

Respiratory Medicine

Series Editor: Sharon I.S. Rounds

Kent E. Pinkerton

William N. Rom *Editors*

Global Climate Change and Public Health

 Humana Press

Respiratory Medicine

Series Editor:

Sharon I.S. Rounds

For further volumes:

<http://www.springer.com/series/7665>

Kent E. Pinkerton • William N. Rom
Editors

Global Climate Change and Public Health

 Humana Press

Editors

Kent E. Pinkerton, Ph.D.
Department of Pediatrics, School
of Medicine
Department of Anatomy, Physiology
and Cell Biology
School of Veterinary Medicine
John Muir Institute of the Environment
University of California, Davis
Davis, CA, USA

William N. Rom, M.D., M.P.H.
Sol and Judith Bergstein Professor of
Medicine and Environmental Medicine
Division of Pulmonary, Critical Care
and Sleep Medicine
Departments of Medicine and
Environmental Medicine
New York University School of Medicine
New York, NY, USA

ISBN 978-1-4614-8416-5 ISBN 978-1-4614-8417-2 (eBook)
DOI 10.1007/978-1-4614-8417-2
Springer New York Heidelberg Dordrecht London

Library of Congress Control Number: 2013948452

© Springer Science+Business Media New York 2014

This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed. Exempted from this legal reservation are brief excerpts in connection with reviews or scholarly analysis or material supplied specifically for the purpose of being entered and executed on a computer system, for exclusive use by the purchaser of the work. Duplication of this publication or parts thereof is permitted only under the provisions of the Copyright Law of the Publisher's location, in its current version, and permission for use must always be obtained from Springer. Permissions for use may be obtained through RightsLink at the Copyright Clearance Center. Violations are liable to prosecution under the respective Copyright Law.

The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

While the advice and information in this book are believed to be true and accurate at the date of publication, neither the authors nor the editors nor the publisher can accept any legal responsibility for any errors or omissions that may be made. The publisher makes no warranty, express or implied, with respect to the material contained herein.

Printed on acid-free paper

Humana Press is a brand of Springer
Springer is part of Springer Science+Business Media (www.springer.com)

Foreword

The effects of global climate change on human health can be grouped into two main categories: the acute impact of extreme weather events on whole communities, but especially the most vulnerable groups within a community, and the increased severity of chronic air pollution exposure in areas where smog and soot are already well-known threats to children, the elderly, and persons with heart and lung ailments.

Little of the public debate about potential action to reduce human-caused release of greenhouse gas emissions has focused on health. The vacuum may be explained partially by concern on the part of established health advocacy groups that global warming is literally too hot to address. Some community-based organizations worry that working on climate change is a distraction from the ongoing efforts to maintain political support for measures to clean up existing sources of air pollution. Further, professionals who treat patients are appropriately cautious about speaking out on an issue that has become so politicized, especially if they are not well equipped to discuss the science.

The fact is that we have plenty of public policy tools—and some genuine success stories—to build on. In California, efforts began with a solid inventory of emissions from major sources as mandated by annual reporting requirement for major emitters. The winning struggle to require manufacturers to reduce emissions of greenhouse gases from new cars and light trucks led to the 2012 adoption by the U.S. Environmental Protection Agency and the National Highway Traffic Safety Administration of linked fuel economy and emission standards. These criteria will save consumers money and double gas mileage for the new car fleet over the next decade.

Measures adopted by California pursuant to ASB32, the Global Warming Solutions Act of 2006, include mandatory industrial audits, a Renewable Portfolio Standard requiring electric utilities to provide 33 % of their electricity from renewable solar, wind, and geothermal generation; a low-carbon fuel standard designed to push for investment in cleaner fuels; and a cap-and-trade regulation that establishes a price on carbon as a way to incent further cleanup.

California's program is more comprehensive than other states, but a recent inventory indicated that almost two-thirds of the states have enacted one or more

measures designed to promote energy efficiency or promote renewable energy, and nearly half the states as well as hundreds of local governments have made explicit commitments to reduce greenhouse gases. While federal government lags behind even voluntary action by many businesses, there are signs that once again the federal administration will pursue measures to promote meaningful reductions.

Many of the actions mentioned above fit the agenda that environmental and clean energy groups have been promoting for many years. Achieving drastic cuts in emissions relies on the same types of measures that are needed to meet health standards for air pollution—burning cleaner fuels and using less energy per unit of output. We need to promote technologies that can be diffused through the developing world to help those societies whose economies are growing rapidly to do so without increasing their carbon footprint. Breakthroughs are needed in both policy and technology if we are to bend the upward emissions curve to something more sustainable.

One lesson that we air pollution regulators have learned from our decades-long, highly effective but always contentious campaign to clean the air is that once the public is persuaded that their own health and that of their families is at risk, they are willing to accept additional cost (a few cents on the price of gasoline) and some inconvenience (annual inspection of older cars and trucks). Even better, we have learned to use both financial and behavioral carrots (access to carpool lanes for the cleanest vehicles) to achieve measurable reductions with lower friction. But what underlies the whole enterprise is that the public understands that the goal is improved health. If the same case can be made for climate action, we will have a real chance to make progress in the next few years on the most significant environmental issue of our time.

This book can help bring a broader range of voices into the discussion and increase the chance of effective action by governments to both mitigate the causes and buffer the impacts of ongoing climate change.

Sacramento, CA

Mary D. Nichols

Contents

1 Introduction: Consequences of Global Warming to the Public's Health	1
William N. Rom and Kent E. Pinkerton	
2 Climate Variability and Change Data and Information for Global Public Health	21
Juli M. Trtanj and Tamara G. Houston	
3 Climate Change: Overview of Data Sources, Observed and Predicted Temperature Changes, and Impacts on Public and Environmental Health	31
David H. Levinson and Christopher J. Fettig	
4 Eyewitness to Global Warming	51
Will Steger and Nicole Rom	
5 California and Climate Changes	71
Rupa Basu	
6 Heat Waves and Rising Temperatures: Human Health Impacts and the Determinants of Vulnerability	85
Helene G. Margolis	
7 Climate, Air Quality, and Allergy: Emerging Methods for Detecting Linkages	121
Patrick L. Kinney, Perry E. Sheffield, and Kate R. Weinberger	
8 The Human Health Co-benefits of Air Quality Improvements Associated with Climate Change Mitigation	137
George D. Thurston and Michelle L. Bell	
9 Asthma, Hay Fever, Pollen, and Climate Change	155
Anthony M. Szema	

10	Dengue Fever and Climate Change	167
	Lauren Cromar and Kevin Cromar	
11	Impact of Climate Change on Vector-Borne Disease in the Amazon	193
	William Pan, OraLee Branch, and Benjamin Zaitchik	
12	Climate Variability and Change: Food, Water, and Societal Impacts	211
	Jonathan Patz	
13	Household Air Pollution from Cookstoves: Impacts on Health and Climate	237
	William J. Martin II, John W. Hollingsworth, and Veerabhadran Ramanathan	
14	Biomass Fuel and Lung Diseases: An Indian Perspective	257
	Rajendra Prasad and Rajiv Garg	
15	The Effects of Climate Change and Air Pollution on Children and Mothers' Health	273
	Roya Kelishadi and Parinaz Poursafa	
16	Climate Change and Public Health in Small Island States and Caribbean Countries	279
	Muge Akpınar-Elci and Hugh Sealy	
17	Global Climate Change, Desertification, and Its Consequences in Turkey and the Middle East	293
	Hasan Bayram and Ayşe Bilge Öztürk	
18	Assessing the Health Risks of Climate Change	307
	Kristie Ebi	
19	Federal Programs in Climate Change and Health Research	319
	Maya Levine and John Balbus	
20	Management of Climate Change Adaptation at the United States Centers for Disease Control and Prevention	341
	Jeremy J. Hess, Gino Marinucci, Paul J. Schramm, Arie Manangan, and George Luber	
21	Public Health and Climate Programs at the U.S. Environmental Protection Agency	361
	Erika N. Sasser and C. Andrew Miller	
22	California's Cap-and-Trade Program	383
	John R. Balmes	
	Index	393

Contributors

Muge Akpinar-Elci, M.D., M.P.H. Department of Public Health and Preventative Medicine, St. George's University Medical School, St. George, Grenada

John Balbus, M.D., M.P.H. National Institute of Environmental Health Sciences, Bethesda, MD, USA

John R. Balmes, M.D. Division of Occupational and Environmental Medicine, Department of Medicine, University of California, San Francisco, CA, USA
Division of Environmental Health Sciences, School of Public Health, University of California, Berkeley, CA, USA

Rupa Basu, Ph.D., M.P.H. Office of Environmental Health Hazard Assessment, Air Pollution Epidemiology Section, Oakland, CA, USA

Hasan Bayram, M.D., Ph.D. Department of Chest Diseases, School of Medicine, University of Gaziantep, Gaziantep, Turkey

Michelle L. Bell, Ph.D. School of Forestry and Environmental Studies, Yale University, New Haven, CT, USA

OraLee Branch, Ph.D. Department of Microbiology, NYU School of Medicine, New York, NY, USA

Kevin Cromar, Ph.D. Department of Environmental Medicine, New York University School of Medicine, Tuxedo, NY, USA

Lauren Cromar, M.S. Tahoe Consulting, LLC, North Salt Lake, UT, USA

Kristie Ebi, Ph.D., M.P.H. Department of Medicine, Stanford University, Los Altos, CA, USA

Christopher J. Fettig, M.S., Ph.D. Pacific Southwest Research Station, USDA Forest Service, Davis, CA, USA

Rajiv Garg Department of Pulmonary Medicine, K.G's. Medical University UP, Lucknow, UP, India

Jeremy J. Hess, M.D., M.P.H., F.A.C.E.P. Emory Schools of Medicine and Public Health, Atlanta, GA, USA

Climate and Health Program, NCEH, CDC, Atlanta, GA, USA

John W. Hollingsworth, M.D. Department of Medicine and Immunology, Duke University Medical Center, Durham, NC, USA

Tamara G. Houston National Oceanic and Atmospheric Administration, National Climatic Data Center, NC, USA

Roya Kelishadi, M.D. Department of Pediatrics, Child Growth and Development Research Center, Isfahan University of Medical Sciences, Isfahan, Iran

Patrick L. Kinney, Sc.D., M.S. Department of Environmental Health Sciences, Mailman School of Public Health, Columbia University, New York, NY, USA

Columbia Climate and Health Program, Mailman School of Public Health, Columbia University, New York, NY, USA

Maya Levine, M.A. National Institute of Environmental Health Sciences, Bethesda, MD, USA

David H. Levinson, M.S., Ph.D. Watershed, Fish, Wildlife, Air and Rare Plants, USDA Forest Service, Fort Collins, CO, USA

George Luber, Ph.D. Climate and Health Program, Division of Environmental Hazards and Health Effects, National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, GA, USA

Arie Manangan, M.A. Climate and Health Program, Division of Environmental Hazards and Health Effects, National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, GA, USA

Helene G. Margolis, Ph.D., M.A. Department of Internal Medicine: General Medicine Center for Healthcare Policy & Research, School of Medicine, University of California, Davis, Sacramento, CA, USA

Gino Marinucci, M.P.H. Climate and Health Program, Division of Environmental Hazards and Health Effects, National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, GA, USA

William J. Martin II, M.D. Disease Prevention and Human Health Promotion, Eunice Kennedy Shriver National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, MD, USA

C. Andrew Miller, Ph.D. Air, Climate, and Energy Research Program, Office of Research and Development, U.S. EPA, Washington, DC, USA

Ayşe Bilge Öztürk, M.D. Adult Allergy Unit, Göztepe Education and Research Hospital, Medeniyet University Goztepe Training and Research, Kadıköy, Istanbul, Turkey

William Pan, P.H., M.S., M.P.H. Nicholas School of Environment and the Duke Global Health Institute, Duke University, Durham, NC, USA

Jonathan Patz, M.D., M.P.H. Global Health Institute, University of Wisconsin in Madison, Madison, WI, USA

