CHEST

Special Features

Lungs in a Warming World

Climate Change and Respiratory Health

Aaron S. Bernstein, MD; and Mary B. Rice, MD

Climate change is a health threat no less consequential than cigarette smoking. Increased concentrations of greenhouse gases, and especially CO_2, in the earth's atmosphere have already warmed the planet substantially, causing more severe and prolonged heat waves, temperature variability, air pollution, forest fires, droughts, and floods, all of which put respiratory health at risk. These changes in climate and air quality substantially increase respiratory morbidity and mortality for patients with common chronic lung diseases such as asthma and COPD and other serious lung diseases. Physicians have a vital role in addressing climate change, just as they did with tobacco, by communicating how climate change is a serious, but remediable, hazard to their patients. ***CHEST 2013; 143(5):1455–1459***

Climate change has gained little traction as a health hazard relevant to health-care professionals in the United States. Yet, according to the American College of Physicians and Surgeons, as well as 13 other national colleges of medicine from around the world, climate change may be the single greatest health threat of this century,[1] especially for people with chronic lung disease.

Those less familiar with climate change science may have suspicions or outright doubts about its validity. This is unfortunate, because the available science concludes with 90% certainty that the earth's climate has warmed over the last half century as a result of greenhouse gas emissions from human activities.[2] In addition, no national academy of science or distinguished professional society of climate scientists has found support for any alternative hypothesis that could explain this warming. Greenhouse gases such as CO_2 come from a variety of sources but mostly from the burning of fossil fuels, and their atmospheric concentration has increased from 280 parts per million at the start of the Industrial Revolution to roughly 395 parts per million today. Greenhouse gas emissions have contributed to the warming of the planet's average temperature by 0.8°C since 1880 (and two-thirds of that since 1980); increased the frequency and severity of heat waves; contributed to poorer air quality; boosted aeroallergen production; and brought about an intensification of the water cycle, which has caused more floods and droughts.[2] All these trends are expected to progress in the coming decades as greenhouse gas concentrations continue to rise.

What do these changes in climate mean for patients with common chronic pulmonary diagnoses, such as asthma or COPD? These patients are likely to bear a disproportionate burden of disease related to the increased heat, temperature variability, and extreme weather events associated with climate change. Their already compromised lung function may be further impaired by other environmental exposures affected by climate change, including air pollution, pollen, and other aeroallergens.

Consider the one in 12 adults and nearly one in 10 children under the age of 18 years with asthma. Patients with asthma are particularly sensitive to changes in weather, and one of the clearest fingerprints of climate change has been an increase in days with extreme heat. Hot, humid days trigger asthma symptoms and have been shown to increase airway resistance

Manuscript received September 26, 2012; revision accepted November 6, 2012.

Affiliations: From the Center for Health and the Global Environment, Harvard School of Public Health (Dr Bernstein); the Division of General Medicine (Dr Bernstein), Boston Children's Hospital; and the Pulmonary Critical Care Unit (Dr Rice), Massachusetts General Hospital, Boston, MA.

Correspondence to: Aaron Bernstein, MD, Division of General Pediatrics, Boston Children's Hospital, 300 Longwood Ave, Hunnewell 2, Boston, MA 02215; e-mail: aaron.bernstein@childrens.harvard.edu

DOI: 10.1378/chest.12-2384

more rapidly than does cold air, most likely by stimulating airway C-fiber nerves.[3,4] Acute rises in temperature and humidity are associated with increased ED visits for asthma, especially in children.[5] Climate models predict that in the coming decades, extreme heat events are likely to become still more frequent, with perhaps as much as a 50% increase in the frequency of the hottest (ie, the top fifth percentile based on historical records) days by midcentury in the United States and Europe.[6,7]

Greenhouse gases may worsen air quality in multiple ways, which is of particular concern to patients with asthma, because air pollution may trigger asthma and lower the bronchoconstrictive threshold to other respiratory antigens.[8] Ground-level ozone is likely to be affected by climate change because warmer temperatures catalyze its production.[9] Ozone is created when oxides of nitrogen (NOx) react with volatile organic compounds (both of which are present in exhaust from motor vehicles and fossil fuel-burning power plants) in the presence of ultraviolet radiation and heat.[10] In animal models and human studies, ozone increases inflammation in bronchial epithelial cells[11-13] and induces bronchial hyperresponsiveness.[14] A large body of evidence associates exposures to ground-level ozone with ED visits and hospitalizations for asthma,[15-17] worse asthma control,[18] and reduced lung function.[19,20] Because of temperature's role in increasing ozone production, global warming has the potential to roll back some of the hard-won ozone pollution reductions achieved in developed countries. In developing countries, where policies regulating ozone precursors may be more lax, the potential of global warming to spur ozone production is of greater concern to asthmatics.[21]

