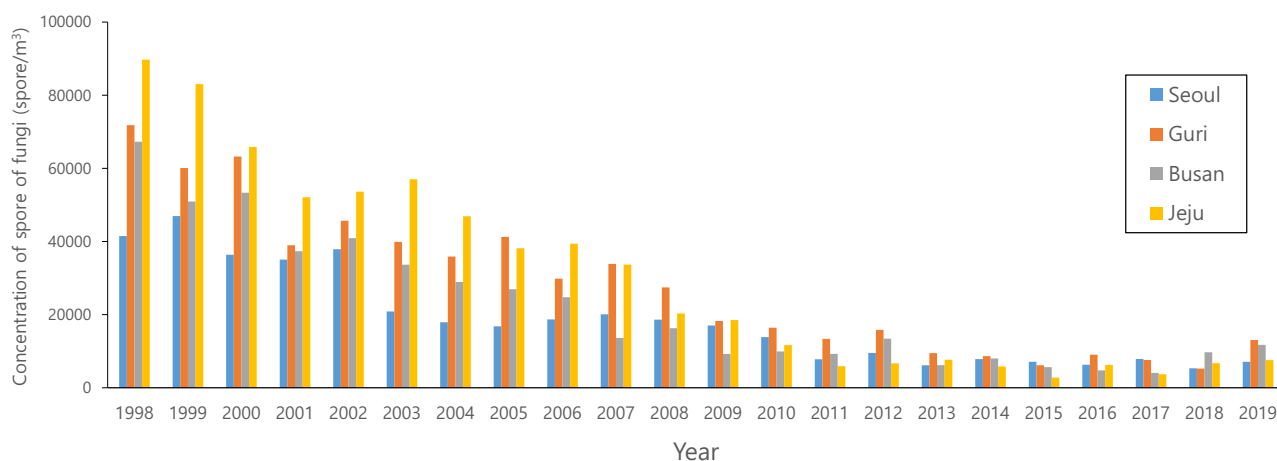


Fig. 1. Annual change of total spore concentration of fungi in Seoul, Guri, Busan, and Jeju of South Korea for the last 22 years. There is shown to decrease them in major 4 cities in Korea annually since 1998 (submitted to Allergy Asthma Immunology Research).

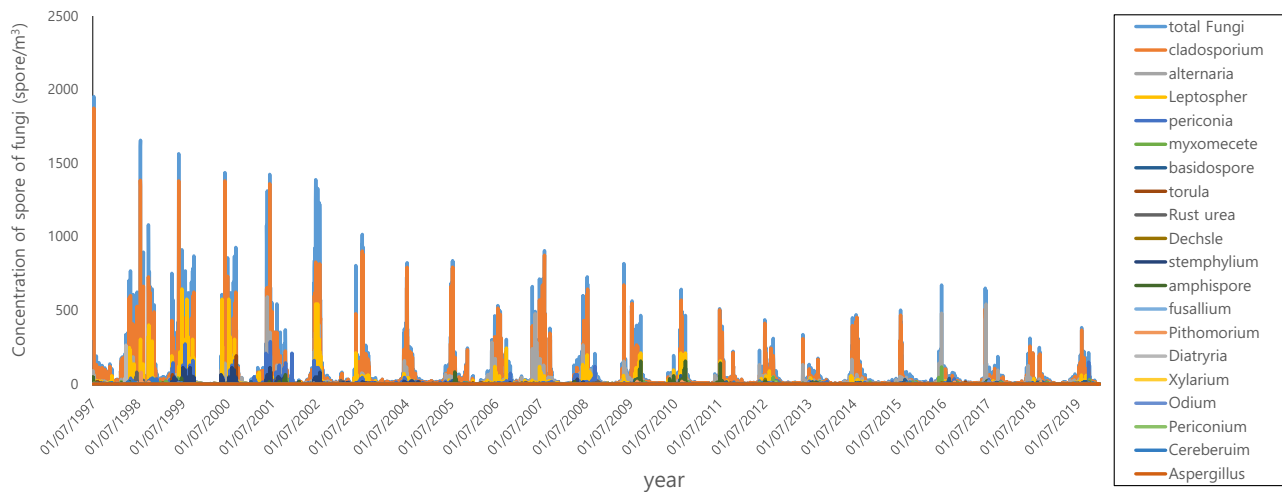


Fig. 2. Annual change of spore concentration of each fungus in Seoul for the last 22 years. It has become apparent that spores of fungi have decreased or dropped away annually since 1998 (submitted to Allergy Asthma Immunology Research).