Kent E. Pinkerton, Ph.D. Department of Pediatrics, School of Medicine, University of California, Davis, CA, USA

Department of Anatomy, Physiology and Cell Biology, School of Veterinary Medicine, University of California, Davis, CA, USA

Center for Health & the Environment, John Muir Institute of the Environment, University of California, Davis, CA, USA

Parinaz Poursafa, Ph.D. Environment Research Center, Isfahan University of Medical Sciences, Isfahan, Iran

Rajendra Prasad, M.D., FAMS, FCCP(USA), FNCCP. Department of Pulmonary Medicine, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi, India

Veerabhadran Ramanathan, M.S., Ph.D. Center for Atmospheric Sciences, Scripps Institution of Oceanography, La Jolla, CA, USA

University of California at San Diego, La Jolla, CA, USA

Nicole Rom, M.S. Will Steger Foundation, Minneapolis, MN, USA

William N. Rom, M.D., M.P.H. Division of Pulmonary, Critical Care and Sleep Medicine, New York University School of Medicine, New York, NY, USA

Erika N. Sasser, Ph.D. Health and Environmental Impacts Division, Office of Air Quality Planning and Standards, Office of Air and Radiation, U.S. EPA, Washington, DC, USA

Paul J. Schramm, M.S., M.P.H. Climate and Health Program, Division of Environmental Hazards and Health Effects, National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, GA, USA

Hugh Sealy, M.Sc., Ph.D. Department of Public Health and Preventive Medicine, St. George's University Medical School, St. George, Grenada

Perry E. Sheffield, M.D., M.P.H. Department of Preventive Medicine and Pediatrics, Mount Sinai School of Medicine, New York, NY, USA

Will Steger, B.S., M.A. Will Steger Foundation, Minneapolis, MN, USA

Anthony M. Szema, M.D., F.C.C.P., F.A.C.P. Department of Medicine and Surgery, Stony Brook University School of Medicine, Three Village Allergy & Asthma, South Setacket, NY, USA

New York State Center for Biotechnology, Stony Brook, NY, USA

George D. Thurston, A.B., Sc.B., S.M., Sc.D Department of Environmental Medicine, New York University School of Medicine, Tuxedo, NY, USA

Juli M. Trtanj National Oceanic and Atmospheric Administration, National Centers for Coastal Ocean Science, Highway, Silver Spring, MD, USA

Kate R. Weinberger, M.A. Department of Environmental Health Sciences, Mailman School of Public Health, Columbia University, New York, NY, USA

Benjamin Zaitchik, Ph.D. Department of Earth and Planetary Science, Johns Hopkins University, Baltimore, MD, USA

Chapter 1

Introduction: Consequences of Global Warming to the Public's Health

William N. Rom and Kent E. Pinkerton

Abstract Global warming from anthropogenic-derived greenhouse gases has consequences including climate change and public health risks. Measurements of these changes began in 1959 with the International Geophysical Year where CO₂ was measured atop Mauna Loa in Hawaii. CO₂ measurements were 316 ppm in 1959, and annual averages have increased until 2010 where it was 389.8 ppm. In 2010 the increase was 2.4 ppm, the largest 1-year increase recorded since 1998. CO₂ represents about 63 % of the greenhouse gases. Greenhouse gases reflect infrared radiation back to the earth's surface causing a warming effect. Global warming has a major effect on climate over time which differentiates climate change from weather which is short-term changes over hours or days. CO₂ has major sinks such as the oceans and peat bogs across the Arctic and taiga, and CO₂ is utilized by plants and forests in metabolism. Anthropogenic sources through burning oil and natural gas for transportation or heating, burning of forest lands for slash and burn agriculture, or burning coal have emitted CO₂ increasing substantially since the industrial revolution. This has been efficiently stored in CO₂ sinks. Only over the past 50 years have anthropogenic sources been prodigious enough to actually exceed the natural sinks and increase the global recordings of greenhouse gases and temperature.

W.N. Rom, M.D., M.P.H. (✉)

Division of Pulmonary, Critical Care and Sleep Medicine, New York University
School of Medicine, 550 First Avenue, New York, NY 10016, USA
e-mail: William.Rom@nyumc.org

K.E. Pinkerton, Ph.D. (✉)

Department of Pediatrics, School of Medicine,
University of California, Davis, CA 95616, USA

Department of Anatomy, Physiology and Cell Biology, School of Veterinary Medicine,
University of California, Davis, CA 95616, USA

Center for Health & the Environment, John Muir Institute of the Environment,
University of California, Davis, CA 95616, USA
e-mail: kepinkerton@ucdavis.edu

Keywords Greenhouse gases • CO₂ measurements • Anthropogenic sources • Radiative forcing • Biosphere consequences • Vector-borne diseases • Human health • Social stability

Greenhouse Gases and Temperature

Global warming from anthropogenic-derived greenhouse gases has consequences including climate change and public health risks. Measurements of these changes began in 1959 with the International Geophysical Year where CO₂ was measured atop Mauna Loa in Hawaii [1]. CO₂ measurements were 316 ppm in 1959, and annual averages have increased until May 10, 2013, when it reached 400 ppm (Fig. 1.1). In 2010 the increase was 2.4 ppm, the largest 1-year increase recorded since 1998. CO₂ represents about 63 % of the greenhouse gases. Greenhouse gases reflect infrared radiation back to the earth's surface causing a warming effect. Global warming has a major effect on climate over time which differentiates climate change from weather which is short-term changes over hours or days. CO₂ has major sinks such as the oceans and peat bogs across the Arctic and taiga, and CO₂ is utilized by plants and forests in metabolism. Anthropogenic sources through burning oil and natural gas for transportation or heating, burning of forest lands for slash and burn agriculture, or burning coal have emitted CO₂ increasing substantially since the industrial revolution. This has been efficiently stored in CO₂ sinks. Only over the past 50 years have anthropogenic sources been prodigious enough to actually exceed the natural sinks and increase the global recordings of greenhouse gases

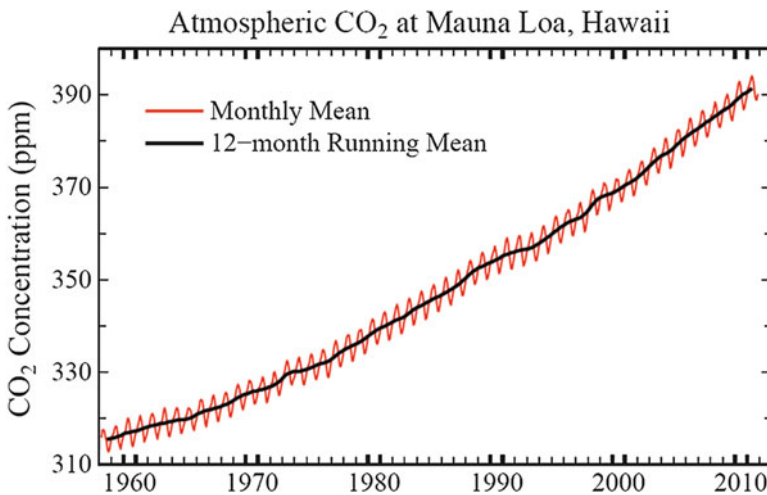


Fig. 1.1 CO₂ concentration on Mauna Loa, Hawaii (Courtesy of the Scripps Institution of Oceanography)

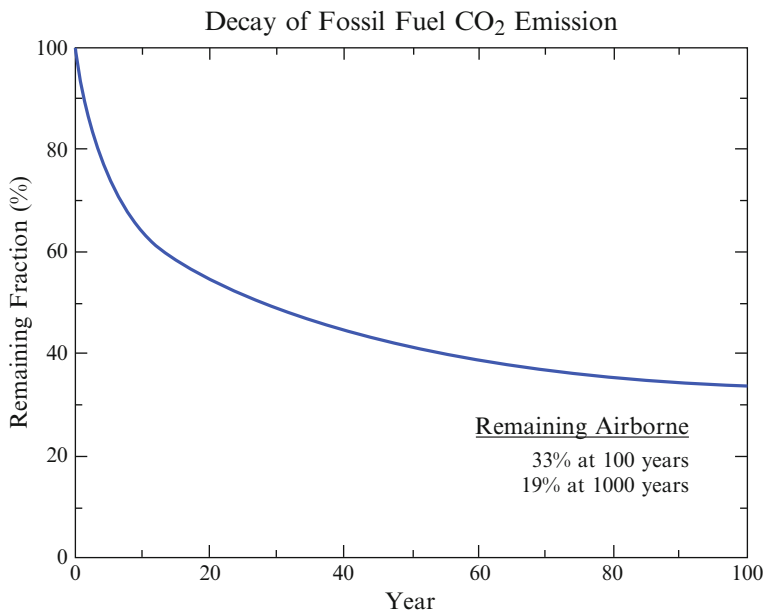


Fig. 1.2 Decay of fossil fuel CO₂ emissions. The fraction of CO₂ remaining in the air, after emission by fossil fuel burning, declines rapidly at first, but 1/3 remains in the air after a century and 1/5 after a millennium (From Hansen J, Sato M, Ruedy R, et al. Dangerous human-made interference with climate: a GISS modelE study. Atmos Chem Phys 2007; 7: 2287–2312 with permission, Copernicus Publications)

and temperature. Primary sources of the CO₂ are the carbon energy sources such as oil, coal, and natural gas which have been the basis of our modern lifestyle; hence, curtailing these sources is inherently political and accomplishing a radical restructuring to a *green* or non-carbon lifestyle requires all stakeholders to agree or *buy in*. Although economists optimistically suggest that this can be accomplished with 1 % of global GDP expenditures such as in the Stern Report, there will be winners and losers. Importantly, the market place externalizes the cost of pollution and is slow to respond especially when government regulation provides no course correction. In the case of CO₂, the gas is long-lived with 33 % remaining at 100 years and 19 % at 1,000 years [2] (Fig. 1.2). This means that once the tipping point of 450–600 ppm CO₂ is reached, the consequences will take years (decades or longer) to reverse [3]. Measurements of CO₂ captured in air bubbles in Antarctic ice show levels ranging between 172 and 300 ppm going as far back as 800,000 years [4]. In this chapter, we will highlight the greenhouse gases and their impacts on climate with consequences in the immediate past and present on human health. Computer models create predictions for the future, but since global warming is an experiment with little precedent, there are uncertainties that only more data will close.

In addition to CO₂, there are other greenhouse gases that have even greater radiative forcing than CO₂ [5]. First, methane has approximately 22 times the radiative forcing of CO₂ and has increased from 1,500 ppt in 1978 to 1,778 ppt in 2007

with a leveling off for the previous 10 years [6]. Methane is far less abundant than CO₂ and comes from carbon-related industrial processes but also agriculture especially rice paddies and the permafrost in the tundra. Methane is released from natural gas pipelines, especially leaks or explosions. A rather exotic release is from farm and range animals' gastrointestinal releases, and interestingly, these releases can be mitigated by changing their diets. There is significant methane in the frozen permafrost, and if this melts due to increased temperatures, release of stored methane would increase the warming trend [7]. Quantifying this risk is challenging [8]. Methane's lifetime in the atmosphere is only 10–12 years. Global water vapor trends have been positive perhaps due to global warming, and water vapor can act as a greenhouse gas, but its role in causing or mitigating climate change is still poorly understood. Nitrous oxide primarily from fertilizers but also coal and gas-fired power plants, nylon production, and vehicle emissions can contribute to radiative forcing. The global concentration of N₂O in 1998 was 314 ppb. Synthetic chemicals including perfluorocarbons, hydrofluorocarbons (HFCs), and sulfur hexafluoride from fire extinguishers, refrigerants, and foam blowers have >200-fold radiative forcing compared to CO₂ and have very long half-lives [9]. These chemicals have been synthesized to replace chlorofluorocarbons that endangered the ozone layer in the stratosphere in the extreme cold of the Antarctic winter. The CFCs have destroyed enough O₃ to result in a seasonal ozone hole over the whole continent of Antarctica; the Montreal Protocol has banned the CFCs providing an opportunity for product substitution, but the HFCs are also greenhouse gases [10]. SO₂ aerosols and organic carbon can provide a small cooling effect and black carbon from diesel emissions and biomass burning contribute to warming [11, 12]. The latter lasts days to weeks providing an opportunity to mitigate warming trends by reducing emission of these small particles.

