



Climate Change and the Impact on Respiratory and Allergic Disease: 2018

Jeffrey G. Demain^{1,2,3}

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Abstract

Purpose of Review The purpose of this paper is to review allergic respiratory disease related to indoor and outdoor exposures and to examine the impact of known and projected changes in climate. The global burden of disease directly attributed to climate change is very difficult to measure and becomes more challenging when the capacity of humans to adapt to these changes is taken into consideration. Allergic respiratory disease, such as asthma, is quite heterogenous, though closely associated with environmental and consequently immunologic interaction. Where is the tipping point?

Recent Findings Our climate has been measurably changing for the past 100 years. It may indeed be the most significant health threat of the twenty-first century, and consequently tackling climate change may be the greatest health opportunity. The impacts of climate change on human health are varied and coming more into focus. Direct effects, such as heatwaves, severe weather, drought, and flooding, are apparent and frequently in the news. Indirect or secondary effects, such as changes in ecosystems and the impact on health, are less obvious. It is these changes in ecosystems that may have the greatest impact on allergic and respiratory diseases.

Summary This review will explore some ways that climate change, current and predicted, influences respiratory disease. Discussion will focus on changing pollen patterns, damp buildings with increased mold exposure, air pollution, and heat stress.

Keywords Climate change · Global warming · Pollination · Pollen · Damp buildings · Mold · Allergy · Asthma · Respiratory disease · Heat stress

Introduction

Climate change may indeed be the most significant health threat of the twenty-first century, and consequently tackling climate change may be the greatest health opportunity [1•]. Global mean surface air temperatures over land and oceans have increased over the last century, reflected by warming oceans, rising sea level, melting glaciers, and shrinking sea

ice [2•]. Global annually averaged surface air temperature has increased by about 1.8 °F (1.0 °C) over the last 115 years (1901–2016) with annual average near-surface air temperatures across Alaska and the Arctic increasing more than twice as fast as the global average temperature [3•]. Globally, this period is now the warmest in the history of modern civilization. The last few years have also seen record-breaking, climate-related weather extremes, and the last 3 years have been the warmest years on record for the globe. These trends are expected to continue over climate timescales [3•]. There are many indicators of climate change, particularly physical responses such as changes in surface temperature, atmospheric water vapor, precipitation, severe weather events, retreat of glaciers, sea level rise, and changes in sea and land ice [2•].

Over the past 20 years, evidence has supported an association between climate change and adverse health outcomes. Changing climate variables and both the potential and realized impacts on health are coming more clearly into focus. Health outcomes associated with heat stress, flooding, crop failure, vector-borne, and waterborne disease have been well reported

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✉ Jeffrey G. Demain
jdemain@allergyalaska.com

¹ Allergy, Asthma and Immunology Center of Alaska, 3841 Piper Street, Suite T4-054, Anchorage, AK 99508, USA

² Department of Pediatrics, University of Washington, 3841 Piper Street, Suite T4-054, Anchorage, AK 99508, USA

³ WWAMI School of Medical Education, University of Alaska, 3841 Piper Street, Suite T4-054, Anchorage, AK 99508, USA

[3••, 4••]. Allergic respiratory disease, such as asthma, is quite heterogenous, though closely associated with environmental and consequently immunologic interaction [5•]. Some of the variables associated with climate that impact respiratory disease include changing pollen patterns, damp buildings with increased mold exposure, and climate impacts on air pollution. Finally, heat stress, especially in combination with pollution, promotes inflammation and lowers the airway hyperreactivity threshold and has been linked with increased mortality rate [6].

Changing Patterns of Pollination

Exposure to outdoor allergens has long been associated with exacerbations of allergic upper and lower respiratory disease. Carbon dioxide (CO₂) is the leading greenhouse gas implicated in global warming. Atmospheric CO₂ levels have topped 400 ppm, up from 280 ppm pre-industrial revolution [2••, 3••].

There is compelling evidence that higher ambient carbon dioxide concentrations and warmer temperatures will result in increased pollen production especially in the urban areas [4••, 6]. Increases in pollen levels associated with increased CO₂ levels have been demonstrated in environmental chambers with controlled variables, as well as in open-field studies [7–10]. Evidence is compelling that when CO₂ levels double, individual ragweed plant pollen production increases 30 to 90% [7–12]. Additionally, ragweed flowers earlier, producing more pollen in urban locations where CO₂ concentrations and temperatures are higher [8].