precipitation gradient [73]. An experiment demonstrated how the exclusion of aerially dispersed fungi and bacteria altered the compositional and functional response of soil microbial and fungal communities to drought. The study presented that aerial dispersal rate alters soil microbial and fungal responses



Fig. 3. Annual concentration of *Alternaria* (a) and *Cladosporium* (b) in 4 cities of South Korea for the last 22 years. It has become apparent that the spore concentration of *Alternaria* and *Cladosporium* has decreased since 1998 (submitted to Allergy Asthma Immunology Research).

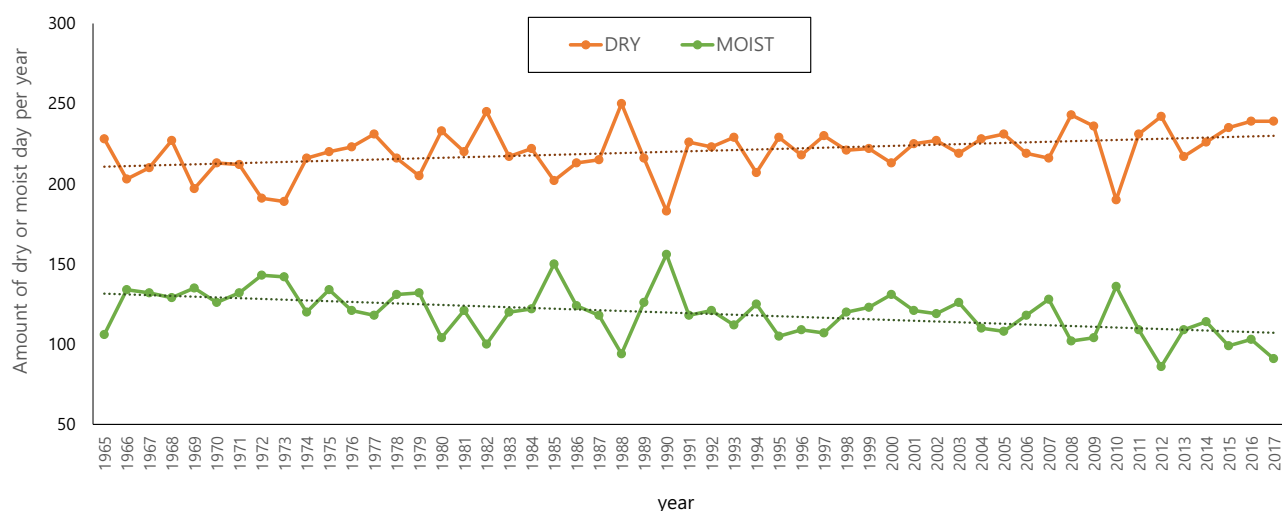


Fig. 4. Weather-type trend of Seoul since 1965. The trend of weather has been changed to a dry moderate weather zone in South Korea during the last 50 years (submitted to Allergy Asthma Immunology Research).

to disturbance. Changes in dispersal rates should be considered when predicting microbial and fungal responses to climate change [74•].

Conclusion

Coupled with air pollution and increases in exposure to aeroallergens, climate change is projected to have potentially serious adverse consequences for human health. Allergenic and immune responses to a variety of environmental factors, such as pollens, fungi, and pollutants, have been clearly demonstrated to be associated with an increased burden of upper and lower respiratory diseases [36]. The U.S. Global Change Research Program, Fourth National Climate Assessment reports that it is extremely likely that human activities, especially emissions of greenhouse gases, are the dominant cause of the observed warming since the mid-twentieth century. There are no convincing alternative explanations supported by observational evidence [31]. Asthma is a significant health and economic burden to patients, their families, and society [75]. Though many factors have contributed to the increase in the prevalence of atopic diseases, observed changes in environmental factors, many related to the changing climate, are an important contributor.