The primary and immediate consequence of greenhouse gas increase in the troposphere is rising surface global temperature [13]. The National Oceanic and Atmospheric Administration (NOAA) within the Department of Commerce collates data on climate at its National Climatic Data Center in Asheville, NC. There are ~25,000 temperature stations around the world and they currently make >1.6 billion daily observations. Data from the World Meteorological Society show annual surface temperatures from 1861 deviate in a positive direction beginning in 1980 and persist and increase from the norm until the present. Data collected from tree rings, corals, ice cores, and historical records corroborate the thermometer recordings. Temperature time series collected from NASA's Goddard Institute for Space Studies and United Kingdom's land series at the University of East Anglia show the same trends. Combining global land ocean measurements, the trend is +0.6 °C/century and +1.0 °F/century. Warming since the 1970s is 0.2 °C/decade (0.36 °F). The 1980s was the warmest decade since the 1880s and every year of the 1990s was warmer than the 1980s average; the 1990s was even warmer than the 1980s and every year of the 2000s was warmer than the 1990s average. Satellites and weather balloons show the troposphere warming similar to the surface temperatures. Rural stations and exclusion of city stations result in the same trend. Global warming is not spatially uniform and greater trends are seen in the northern hemisphere and in high Arctic latitudes.

The Intergovernmental Panel on Climate Change (IPCC) in its Fourth Assessment Report declared, "Warming of the climate system is unequivocal, as is now evident from observations of increase in global average air and ocean temperatures, widespread melting of snow and ice, and rising global average sea level. Most of the observed increase in globally averaged temperatures since the mid-twentieth century is very likely due to the observed increase in anthropogenic greenhouse gas concentrations [14]."

Consequences of Climate Change on the Biosphere

Warming will first affect the ice—in the Arctic there is a decline in the average multiyear ice, and the extent of Arctic sea ice hit a new minimum in 2011. Data acquired by researchers at the University of Bremen in Germany from high-resolution microwave sensors on board NASA's Aqua satellite showed only 4.24 million km² of sea ice on September 8, 2011, which was 27,000 km² smaller than the previous record observed in 2007. In the mid-1980s, roughly 75 % of the Arctic ice pack at the yearly maximum in March had survived at least one summer melt season; today only 45 % has [15]. Since the record low sea ice extent that occurred in summer 2007, no very old ice (9 or more years old) is left in the central Arctic basin. Only a thin ribbon remains tucked up against the islands of the Canadian Arctic. The loss of the multiyear ice is both a result of climate change and, ultimately, an accelerator of it. The less old ice there is in the ice pack, the more easily the ice melts in the summer. The more the ice melts, more ocean is exposed to the 24-h summer sun. Bright white ice reflects incoming sunlight, but dark ocean water absorbs it, heating the ocean and accelerating warming. In a conversation with Ikou Oshima in Siorapaluk, the furthest north village occupied by the Thule Inuit, he lamented the late freezing of the sea ice from October to December making it difficult to hunt in the late fall when complete darkness descends. Furthermore, the earlier melting in the spring moving from the traditional August to as early as May makes the late winter ice slushier and more dangerous. The Inuit have a difficult time compiling sufficient meat for their culture as hunters to survive (Fig. 1.3). The polar bear, numbering almost 20,000, will be unlikely to survive as a species with wider water gaps and less frozen ice to hunt seals. In Antarctica, there has been loss of ice shelves such as the Larsen B in 2002 [16] (Fig. 1.4). Loss or reduction of the size of Antarctic ice shelves adversely affects the population of krill that are the basis of Antarctic biodiversity. The Adelie and emperor penguins on Antarctica and the huge population of king penguins on South Georgia and other Antarctic islands are at risk because of potential declines in krill [17]. Winters with extensive sea ice enhance krill abundance, and emperor penguins mainly feed on fish species that in turn depend on krill and other crustaceans.

Glaciers are in retreat across the globe with Glacier National Park in Montana predicted to be glacier-free by mid-century. Temperate glaciers near the equator are

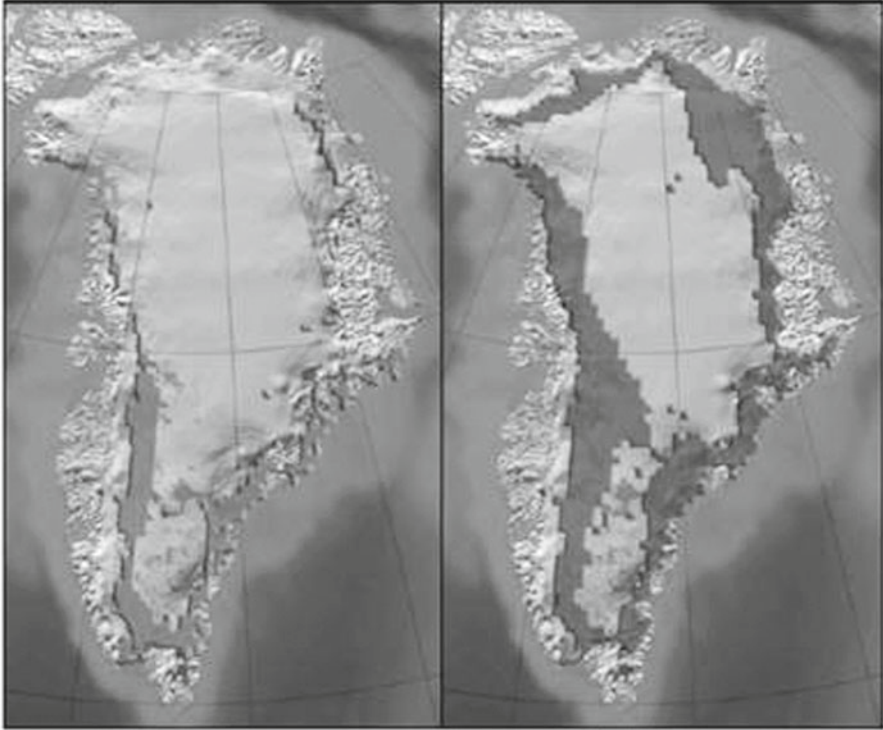
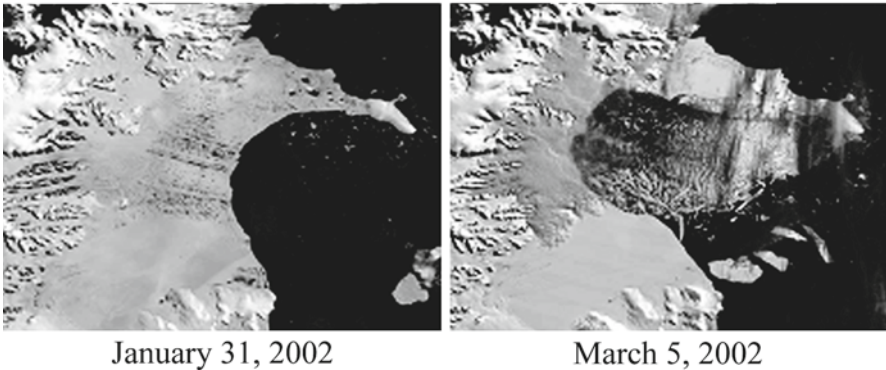


Fig. 1.3 Increase from 1992 (*left*) to 2002 (*right*) in the amount of the Greenland Ice Sheet melted in the summer (From the National Aeronautics and Space Administration, Scientific Visualization Studio, with permission)

Collapse of the Larsen B Ice Shelf, Antarctica



January 31, 2002

March 5, 2002

Fig. 1.4 Breakup of Larsen B Ice Shelf on Antarctic Peninsula January–March 2002 (MODIS images from NASA's Terra satellite. Courtesy of the National Snow and Ice Data Center, University of Colorado, Boulder, CO)



Fig. 1.5 Uhuru Point, Kilimanjaro Summit, William N. Rom M.D. in 1970 (*left*) and daughter Nicole in 1999 (*right*). Notice disappearance of Kilimanjaro ice fields over 29 years (Courtesy of the author)

at immediate risk, e.g., glaciers on Kilimanjaro have declined from 12.5 to 1.8 km² from 1912 to 2000 (Fig. 1.5). Glaciers are the source of drinking water and hydropower for Latin American cities like Lima, Peru, and La Paz, Bolivia, creating a potential cause for concern about their future demise. The seven great rivers arising out of the Himalayas and Kun Lun Ranges from glacial melt serve nearly 40 % of the world's population. Increasing glaciers' melting also produces lakes at the terminus of their moraines; increased meltwater can rupture these enlarging lakes and flood downstream communities. In 2005, photojournalist David Arnold retraced Bradford Washburn's footsteps photographing glaciers at the same date, time, and altitude/position as a half century earlier. Bradford Washburn had taken 8,000 black-and-white photographs chronicling mountains and glaciers of Alaska. The before-and-after photographic project illustrates the rate of change in glaciers from global warming (Fig. 1.6).

Changes in the Arctic regions include warming that could melt permafrost or perennial frozen ground releasing CO₂ and methane gas [18]. Permafrost underlies 24 % of the terrestrial northern hemisphere and 80 % of Alaska. Permafrost is mostly discontinuous and much of the known regions is in disequilibrium with the current climate. Melting of the permafrost could allow peat and attendant water to increase plant species taking up carbon, but models predict a greater release of carbon over time. Melting permafrost disrupts forests and man-made structures including buildings, pipelines, roads, and other infrastructures. The boreal and mountain forests are at risk due to climate change. The mountain pine bark beetle, lethal to spruce and pine trees, is normally killed by extreme cold 20–40° below zero. With global warming, the mountain pine bark beetle is thriving putting extensive forests in the sub-Arctic and US Rocky Mountain West at risk. Bark beetles in Alaska's Kenai Peninsula have killed spruce across three million acres, nearly half of the peninsula. An outbreak has consumed millions of acres in British Columbia spreading north and east into Alberta and into higher altitudes. Unfortunately, there is evidence that it has spread to jack pine, which is a common species throughout the boreal forest. Complicating the mountain beetle infestation is the more rapid melting of the winter

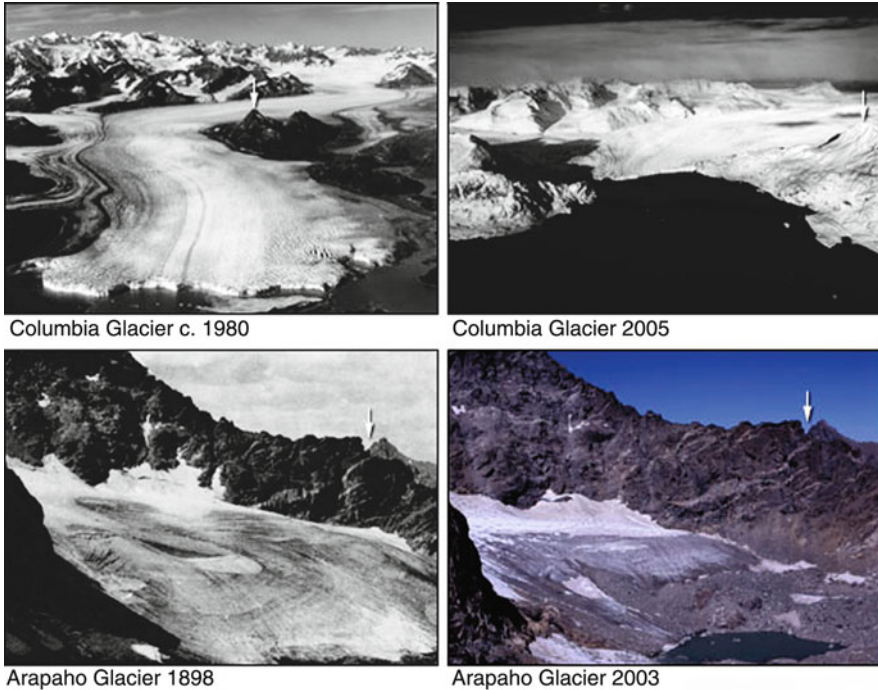


Fig. 1.6 Columbia Glacier, Alaska, c. 1980 by Austin Post, US Geological Survey; Columbia Glacier 2005 by Tad Pfeffer, Institute of Arctic and Alpine Research, University of Colorado (From from the National Aeronautics and Space Administration. Arapaho Glacier 1898 and 2003 courtesy of USGS Repeat Photography Project <http://nrmssc.usgs.gov/repeatphoto/>)

snowpack and drying of the climate leading to water-challenged forests leaving root structures of such species as aspens unable to support the forest. These forests are tinderboxes that serve to enable forest fires that are increasingly common in the US Southwest. Deforestation, usually to make way for agriculture, has been under way for decades, with Brazil and Indonesia being hotspots. The burning of tropical forests not only ends their ability to absorb carbon but also produces an immediate flow of carbon back to the atmosphere, making it one of the leading sources of greenhouse gas emissions. The world's forests cover ten billion acres and absorb one-quarter of human emissions of CO_2 . Deforestation of the Amazon is proceeding at a pace 5.8 million acres per year due to roads, hydroelectric plants, forest burning, and soybean farming. Second-growth forest may be able to keep pace with CO_2 absorption, although this needs study. Species may likely change as global warming proceeds with southern species extending their range northward. In this regard, the sugar maple of Vermont may be at risk for replacement by oak, hickory, or pine; already maple sap is running up to 2 weeks earlier.