Pollen seasons have lengthened in North America, correlating with increased temperatures and lengthening frost-free periods [13•]. Elevated CO₂ levels appear to also increase Amb a 1, the major allergenic peptide of ragweed [9]. Interestingly, Bet v 1, the major allergenic peptide of birch, has been demonstrated to be stronger in samples collected from trees grown in a garden with higher daily mean temperatures [14].

As stated earlier, allergic respiratory disease is heterogeneous, making it difficult to determine cause and effect on any one variable. In an Italian study, Ariana [15•] monitored pollen counts and meteorological data, in conjunction with allergen sensitization patterns of his patients. Over that 27-year period, there was a progressive increase in pollen load of approximately 25% (excluding grasses). These pollen cycle changes correlated with increasing temperature and increasing days > 30 °C. During this same period, there was an observed increased incidence of pollen sensitivity, while there was no change in perennial sensitivity, such as dust mite. Concluding that progressive climate changes, with increased temperatures, may modify the global pollen load and affect the rate of allergic sensitization across extended periods [16].

In summary, warmer temperatures and later fall frosts allow ragweed plants to produce pollen later into the year, potentially intensifying and prolonging the allergy season for millions of people [13•, 16]. Warming temperatures and increasing CO₂ levels increase pollen load, prolong pollen seasons, and intensify allergenicity in studied weed and tree pollens. Moreover, as the climate changes, plant species may adapt and undergo shifts in demographic distribution, expanding the zone of some allergenic plants.

Increasing Extreme Weather Events and Sea Level Rise

Climate change is expected to drive more extreme weather events, which will likely impact respiratory morbidity and mortality. Increased intensity and frequency of hurricanes and thunderstorms will bring more rainwater. Water damage from flooding promotes damp buildings and mold growth. The increased rainwater from storms will also rupture pollen grains, releasing respirable aeroallergens [17••]. Thunderstorms can concentrate pollen grains at ground level, releasing allergenic particles of respirable size in the atmosphere after their rupture by osmotic shock. During the first 20–30 min of a thunderstorm, patients suffering from pollen allergies are at risk of inhaling high concentrations of the allergenic peptides, which can induce potentially severe asthmatic exacerbations [18•].

Global average sea level has risen by about 7 to 8 inches since 1900, with almost half (approximately 3 inches) of that rise occurring since 1993 [3••]. Although there is widespread variability in these forecasts, recent estimates raise sea level 1.8 mm per year for the past century and between 2.8 and 3.1 mm per year over the past decade [19]. Sea level rises are expected to cause greater indoor moisture in seaside communities and contribute to wet housing and associated respiratory problems [20]. Over the past 50 years, there has been population growth in low-lying coastal areas and along the ocean fronts. It has been suggested there is the potential for an increase in water exposure for an estimated 3.3 billion people around the world who live near coastal regions [21]. Damp buildings have been clearly associated with respiratory disease [20], and a rise in sea level would expose the sizable percentage of the US population who live in coastal and wetland areas to additional wet housing conditions, posing increased risk of indoor mold contamination and resultant exposure. Allergic rhinitis and asthma both have been associated with exposure to fungal contamination in homes [22]. A quantitative meta-analysis of 33 epidemiologic studies showed an increase of 30 to 50% in adverse respiratory health outcomes in occupants because of dampness and mold exposure [23].

Air Pollution

Ground level ozone (O₃) is a pollutant gas with direct impact on the human respiratory system, increasing the frequency of asthma exacerbations, increasing susceptibility to infection, and promoting chronic obstructive pulmonary disease (COPD) [24]. O₃ is generated at ground level by photochemical reactions involving nitrogen dioxide (NO₂), hydrocarbons, heat and ultraviolet radiation. Ozone concentration in the lower atmosphere has increased since pre-industrial times. Although by some measures, air quality in the USA has improved over recent decades, however ozone remains a problem related to increasing emissions of methane, carbon monoxide, and nitrogen oxides produced chiefly by transportation related activities [5•, 25]. Ground level O₃ levels strongly correlate with summers having the highest number of hot days (> 32 °C) [4••]. Exposure to O₃ induces airway inflammation, induces increased airway permeability, reduces lung function, and increases risk of asthma exacerbations [25, 26]. Beck et al. demonstrated that exposure to elevated levels of O₃ enhances the allergic response to birch pollens in asthmatics [27]. Long-term exposure to ozone promotes inflammation and impairs pulmonary function, especially in those predisposed to respiratory disease along with an increased risk of death from respiratory causes [28]. In the Children's Health Study, nitrogen oxide, nitrogen dioxide, and PM_{2.5} were associated with a worsening in lung function, demonstrated by decreases in FEV₁ [29].

