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# Climate change, the environment, and rhinologic disease

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## Abstract

**Background:** The escalating negative impact of climate change on our environment has the potential to result in significant morbidity of rhinologic diseases.

**Methods:** Evidence based review of examples of rhinologic diseases including allergic and nonallergic rhinitis, chronic rhinosinusitis, and allergic fungal rhinosinusitis was performed.

**Results:** The lower socioeconomic population, including historically oppressed groups, will be disproportionately affected.

**Conclusions:** We need a systematic approach to improve healthcare database infrastructure and funding to promote diverse scientific collaboration to address these healthcare needs.

**KEYWORDS**

air pollution, allergic fungal rhinosinusitis, allergic rhinitis, chronic rhinosinusitis, climate change, health disparities, nonallergic rhinitis

## 1 | INTRODUCTION

There is growing evidence for climate change as a significant driver of human diseases.[1,2] Increased human fossil fuel use in the last half-century produces $CO_2$ and methane greenhouse gases. These gases absorb heat and result in global warming of the earth's surface. This warming is accompanied by regional changes in precipitation and increases in many types of weather extremes. Mounting evidence suggests that this results in exacerbation of human diseases, including cardiovascular, allergic, infectious, and mental health diseases.[1] These effects preferentially impact the lower socioeconomic populations, reflecting the fact that climate risks are a product of hazard, exposure, vulnerability, and response (Figure 1). The multifactorial character of climate-induced health risk is further complicated by the multifactorial processes that link climatic change to a health outcomes. Where some climate impacts may have a relatively direct physical impact on the body (e.g., outdoor heat exposure), others are mediated by ecological processes (e.g., vector-borne disease), and still others emerge from social or institutional mediation (e.g., malnutrition). Distinguishing the mediating processes can be important when projecting future health impacts. Herein we explore the evidence for the impact of climate change on several examples of rhinologic diseases and the consequences on the demographics of the afflicted population.

## 2 | EFFECT ON ALLERGIC RHINITIS

### 2.1 | Evidence

Over the past century, allergic disease has increased at an astounding epidemic rate and is currently estimated to affect from 10% to 30% of the world's population.[3–5] The basis of allergic disease is thought to be due to underlying genetic predisposing factors that are modified by environmental risk factors.[5] These factors include genetic mutational changes in the individual's DNA, the hereditary ability to carry disease to the next generation, race, sex, and age. However, environmental factors are now appreciated as significant major drivers of the accelerated increase in the prevalence of allergic disease, which appear to be independent of evolution of the natural genetic drift by host factors.[5] The hygiene hypothesis alone cannot explain the recent exponential growth of allergic disease.[3–5] Climate change and climate events have changed how individuals experience allergic rhinitis (AR). Spring has inched earlier in the year leading to earlier and prolonged release of tree pollens.[6] Conversely, there has been a progressive delay in the onset of fall and the associated first frost, increasing the length of ragweed pollination.[7] The primary carbon source for photosynthesis, atmospheric $CO_2$, has continued to increase providing more fuel for pollen production from ragweed and timothy grass.[8,9] Prolonged growing seasons and increased fuel supply has resulted in an overall increase in plant biomass, which serves as a nutrient source for mold.[10] As such, higher levels of mold spores are present every fall and spring.[10,11] Extremes in weather conditions induced by global warming modify pollination patterns and distribution resulting in growth of allergic microbes (mold and fungus), and promotion of allergic rhinitis and asthma exacerbations. Increases in air pollution from weather events then further compounds the exacerbation of allergen mediated respiratory diseases, such as chronic rhinosinusitis (CRS).[12]