Otherwise, the sporulation of allergenic fungi is likely to be amplified as atmospheric CO₂ concentration increases with climate change, and is also likely to contribute to the increasing prevalence and severity of asthma and other respiratory allergies. Furthermore, global warming to drought and atmospheric dryness significantly altered the community composition and biochemical transformation of atmospheric allergic fungi [73].

Recognition of environmental changes and their impact on respiratory allergy is paramount, as is working to lessen the impact on patients and adapting practices to meet those needs.

Compliance with Ethical Standards

Conflict of Interest

Jeffrey Demain reports financial activity outside the submitted work, including serving as a contributor for UpToDate and as an editor for Ask the Expert (AAAAI). Young-Jin Choi declares no conflict of interest. Jae-Won Oh declares no conflict of interest.

Human and Animal Rights and Informed Consent

This article does not contain any studies with human or animal subjects performed by any of the authors.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

1. Beggs PJ. Adaptation to impacts of climate change on aeroallergens and allergic respiratory diseases. *Int J Environ Res Public Health*. 2010;7(8):3006–21. <https://doi.org/10.3390/ijerph7083006>.
 - 2.•• Watts N, Adger WN, Agnolucci P, Blackstock J, Byass P, Cai W, et al. Health and climate change: policy responses to protect public health. *Lancet*. 2015;386(10006):1861–914. [https://doi.org/10.1016/s0140-6736\(15\)60854-6](https://doi.org/10.1016/s0140-6736(15)60854-6)
- The 2015 Lancet Commission on Health and Climate Change discusses the impacts of climate change and proposes policy addressing health of worldwide population. This is a very comprehensive review that summarizes the global impact of climate change and consequential potential health effects.
3. Stocker TF, Qin D, Plattner GK, Alexander LV, Allen SK, Bindoff NL, et al. Technical summary. In: Stocker TF, Qin D, Plattner GK, Tignor M, Allen SK, Boschung J, et al., editors. *Climate change 2013: the physical science basis*. Contribution of Working Group I to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge, United Kingdom and New York, NY, USA: Cambridge University Press; 2013.
 4. U.S. Global Change Research Program. *Climate Science Special Report: Fourth National Climate Assessment, Volume I*. In: Wuebbles DJ, Fahey DW, Hibbard KA, Dokken DJ, Stewart BC, Maycock TK, editors. . Washington, DC; 2017. p. 470. <https://doi.org/10.7930/J0J964J6>.
 5. Patz JA, Frumkin H, Holloway T, Vimont DJ, Haines A. Climate change: challenges and opportunities for global health. *Jama*. 2014;312(15):1565–80. <https://doi.org/10.1001/jama.2014.13186>.
 - 6.• 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(5):434–40. <https://doi.org/10.1097/ac.0000000000000301>
- An important paper addressing weather and climate change impacts on respiratory health.
7. Witt C, Schubert AJ, Jehn M, Holzgreve A, Liebers U, Endlicher W, et al. The effects of climate change on patients with chronic lung disease. A systematic literature review. *Dtsch Arztebl Int*. 2015;112(51–52):878–83. <https://doi.org/10.3238/arztebl.2015.0878>.
 - 8.•• Ziska LH, Gebhard DE, Frenz DA, Faulkner S, Singer BD, Straka JG. Cities as harbingers of climate change: common ragweed, urbanization, and public health. *J Allergy Clin Immunol*. 2003;111(2):290–5. <https://doi.org/10.1067/mai.2003.53>
- While dated, this remains one of the best studies addressing the difference of ragweed biomass levels between urban and rural Baltimore. This further elaborates the impact of CO₂ and heat on ragweed pollination.
9. Ziska LH, Bunce JA, Goins EW. Characterization of an urban-rural CO₂/temperature gradient and associated changes in initial plant productivity during secondary succession. *Oecologia*. 2004;139(3):454–8. <https://doi.org/10.1007/s00442-004-1526-2>.
 10. 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(3):279–82. [https://doi.org/10.1016/s1081-1206\(10\)62009-1](https://doi.org/10.1016/s1081-1206(10)62009-1).
 11. Singer BD, Ziska LH, Frenz DA, Gebhard DE, Straka JG. Increasing Amb a 1 content in common ragweed (*Ambrosia artemisiifolia*) pollen as a function of rising atmospheric CO₂ concentration. *Funct Plant Biol*. 2005;32(7):667–70.
 12. Rogers CA, Wayne PM, Macklin EA, Muilenberg ML, Wagner CJ, Epstein PR, et al. Interaction of the onset of spring and elevated atmospheric CO₂ on ragweed (*Ambrosia artemisiifolia* L.) pollen production. *Environ Health Perspect*. 2006;114(6):865–9. <https://doi.org/10.1289/ehp.8549>.

