

Readers are invited to submit letters for publication in this department. Submit letters online at <http://joem.edmgr.com>. Choose "Submit New Manuscript." A signed copyright assignment and financial disclosure form must be submitted with the letter. From available at www.joem.org under Author and Reviewer information.

Climate Change, Allergies, and Asthma

To the Editor:

Climate change, if present, is associated with atmospheric warming—so-called "global warming"—as well as volatility in weather patterns, leading to more severe winters at a given latitude (since cold air typically further north in latitude is pushed south) and hotter summer months, when the earth is closer to the sun.¹ Hot weather generates more pollen from plants.² Cold weather is associated with asthma emergency department visits in New York City.³ More pollen causes more disease, not only allergically induced, but also nonallergic, since ragweed, for example, produced more reactive oxygen species (ROS), so it may produce inflammation, leading to upper and lower respiratory tract symptoms, even in those persons without allergic asthma, rhinitis, or conjunctivitis.⁴ Clean Air Act enforcement may reduce sources of anthropogenic heat.⁵

Does climate change have 2 potentially deleterious effects on human health: (1) prolonged and more severe pollen seasons, leading to (2) worsened asthma and allergies? If true, these downstream consequences may pose significant risks in terms of patient care costs, lost time from work, morbidity, and possible mortality.

1. For the first question, supporting the possible concept of prolonged and more severe pollen seasons from hot weather, the duration of ragweed pollen season has been increasing as a function of latitude in North America—associated with delay in first frost by 27 days and lengthening of the frost-free period at latitudes above 44°N since 1995.² In Turkey, daily mean temperature and levels of sunshine are associated with more severe pollen counts.⁶
2. Our research group determined that for the latter question—cold weather and asthma—atmospheric temperature indeed impacts New York City asthma emergency department visits. Temperature data were recorded in Central Park from 2000 to 2007. Particulate matter

(PM_{2.5}) air pollution data, the concentration of 2.5 micron-sized particles per cubic meter of air were collected from the Bronx, Queens, and Manhattan from 2006 to 2009. Pollen counts were measured in Brooklyn in 2008. We obtained New York City asthma emergency department visit data from NYC.GOV from 2000 to 2007. Relations among these data were determined on the basis of correlation coefficients. There was a reverse relationship between asthma indicators and temperature, that is, extremely low temperature was associated with higher asthma discharge rates in the Bronx.³

3. *Does more pollen cause more disease?* Ragweed, known as *Artemisia* species, pollen represents a major cause of allergy in Central Europe. Variations in the pollen season, the influence of climate variables and the prevalence of pollinosis to it, were analyzed in Poznan, in western Poland between 1995 and 2004. The *Artemisia* species pollen season grew longer because of a clear advance in the starting day and only a slightly earlier end point; the peak day also came slightly earlier. *Temperature was directly correlated with daily Artemisia species pollen levels; relative humidity was inversely correlated.* Twelve percent of patients had a positive skin-prick test reaction to *Artemisia* species. Their symptoms were rhinitis and conjunctivitis (15%), atopic dermatitis (15%), chronic urticaria (14.3%), bronchial asthma (2.4%), and facial and disseminated dermatitis (1.3%). Elevated specific IgE concentrations were detected in the sera of 10.1% of patients. Pollen season intensity was also found to be highly influenced by rainfall in the previous weeks. Trends toward earlier season starts and longer duration, possibly caused by climate change, may have had an impact on this allergic Polish population.⁴

Another study relates geo-climate effects on asthma and allergic diseases in adults in Turkey (PARFAIT study). Evaluation of 25,843 questionnaires from parents of 25,843 primary schoolchildren in 14 cities indicated that mean annual temperature was significantly associated with the prevalence of asthma and wheezing in both genders. Eczema and temperature were associated in female subjects. Asthma in women was associated with mean annual

humidity in the air. Annual number of days with snow was associated with wheezing.⁷

4. *Is there an additional effect of nonallergenic air pollution acting in concert with aeroallergens?*

Ragweed has physicochemical properties to release ROS to cause disease, so that a 2 hit hypothesis may come into play. For those not sensitized, ROS may play a role; for those with allergies, ROS plus IgE-mediated disease would enhance the inflammation.⁸