Although higher temperatures are, in general, associated with higher pollen and aeroallergen counts, the relationship between climate and these allergens is mediated by other climate factors and ecological processes, including effects of altered precipitation patterns and changes in the geographic range of allergenic species. Techniques for measuring pollen rely upon either particles settling on a surface or being impacted in a trap.[13] Modern pollen counting techniques, introduced in the 1950s, have reliably shown a steady increase in pollen load

and length of pollen season[14] because temperatures have climbed across the globe. But global warming has also brought shifts in air movement and atmospheric properties. For example, higher humidity has been associated with lower concentrations of some allergens such as tree pollen, but increases in others such as mold spores.[15–18] Warmer air is able to hold more moisture, but this leads to lower relative humidity (defined as the amount of water vapor in the air relative to what the air can hold) in many regions.[19] A regional change in these humidity conditions, then, could have diverse impacts on allergens in the environment.

The distribution of allergenic species has also been in flux, due to regional changes in temperature and precipitation. Hardiness zones, defined as regions where temperature sensitive plant species thrive, have shifted dramatically from 1990 to 2015.[20] North American climates have become hospitable for known allergenic species like oak and hickory, at the expense of less allergenic pine and fir.[21] In regions where precipitation has increased, and/or are prone to extreme weather events, mold has thrived. Hurricane Katrina served as a prime example of weather-related changes to spore production. Spore counts increased while hospitals reported a rise in cold and allergy symptoms, which providers suggested was related to increased mold exposure.[22,23] As is the case in many health impacts of extreme events, this phenomenon reflects an interaction of a climate hazard with ecological processes that play out in the built environment. The health risk, then, is a product of the climate hazard, the ecologically mediated exposure to the allergen, and the vulnerability of people on account of their background health conditions and housing quality. The example also highlights the importance of the health system response as a risk mediator, as the ability of providers to infer mold exposure offered a path to address the source of the health risk (Figure 1).

### 2.2 | Summary and recommendations

The prevalence of AR has progressively increased throughout the world. This trend has been seen in Finland (0.6% prevalence to 8.88%), Sweden (21% to 31%), United Kingdom (5.8 to 19.9%), Northern Europe (19.7% to 24.7%), Italy (16.2% to 37.4%), South Korea (13.5% to 17.1%), and Western Australia (21.9% to 46.7%).[24–32] As would be expected, as the prevalence of AR has increased, so too has the incidence of allergic asthma.[33] Asthma hospitalizations similarly correlate with both pollen load and climate events such as floods and fires.[33] These data highlight the fact that no corner of the world is exempt from the effects of climate change on allergic disease. This further emphasizes that we, as health care providers, have both the opportunity and an obligation to urgently raise public awareness of the negative impact of climate change on upper airway health.

## 3 | EFFECT ON NONALLERGIC RHINITIS

### 3.1 | Evidence

About 200 million people worldwide, or approximately 3% of the global population, are estimated to suffer from nonallergic rhinitis (NAR).[34,35] NAR encompasses a broad range of nasal inflammatory conditions that are independent of typical infectious and allergic mechanisms. Environmental NAR is characterized by nasal hyperreactivity to various environmental triggers, including cold air, tobacco smoke, and chemical products.[34,36–38]

The hallmark symptoms of NAR are nasal congestion, rhinorrhea, and various pain syndromes, including headaches and atypical facial pain, which are thought to occur through shared neurologic pathways of the sphenopalatine ganglion.[39] As an example of the impact on climate change on NAR related symptoms, results from a recent study of ~10,000 chronic pain sufferers from the United Kingdom demonstrated a direct and significant association between prevalence of pain with low atmospheric pressure, high humidity, high precipitation rate, and stronger wind.[40] NAR reduces quality-of-life (QOL) measures, including sleep, mood, and attention.[36] A comparison in QOL measures between NAR and AR demonstrates equally significant impairment, with greater degrees of nasal stuffiness and rhinorrhea, fatigue, and diminished sleep quality for NAR.[41] A majority of NAR patients further remain dissatisfied with the treatment of their symptoms, highlighting the lack of effective treatment options.[41] Pathophysiologic studies suggest that NAR symptoms occur through the activation of exaggerated autonomic reflexes by environmental exposures, such as shifts in temperature, pressure, and humidity.[42,43] This neurogenic response is supported by the presence of nociceptive neurons in nasal mucosa and increased cholinergic activity of the nasal glands.[36] Air pollutants, such as ozone and particulate matter, also contribute as independent triggers to NAR exacerbations and result in histopathologic metaplasia in the nasal epithelium.[42,44,45] Conversely, reduction in exposures to environmental irritants can subjectively and objectively improve NAR symptoms.[46] Thus, despite the heterogeneity of NAR, there appears to be a consistent correlation between ozone and particulate matter sensitivity with manifestation of symptoms of NAR.