13. Ziska L, Knowlton K, Rogers C, Dalan D, Tierney N, Elder MA, et al. Recent warming by latitude associated with increased length of ragweed pollen season in central North America. *Proc Natl Acad Sci U S A*. 2011;108(10):4248–51. <https://doi.org/10.1073/pnas.1014107108>.
 14. Ahlholm JU, Helander ML, Savolainen J. Genetic and environmental factors affecting the allergenicity of birch (*Betula pubescens* ssp. *czerepanovii* [Orl.] Hamet-ahiti) pollen. *Clin Exp Allergy*. 1998;28(11):1384–8. <https://doi.org/10.1046/j.1365-2222.1998.00404.x>.
 15. Ariano R, Canonica GW, Passalacqua G. Possible role of climate changes in variations in pollen seasons and allergic sensitizations during 27 years. *Ann Allergy Asthma Immunol*. 2010;104(3):215–22. <https://doi.org/10.1016/j.anai.2009.12.005>
- A 27-year study monitoring variables, such as temperature, prolongation of seasons, timing of frost, and association of start, duration, and level of pollination. All the variables are applied to observed increased incidence of developing pollen allergies compared to no change in perennial allergies. This represents a well-done observational study in a real-world setting.
16. Ziska LH, Makra L, Harry SK, Bruffaerts N, Hendrickx M, Coates F, et al. Temperature-related changes in airborne allergenic pollen abundance and seasonality across the northern hemisphere: a retrospective data analysis. *Lancet Planet Health*. 2019;3(3):e124–e31. [https://doi.org/10.1016/s2542-5196\(19\)30015-4](https://doi.org/10.1016/s2542-5196(19)30015-4)
- This study presented showed a significant increase in pollen season duration over time, increasing, on average, 0.9 days per year. The ongoing increase in temperature extremes might already be contributing to extended seasonal duration and increased pollen load for multiple aeroallergenic pollen taxa in diverse locations across the northern hemisphere.
17. Malayeri BE, Noori M, Jafari M. Using the pollen viability and morphology for fluoride pollution bio-monitoring. *Biol Trace Elem Res*. 2012;147(1–3):315–9. <https://doi.org/10.1007/s12011-011-9290-8>.
 18. Schiavoni G, D'Amato G, Afferni C. The dangerous liaison between pollens and pollution in respiratory allergy. *Ann Allergy Asthma Immunol*. 2017;118(3):269–75. <https://doi.org/10.1016/j.anai.2016.12.019>.
 19. D'Amato G, Holgate ST, Pawankar R, Ledford DK, Cecchi L, Al-Ahmad M, 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(1):25. <https://doi.org/10.1186/s40413-015-0073-0>
- This is a statement of the World Allergy Organization (WAO) which raises the importance of climate-related health impacts. The health impacts are illuminated, including frequency of acute cardiorespiratory events due to higher concentrations of ground-level ozone; changes in the frequency of respiratory diseases due to particulate pollution; allergic impacts of altered distribution of pollen and mold allergens; and some infectious disease vectors.
20. U.S. Environmental Protection Agency. Ground-level ozone basics. In: Ground-level ozone pollution. Washington DC: U.S. Environmental Protection Agency. 2017. <https://www.epa.gov/ground-level-ozone-pollution/ground-level-ozone-basics/>. Accessed May 8, 2020.
 21. World Health Organization. Occupational and Environmental Health Team. (2006). WHO air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide: global update 2005: summary of risk assessment. World Health Organization. <https://apps.who.int/iris/handle/10665/69477/>. Accessed May 8, 2020.
 22. D'Amato G, Vitale C, De Martino A, Viegi G, Lanza M, Molino A, et al. Effects on asthma and respiratory allergy of climate change and air pollution. *Multidiscip Respir Med*. 2015;10:39. <https://doi.org/10.1186/s40248-015-0036-x>.
 23. Beck I, Jochner S, Gilles S, McIntyre M, Buters JT, Schmidt-Weber C, et al. High environmental ozone levels lead to enhanced allergenicity of birch pollen. *PLoS One*. 2013;8(11):e80147. <https://doi.org/10.1371/journal.pone.0080147>.
 24. Jerrett M, Burnett RT, Pope CA 3rd, Ito K, Thurston G, Krewski D, et al. Long-term ozone exposure and mortality. *N Engl J Med*. 2009;360(11):1085–95. <https://doi.org/10.1056/NEJMoa0803894>.
 25. McGeachie MJ, Yates KP, Zhou X, Guo F, Sternberg AL, Van Natta ML, et al. Patterns of growth and decline in lung function in persistent childhood asthma. *N Engl J Med*. 2016;374(19):1842–52. <https://doi.org/10.1056/NEJMoa1513737>.
 26. McConnell R, Berhane K, Yao L, Jerrett M, Lurmann F, Gilliland F, et al. Traffic, susceptibility, and childhood asthma. *Environ Health Perspect*. 2006;114(5):766–72. <https://doi.org/10.1289/ehp.8594>.
 27. Gordian ME, Haneuse S, Wakefield J. An investigation of the association between traffic exposure and the diagnosis of asthma in children. *J Expo Sci Environ Epidemiol*. 2006;16(1):49–55. <https://doi.org/10.1038/sj.jea.7500436>.
 28. Jerrett M, Finkelstein MM, Brook JR, Arain MA, Kanaroglou P, Stieb DM, et al. A cohort study of traffic-related air pollution and mortality in Toronto, Ontario, Canada. *Environ Health Perspect*. 2009;117(5):772–7. <https://doi.org/10.1289/ehp.11533>.
 29. NASA Global Climate Change. Lightning sparking more boreal forest fires. 2017. <https://climate.nasa.gov/news/2602/lightning-sparking-more-boreal-forest-fires/>. Accessed May 8, 2020.
 30. Poole JA, Barnes CS, Demain JG, Bernstein JA, Padukudru MA, Sheehan WJ, 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(5):1702–10. <https://doi.org/10.1016/j.jaci.2019.04.010>.