Changes in production, dispersion, and allergen content of pollen and spores, both region- and species-specific may have been influenced by urban air pollutants interacting directly with pollen.⁹

While the incidence of allergy and asthma appears to be increasing worldwide, residents of urban areas more frequently experience these conditions than rural dwellers. Outdoor air pollution concentrations result from intense energy consumption and exhaust emissions from automobiles. Urban air pollution is a serious public health hazard. Laboratory studies have confirmed epidemiologic evidence that air pollution adversely affects lung function in patients with asthma. Damage to airway mucous membranes and impaired mucociliary clearance caused by air pollution may facilitate access of inhaled allergens to the immune cells in the airway, thus promoting sensitization of the airway. Consequently, a more severe allergic antibody (immunoglobulin IgE-mediated) response to aeroallergens and airway inflammation could account for increasing prevalence of allergic respiratory diseases in polluted urban areas. The most abundant components of urban air pollution entail high levels of vehicle traffic with airborne particulate matter called PM₁₀ and PM_{2.5}, nitrogen dioxide, and ozone.⁵ Diesel exhaust is particularly troublesome, since it increases the production of allergic IgE antibodies.¹⁰

If the earth's temperature is increasing—from fossil fuel combustion, greenhouse gas emissions from energy supply, transport, industry, and agriculture—then climate change altering the concentration and distribution of air pollutants, and interfering with the seasonal presence of allergenic pollens in the atmosphere, will significantly prolong these periods.¹¹

5. The Clean Air Act gives Americans the opportunity to attenuate anthropogenic climate change like industrial

air pollution, thereby alleviating a man-made scourge of heat-induced increased aeroallergen concentrations. I testified before Congress about the need to fund the EPA and the Clean Air Act, and in this letter, I reaffirm my position.⁵

**Anthony M. Szema, MD, FCCP,
FACAAI, FAAAAI, FACP**

Assistant Professor of
Medicine and Surgery
Stony Brook University
School of Medicine
Stony Brook Allergy & Asthma
Stony Brook, New York

REFERENCES

- Petoukhov V, Semenov VA. A link between reduced Barents-Kara sea ice and cold winter extremes over northern continents. *J Geo Phys Res*. 2010;115,D21111.
- Ziska L, et al. Recent warming by latitude associated with increased length of ragweed pollen season in Central North America. *Proc Natl Acad Sci USA*. 2011;108(10):4248–4251.
- Chen Y, Forsyth E, Pan K, Szema A, Szema K, Chenrachasit P. Atmospheric temperature and pollen counts impact New York City asthma ER visits. *J Allergy Clin Immunol*. 2010;125(2,suppl 1):Ab208.
- Stach A, et al. Prevalence of *Artemisia* species pollinosis in western Poland: impact of climate change on aerobiological trends, 1995–2004. *J Investig Allergol Clin Immunol*. 2007;17(1):39–47.
- Statement by American Thoracic Society by Anthony Szema before U.S. House of Representatives Committee on Appropriations, Subcommittee on Interior, Environment, and Related Agencies. Fiscal Year 2012, U.S. Environmental Protection Agency, April 5, 2011. Available at: http://appropriations.house.gov/_files/041511WrittenTestimonyPublicWitnessesPartI.pdf. Accessed October 6, 2011.
- Kizilpinar I, et al. Pollen counts and their relationship to meteorological factors in Ankara, Turkey during 2005–2008. *Int J Biometeorol*. 2011;55(4):623–631.
- Metintas S, Kurt E. Geo-climate effects on asthma and allergic diseases in adults in Turkey: results of PARFAIT study. *Int J Environ Health Res*. 2010;20(3):189–199.
- Dharajjiya N, Boldogh I, Cardenas V, Sur S. Role of pollen NAD(P)H oxidase in allergic inflammation. *Curr Opin Allergy Clin Immunol*. 2008;8(1):57–62.
- Cecchi L, et al. Projections of the effects of climate change on allergic asthma: the contribution of aerobiology. *Allergy*. 2010;65(9):1073–1081.
- Diaz-Sanchez D, Dotson AR, Takenaka H, Saxon A. Diesel exhaust particles induce local IgE production in vivo and alter the pattern of IgE messenger RNA isoforms. *J Clin Invest*. 1994;94(4):1417–1425.
- D'Amato G, Cecchi L, D'Amato M, Liccardi G. Urban air pollution and climate change as environmental risk factors of respiratory allergy: an update. *J Investig Allergol Clin Immunol*. 2010;20(2):95–102; quiz following 102.