NAR, then, is a case in which physical atmospheric conditions can have a direct influence on symptoms: climate conditions impact patient health without any requisite

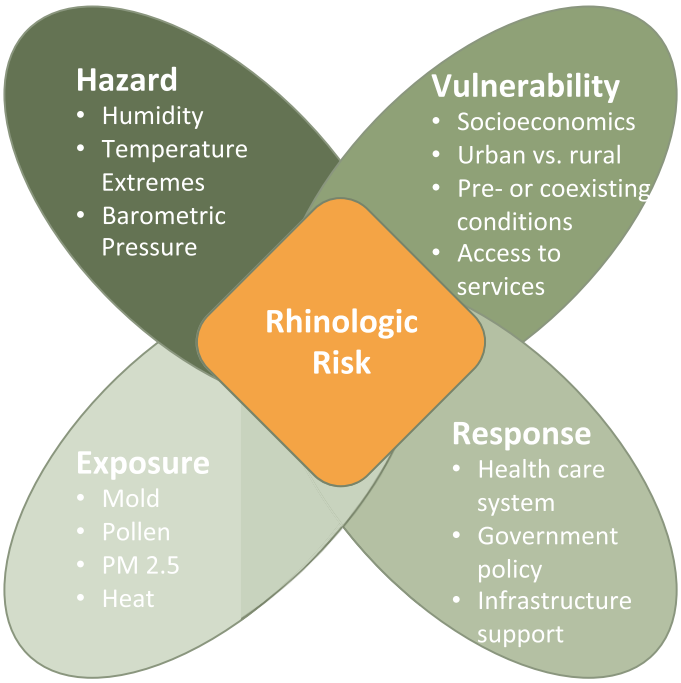


**FIGURE 1** The "risk propeller" applied to rhinologic disease. The "risk propeller" frequently used in Intergovernmental Panel on Climate Change reports, shown here with a four-blade structure was first introduced by Simpson et al.[108] Risk is understood to be a product of interactions between climate hazards, exposures related to factors that put people in harm's way, vulnerabilities that predispose people or groups to adverse impacts, and responses that either mitigate or inadvertently exacerbate the risk of negative outcomes.

ecological or social mediation. At the same time, the potential for pollutants to serve as triggers for NAR indicates that even in this case of direct climate influence on the body, there are potentially significant influences of social factors such as pollution exposure, with associated environmental justice implications for who is most vulnerable and faces greatest risk. Global warming is associated with an increase in weather variability and weather extremes,[47] which will have negative effects for NAR-susceptible individuals who have symptoms triggered by weather-related pressure or humidity changes.[19]