2019.02.018

AAAAI Work Group report addressing many factors associated with climate change, pollution, and weather. Addresses clinical impacts with focus on respiratory impacts.

31. U.S. Global Change Research Program, 2016. The impacts of climate change on human health in the United States: a scientific assessment. Crimmins A, Balbus J, Gamble JL, Beard CB, Bell JE, Dodgen D et al., editors. U.S. Global Change Research Program, Washington, DC. https://health2016.globalchange.gov/low/ClimateHealth2016_FullReport_small.pdf/. Accessed May 8, 2020.
 32. D'Amato G, Annesi-Maesano I, Vaghi A, Cecchi L, D'Amato M. How do storms affect asthma? *Curr Allergy Asthma Rep.* 2018;18(4):24. <https://doi.org/10.1007/s11882-018-0775-9>.
 33. D'Amato G, Vitale C, D'Amato M, Cecchi L, Liccardi G, Molino A, et al. Thunderstorm-related asthma: what happens and why. *Clin Exp Allergy.* 2016;46(3):390–6. <https://doi.org/10.1111/cea.12709>.
 34. Bindoff NL, Willebrand J, Artale V, Carillo A, Gregory J, Gulev S et al. Observations: oceanic climate change and sea level. 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*. In: Solomon S, Qin D, Manning M, Chen Z, Marquis M, Averyt KB et al., editors. Cambridge University Press, Cambridge, United Kingdom; New York, NY, USA. 2007. <https://www.ipcc.ch/site/assets/uploads/2018/02/ar4-wg1-chapter5-1.pdf>. Accessed May.
 35. Institute of Medicine (US) Committee on the Assessment of Asthma and Indoor Air. *Clearing the air: asthma and indoor air exposures*. Washington, DC: National Academies Press; 2000; <https://www.ncbi.nlm.nih.gov/pubmed/25077220>.
 36. Barnes CS, Alexis NE, Bernstein JA, Cohn JR, Demain JG, Levetin E. Reply: To PMID 23687635. *J Allergy Clin Immunol Pract.* 2013;1(5):543–4. <https://doi.org/10.1016/j.jaip.2013.05.015>.
 37. Knutsen AP, Bush RK, Demain JG, Denning DW, Dixit A, Fairs A, et al. Fungi and allergic lower respiratory tract diseases. *J Allergy Clin Immunol.* 2012;129(2):280–91; quiz 92–3. <https://doi.org/10.1016/j.jaci.2011.12.970>.
 38. Fisk WJ, Lei-Gomez Q, Mendell MJ. Meta-analyses of the associations of respiratory health effects with dampness and mold in homes. *Indoor Air.* 2007;17(4):284–96. <https://doi.org/10.1111/j.1600-0668.2007.00475.x>.
 39. Guo Y, Gasparrini A, Li S, Sera F, Vicedo-Cabrera AM, de Sousa Zanotti Stagliorio Coelho M, et al. Quantifying excess deaths related to heatwaves under climate change scenarios: a multicountry time series modelling study. *PLoS Med.* 2018;15(7):e1002629. <https://doi.org/10.1371/journal.pmed.1002629>.
 40. McMichael AJ. The urban environment and health in a world of increasing globalization: issues for developing countries. *Bull World Health Organ.* 2000;78(9):1117–26.