Climate change can also worsen air pollution by fostering conditions favorable to forest fires, which can generate immense volumes of air toxics. Climate models indicate that with 1°C of warming, wildfire risk may increase two- to sixfold over the 1950 to 2003 baseline in most of the continental United States west of the Mississippi.[22] When forests burn, they release a range of pollutants, from particulate matter and acrolein (a respiratory irritant) to carcinogens such as formaldehyde and benzene. In multiple studies, air pollution from wildfire smoke has been associated with more asthma exacerbations, ED visits, and hospitalizations.[23-26] Although forest fires may ignite only in certain regions, such as the western United States, their smoke plumes may extend over great distances. During the Russian heat wave of 2010, for instance, smoke from >500 fires stretched across about 3,000 km, roughly the distance from San Francisco to Chicago.[27]

Higher atmospheric concentrations of CO_2 and a warmer climate act synergistically to increase pollen levels, which will be problematic for the majority of asthmatics who are sensitized to pollen. Warming prolongs the pollen season as plants bloom earlier in the spring. Between 1995 and 2009 alone, the pollen season lengthened considerably, from 13 to 27 days, above 44°N in the United States.[28] CO_2 itself can stimulate pollen production. For example, experimental studies on ragweed have shown that a doubling of ambient CO_2 concentrations can boost the pollen output of the plant by >60%.[29] Ragweed and other allergenic plants have been shown to produce more allergenic pollen when grown at elevated CO_2 levels as well.[30,31] Experimental studies have found reduced peak expiratory flow rates in allergic asthmatics exposed to pollen[32] and increases in epithelial shedding and neutrophils in bronchial biopsies of asthmatics in association with natural pollen exposure.[33] Marked increases in asthma and wheeze-related ED visits have been linked to high pollen concentrations in several studies,[34,35] and a dose-response relationship has been found between asthma ED visits and grass pollen levels.[36] There is little doubt that pollen levels will continue to rise as more CO_2 is released into the atmosphere and that they will contribute to asthma morbidity.

Many effects of climate change (more heat, more extreme weather events, and higher ozone levels, among others) are more ominous for the roughly 12 million people who have COPD and other serious lung diseases in the United States. Patients with COPD may not only have more frequent exacerbations and hospitalizations; they are also more likely to die as a result of climate change. The relationship between heat and all-cause mortality has been well established, and heat wave deaths predominantly affect the elderly and those with chronic respiratory or cardiovascular disease.[37,38] Sixty to seventy percent of deaths that occurred in heat waves in the United States resulted in true life-years lost and not short-term mortality displacement (ie, these deaths would not have occurred in the next 1 to 2 months without the exposure to the heat wave).[39] As average temperatures increase, populations will adjust to a higher temperature range but they will continue to be vulnerable at temperature extremes.[40]

Numerous recent studies have found excess hospital admissions for respiratory diseases and mortality as a result of heat waves in Europe and the United States. For each 1°C increase in mean temperature during the 2006 heat wave in Portugal, for example, all-cause mortality increased by 2.7% and COPD morbidity increased by 5.4%, with even greater increases in COPD morbidity for women and people over the age of 75 years.[41] A study of New York City summers from 1991 to 2004 found that each 1°C above an empirically derived temperature-health effect threshold for

the population resulted in a 2.7% to 3.1% increase in same-day hospitalizations for respiratory diseases, with a marked increase in COPD and asthma admissions.[37] In temperate climates, such as those of the United States and Europe, the frequency of summer heat waves is expected to double for every 2°C to 3°C increase in average temperature.[2] Urban populations will likely be disproportionately affected because of the urban heat island effect, which amplifies temperature rise.[39]

Although summer heat waves will cause excess mortality and hospital admissions for patients with COPD, the increased variability of temperature may be of even greater consequence. The variation in summertime temperature is forecast to increase in temperate regions as a result of climate change. Zanobetti et al[38] studied temperature variability and long-term survival between 1985 and 2006 among patients over age 65 with COPD and three other chronic diseases in 135 cities in the United States. They found increased mortality hazard ratios, indicating 5% to 7% increased mortality, with each 1°C increase in the SD of summertime temperature in patients with COPD and the other groups. The mortality effect sizes increased when the number of heat waves in each summer was controlled for, indicating that the results were not driven by heat waves, but by temperature variability itself.