### 3.2 | Summary and recommendations

Thus, NAR may represent a sentinel event that predicts weather-related events. Affected domains such as sleep, emotion, and function directly impact on depression, productivity, lost days of work, and restriction of functional activity.[48–50] If global warming continues to accelerate at the present exponential rate, decline in QOL observed with NAR has the potential to result in major negative impact on our current population's socioeconomic status quo. Despite the negative impact of NAR on QOL, the exact socioeconomic burden of NAR is not well studied and varies across epidemiological studies due to the clinical and geographic heterogeneity of disease. The global prevalence and QOL burden of NAR highlights the need to first recognize NAR as a disease entity that requires attention and investigation. This will require collaboration among experts across multiple scientific disciplines in the United States and worldwide to advance understanding of the association between NAR and climate change. As an example of the power of collaboration, our European colleagues have commendably moved to systematically study CRS health outcomes by initiating establishment an international outcome registry Chronic Rhinosinusitis Outcome Registry (CHRINOSOR).[51] If our European colleagues can work to overcome logistical impediments to data collection from 10 independent countries, surely, we should be able to emulate their efforts in the United States. Clearly more research on the pathophysiology, epidemiology, and socioeconomic impact of NAR itself is needed, in addition to studying the effect of climate change on NAR.

## 4 | EFFECT ON CHRONIC RHINOSINUSITIS: EFFECT OF AIR POLLUTION ON THE UPPER RESPIRATORY TRACT

### 4.1 | Evidence

CRS affects approximately 5% to 12% of the US population or up to ~ 40 million Americans.[52–55] Exposure to airborne pollutants including particulate matter (PM), nitrogen dioxide ($NO_2$), and ozone has been shown to contribute to the development and/or exacerbation of several common upper respiratory tract diseases including AR and CRS.[56–66] PM2.5 in the atmosphere is comprised of organic compounds, metals, and products of combustion that are 2.5 microns in diameter or approximately 1/20th the diameter of a human hair particle.[67] Clinically, long-term air pollution exposure has been found to be associated with

the development of CRS in a powered case-control study and a recent systematic review.[68,69] In addition, air pollutant exposure has been correlated with increased need for revision sinus surgery.[61,70] Furthermore, US veterans who were exposed to pollutants on deployment overseas have been identified to have a higher prevalence of CRS than a similar cohort of nondeployed veterans.[71–73] These findings also extend globally to other continents. Urban areas in Cologne, Germany, with above average air pollution levels were associated with higher rates of CRS.[74] Significant correlation between particulate matter exposure and CRS prevalence was found by the South Korean National Health and Nutritional Examination Survey[75] and in a rhinologic outpatient cohort in Xinxiang, China.[76]

Mechanistic studies have shown that PM results in increased epithelial permeability, airway hyperresponsiveness, oxidative stress, immune dysregulation, and epigenetic changes.[62–66] Throughout a series of experiments using human sinonasal epithelial cells, a transition toward a proinflammatory state has been identified upon exposure to PM.[57,58,66] A recently published study by Patel et al.[65] sought to identify associations between sinonasal histopathology specimens and levels of air pollutants at the patients' place of residence. Within the CRS with nasal polyposis (CRSwNP) cohort, increased inflammation, Charcot-Leyden crystals, and eosinophil aggregates were associated with increased ozone exposure. In fact, for each 1–part per billion (1-ppb) increase in ozone exposure, there was an 81% increased likelihood of having eosinophil aggregates.

Climate change has both direct and indirect impacts on air pollutants (relevant for CRS and NAR). The chemical reactions that lead to creation or destruction of ozone and PM depend on both temperature and humidity, some natural sources of ozone precursors are temperature dependent, and changes in weather patterns can change the transport of pollutants.[77,78] However, the future trajectories of these pollutants are expected to depend more on environmental policies to reduce air pollution or climate change, than the direct impacts of a changing climate. For example, scenarios with high emissions of methane (i.e., little effort to reduce greenhouse gas emissions) project rising ozone concentrations primarily because of methane's role as an ozone precursor, whereas scenarios with methane mitigation project reduced ozone levels.[79] Exceptions could be regions where wildfires or dust storms occur. Global warming is associated with increased wildfire risk,[80,81] and increased droughts[82] in many regions, which leads to an associated increase in regional atmospheric concentrations of PM and other pollutants.[82,83] The US Environmental Protection Agency is conducting research specifically to address this question. In the Wildfire ASPIRE Study (Wildfire Advancing Science Partnerships for Indoor Reductions of Smoke Exposures), Missoula City-County Health Department in Montana, University of Montana, and the Hoopa Valley Tribe in California aim to compare indoor and outdoor PM2.5 concentrations and develop strategies for reducing indoor pollutant in public buildings during wildland fire smoke events (https://www.epa.gov/air-research/wildland-fire-research-health-effects-research). However, these studies are ongoing and results pending.