 41. Cheng J, Xu Z, Bambrick H, Prescott V, Wang N, Zhang Y, et al. Cardiorespiratory effects of heatwaves: a systematic review and meta-analysis of global epidemiological evidence. *Environ Res.* 2019;177:108610. <https://doi.org/10.1016/j.envres.2019.108610>.
 42. Hansel NN, McCormack MC, Kim V. The effects of air pollution and temperature on COPD. *Copd.* 2016;13(3):372–9. <https://doi.org/10.3109/15412555.2015.1089846>.
 43. McCormack MC, Belli AJ, Waugh D, Matsui EC, Peng RD, Williams DL, et al. Respiratory effects of indoor heat and the interaction with air pollution in chronic obstructive pulmonary disease. *Ann Am Thorac Soc.* 2016;13(12):2125–31. <https://doi.org/10.1513/AnnalsATS.201605-329OC>.
 44. Lin S, Luo M, Walker RJ, Liu X, Hwang SA, Chinery R. Extreme high temperatures and hospital admissions for respiratory and cardiovascular diseases. *Epidemiology.* 2009;20(5):738–46. <https://doi.org/10.1097/EDE.0b013e3181ad5522>.
 45. Prillinger H, Lopandic K, Schweigkofler W, Deak R, Aarts HJ, Bauer R, et al. Phylogeny and systematics of the fungi with special reference to the Ascomycota and Basidiomycota. *Chem Immunol.* 2002;81:207–95. <https://doi.org/10.1159/000058868>.
 46. Delfino RJ, Zeiger RS, Seltzer JM, Street DH, Matteucci RM, Anderson PR, et al. The effect of outdoor fungal spore concentrations on daily asthma severity. *Environ Health Perspect.* 1997;105(6):622–35. <https://doi.org/10.1289/ehp.97105622>.
 47. Barnes C. Fungi and atopy. *Clin Rev Allergy Immunol.* 2019;57(3):439–48. <https://doi.org/10.1007/s12016-019-08750-z>.
 48. D'Amato G, Spiekma FT. Aerobiologic and clinical aspects of mould allergy in Europe. *Allergy.* 1995;50(11):870–7. <https://doi.org/10.1111/j.1398-9995.1995.tb02492.x>
- This article is a position paper in European Academy of Allergy and Clinical Immunology for mold allergy. Fungal spores such as *Alternaria* can be carried deeper into the respiratory tract and to induced asthma.
49. Rodriguez-Rajo FJ, Iglesias I, Jato V. Variation assessment of airborne *Alternaria* and *Cladosporium* spores at different bioclimatical conditions. *Mycol Res.* 2005;109(Pt 4):497–507. <https://doi.org/10.1017/s0953756204001777>.
 50. Caillaud D, Cheriaux M, Charpin D, Chaabane N, Thibaudon M. Outdoor moulds and respiratory health. *Rev Mal Respir.* 2018;35(2):188–96. <https://doi.org/10.1016/j.rmr.2018.01.001>.
 51. Bernardis P, Agnoletto M, Puccinelli P, Parmiani S, Pozzan M. Injective versus sublingual immunotherapy in *Alternaria tenuis* allergic patients. *J Invest Allergol Clin Immunol.* 1996;6(1):55–62.
 52. Darke CS, Knowelden J, Lacey J, Milford WA. Respiratory disease of workers harvesting grain. *Thorax.*