Air pollution is of particular concern to patients with COPD because it can be fatal.[42-44] Temperature and air pollution have additive effects on mortality and respiratory disease. Several studies have found that air pollutants (particularly ozone and particulate matter) and high temperatures interact to cause excess overall mortality and hospital admissions for respiratory causes.[45,46] Thus, even if air pollution levels do not rise and levels of ozone, particulates, and other pollutants remain constant despite a changing climate, higher mean temperatures may cause more admissions and deaths for patients with COPD than would otherwise be expected from ambient air pollution.

Extreme weather events such as floods, hurricanes, droughts, and forest fires, like heat waves, will disproportionately affect patients with COPD and other chronic lung diseases. Natural disasters can increase the transmission of communicable diseases, including respiratory viruses and bacterial pneumonia, which carry a particularly high mortality rate in this patient population.[47] Although these extreme events may affect a minority of the population, the consequences for those affected, such as the residents of New Orleans after Hurricane Katrina or those of Colorado after the summer 2012 wildfires, can be devastating.[48,49] During the Russian drought and heat wave of 2010, for instance, an estimated 50,000 people were killed by the combination of heat and forest fire smoke in just over 1 month.[50]

Given all that is at stake with climate change regarding the health of patients with chronic lung diseases, much can be gained from actions to reduce greenhouse gas emissions. Burning fewer fossil fuels will yield immediate dividends because the major pollutants known to worsen asthma, COPD, and other respiratory diseases (air particulates, nitrogen oxides, sulfates, and ozone among them) come from fossil fuel combustion. CO_2 stays in the atmosphere for hundreds of years, which means its effects on the climate are long lived, but also that reducing emissions today will yield enormous health benefits in the future. The best available science indicates that unless significant action is taken to reduce greenhouse gas emissions, the planet may warm 2°C to 3°C above the preindustrial threshold by the end of the century, which is an increment that carries with it substantial health ramifications, some of which have been presented here.[2]

Starting in the 1970s, in response to robust scientific evidence and profound health consequences, physicians helped lead antismoking campaigns that made clear the connection between cigarette smoking and lung cancer and advocated smoking cessation and stricter tobacco controls. Today, the benefits of those campaigns are evident in lower rates of tobacco-related morbidity and mortality.

Too many people remain confused about climate science and what it means for their health. Physicians, and in particular pulmonologists, whose patients are among those expected to suffer most from climate change, can help change this. Because of their unique position in society as hands-on healers who apply the best available scientific knowledge to make their decisions, physicians can have a vital role in communicating the health risks of climate change to the public and policymakers, just as they did with tobacco, and help us secure a path that leads to a healthier, lower-carbon, future.

Acknowledgments

Financial/nonfinancial disclosures: The authors have reported to *CHEST* that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

References

1. Lim V, Stubbs JW, Nahar N, et al. Politicians must heed health effects of climate change. *Lancet*. 2009;374(9694):973.
2. IPCC. Climate change 2007: synthesis report. Contribution of Working Groups I, II and III to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. 2007. http://www.ipcc.ch/publications_and_data/ar4/syr/en/contents.html. Accessed September 22, 2010.
3. Hayes D Jr, Collins PB, Khosravi M, Lin R-L, Lee L-Y. Bronchoconstriction triggered by breathing hot humid air in patients with asthma: role of cholinergic reflex. *Am J Respir Crit Care Med*. 2012;185(11):1190-1196.