### 4.2 | Summary and recommendations

Although our government continues to debate policy on ways to decrease greenhouse gases, declining air quality, increasing wildfires, and increasing droughts continue to exacerbate CRS. Our academic societies in the United States and the world need to come together in vocal unity to highlight the importance for the need for research and research funding. We need to better understand the basic mechanisms involved in the pathogenesis of CRS that evolve from climate change that are both independent of and also interact with allergic mechanisms. The National Institutes of Health (NIH) purports to have interest in supporting CRS research. For example, the most recent funding National Institute of Allergy and Infectious Diseases (NIAID) newsletter purports a U19 mechanism that has an interest in funding studies on CRS as a programmatic priority in four out of six areas.[84] However, these four areas each pertain to CRS, as it is related to allergic mechanisms. But if we set aside our biases stemming from historical evolution of disease understanding, nonallergic alternate immune mechanisms also need to be considered. Interestingly, the specific program area where studies of pollution on the immune response is listed, there is an explicit absence CRS as a disease of interest: "The impact of the microbiome and of pollution on immune responses as they pertain to developing, preventing, and managing asthma, allergic rhinitis, food allergy, and atopic dermatitis."[84] This highlights the need for our government funding agencies to recognize that CRS is a disease area that requires additional and serious funding consideration with a diverse programmatic perspective, irrespective of whether traditionally appreciated allergic mechanisms are proposed in the application.

## 5 | EFFECTS ON ALLERGIC FUNGAL RHINOSINUSITIS

### 5.1 | Evidence

Allergic fungal rhinosinusitis (AFRS), a distinct subtype of CRSwNP, is an example of a "poster child" disease exacerbated by climate change. From an epidemiology

standpoint, changes in the environment, including increases in temperature and humidity, have the potential to directly affect prevalence and severity of AFRS. Increases in molds, caused by heavier rainfall and higher temperatures, can cause respiratory and asthma-related conditions as well as allergic bronchopulmonary aspergillosis, AFRS, and hypersensitivity pneumonitis.[85] Climate change induced increases in both temperature and precipitation extremes, then, could increase AFRS risk. It is widely accepted that AFRS is more commonly diagnosed in warm, humid environments within the United States and around the world. Ferguson et al.[86] reported higher incidence of AFRS along the Mississippi basin and the south-central part of the United States. A systematic review of AFRS studies across five continents identified that cities with higher mean temperatures were found to have higher prevalence of AFRS and that housing humidity could be related to the development of AFRS.[87] The distinction between housing humidity and ambient humidity is important, as it points to the interactions between climate conditions and the built environment. These correlations suggest that environmental exposure to fungi is important in the development of AFRS. The correlation of exposure and increased environmental fungal load in warmer climates is concerning in the context of global warming associated with climate change. Initial case control studies examining the correlation of household mold exposure with prevalence of AFRS found no statistically significant association of AFRS as compared to the CRSwNP group.[88] However, this was a relatively small study ($n = 31$ vs. $n = 39$, respectively) conducted at a single institution, and within a single geographic location. This highlights an opportunity and the need for more studies at multiple institutions, at multiple geographic sites throughout the United States and the world. Because individual patients demonstrate unique and varied levels of sensitivity to atmospheric allergens, it will also be important to understand how environmental mold growth patterns are changing over time.[89]