The gradual rise of the sea level is of concern for low-lying nations and small island states like the Maldives in the Indian Ocean and Kiribati, Tuvalu, Cook Islands, and Marshall Islands in the Pacific. The sea-level rise is based on two mechanisms: first, the global warming exerts a steric force by thermal expansion of the volume of water and second, a eustatic force by increased mass of water from melting of sea ice, polar ice shelves, and polar glaciers. The Institute of Arctic and Alpine Research calculated that with the most likely glacial melting scenarios estimated a range of sea-level rise of 0.8–2.0 m by 2100 which was higher than the Fourth Assessment Report of the IPCC [8]. These calculations are difficult because the rate of rise in greenhouse gases and temperature is increasing 1990–2010 accelerating the melting of polar ice each year, although there are vast amounts of ice remaining.

Bleaching of coral reefs is occurring worldwide, and most experts attribute this to global warming from increased temperature and acidity of the oceans, although local pollution is another contributing factor [19]. Coral reefs are critical to biodiversity of the ocean, e.g., there are up to 800 types of coral, and 4,000 fish species live and propagate on coral reefs. Seaside communities in developing countries depend on coral fish populations for food, and as the coral reefs bleach and disappear, the health and survival of these people are at risk. Coral reef ecosystems are a symbiotic relationship between various coral species and algae, e.g., crustose coralline algae. Algae are known as dinoflagellates because they use irradiance for photosynthesis. These zooxanthellae supply coral reefs with essential nutrients produced by photosynthesis, especially carbon, in return for shelter and access to sunlight provided by the reefs. The algae impart color to the reefs, but they are sensitive to increase in temperature, CO₂, and acidity causing them to die and consequently starving the reefs turning them white which is known as coral bleaching (Fig. 1.7). The oceans serve as a sink for CO₂ with >30 % of CO₂ emitted to the atmosphere by human activities taken up by the ocean; the resultant carbonic acid has lowered the pH from 8.16 to 8.05 over the past 2 decades [20]. The acidity prevents calcium carbonate accretion by reef corals. Experimental aquae on the Great Barrier Reef in Australia with several CO₂ and warming scenarios show striking bleaching up to 50 % after 8 weeks of exposure to CO₂ 520–1,300 ppm (IPCC categories IV–VI) and irradiance [21]. Acidification was an additional effect, and any potential adaptation and acclimatization by coral reef organisms to thermal stress may be offset or overridden by CO₂ effects. Thus the authors concluded that CO₂ triggers bleaching in synergy with warming under high light.

Consequences on Human Health

The World Health Organization estimates that the warming and precipitation trends due to anthropogenic climate change over the past 30 years already claim 150,000 lives annually [22, 23].

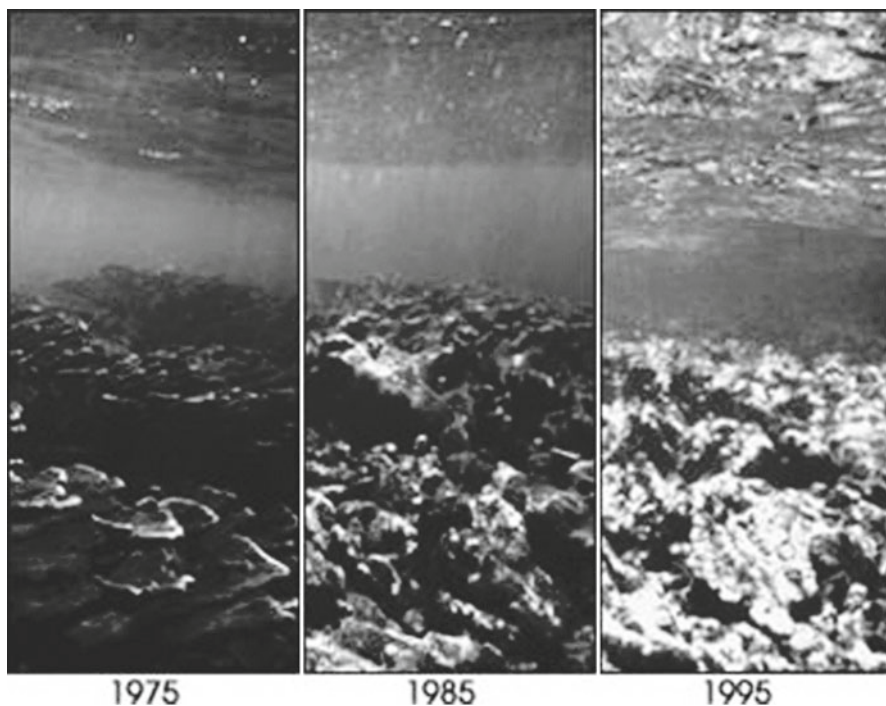


Fig. 1.7 The health of Carysfort Reef off the coast of Florida has declined dramatically in the past 25 years. The photographs show this decline. Coral that was healthy in 1975 is visibly sick by 1985, and dead and broken by 1995 (Courtesy of Phillip Dustan, College of Charleston, Charleston, SC)

Heat Waves

The first consequence of global warming will be increased heat stress, particularly in urban centers that already can serve as heat islands [24, 25]. Increased temperature in urban islands will occur not only in the daytime but also at night preventing any nocturnal relief. Heat waves will be accompanied by increased mortality due to cardiorespiratory diseases but also diabetes, accidents, homicides, and suicides [26]. Mortality also goes up for heat stroke with its attendant dehydration. Heat stroke is defined clinically as core body temperature $>40.6^{\circ}\text{C}$ accompanied by hot, dry skin and central nervous system abnormalities. The more rare hyperthermia is a medical emergency due to failed thermoregulation by the body and core body temperatures exceeds $>41\text{--}42^{\circ}\text{C}$. Extreme heat events vary by region and adaptation, e.g., a temperature of 102°F would create a negative health outcome in Cleveland, whereas the same temperature would have little additional effect on people in Phoenix.

The most famous heat wave occurred in Europe in August 2003 resulting in 32,000 excess deaths and adverse environmental effects on crops, forest fires, and loss of glacial mass [27]. France experienced a loss of 15,000 deaths with 2,000

heat-related deaths in 1 day [28, 29]. Hospitals, retirement, and nursing homes without air conditioning were especially vulnerable. Heat effects may be lingering where the 1-month and 2-year mortality in Lyon, France, among 83 patients admitted for heat stroke August 1–20, were 58 % and 71 %, respectively. A positive association has been noted between heat waves and mortality in the elderly, especially elderly women in social isolation [30]. In the Assessment and Prevention of Acute Health Effects of Weather Conditions in Europe project, a 1 °C increase in maximum apparent temperature above a threshold increased respiratory admissions by +4.5 % (95 % CI 1.9–7.3) in Mediterranean and North-Continental cities [31]. In the EuroHEAT project, heat wave-related mortality ranged from +7.6 % in Munich to +33.6 % in Milan 1990–2004 [32]. The increase was up to three-times greater during episodes of long duration and intensity. The highest effect was observed for respiratory diseases and among women aged 75–84 years. In 2003 the highest impact was observed in cities where the heat wave episode was characterized by unusual meteorological conditions.

Higher surface temperatures, especially in urban areas, promote increased ground-level ozone with a synergistic effect on mortality. Data from nine French cities regressing temperature and ozone on mortality found a significant effect of 1 % per 10 $\mu\text{g}/\text{m}^3$ in ozone level [33]. The US epidemiological studies show that a 10 °C increase in temperature on the same summer day increased cardiovascular mortality by 1.17 %, and there was an 8.3 % difference comparing the highest level of ozone to the lowest among the 95 cities in the National Morbidity and Mortality Study [34]. Schwartz and colleagues found an association between elevated temperatures and short-term increases in cardiovascular-related admissions for 12 US cities [35, 36]. PM_{10} has been associated with increased cardiovascular and chronic pulmonary disease deaths in Wuhan, China, located in a deep valley susceptible to trapping air pollutants, where a dose response has been observed with the highest mortality on the days of extremely high temperature exceeding 33.1 °C [37]. Behavioral conditions have also been associated with higher temperatures in urban areas [38]. A recent study of 40 US cities projected extreme heat events to increase fivefold by mid-century resulting in 32,934 more deaths and eightfold with business as usual by 2100 and 150,322 more deaths [39]. A recent study of Medicare hospital data 1985–2006 for 135 cities evaluated mortality for congestive heart failure, myocardial infarction, COPD, and diabetes [40]. A Cox proportional hazard model for each cohort within each city was correlated to summer temperature variation. Mortality hazard ratios ranged from 1.028 to 1.040 per 1 °C increase with higher associations for those >74 years. They were lower in cities with a higher percentage of land with green surface. Based on an average of 270,000 deaths per year across all four cohorts, a 5 % increase in mortality would correspond to ~14,000 additional deaths per year due to an increase in temperature variability in the United States.

It has been postulated that allergic diseases including hay fever and asthma will increase with urban global warming due to increases in pollen [41, 42]. Increases in CO_2 from 350 to 700 ppm in laboratory conditions can increase ragweed mass and pollen output from 40 to 60 % [43]. The major ragweed allergen, Amb a 1, was also noted to increase in laboratory experiments [44]. The US Department of Agriculture

has performed field experiments with ragweed plots in Baltimore demonstrating an urban island heat and CO₂ effect on pollen release compared to suburban or rural plots. More than 40 million Americans complain of hay fever and 16 million have asthma defined by the Centers for Disease Control and Prevention, and the trend for asthma has been increasing over the past 2 decades [45].

Vector-Borne Diseases

Global warming may alter the distribution of vector-borne diseases with malaria and dengue fever expanding their ranges by moving north from tropical to midlatitude regions including the United States [46]. Malaria continues to plague African children with 880,000 deaths and approximately 250 million cases per year globally [47]. The epidemic potential of malarial transmission has been projected to increase 12–27 % as a result of climate change. The more compelling data comes from records of illnesses kept in health dispensaries on tea plantations stemming from the British colonial era in Kenya [48]. The cases of malaria were projected for the tea highlands with temperature and rainfall over 3 decades showing a nonlinear correlation with actual cases exceeding predicted suggesting an effect already from climate change [49].