- 1976;31(3):294–302. <https://doi.org/10.1136/thx.31.3.294>.
53. Bousquet J, Heinzerling L, Bachert C, Papadopoulos NG, Bousquet PJ, Burney PG, et al. Practical guide to skin prick tests in allergy to aeroallergens. *Allergy*. 2012;67(1):18–24. <https://doi.org/10.1111/j.1398-9995.2011.02728.x>.
 54. Lyons TW, Wakefield DB, Cloutier MM. Mold and *Alternaria* skin test reactivity and asthma in children in Connecticut. *Ann Allergy Asthma Immunol*. 2011;106(4):301–7. <https://doi.org/10.1016/j.anai.2010.12.009>.
 55. Park HJ, Lee JH, Park KH, Ann HW, Jin MN, Choi SY, et al. A nationwide survey of inhalant allergens sensitization and levels of indoor major allergens in Korea. *Allergy, Asthma Immunol Res*. 2014;6(3):222–7. <https://doi.org/10.4168/aair.2014.6.3.222>.
 56. D'Amato G, Chatzigeorgiou G, Corsico R, Gioulekas D, Jager L, Jager S, et al. Evaluation of the prevalence of skin prick test positivity to *Alternaria* and *Cladosporium* in patients with suspected respiratory allergy. A European multicenter study promoted by the Subcommittee on Aerobiology and Environmental Aspects of Inhalant Allergens of the European Academy of Allergy and Clinical Immunology. *Allergy*. 1997;52(7):711–6. <https://doi.org/10.1111/j.1398-9995.1997.tb01227.x>.
 57. de Benedictis FM, Franceschini F, Hill D, Naspitiz C, Simons FE, Wahn U, et al. The allergic sensitization in infants with atopic eczema from different countries. *Allergy*. 2009;64(2):295–303. <https://doi.org/10.1111/j.1398-9995.2008.01779.x>.
 58. Arbes SJ Jr, Gergen PJ, Elliott L, Zeldin DC. Prevalences of positive skin test responses to 10 common allergens in the US population: results from the third National Health and Nutrition Examination Survey. *J Allergy Clin Immunol*. 2005;116(2):377–83. <https://doi.org/10.1016/j.jaci.2005.05.017>.
 59. Mari A, Schneider P, Wally V, Breitenbach M, Simon-Nobbe B. Sensitization to fungi: epidemiology, comparative skin tests, and IgE reactivity of fungal extracts. *Clin Exp Allergy*. 2003;33(10):1429–38. <https://doi.org/10.1046/j.1365-2222.2003.01783.x>.
 60. Heinzerling L, Frew AJ, Bindslev-Jensen C, Bonini S, Bousquet J, Bresciani M, et al. Standard skin prick testing and sensitization to inhalant allergens across Europe—a survey from the GALEN network. *Allergy*. 2005;60(10):1287–300. <https://doi.org/10.1111/j.1398-9995.2005.00895.x>.
 61. Targonski PV, Persky VW, Kelleher P, Addington W. Characteristics of hospitalization for asthma among persons less than 35 years of age in Chicago. *J Asthma*. 1995;32(5):365–72. <https://doi.org/10.3109/02770909509082761>.
 62. Tariq SM, Matthews SM, Stevens M, Hakim EA. Sensitization to *Alternaria* and *Cladosporium* by the age of 4 years. *Clin Exp Allergy*. 1996;26(7):794–8.
 63. Halonen M, Stern DA, Wright AL, Taussig LM, Martinez FD. *Alternaria* as a major allergen for asthma in children raised in a desert environment. *Am J Respir Crit Care Med*. 1997;155(4):1356–61. <https://doi.org/10.1164/ajrccm.155.4.9105079>.
 64. Kim KR, Oh JW, Woo SY, Seo YA, Choi YJ, Kim HS, et al. Does the increase in ambient CO₂ concentration elevate allergy risks posed by oak pollen? *Int J Biometeorol*. 2018;62(9):1587–94. <https://doi.org/10.1007/s00484-018-1558-7>
- This study is first report the impact of increased CO₂ concentration on the exposure risk of allergenic oak pollen is quantified.