Factors affecting social determinants of health have a significant impact on prevalence of AFRS. Using a North Carolina state database, AFRS was found to be associated with those that self-identify as African American race, lower socioeconomic status, and less access to primary care providers, as compared to other phenotypes of CRSwNP and CRS without nasal polyposis (CRSsNP).[90] Similarly, another retrospective review identified that their AFRS cohort was younger at presentation with a higher proportion of uninsured/Medicaid status and those identified as African Americans, as compared to other CRSwNP and CRSsNP cohorts.[91,92] Furthermore, markers of disease severity within AFRS patient cohorts have been found to be associated with lower income and rural housing.[93]

### 5.2 | Summary and recommendations

The rates of AFRS diagnoses over time have not been well documented or examined, which directly hinders our ability to assess the impact of this disease on our healthcare system. This may be due to several reasons including challenges with meeting diagnostic criteria for AFRS. Phenotypic variability of clinical presentation and lack of a specific International Classification of Diseases, 9th revision (ICD-9) or International Statistical Classification of Diseases and Related Health Problems, 10th revision (ICD-10) code for AFRS contribute to diagnostic confusion.[90] This can result in inconsistencies in disease definitions and study methodologies between institutions and geographic regions. For example, if pathologists are not directed to review for specific histopathologic findings, this diagnosis may be missed. Other studies have utilized a strategy of classifying patients as AFRS patients when only three of five diagnostic criteria are met.[90] Given the present lack of consistency in diagnosis, it will be necessary to further define the criteria for establishing a diagnosis of AFRS in order to streamline and standardize future research efforts.[94] Recent studies have identified a significant number of patients with local nasal immunoglobulin E (IgE) reactivity in the absence of systemic findings of hypersensitivity, which raises additional questions about the need to further clarify and refine diagnostic criteria.[95] The current lack of consensus presents an opportunity for rhinologists to collaborate with our allergy and pulmonary medicine colleagues to standardize diagnostic criteria and coding so that meaningful data can be collected and mined for much needed studies.

## 6 | EFFECT ON HEATH DISPARITIES

### 6.1 | Evidence

Historically vulnerable and oppressed populations are at higher risk of harm from climate change, extreme weather events, and pollution. The National Academies of Science, Engineering and Medicine have focused on the intersection of climate change and equity. In their Environmental Health Matters Initiative, they stated that, "Communities disadvantaged by a legacy of racial segregation and environmental injustice struggle with disparate health outcomes, are vulnerable to the effects of climate change…and lack sufficient resources to recover from and rebuild for resilience against future events."[96]

Efforts to investigate the intersection of climate change and rhinologic disease need a health equity approach regardless of whether equity is the primary topic of investigation. Megwalu et al.[97] recently published a

resource for otolaryngologists summarizing evidence-based research frameworks and approaches for health equity research in otolaryngology. Research in equity has three phases: (1) detecting, (2) understanding, and (3) reducing disparities.[98] In otolaryngology, research has focused primarily on detecting disparities and needs to progress toward understanding the underlying mechanisms and ameliorating disparities.[99] Mechanisms help elucidate the specific causal pathway between sociodemographic characteristics and disparities in health, healthcare utilization, or healthcare outcomes. For example, housing or occupational health policies affecting patients' home or work environments may be important mechanisms for mitigating environmental exposures. The National Institute on Minority Health and Health Disparities (NIMHD) research framework lists additional categories for consideration in the study of health disparities and social determinants of health.[100] Climate events are anticipated to increase the risk of certain types of rhinologic disease and disrupt healthcare access and care continuity, and these effects are expected to be worse for disadvantaged communities.

Health services research on disparities often utilizes administrative datasets such as Medicare data or Healthcare Cost and Utilization Project data. However, CRS is challenging to study in administrative datasets given the poor reliability of administrative codes and lack of consistent documentation of patient reported outcomes data.[101] The Cochrane Collaborative research organization recommends standardized collection of sociodemographic characteristics and social determinants of health in disparities research.[102,103] Registries and single- or multi-institutional cohort studies therefore play a critical role in rhinologic research. For high-quality and generalizable research related to climate change, we need large-scale efforts to diversify of our registry cohorts to focus on the underserved population and include social determinants of health, geographic location, and climate pressures such as flooding, drought, heat, and fires. Collaboration between institutions and with community groups will be helpful.