Dengue fever is primarily transmitted by *Aedes aegypti* (now named *Stegomyia aegypti*) and secondarily by *Aedes (Stegomyia) albopictus*. The WHO chronicles a 30-fold increase over recent decades and 50–100 million cases globally. These mosquitoes bite during the daytime making bednets a less useful preventative compared to household spraying. Oviposition or the number of eggs laid per female increases dramatically with temperature with a doubling per 5 °C. Oviposition also increases when humidity climbs above 60 %. The eggs need standing water to hatch and increased rainfall will assist moving the mosquito life cycle. Increasing temperature shortens the incubation time for the egg inside the mosquito and can increase mosquito abundance. At higher temperatures there is a reduced size, weight, and wing span of the mosquito which requires more frequent biting to complete one gonotrophic cycle. Higher temperatures require unfed females to feed sooner for the sake of their own survival than do lower temperatures.

Dengue fever is characterized by high fever, headache, skin rash, and muscle and joint pains with the name breakbone fever. A more severe form, dengue hemorrhagic fever occurring in about 5 % of cases, is characterized by shock with increased vascular permeability, internal bleeding and disseminated intravascular coagulation, and circulatory failure. It is caused by a RNA flavivirus, and there are four distinct serotypes. The Gates Foundation is funding efforts for a multivalent vaccine against the four serotypes. The entire *Stegomyia* genome has been sequenced with 14,519 protein coding sequences arising from 1.38 billion base pairs. Computer modeling predicts 5–6 billion people at risk of dengue transmission by 2085, but if CO₂ were controlled at current projected trends, then only 3.5 billion (35 % of the world's population) would be at risk [50]. Dengue fever time-series studies correlate outbreaks with temperature, rainfall, and humidity.

Implications for Social Stability

Global warming leads to climate change with potential effects on intensity and frequency of hurricanes, cyclones, and storms; drought with effects on food production and famine, population migrations, and potential war; increased precipitation with attendant flooding; and adverse financial impacts on insurance companies and governments' ability to respond to disasters [51]. September 2011 brought a mix of wet and dry conditions around the globe. Tropical cyclones Talas and Roke impacted Japan and nearby regions with intensive precipitation; Nesat brought extremely heavy rainfall to the Philippines; and Irene and Lee drenched the northeastern United States. Irene also dumped heavy rain over the Dominican Republic. The southwest Asian monsoon brought heavy precipitation to Pakistan and eastern India. Other regions with much higher-than-normal precipitation included Colombia in South America and part of southeastern Africa around Mozambique, Zimbabwe, and Tanzania. Hurricane Katrina highlighted the 2005 hurricane season that was the costliest in the United States at \$81 billion in property damage. Katrina, a category 3 hurricane, was the fifth worst hurricane in the history of the United States causing 1,836 deaths from the hurricane and its attendant floods. Over 80 % of New Orleans flooded after the failure of its levee system.

Below-average precipitation anomalies across the southern tier of the United States are indicative of ongoing major drought conditions. It was also exceptionally dry across the western United States; much of eastern and southern South America, particularly eastern Brazil; much of central Asia, including nearly all of Mongolia; and much of Australia. These hot, dry conditions exacerbate intensity and frequency of forest fires. In 2010, Australian blazes occurred after a record heat wave and hot, dry winds in southern Victoria state. The fires have swept nearly 500,000 acres. At least 170 people were killed in the disaster, and more than 3,000 people were displaced.

Lack of rainfall over several seasons is the most immediate and most visible cause of the current humanitarian crisis in the Horn of Africa. Climate change is only one of several factors that have led to the crisis; other factors are longer term. They include a very large population that depends on rain-fed agriculture and pastoralism for their livelihoods and sustenance. Environmental degradation—soil degradation and water degradation—and rapid population growth have compounded the problem. The climate is changing, which is changing the frequency of extreme events, such as the current drought, in ways that can only partially be anticipated. Much of sub-Saharan Africa has neglected agricultural development, and a recent phenomenon has been the purchase of large tracts of land to produce export food commodities. As a result, rural communities across Africa are trapped in worse and worse poverty, vulnerability, and dependence and have become more and more vulnerable to the impacts of shocks such as the current drought, increasing dependence on external humanitarian assistance. Population growth and the desire by a wealthier middle class in developing countries have created the need for more energy sources, supplied largely from the burning of fossil fuels. Urban expansion has resulted in the net loss of agricultural land. Migration to urban centers from rural

areas by peasants seeking a better life has increased the stress on food production. Some of the most fertile and productive farmlands are near cities. Agricultural lands can have reduced production due to overdrawn aquifers without replenishing the water. The combined effects of climate change, population increase, and expectations of a higher standard of living that lead to land and water scarcity for food production will affect the quantity of food and quality of the diet that can portend adverse effects on nutrition. Although increased CO₂ would expect to enhance crop growth, more likely there will be numerous other factors including fertility of the soil, insect, and other pests that flourish in warmer climate that will mitigate or eliminate any positive effects.

The world experiences about 500 weather-related disasters a year compared to about 100 per year in 1980. This adversely affects the insurance and reinsurance industries including Swiss Re, AIG, and others. Swiss Re estimates 3.4 billion people, primarily in the developing world, are at risk from storms, droughts, and floods creating a risk pool for innovative insurance solutions. Insured losses have jumped from an annual \$5 billion to an annual \$5–27 billion over the last 40 years. In 2011, Americans experienced 14 record-breaking weather and climate disasters that each caused \$1 billion or more in damages, in total costing approximately \$53 billion. In March 2012, 15,292 warm temperature records were broken across the United States. Climate risks are estimated to cost up to 19 % of annual GDP by 2030 with the potential of setting back development gains by years. The United Nations estimates that by 2030 the world should be spending an additional \$36–135 billion each year to address the effects of climate change. Companies such as Swiss Re are offering commercial insurance solutions as pre-disaster planning for developing countries to offset public budgets, but the countries must adapt climate-mitigation policies. At the World Economic Forum, it was estimated that moving to a low-carbon energy infrastructure and restricting warming to below 2 °C would require global investment in clean energy of roughly \$500 billion per year by 2020. However, public and private investment in clean energy in 2009 was only \$145 billion, far below needed levels. Private sector investors are critical to global efforts to stimulate a low-carbon economy, adapt to the unavoidable impacts of climate change, and close the climate investment gap. They require risk-adjusted long-term certainty from governments and international institutions about the direction of clean energy and climate policies and financing. Capital is not flowing to low-carbon investments at the scale required because of a lack of investor confidence in their climate and clean energy policy framework.

The United States' military is assessing risks for future conflict around the world relating to climate change. Recent war games and intelligence studies conclude that over the next 20–30 years, vulnerable regions, particularly sub-Saharan Africa, the Middle East, and South and Southeast Asia, will face the prospect of food shortages, water crises, and catastrophic flooding driven by climate change that could demand an American humanitarian relief or military response. As an example, Bangladesh will lose about 20 % of its land mass, creating a major refugee population since it is already densely populated. There will be a spill over migration or an exodus of people walking toward India. The Indians have built a fence around

Bangladesh and are in the process of electrifying it. This will be one potential site for armed conflict with different religions, damage to infrastructure from flooding, and the spread of contagious diseases. The US military has seen damage to infrastructure such as its Pensacola naval station in Florida from hurricanes, the potential loss of bases such as Diego Garcia in the Indian Ocean, and new Arctic sea lanes to defend with the melting of the Arctic ice cap. They have been particularly innovative in creating fuel cells, solar panels for Afghan outposts, and alternative fuels for aircraft and vehicles since supply lanes are vulnerable to attack.

Efforts at Mediation and Regulation

The United Nations has been the central focus on developing international consensus for climate change science and mitigation. Stockholm, Sweden, was the host for the first United Nations Conference on the Human Environment in 1972 and led to the establishment of the United Nations Environment Program (UNEP). The purpose of the conference was to unite the countries of the world against a common enemy, which was environmental degradation. Following this, the UN set up a commission of environment and development that issued a report using the term “sustainable development” as the way to ensure that economic development would not endanger the ability of future generations to enjoy the fruits of the earth. The twentieth anniversary of this conference was held in Rio de Janeiro in 1992 and called the “Earth Summit” which was attended by leaders of 105 nations demonstrating their commitment to sustainable development. The framework convention on climate change encouraged adoption of national policies that mitigate climate change by limiting anthropogenic emissions of greenhouse gases and protecting and enhancing their greenhouse gas sinks and reservoirs.

Since the 1992 agreement set no mandatory limits on greenhouse gas emissions for individual countries and contained no enforcement mechanisms, it was considered nonbinding. It did establish national greenhouse gas inventories of emissions and removals and set up the Conferences of the Parties (COP). In 1997 the Kyoto Protocol established legally binding obligations for developed countries to reduce their greenhouse gas emissions. Most industrialized countries and some central European economies in transition agreed to legally binding reductions in greenhouse gas emissions of an average of 6–8 % below 1990 levels between the years 2008 and 2012. The United States would be required to reduce its total emissions an average of 7 % below 1990 levels. Despite the negotiations on behalf of the US government by Vice President Al Gore and the President's signature, the US Senate refused to consider ratification because developing countries such as India, China, and Brazil were not bound to reduce their greenhouse gas emissions. The Byrd–Hagel Senate Resolution agreed to by 95 senators mandated that developing countries had to be included before the United States would ratify the treaty. China, India, Brazil, and other developing countries already emit half of the greenhouse gas emissions and are not subject to Kyoto reductions.

The details of this treaty were gradually agreed to at the COP meetings. After the 2001 rejection of the Kyoto Treaty by the Bush Administration, the United States was reduced to observer status at the COP meetings. The flexibility mechanisms advocated by the United States were agreed to which allowed industrialized countries to fund emissions reduction activities in developing countries. The Joint Implementation projects fund clean energy projects in countries that are industrialized but not required to contribute to the costs of developing countries; the advantage of these projects is that they are cheaper but still satisfy greenhouse gas reduction targets. The Clean Development Mechanism allows industrialized countries to invest in renewable energy, energy efficiency, and fuel switching in developing countries to meet their CO₂ limits and invest more cheaply to achieve the target reduction of 1.5 billion tons of CO₂ equivalents. A Program of Activities was developed to bundle CDM efforts such as distributing compact fluorescent lamps, efficient cook stoves, building refurbishment, or solar water heaters. The COP also agreed that credit would be granted for broad activities that absorb CO₂ from the atmosphere or store it, including forest and cropland management and revegetation. The COP agreed to compliance issues such as a “make-up” requirement for shortfalls at 1.3 tons to 1 and/or suspension of the right to sell credits for surplus emissions reductions.

Ministers and officials from 192 countries met at COP 15 in Copenhagen, Denmark, in 2009 to establish an ambitious global climate agreement for the period from 2012. President Obama decided to put off the difficult task of reaching a climate change agreement and instead pursued a less specific political accord to limit the growth in CO₂ emissions with a temperature increase limited to 2.0 °C. The accord was notable in that it referred to a collective commitment by developed countries for \$30 billion 2010–2012 for forestry and investments through international institutions. In Cancún, Mexico, COP 16 confirmed the goal of limiting global warming to no more than 2 °C above preindustrial levels and agreed to set up a new climate green fund to transfer money to developing countries. They also gave backing to the UN’s deforestation scheme and defined the building blocks for a framework to help countries design and implement effective adaptation strategies, explicitly mentioning risk management and insurance. The agreement also noted that addressing climate change required a paradigm shift toward building a low-carbon society. The agreement included a “Green Climate Fund” of \$100 billion a year by 2020 to assist poorer countries in financing emissions reductions and adaptation. There was no specific agreement on how this fund will be raised, and the decisions of the legal form and level of emissions reductions were once again deferred. They did develop a time frame for implementation of efforts to reduce emissions from deforestation and forest degradation (REDD); robust measurement, reporting, and verification (MRV), to increase confidence in national climate policies; and support for the creation of well-functioning markets in developing countries for energy efficiency and renewable energy to accelerate the effective deployment and diffusion of these technologies at scale.