Fine particulate matter (PM_{2.5}) is a mixture of organic and inorganic solid particles and liquid particles of varying sizes and composition. Fine particulates are formed from a variety of sources, such as diesel exhaust, forest fire smoke, airborne dusts, volcanic eruptions, glacial silt, and industry [1••, 4••]. High exposures to particulates pose a significant health risk, particularly to the respiratory and cardiovascular system. The association between living near high-traffic areas and diesel exhaust exposure and increased risk and severity of asthma and allergic disease has been well studied and reported [28, 30–32]. As continued warming and drier conditions continue in northern latitudes, forest fires are becoming more frequent and severe. The incidence of large forest fires in western USA and Alaska has increased since the early 1980s and is projected to further increase in those regions as climate warms, with profound changes in ecosystems [3••]. Higher temperatures spur more thunderstorms while drier conditions compromise containment. In turn, forest fires increase airborne particulates [33]. Exposure to these elevated levels of particulates and ozone has not only been attributed to worsening respiratory disease, but has been correlated with increased mortality [28].

Heat Waves

The link between heatwaves and adverse health impacts, including heat-related mortality, has been well established. The frequency and intensity of heatwaves will likely increase over the coming century as world temperatures rise [2••]. The impact of heatwaves on mortality is typically greatest in urban areas, largely due to the “heat island” effect [34]. Heat island effect is largely caused by large expanses of treeless asphalt and large heat retaining structures that block cooling breezes. Relatively few studies have been done, specifically addressing respiratory death due to heatwaves. In a review of 33 relevant articles, Witt et al. [6] reported that the risk of dying because of chronic lung disease during a heat wave was 1.8 to 8.2% higher than during average summer temperature. Heat stress causes fluid loss, disrupting pulmonary perfusion. This in combination with raised concentrations of pollutants leads to bronchial inflammation and decreased threshold for bronchoconstriction. In a study by McCormack et al. [35], in participants with moderate to severe COPD, increases in maximal indoor temperature resulted in worsening breathlessness, cough, and sputum production. A positive interaction between indoor temperature and indoor air pollution, including PM_{2.5} and NO₂, was also demonstrated. Lin et al. [36] reported a 7.6% increase in hospitalization rate for patients with COPD for every 1 °C increase in temperatures above 29 °C, in New York [36]. Vulnerable populations include the elderly and those with co-morbidities, such as respiratory disease. Projections predict worsening heatwaves in the future and likewise increasing heat-related deaths.

Conclusion

The US Global Change Research Program, Fourth National Climate Assessment [3••], concluded “...based on extensive evidence, that it is extremely likely that human activities, especially emissions of greenhouse gases, are the dominant cause of the observed warming since the mid-20th century. For the warming over the last century, there is no convincing alternative explanation supported by the extent of the observational evidence.”

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 disease [37•]. Coupled with air pollution and increased exposure to aeroallergens climate change may have potentially serious adverse consequences for human health. In 1980, the CDC reported that 3% of the US population were asthmatic, and in 2010 that number increased to > 8%. Based on CDC data, in the USA, asthma affects 25.7 million people, including 7.0 million children under 18 and is a significant health and economic burden

to patients, their families, and society. In 2010, 1.8 million people visited an ED for asthma-related care and 439,000 people were hospitalized because of asthma [38]. Though many factors have contributed to the increase in prevalence of atopic disease, observed changes in environmental factors related to the changing climate are an important consideration. Physicians, particularly allergist and pulmonologists, are on the front line in assisting those impacted by environmental factors, such as air quality and allergens. Physicians need to continue to maintain a vigilance, interpreting causes and triggers, working to lessen the impact on our patients, and adapting practices to meet those needs.

Our understanding of the impact of our changing climate on respiratory health is becoming clearer, as should our approach for adaptation and optimally mitigation. As stewards of our planet, choices and actions today will influence the impact changing climate will have on human health for decades to come. Where is the tipping point?

Compliance with Ethical Standards

Conflict of Interest The author declares no conflicts of interest relevant to this manuscript.

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.

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