### 6.2 | Summary and recommendations

Translational science and clinical trial efforts should include equity in their scientific approach and methods.[104] For clinical trials, the Consolidated Standards of Reporting Trials statement was updated in 2017 (CONSORT-Equity 2017) to improve reporting of randomized trials from an equity standpoint.[105] The standards improve the inclusiveness, interpretability, and generalizability of clinical trials.

Inclusion of equity in any scientific approach is an essential part of the research. Equity needs to be integrated into the development of our scientific questions, our methodological approaches, the composition of our research teams and the patients and communities included in our research. Ultimately these efforts would greatly benefit from enhanced funding. Work is needed to define risk factors and disparate exposure to climate pressures, and urgent efforts to mitigate those effects.

As healthcare providers, we have worked diligently to earn the public trust in our quest to mitigate disease-induced morbidity, prevent untimely death, and improve the QOL for our patients. However, healthcare industry itself contributes ~8.5% of total greenhouse gas emissions in the United States.[106] This is an alarming wake up call. Therefore, to maintain public trust, we will also need some deep introspection and support a collaborative and actionable plan to reduce healthcare industry's contribution to greenhouse gas emission as proposed by the National Academy of Medicine's Action Collaborative on Decarbonizing the US Health Sector.[107]

## 7 | CONCLUSION

There is mounting evidence to suggest that climate change mediated effects on our environment have the potential to induce and exacerbate significant morbidity of rhinologic diseases. This will disproportionately affect the lower socioeconomic population and historically oppressed groups, and further worsen the healthcare disparities already present. Thus, the hot get hotter, the wet get wetter, the dry get dryer, and the poor get poorer with respect to health. On a practical level, this will impact the demographics and number of patients that we can anticipate treating in our daily clinical practices. The question now becomes: Are we ready? At first glance, it seems as though we are not. To address the negative impact of climate change on rhinologic health, we need improved infrastructure support of databases that contain (but is not limited to) critical information regarding: unambiguous and established rhinologic diagnoses, social and environmental determinants of health, health outcomes, and healthcare cost/utilization. This will complement the much needed research support on elucidating the pathophysiology of disease examples outlined above. Major funding for focused research using diverse and inclusive approaches on multiple levels is needed. This includes diversity in the spectrum of scientific collaboration between climate environmental, epidemiologic, basic biological, and medical scientists. This also implies a need for multi-institutional collaboration to study the diversely afflicted population within multiple geographic locations.

This call for engagement is not limited to academia, but should also include biopharmaceutical and medical device industry, healthcare services industry, nonprofits, and the federal government. The goal then will be to develop a better understanding of the mechanisms of environmental effects of climate change on rhinologic health such that we will be better prepared to mitigate these negative outcomes.

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## CONFLICT OF INTEREST

Jean Kim has grant funding from Genentech and GSK and has been a consultant for GSK. Regan Bergmark has grant funding from I-Mab Biopharma. Amber Luong is a consultant for Lyra Therapeutics, Medtronics, Sanofi, and Stryker; and has served on the advisory board for ENTvantage Cx, Soundhealth, and Maxwell Biosciences. Kent Lam is a speaker for Optinose. Lauren Roland serves on the advisory board of GSK. Joshua Levy serves on the advisory board for GSK, Regeneron, and Honeywell International. Jiviannee Lee is a consultant for Medtronic, Stryker, Aerin, and Sanofi. Mas Takashima is a consultant for Aerin, Acclarent, Medtronic, and LivaNova. Mohamad Chaaban serves on the advisory board for Optinose. All other authors declare no conflicts of interest.

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