The United States was stymied to develop a national carbon policy with the Senate only mustering 44 votes for the first McCain–Lieberman Climate Bill that would set up a modified cap-and-trade program in 2003; nothing further was debated on the floor for the next 8 years. The US House of Representatives passed the first

climate change bill in 2009 named after Representatives Waxman and Markey. It was based on cap and trade, with a goal of reducing greenhouse gas emissions 17 % below 2005 levels by 2020 and 83 % by 2050. This bill prohibited the EPA from regulating CO₂ under the Clean Air Act. In 2007 the US Supreme Court decided that EPA had statutory authority under the Clean Air Act to regulate CO₂ (Massachusetts et al. vs. EPA). The EPA also announced the Greenhouse Gas Reporting Rule, which affects entities with more than 25,000 tons/year (about 70 % of all US emitters). The EPA also found that CO₂ endangered public health and welfare allowing it to regulate CO₂ under the National Ambient Air Quality Standards. EPA regulations are based on science to protect the public health with an adequate safety margin to protect susceptible subgroups; however, political considerations at the White House mitigate what can be achieved in regard to pressure groups who bear the burden of cleanup costs.

California is the first state in the United States to adopt a cap-and-trade CO₂ regulation with a target to reduce CO₂ emissions by 15 % by 2020 compared to 1990 baseline implementing AB 32, California's historic climate change law. The California Air Resources Board will implement regulations covering 360 businesses representing 600 facilities mandating caps or credits in 2013 and by 2015 will cover distribution of transportation fuel and natural gas. Under the program, companies are not given a specific limit on their greenhouse gas emissions but must supply a sufficient number of allowances to cover their annual emissions. As a state-wide cap declines annually, the total number of allowances issued also declines. The allowances given to electric utilities are to be sold at auction, with the proceeds distributed to ratepayers. California joins Europe and Australia with a cap-and-trade program. Pacala and Socolow's 7/15 stabilization wedges required to solve the climate problem over the next 50 years with current technologies projected mind-numbing options that humankind has not even approached considering [52].

Canada has been increasing its production of oil from its Alberta tar sands (sand saturated with bitumen) that contain twice the amount of CO₂ emitted by global oil use in our entire history. The mineable area encompasses approximately 700,000 acres (size of Rhode Island) and ten mines are operating on almost one-third of this area [53]. About two-thirds of this boreal forest is peatland habitat, and this will not be restored by reclamation. Reclamation will create upland forest and scraping off peatlands onto mined areas does not remain alive. Landscape changes caused by currently approved mines will release 11–47 million tons of stored carbon and will reduce carbon sequestration potential by 6–7 metric tons carbon/year. This is an energy-intensive industry with natural gas used to heat bitumen and refine it into a liquid that can be transported by pipeline to Midwest refineries. Additional pipelines to Gulf Coast refineries and across British Columbia for export to the Far East have been controversial. The tar sands contain enough carbon, 240 gigatons, to add 120 ppm to the 393 ppm CO₂ currently in the atmosphere. Hansen's approach to solve this crisis is a carbon fee placed at the source of fossil fuel industries with the proceeds distributed on a per capita basis to individuals to invest [2]. Economic forecasts suggest that Americans would get back more than what they would pay in higher prices, and the reduction in oil use resulting from the carbon price would be nearly six times as great as the oil supplied from the proposed pipeline from Canada.

References

1. Keeling CD, Whorf TP. Atmospheric CO₂ concentrations derived from flask air samples at sites in the SIO network. In: Trends: a compendium of data on global change. Oak Ridge, TN: Carbon Dioxide Information Analysis Center, Oak Ridge National Laboratory, U.S. Department of Energy; 2004. <http://cdiac.ornl.gov/>
2. Hansen J, Sato M, Ruedy R, et al. Dangerous human-made interference with climate: a GISS model E study. *Atmos Chem Phys*. 2007;7:2287–312.
3. Lenton TM, Held H, Kriegler E, Hall JW, Lucht W, Rahmstorf S, et al. Tipping elements in the Earth's climate system. *Proc Natl Acad Sci USA*. 2008;105:1786–93.
4. Luthi D, Le Floch M, Bereiter B, Blunier T, Barnola JM, Siegenthaler U, et al. High-resolution carbon dioxide concentration record 650,000–800,000 years before present. *Nature*. 2008;453:379–82.
5. Montzka SA, Dlugokencky EJ, Butler JH. Non-CO₂ greenhouse gases and climate change. *Nature*. 2011;476:43–50.
6. Hansen J, Nazarenko L, Ruedy R, Sato M, Willis J, Del Genio A, et al. Earth's energy imbalance: confirmation and implications. *Science*. 2005;308:1431–5.
7. Smith LC, MacDonald GM, Velichko AA, Beilman DW, Borisova OK, Frey KE, et al. Siberian peatlands a net carbon sink and global methane source since the early Holocene. *Science*. 2004;303:353–6.
8. Oppenheimer M, O'Neill BC, Webster M, Agrawala S. The limits of consensus. *Science*. 2007;317:1505–6.
9. Hansen J, Sato M. Greenhouse gas growth rates. *Proc Natl Acad Sci USA*. 2004;101:16109–14.
10. Velders GJM, Andersen SO, Daniel JS, Fahey DW, McFarland M. The importance of the Montreal Protocol in protecting climate. *Proc Natl Acad Sci USA*. 2007;104:4814–9.
11. Hansen J, Nazarenko L. Soot climate forcing via snow and ice albedos. *Proc Natl Acad Sci USA*. 2004;101:423–8.
12. Ramanathan V, Carmichael G. Global and regional climate changes due to black carbon. *Nat Geosci*. 2008;1:221–7.
13. Ramanathan V, Feng Y. On avoiding dangerous anthropogenic interference with the climate system: formidable challenges ahead. *Proc Natl Acad Sci USA*. 2008;105:14245–50.
14. Intergovernmental Panel on Climate Change. www.ipcc.ch/
15. Serreze MC, Holland MM, Stroeve J. Perspectives on the Arctic's shrinking sea-ice cover. *Science*. 2007;315:1533–6.
16. Steig EJ, Schneider DP, Rutherford SD, Mann ME, Comiso JC, Shindell DT. Warming of the Antarctic ice-sheet surface since the 1957 International Geophysical Year. *Nature*. 2009;457:459–62.
17. Jenouvrier S, Caswell H, Barbraud C, Holland M, Stroeve J, Weimerskirch H. Demographic models and IPCC climate projections predict the decline of an emperor penguin population. *Proc Natl Acad Sci*. 2009;106(6):844–7.
18. Rosenzweig C, Karoly D, Vicarelli M, Neofotis P, Wu Q, Casassa G, et al. Attributing physical and biological impacts to anthropogenic climate change. *Nature*. 2008;453:353–7.
19. Carpenter KE, Abrar M, Aeby G, Aronson RB, Banks S, Bruckner A, et al. One-third of reef-building corals face elevated extinction risk from climate change and local impacts. *Science*. 2008;321:560–3.
20. Anthony KRN, Kline DI, Diaz-Pulido G, Dove S, Hoegh-Guldberg O. Ocean acidification causes bleaching and productivity loss in coral reef builders. *Proc Natl Acad Sci USA*. 2008;105:17442–6.
21. Hoegh-Guldberg O, Mumby PJ, Hooten AJ, Steneck RS, Greenfield P, Gomez E, et al. Coral reefs under rapid climate change and ocean acidification. *Science*. 2007;318:1737–42.
22. Patz JA, Campbell-Lendrum D, Holloway T, Foley JA. Impact of regional climate change on human health. *Nature*. 2005;438:310–7.

23. Frumkin H, Hess J, Luber G, Malilay J, McGeehin M. Climate change: the public health response. *Am J Public Health*. 2008;98:435–45.
24. Ebi KL, Mills DM, Smith JB, Grambsch A. Climate change and human health impacts in the United States: an update on the results of the U.S. national assessment. *Environ Health Perspect*. 2006;114:1318–24.
25. Epstein PR. Climate change and human health. *N Engl J Med*. 2005;353:1433–6.
26. Rom WN, Pinkerton KE, Martin WJ, Forastiere F. Global warming: a challenge to all American Thoracic Society members. *Am J Respir Crit Care Med*. 2008;177:1053–7.
27. Fouillet A, Rey G, Laurent F, Pavillon G, Bellec S, Guihenneuc-Jouyaux C, et al. Excess mortality related to the August 2003 heat wave in France. *Int Arch Occup Environ Health*. 2006;80:16–24.
28. Argaud L, Ferry T, Le Q-H, Marfisi A, Ciorba D, Achache P, et al. Short- and long-term outcomes of heatstroke following the 2003 heat wave in Lyon, France. *Arch Intern Med*. 2007;167:2177–83.
29. Dhainaut JF, Claessens Y-E, Ginsberg C, Riou B. Unprecedented heat-related deaths during the 2003 heat wave in Paris: consequences on emergency departments. *Crit Care*. 2004;8:1–2.
30. Stafoggia M, Forastiere F, Berti G, Bisanti L, Cadum E, Caranci N, et al. Factors associated with heat-related in-hospital mortality: a multicity case-crossover analysis. *Epidemiology*. 2006;17:S163–4.
31. Michelozzi P, Accetta G, De Sarlo M, D'Ippoliti D, Marino C, Baccini M, et al. High temperature and hospitalizations for cardiovascular and respiratory causes in 12 European cities. *Am J Respir Crit Care Med*. 2009;179:383–9.
32. D'Ippoliti D, Michelozzi P, Marino C, de'Donato F, Menne B, Katsouyanni K, et al. The impact of heat waves on mortality in 9 European cities: results from the EuroHEAT project. *Environ Health*. 2010;9:37.
33. Filleul L, Cassadou S, Medina S, Fabres P, Lefranc A, Eilstein D, et al. The relation between temperature, ozone and mortality in nine French cities during the heat wave of 2003. *Environ Health Perspect*. 2006;114:1344–7.
34. Ren C, Williams GM, Morawska L, Mengersen K, Tong S. Ozone modifies associations between temperature and cardiovascular mortality: analysis of the NMMAPS data. *Occup Environ Med*. 2008;65:255–60.
35. Schwartz J, Samet JM, Patz JA. Hospital admissions for heart disease: the effects of temperature and humidity. *Epidemiology*. 2004;15:755–61.
36. Medina-Ramon 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;114:1331–6.
37. Qian Z, He Q, Lin HM, Kong L, Bentley CM, Liu W, et al. High temperatures enhanced acute mortality effects of ambient particle pollution in the “oven” city of Wuhan, China. *Environ Health Perspect*. 2008;116:1172–8.
38. Hansen A, Bi P, Nitschke M, Ryan P, Pisaniello D, Tucker G. The effect of heat waves on mental health in a temperate Australian city. *Environ Health Perspect*. 2008;116:1369–75.
39. Greene S, Kalkstein LS, Mills DM, Samenow J. An examination of climate change on extreme heat events and climate-change mortality relationships in large U.S. cities. *Weather Clim Soc*. 2011;3:281–91.
40. 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 USA*. 2012;109:6608–13.
41. Cecchi L, D'Amato G, Ayres JG, Galan C, Forastiere F, Forsberg B, et al. Projections of the effects of climate change on allergic asthma: the contribution of aerobiology. *Allergy*. 2010;65:1073–81.
42. D'Amato G, Cecchi L. Effects of climate change on environmental factors in respiratory allergic diseases. *Clin Exp Allergy*. 2008;38:1264–74.