4. Aitken ML, Marini JJ. Effect of heat delivery and extraction on airway conductance in normal and in asthmatic subjects. *Am Rev Respir Dis*. 1985;131(3):357-361.
5. Mireku N, Wang Y, Ager J, Reddy RC, Baptist AP. Changes in weather and the effects on pediatric asthma exacerbations. *Ann Allergy Asthma Immunol*. 2009;103(3):220-224.
6. Duffy PB, Tebaldi C. Increasing prevalence of extreme summer temperatures in the U.S. *Clim Change*. 2012;111(2): 487-495.
7. Meehl GA, Tebaldi C. More intense, more frequent, and longer lasting heat waves in the 21st century. *Science*. 2004; 305(5686):994-997.
8. Leikauf GD. Hazardous air pollutants and asthma. *Environ Health Perspect*. 2002;110(suppl 4):505-526.
9. Bloomer BJ, Stehr JW, Piety CA, Salawitch RJ, Dickerson RR. Observed relationships of ozone air pollution with temperature and emissions. *Geophys Res Lett*. 2009;36(9):1-5.
10. US Environmental Protection Agency. What is ozone? US Environmental Protection Agency website. http://www.epa.gov/apti/ozonehealth/what.html. Published 2012. Accessed August 20, 2012.
11. Alexis NE, Lay JC, Hazucha M, et al. Low-level ozone exposure induces airways inflammation and modifies cell surface phenotypes in healthy humans. *Inhal Toxicol*. 2010;22(7): 593-600.
12. Scannell C, Chen L, Aris RM, et al. Greater ozone-induced inflammatory responses in subjects with asthma. *Am J Respir Crit Care Med*. 1996;154(1):24-29.
13. Song H, Tan W, Zhang X. Ozone induces inflammation in bronchial epithelial cells. *J Asthma*. 2011;48(1):79-83.
14. Larsen ST, Matsubara S, McConville G, Poulsen SS, Gelfand EW. Ozone increases airway hyperreactivity and mucus hyperproduction in mice previously exposed to allergen. *J Toxicol Environ Health A*. 2010;73(11):738-747.
15. Moore K, Neugebauer R, Lurmann F, et al. Ambient ozone concentrations cause increased hospitalizations for asthma in children: an 18-year study in Southern California. *Environ Health Perspect*. 2008;116(8):1063-1070.
16. Glad JA, Brink LL, Talbott EO, et al. The relationship of ambient ozone and PM(2.5) levels and asthma emergency department visits: possible influence of gender and ethnicity. *Arch Environ Occup Health*. 2012;67(2):103-108.
17. Babin S, Burkom H, Holtry R, et al. Medicaid patient asthma-related acute care visits and their associations with ozone and particulates in Washington, DC, from 1994-2005. *Int J Environ Health Res*. 2008;18(3):209-221.
18. Meng Y-Y, Wilhelm M, Rull RP, English P, Ritz B. Traffic and outdoor air pollution levels near residences and poorly controlled asthma in adults. *Ann Allergy Asthma Immunol*. 2007;98(5):455-463.
19. Chan C-C, Wu T-H. Effects of ambient ozone exposure on mail carriers' peak expiratory flow rates. *Environ Health Perspect*. 2005;113(6):735-738.
20. Chen PC, Lai YM, Chan CC, Hwang JS, Yang CY, Wang JD. Short-term effect of ozone on the pulmonary function of children in primary school. *Environ Health Perspect*. 1999; 107(11):921-925.
21. Fowler D. *Ground-level Ozone in the 21st Century: Future Trends, Impacts and Policy Implications*. London, England: The Royal Society; 2008.
22. Solomon S, Battisti DS, Doney SC, et al. *Climate Stabilization Targets: Emissions, Concentrations, and Impacts over Decades to Millennia*. Washington, DC: National Academy Press; 2011:180.
23. Rappold AG, Stone SL, Cascio WE, et al. Peat bog wildfire smoke exposure in rural North Carolina is associated with cardiopulmonary emergency department visits assessed through syndromic surveillance. *Environ Health Perspect*. 2011; 119(10):1415-1420.
24. Henderson SB, Brauer M, Macnab YC, Kennedy SM. Three measures of forest fire smoke exposure and their associations with respiratory and cardiovascular health outcomes in a population-based cohort. *Environ Health Perspect*. 2011; 119(9):1266-1271.
25. Henderson SB, Johnston FH. Measures of forest fire smoke exposure and their associations with respiratory health outcomes. *Curr Opin Allergy Clin Immunol*. 2012;12(3):221-227.
26. Delfino RJ, Brummel S, Wu J, et al. The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003. *Occup Environ Med*. 2009; 66(3):189-197.
27. NASA Earth Observatory. Fires and smoke in Russia. 2010. Earth Observatory website. http://earthobservatory.nasa.gov/IOTD/view.php?id=45046. Accessed October 17, 2010.
28. Ziska L, Knowlton K, Rogers C, 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):4248-4251.
29. Wayne P, Foster S, Connolly J, Bazzaz F, Epstein P. Production of allergenic pollen by ragweed (Ambrosia artemisiifolia L.) is increased in CO_2-enriched atmospheres. *Ann Allergy Asthma Immunol*. 2002;88(3):279-282.
30. Reid CE, Gamble JL. Aeroallergens, allergic disease, and climate change: impacts and adaptation. *EcoHealth*. 2009; 6(3):458-470.
31. 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):667-670.
32. Higgins BG, Francis HC, Yates C, et al. Environmental exposure to air pollution and allergens and peak flow changes. *Eur Respir J*. 2000;16(1):61-66.
33. Boulet L-P, Turcotte H, Boutet M, Montminy L, Laviolette M. Influence of natural antigenic exposure on expiratory flows, methacholine responsiveness, and airway inflammation in mild allergic asthma. *J Allergy Clin Immunol*. 1993;91(4): 883-893.
34. Darrow LA, Hess J, Rogers CA, et al. Ambient pollen concentrations and emergency department visits for asthma and wheeze. *J Allergy Clin Immunol*. 2012;130(3):630–638.
35. Héguy L, Garneau M, Goldberg MS, Raphoz M, Guay F, Valois MF. Associations between grass and weed pollen and emergency department visits for asthma among children in Montreal. *Environ Res*. 2008;106(2):203-211.
36. Erbas B, Akram M, Dharmage SC, et al. The role of seasonal grass pollen on childhood asthma emergency department presentations. *Clin Exp Allergy*. 2012;42(5):799-805.
37. 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): 738-746.
38. Zanobetti A, O'Neill MS, Gronlund CJ, Schwartz JD. Summer temperature variability and long-term survival among elderly people with chronic disease. *Proc Natl Acad Sci U S A*. 2012;109(17):6608-6613.
39. McMichael AJ, Woodruff RE, Hales S. Climate change and human health: present and future risks. *Lancet*. 2006; 367(9513):859-869.
40. Curriero FC, Heiner KS, Samet JM, Zeger SL, Strug L, Patz JA. Temperature and mortality in 11 cities of the eastern United States. *Am J Epidemiol*. 2002;155(1):80-87.
41. Monteiro A, Carvalho V, Oliveira T, Sousa C. Excess mortality and morbidity during the July 2006 heat wave in Porto, Portugal. *Int J Biometeorol*. 2012;57(1):155-167.