Work Prognosis of Complex Regional Pain Syndrome Type I: Multicenter Retrospective Study on the Determinants and Time to Return to Work

To the Editor:

Complex regional pain syndrome type I (CRPS I) is a persistent functional disorder of the control of microcirculation of one or several joints by the autonomic nervous system, usually secondary to trauma.¹ This unpredictable disease^{2,3} is responsible for disabling sequelae with an impact on the patient's ability to return to work,⁴ as about 30% of the patients are unable to return to work. No published case series has assessed the work prognosis of patients with advanced CRPS I.⁴

We aimed to assess the medical and work outcome of patients managed in one of the occupational health centers in the Paris region following CRPS I and to analyze the factors associated with a favorable or unfavorable prognosis.

This was a multicenter observational study conducted in occupational health centers of the Paris region (Raymond Poincaré, Cochin-Hôtel-Dieu and Fernand Widai public hospitals, and the Centre Hospitalier Intercommunal de Créteil).

All patients, for whom the presenting complaint (principal or comorbidity) was CRPS I, were included. Clinical criteria used were described by Veldman et al in 1993,⁵ considering that no consensus had been reached concerning those described by Bruehl et al and Galer et al.^{6–8}

Data were extracted from their medical charts for visits held between 2001 and 2006. Missing data were collected by means of a patient telephone questionnaire (with a maximum of three reminder calls to the patient) or by calling their respective doctors (attending physician or occupational health physician).

Patients unable to answer the telephone questionnaire (invalid telephone number, poor comprehension, impaired cognitive function), patients not in contact with their doctors, and patients refusing to

give their informed consent to participate were excluded.

Several types of variables were investigated in the medical charts: *sociodemographic variables* (gender; age [in three classes]; level of education), *medical variables* (site, upper limb versus lower limb; clinical signs, treatments received and comorbidities [dyslipidemia, hyperuricemia, diabetes, smoking habits]) and *work variables* (size of current company, a nonsedentary job [job involving walking]).

Two endpoints were considered: return to work at the time of the study and the interval between the accident and return to work. The association between the variables studied and return to work was studied by univariate (Fisher exact test) and multivariate analyses (logistic model). In view of the power of the study, only univariate analyses were performed on the time to return to work based on significant variables identified by the logistic model (log-rank test).

In 6 years, 55 subjects were selected (six patients without CRPS I were not included), and no patients were excluded. A total of 22 men and 33 women with a mean age of 48 years (range: 24–64 years) participated in this study. Return to work was observed in 68% of the patients of this population.

The upper limb was affected significantly more frequently, especially the wrist (78%). Edema was the most specific positive clinical sign (Table). The treatments most often prescribed were analgesics and physiotherapy (66% of the patients who took this treatment returned to work).

On univariate analysis, criteria predictive of return to work were young age, a sedentary job, upper limb involvement, high level of education probably allowing a change of job more adapted to the patient's disability or a more sedentary job, and early introduction of analgesic treatment associated with physiotherapy (Table).

Lower limb involvement was significantly negatively correlated with return to work after adjustment for age, gender, sector of activity, and level of education. The average time to return to work of 14.8 months (± 11.5 , 9 missing data) was also significantly longer for patients with lower limb involvement.

The results of the descriptive analysis of this population in terms of return to work are consistent with the rare data of the literature on this subject, despite the small number of subjects. The occupational health centers only see patients presenting diagnostic difficulties of work-related diseases and problems concerning maintenance of employment and they therefore tend to overestimate the medical and social severity of the lesions observed. However, perfectly comparable data concerning return to work were

Copyright © 2011 by American College of Occupational and Environmental Medicine
DOI: 10.1097/JOM.0b013e318239e0ef