43. Wayne P, Foster S, Connelly J, Bazzaz FA, Epstein PR. Production of allergenic pollen by ragweed (*Ambrosia artemisiifolia*) is increased in CO₂ enriched atmospheres. *Ann Allergy Asthma Immunol.* 2002;88:279–82.
44. 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;32:667–70.
45. Beggs PJ, Bambrick HJ. Is the global rise of asthma an early impact of anthropogenic climate change? *Environ Health Perspect.* 2005;113:915–9.
46. Haines A, Patz JA. Health effects of climate change. *JAMA.* 2004;291:99–103.
47. Patz JA, Olson SH. Malaria risk and temperature: influences from global climate change and local land use practices. *Proc Natl Acad Sci USA.* 2006;103:5635–6.
48. Pascual M, Ahumada JA, Chaves LF, Rodo X, Bouma M. Malaria resurgence in the East African highlands: temperature trends revisited. *Proc Natl Acad Sci USA.* 2006;103:5829–34.
49. Alonso D, Bouma MJ, Pascual M. Epidemic malaria and warmer temperatures in recent decades in an East African highland. *Proc R Soc B.* 2010;278(1712):1661–9.
50. Hales S, de Wet N, Maindonald J, Woodward A. Potential effect of population and climate changes on global distribution of dengue fever: an empirical model. *Lancet.* 2002;360:830–4.
51. Costello A, Abbas M, Allen A, Ball S, Bellamy R, Friel S, et al. Managing the health effects of climate change. *Lancet.* 2009;373:1693–733.
52. Pacala S, Socolow R. Stabilization wedges: solving the climate problem for the next 50 years with current technologies. *Science.* 2004;305:968–72.
53. Rooney RC, Bayley SE, Schindler DW. Oil sands mining and reclamation cause massive loss of peatland and stored carbon. *Proc Natl Acad Sci USA.* 2012;109:4933–7.

Chapter 2

Climate Variability and Change Data and Information for Global Public Health

Juli M. Trtanj and Tamara G. Houston

Abstract Using climate data correctly is a critically important challenge that underpins robust science and decision making about the health effects of climate change. Researchers in this interdisciplinary field must be informed enough to ask the right questions, to find and understand the right data that ultimately provide scientifically sound information to help people make the right decision. This requires active recognition of the need to really understand the caveats and best uses of a particular dataset or product. Some more widely used data and products such as those developed for the Intergovernmental Panel on Climate Change may have well-defined tutorials and use parameters. In most cases, however, it is wiser to find the owner or originator of the data, and work with them to ensure appropriate use of the data and therefore robust scientific findings that inform decisions and move this interdisciplinary field forward in both science and policy contexts.

Keywords Climate variability and health • Climate and Health • Global public health • Health consequences of climate variability • Climate data • Global Ocean Observing System • National Oceanic and Atmospheric Administration

One of the great challenges in understanding the health consequences of climate variability and change is the paucity of temporally and spatially compatible data to underpin evidence-based scientifically sound knowledge and action. Robust results

J.M. Trtanj (✉)

National Oceanic and Atmospheric Administration,
National Centers for Coastal Ocean Science, 1305 East-West Highway,
SSMC4-9143, Silver Spring, MD 20910, USA
e-mail: juli.trtanj@noaa.gov

T.G. Houston

National Oceanic and Atmospheric Administration,
National Climatic Data Center, 151 Patton Avenue Asheville, NC 28801, USA
e-mail: Tamara.Houston@noaa.gov

K.E. Pinkerton and W.N. Rom (eds.), *Global Climate Change and Public Health*,
Respiratory Medicine 7, DOI 10.1007/978-1-4614-8417-2_2,
© Springer Science+Business Media New York 2014

require data from many different disciplines, ranging from medical, epidemiology, social science, environment, oceanography, to climate. Within each of those disciplines, there is yet greater granularity, variability, and quality of data. The key is to have a well-defined problem, ask the right questions to identify the most appropriate data, and find out as much as you can about the data, preferably by reaching the person who owns, collected, or processed the data, and at the very least the metadata manager. This level of data familiarity is critical to continually improve the quality of research in this field, and support greater knowledge about health consequences and adaptation options. Too often those in a specific discipline think their data are the most complex or difficult and will think it straightforward to simply download or use data from another discipline, do their analysis, and publish the results without a clear understanding of the data and its limitations. The reality is that most datasets are complex and have significant strengths and weaknesses. Knowing how and when to use them appropriately is critical. Otherwise, the result is often erroneous conclusions about causality, or mechanism, which fundamentally detracts from the scientific rigor that underpins this interdisciplinary community.

Climate Data

This chapter is designed to provide a common understanding of climate terminology, climate data, and to highlight the major, long-standing data and modeling centers through which climate data and models are available. Even within the climate and weather community there is often not consensus about the definitions that follow. This chapter is intended to provide general guidelines and definitions that harmonize terminology across the physical and biological sciences to facilitate more fruitful interactions.

Data Cultures

Just as epidemiology is the study of patterns of disease in a specific population at a specific location over a specific period of time; person, place, and time, climatology is somewhat similar in concept, but differs greatly in approach. One of the biggest differences—and opportunities—between the climate and health communities is the approach to data—volume, scale, scope, frequency, continuity, and treatment. The climate community has a culture of voluminous data collection through targeted and sustained in situ, space-based and airborne platform observations, data management, archiving, reanalysis, and creating modeled datasets. Data management is a highly respectable career; Entire highly respectable careers are spent on data management. International cooperation is built around data sharing (see GEO). Supercomputer power is critical to manage and model it. In contrast, health data tend to be event and illness specific, often without continuous collection over long time periods that establish to baseline conditions, and usually without any geo-referenced environmental parameters. Actual health outcome data may even be more sparse, or due to privacy

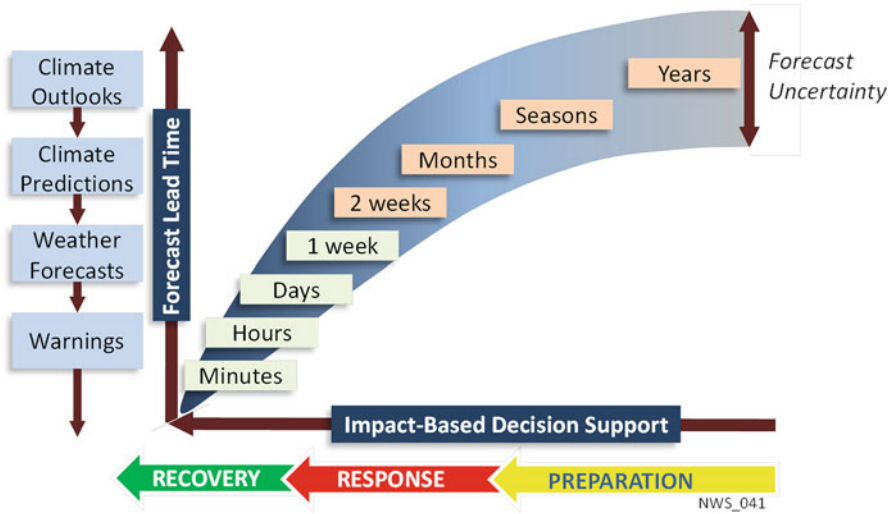


Fig. 2.1 NOAA seamless suite of forecasts (Courtesy of National Oceanic and Atmospheric Administration, www.noaa.gov/)

issues, unavailable at all. Multidisciplinary collaboration will require each of us to learn from the other to improve data collection, access and ultimately the public health usefulness, let’s learn from each other and together tackle this data disconnect.

Defining Terms

Understanding and using the correct terminology will greatly facilitate communication across disciplines, the development of a robust problem statement, and identification of appropriate data to use in answering that problem. Climate is a continuum encompassing short-term weather to seasonal, decadal, and long-term changes in the climate system. On top of this is layered the operative functional capacity, i.e., forecast, early warning, prediction, and scenario, with each having associated levels of uncertainty based on the lead time and model error. To really understand the complexity inherent in these coupled human and natural systems requires the consideration of other social and economic factors. Figure 2.1 provides an overview of the relationship between time scale and uncertainty.

Weather is the day-to-day state of the atmosphere, at a specific place and time, and its short-term (minutes to days) variation. Weather is described as the combination of temperature, humidity, precipitation, cloudiness, visibility, and wind speed and direction. We talk about the weather in terms of “What will it be like today?” “How hot is it right now?” and “When will that storm hit our section of the country?” [1].

Climate is the slowly varying aspect of the atmosphere-hydrosphere-land surface system, defined as statistical weather information that describes the variation of weather at a given place for a specified interval. It is typically characterized in terms of averages of

specific states of the atmosphere, ocean, and land, including variables such as temperature (land, ocean, and atmosphere), salinity (oceans), soil moisture (land), wind speed and direction (atmosphere), and current strength and direction (oceans). In popular usage, it represents the synthesis of weather; more formally it is the weather of a locality averaged over some period (usually 30 years) plus statistics of weather extremes [2].

Local or regional climate is in terms of the averages of weather elements, such as temperature and precipitation, derived from observations taken over a span of many years. In this empirically based context, climate is defined as weather (the state of the atmosphere) at some locality averaged over a specified time interval. Climate must be specified for a particular place and period because, like weather, climate varies both spatially and temporally [3].

In the most general sense, the term *climate variability* denotes the inherent characteristic of climate which manifests itself in changes on seasonal, interannual, decadal and multidecadal time scales. These climate variability phenomena which affect weather includes regimes such as the El Niño-Southern Oscillation (ENSO), Madden-Julien Oscillation (MJO), Atlantic Oscillation (AO), North Atlantic Oscillation (NAO), Pacific North American Oscillation (PNA). A suite of weather and climate forecast products can be found at <http://www.cpc.ncep.noaa.gov/products/forecasts/>. The degree of climate variability can be described by the differences between long-term statistics of [meteorological elements](#) calculated for different periods. The term *climate variability* is often used to denote deviations of climate statistics over a given period of time (such as a specific month, season, or year) from the long-term climate statistics relating to the corresponding calendar period. In this sense, climate variability is measured by those deviations, which are usually termed anomalies [4].

Climate change is a change in the statistical distribution of weather over periods of time that range from decades to millions of years [5]. Climate change is expressed in terms of years, decades, or even centuries—but its impacts can be felt in the present. Scientists study climate to look for trends or cycles of variability (such as the changes in wind patterns, ocean surface temperatures, and precipitation over the equatorial Pacific that result in El Niño and La Niña) and also to place cycles or other phenomena into the bigger picture of possible longer term climate changes [2]. Epidemiologists too look for trends or cycles of disease incidence or patterns of outbreak.

Global warming is the gradual increase in the average temperatures of Earth's near-surface air and oceans since the mid-twentieth century and its projected continuation [5].

Early Warning, Outlook, Prediction, Forecast, Projection, and Scenario

In addition to the basic definitions, the application of those terms to a suite of predictive tools across time scales warrants similar clarification.

Early warning can mean basic monitoring, forecasts, or predictions that provide advance notice to decision makers that allow preventive action to take place. This can cross time scales ranging from tornado warning to a risk map of potential pathogenic vibrio affecting shellfish, or using an El Niño forecast to help manage malaria risk.

Outlooks are typically on of for one to thirteen months in the future. Extended range outlooks for 6-10 and 8-14 days also exist for degree days, drought, and soil moisture. (cite <http://www.cpc.ncep.noaa.gov/products/forecasts/>) weeks to monthly to seasonal time scales.

Climate prediction is generally intraseasonal to seasonal to interannual. A *prediction* is a probabilistic statement that something will happen in the future based on what is known today and is most influenced by the initial, or current, conditions. A prediction generally assumes that future changes in related conditions will not have a significant influence. For example, a weather prediction indicating whether tomorrow will be clear or stormy is based on the state of the atmosphere today (and in the recent past) and not on unpredictable changes in “boundary conditions” such as how ocean temperatures or even society may change between today and tomorrow. For decision makers, a prediction is a statement about an event that is likely to occur no matter what they do [6].

Climate predictions are usually expressed in probabilistic terms (e.g., probability of warmer or wetter than average conditions) for periods such as weeks, months, or seasons. A prediction is a probabilistic statement of something that could happen in the future based only on what is known today. *Climate projections* are long-range predictions of the future climate based on changing atmospheric conditions, such as increased or decreased pollutants due to emissions from the burning of fossil fuels (coal, oil, gas) [7].

Forecasts are typically on weather time scales (daily and out 7–10 days). In cases of extreme weather events such as hurricanes or tornados, the forecasts can be less than hourly with frequent updates. Related to a prediction is a *forecast*, which I would suggest is a best prediction made by a particular person or with a particular technique or representation of current conditions. An example of a forecast is a statement by a weather forecaster that it will rain at 3:30 PM tomorrow—that is, that individual’s best judgment, perhaps drawn from a prediction that there is a 70 % chance of rain tomorrow afternoon. For a decision maker, the credibility of the forecast depends critically on the credibility of the forecaster (or forecasting technique) as well as on the inevitability of the event. The recent development of “ensemble forecasts” (i.e., assembly of a set of forecasts that are each based on a separate technique or set of initial conditions) can be considered a step toward transforming forecasts into predictions.