42. Ko FWS, Hui DSC. Air pollution and chronic obstructive pulmonary disease. *Respirology*. 2012;17(3):395-401.
43. Medina-Ramón M, Zanobetti A, Cavanagh DP, Schwartz J. Extreme temperatures and mortality: assessing effect modification by personal characteristics and specific cause of death in a multi-city case-only analysis. *Environ Health Perspect*. 2006;114(9):1331-1336.
44. Sunyer J. Geographical differences on the mortality impact of heat waves in Europe. *Environ Health*. 2010;9(38):1-2.
45. Katsouyanni K, Pantazopoulou A, Touloumi G, et al. Evidence for interaction between air pollution and high temperature in the causation of excess mortality. *Arch Environ Health*. 1993;48(4):235-242.
46. Ren C, Williams GM, Mengersen K, Morawska L, Tong S. Does temperature modify short-term effects of ozone on total mortality in 60 large eastern US communities? An assessment using the NMMAPS data. *Environ Int*. 2008;34(4):451-458.
47. Ivers LC, Ryan ET. Infectious diseases of severe weather-related and flood-related natural disasters. *Curr Opin Infect Dis*. 2006;19(5):408-414.
48. Anon. Public health response to Hurricanes Katrina and Rita - United States, 2005. *MMWR. Surveillance Summaries: Morbidity and Mortality Weekly Report.* 2006;55(9):229-231.
49. National Oceanic and Atmospheric Administration National Climactic Data Center. Wildfires, August 2012. State of the climate. NOAA website. http://www.ncdc.noaa.gov/sotc/fire/2012/8. Accessed December 21, 2012.
50. Topics geo: natural catastrophes 2010. Munich Re website. www.munichre.com/publications/302-06735_en.pdf. 2011. Accessed September 22, 2012.