65. Choi YJ, Oh HR, Oh JW, Kim KR, Kim MJ, Kim BJ, et al. Chamber and field studies demonstrate differential Amb a 1 contents in common ragweed depending on CO₂ levels. *Allergy, Asthma Immunol Res*. 2018;10(3):278–82. <https://doi.org/10.4168/aair.2018.10.3.278>.
 66. Wolf J, O'Neill NR, Rogers CA, Muilenberg ML, Ziska LH. Elevated atmospheric carbon dioxide concentrations amplify *Alternaria alternata* sporulation and total antigen production. *Environ Health Perspect*. 2010;118(9):1223–8. <https://doi.org/10.1289/ehp.0901867>.
 67. Ainsworth EA, Long SP. What have we learned from 15 years of free-air CO₂ enrichment (FACE)? A meta-analytic review of the responses of photosynthesis, canopy properties and plant production to rising CO₂. *New Phytol*. 2005;165(2):351–71. <https://doi.org/10.1111/j.1469-8137.2004.01224.x>
- This review may provide the most plausible estimates of how plants in their native environments and field-grown crops will respond to rising atmospheric [CO₂].
68. Ainsworth EA, Lemonnier P. Phloem function: a key to understanding and manipulating plant responses to rising atmospheric [CO₂]? *Curr Opin Plant Biol*. 2018;43:50–6. <https://doi.org/10.1016/j.pbi.2017.12.003>.
 69. Taub DR, Wang X. Why are nitrogen concentrations in plant tissues lower under elevated CO₂? A critical examination of the hypotheses. *J Integr Plant Biol*. 2008;50(11):1365–74. <https://doi.org/10.1111/j.1744-7909.2008.00754.x>.
 70. Tu Q, Yuan M, He Z, Deng Y, Xue K, Wu L, et al. Fungal communities respond to long-term CO₂ elevation by community reassembly. *Appl Environ Microbiol*. 2015;81(7):2445–54. <https://doi.org/10.1128/aem.04040-14>.
 71. Dai Z, Liu X, Wu J, Xu J. Impacts of simulated acid rain on recalcitrance of two different soils. *Environ Sci Pollut Res Int*. 2013;20(6):4216–24. <https://doi.org/10.1007/s11356-012-1288-z>.
 72. Schwalm CR, Anderegg WRL, Michalak AM, Fisher JB, Biondi F, Koch G, et al. Global patterns of drought recovery. *Nature*. 2017;548(7666):202–5. <https://doi.org/10.1038/nature23021>
- This article provides two key insights into the spatiotemporal patterns of drought recovery time: first, that recovery is longest in the tropics and high northern latitudes (both vulnerable areas of earth's climate system) and second, that drought

impacts have increased over the twentieth century. If droughts become more frequent, the time between droughts may become shorter than drought recovery time, leading to permanently damaged ecosystems and widespread degradation of the land carbon sink.

73. Ochoa-Hueso R, Collins SL, Delgado-Baquerizo M, Hamonts K, Pockman WT, Sinsabaugh RL, et al. Drought consistently alters the composition of soil fungal and bacterial communities in grasslands from two continents. *Glob Chang Biol.* 2018;24(7):2818–27. <https://doi.org/10.1111/gcb.14113>.
74. • Evans SE, Bell-Dereske LP, Dougherty KM, Kittredge HA. Dispersal alters soil microbial community response to drought. *Environ Microbiol.* 2020;22(3):905–16. <https://doi.org/10.1111/1462-2920.14707>

This study presented that aerial dispersal rate alters soil

microbial and fungal responses to disturbance. Changes in dispersal rates should be considered when predicting microbial and fungal responses to climate change.

75. Sheehan WJ, Gaffin JM, Peden DB, Bush RK, Phipatanakul W. Advances in environmental and occupational disorders in 2016. *J Allergy Clin Immunol.* 2017;140(6):1683–92. <https://doi.org/10.1016/j.jaci.2017.09.032>.

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