Climate projections are generally decadal to centennial. In contrast to a prediction, a *projection* specifically allows for significant changes in the set of “boundary conditions” that might influence the prediction, creating “if this, then that” types of statements. Thus, a *projection is a probabilistic statement that it is possible that something will happen in the future if certain conditions develop*. The set of

boundary conditions that is used in conjunction with making a projection is often called a scenario, and each scenario is based on assumptions about how the future will develop. For example, the IPCC recently *projected* a range of possible temperature changes that would likely occur for a range of plausible emissions scenarios and a range of model-derived estimates of climate sensitivity (the temperature change that would result from a CO₂ doubling). This is clearly a projection of what *could* happen *if* certain assumed conditions prevailed in the future—it is neither a prediction nor a forecast of what will happen independent of future conditions. For a decision maker, a projection is an indication of a possibility and normally of one that could be influenced by the actions of the decision maker [6].

A *scenario* is a coherent, internally consistent, and plausible description of a possible future state of the world. It is not a forecast; rather, each scenario is one alternative image of how the future can unfold. A projection may serve as the raw material for a scenario, but scenarios often require additional information (e.g., about baseline conditions). A set of scenarios is often adopted to reflect, to the extent possible, the range of uncertainty in projections. For instance, the Intergovernmental Panel on Climate Change will run several scenarios with different boundary conditions such as emissions and economic growth rates. Other terms that have been used as synonyms for scenario are “characterization,” “storyline,” and “construction.”

Scenarios are best thought of as “plausible alternative futures—each an example of what might happen under particular assumptions”; scenarios are not predictions or forecasts because they depend on assumed changes in key boundary conditions (like emissions) and scenarios are not fully projections of what is likely to happen because they have considered only a limited set of possible future boundary conditions (e.g., emissions scenarios). For the decision maker, scenarios provide an indication of possibilities, but not definitive probabilities. For instance, the Intergovernmental Panel on Climate Change will run several scenarios with different boundary conditions such as emissions and economic growth rates [8]. In a public health context, a scenario may be an attempt to simulate a certain event or decision-making exercise, which is very different from how scenario is used in the climate context.

How to Think About Climate Data: or When to Use What

Climate data are comprised of many different types, scales, and resolution of data, derived from multiple sources (satellite or in situ) and made available through a number of products and service modes.

Scale

Climate data can be global, regional, or local in scale and is comprised of oceanic, atmospheric, and terrestrial data. Within that are mostly physical parameters such as precipitation, temperature (atmospheric and oceanic), sea level, waves, and winds. While collected separately, the data streams can be part of the same satellite or field

collection effort. The different data streams are then combined to make climate data products and models. Scale is largely dependent on the means by which the data are collected (satellite or in situ observations), the area of coverage, and density of collection sites for in situ observations or grid size for satellites.

Source

Data are collected or provided from multiple sources: satellite or space-based sensors, airborne platforms, in situ, modeled, reanalyzed, and projections. Satellites provide periodic but global coverage from polar orbital satellites or consistent coverage over specific parts of the globe through geostationary satellites. Polar orbital satellites provide total earth coverage but will measure the same place twice each day at the same local time, every 12 hours, as part of their low earth orbit (approximately 500 miles altitude) moving from North Pole to South Pole. Because of their lower altitude, polar orbital satellites can use microwave radiometers which allows them to measure through clouds to sense precipitation, temperature in different layers of the atmosphere, and surface characteristics like ocean surface winds. Geostationary satellites are fixed high above the equator (approximately 22,000 miles altitude) providing continuous coverage of the same area, but the resolution is generally 1 km at best, and coverage is not global. In general, for climate and weather purposes, the National Aeronautics and Space Administration (NASA) launches research satellites mostly in polar orbital and in lower earth orbit. The National Oceanic and Atmospheric Administration (NOAA) operates the satellites needed for weather and climate predictions which include geostationary satellites.

In situ data are collected from ground, water-based, or airborne instruments and sensors. Availability varies by country, both in temporal and spatial coverage, and access. The quality varies according to the instrumentation and human skill in collection and recording. Metadata may or may not be available, and upkeep, updates, and archiving are problematic for many countries. In situ data are useful alone, can be combined with other data into more comprehensive products, and can be used to validate and enhance satellite data. The networks and instruments for in situ data collection vary widely and include everything from permanent weather stations, to tide gauges, to drifting buoys in the ocean and ships of convenience, to the atmospheric radiation, temperature and carbon dioxide measurements at Mauna Loa Observatory in Hawaii which has tracked CO₂ since the 1950s.

Products

Data can also be processed into products such as sea surface temperature (SST), SST anomalies (commonly depicted during El Niño and La Niña events), vegetation indices, and sea ice (see <http://www.realclimate.org/index.php/data-sources/> for additional products). A suite of climate and weather forecast products can be found at <http://www.cpc.ncep.noaa.gov/products/forecasts/>. One of the most well-known

Table 2.1 Global Ocean Observing System (GOOS) in situ measurements

3,000 Argo floats collect high-quality temperature and salinity profiles from the upper 2,000 m of the ice-free global ocean and currents from intermediate depths
1,250 drifting buoys record the currents of surface, the temperature, and the atmospheric pressure
350 embarked systems on commercial or cruising yachts which collect the temperature, salinity, the oxygen and the carbon dioxide (CO ₂) in the ocean and the atmosphere, and the atmospheric pressure
100 research vessels measure all the physical, chemical, and biological parameters, between the surface of the sea and the ocean floors every 30 nautical miles out of 25 transoceanic lines
200 marigraphs and holographs which transmit information in quasi real time, thus providing the possibility of detecting tsunamis
50 commercial ships which launch probes measuring the temperature and salinity between the surface and the ocean floor on their transoceanic ways
200 moorings in open sea which are used as long-term observatories, recording weather, chemical, and biological parameters on a fixed site between the surface and the bottom

datasets is the Global Historical Climatology Network (GHCN) dataset, which is a global, daily in situ dataset derived from multiple sources, approximately 25,000 temperature stations, 44,000 precipitation stations, and 25,000 snowfall or snow depth stations, and currently ingests more than 1.6 billion daily observations with the earliest value from January 2, 1833 and the latest value from yesterday.

The scientific community has established three global networks for terrestrial, oceanographic, and climate data. The Global Ocean Observing System (GOOS) is a permanent global system for observations, modeling, and analysis of marine and ocean variables to support operational ocean services worldwide (Table 2.1). GOOS is comprised of a network of ocean-based observations and satellite observations and together with the Global Climate Observing System (GCOS), and the Global Terrestrial Observing System (GTOS) comprise a global network of monitoring to understand and predict climate, among other things.

Reanalysis

In order to create consistent and comparable global datasets, major efforts are made by the climate community to create reanalysis datasets. These are weather models which have the real-world observations assimilated into the solution to provide a “best guess” of the evolution of weather over time (although pre-satellite era estimates before 1979 are less accurate). The newest as of this writing is the NCEP/NCAR reanalysis with 6-h, daily, and monthly data available [9].

Projections

Data are also generated through climate projections and scenarios. A climate projection is a model-derived estimate of the future and the pathway leading to it. When the certainty around a projection is determined, with levels of certainty assigned such as ‘most likely’, the projection can become a forecast or prediction.

A forecast is often obtained using deterministic models, possibly a set of these, outputs of which can enable some level of confidence to be attached to projections. General Circulation Models (GCMs) are numerical models that represent the physical processes in the atmosphere, ocean, cryosphere, and land surface are the most advanced tools currently available for simulating the response of the global climate system to increasing greenhouse gas concentrations. While simpler models have also been used to provide globally or regionally averaged estimates of the climate response, only GCMs, possibly in conjunction with nested regional models, have the potential to provide geographically and physically consistent estimates of regional climate change which are required in impact analysis; GCMs depict the climate using a three-dimensional grid over the globe typically having a horizontal resolution of between 250 and 600 km, 10–20 vertical layers in the atmosphere, and sometimes as many as 30 layers in the oceans. Many physical processes and feedback mechanisms such as water vapor and warming, or clouds and radiation, occur at smaller scales and cannot be properly modeled. Instead, their known properties must be averaged over the larger scale in a technique known as parameterization, which are sources of uncertainty in GCM-based simulations of future climate.

Assessing Climate Data Partners

NOAA not only houses much of the climate, weather, and ocean data for the United States but also serves as the main repository for the World Meteorological Organization and other international bodies. In the United States there is a three-tiered climate services support program. The partners of this program include NOAA's National Climatic Data Center (NCDC—<http://www.ncdc.noaa.gov/>), six Regional Climate Centers (RCCs—<http://www.ncdc.noaa.gov/oa/climate/regionalclimatecenters.html>), and individual State Climate Offices (SCO—<http://www.stateclimate.org/>). NCDC is the world's largest active archive of weather data with over 150 years of in situ, radar, and satellite data available for use in a wide variety of applications. The RCCs are a federal-state cooperative effort that is managed by NCDC. The RCCs are engaged in the timely production and delivery of useful climate data, information, and knowledge for decision makers and other users at the local, state, and national level. The RCCs support NOAA's efforts to provide operational climate services while leveraging improvements in technology and collaborations with partners to expand quality data dissemination capabilities. State Climatologists have the best understanding of the climate of their state and the ability and knowledge to provide climate data and information to local users. Additional NOAA climate partners include the National Weather Service Climate Services Division (<http://www.nws.noaa.gov/os/csd/index.php>), the Climate Prediction Center (<http://www.cpc.ncep.noaa.gov/>), the Climate Diagnostics Center (<http://cires.colorado.edu/science/centers/cdc/>), the Climate Program Office (<http://www.climate.noaa.gov/>), and six Regional Climate Service Directors that are located at the NWS Regional Headquarters.

Some applications require data and information for areas outside of the United States. While the agencies mentioned above focus primarily at the national, regional, and local level, some do participate in international activities as well. For example, NCDC

operates a World Data Center for Meteorology (<http://www.ncdc.noaa.gov/oa/wdc/index.php>) and a World Data Center for Paleoclimatology. The World Data Centers are part of a global network of discipline subcenters that facilitate international exchange of scientific data. The World Meteorological Organization also maintains a list of member National Meteorological or Hydrometeorological Services (http://www.wmo.int/pages/members/members_en.html) in which users can go directly to the country of interest in order to obtain weather and climate data and information for their application.

Global Observing Systems Information Center is a one-stop shop for the GOOS, GCOS, and GTOS (<http://gosic.org/goos>).

Conclusion

In summary, climate data comes from multiple sources, can be observed data or modeled, covers time scales from weeks, to decades to centuries, and can provide a powerful tool for enhanced decision making. Researchers in this interdisciplinary field must be well-versed enough to ask the right questions that lead them to find and understand the right data, and which ultimately to provide scientifically sound information to help people make the right decision. This requires active recognition of the need to really understand the caveats and best uses of a particular dataset or product. In general, while some more widely used data and products such as those developed for the Intergovernmental Panel on Climate Change may have well-defined tutorials and use parameters, in general it is wiser to find the owner or originator of the data and work with them to ensure appropriate use of the data and therefore robust scientific findings that both inform decisions and move this interdisciplinary field forward in both science and policy contexts.

References

1. http://nsidc.org/arcticmet/basics/weather_vs_climate.html), <http://www.ametsoc.org/amsedu/online/climateinfo/samplecourse/Ch01-1stEd.pdf>
2. http://nsidc.org/arcticmet/basics/weather_vs_climate.html
3. <http://www.ametsoc.org/amsedu/online/climateinfo/samplecourse/Ch01-1stEd.pdf>
4. http://nsidc.org/arcticmet/glossary/climate_variability.html
5. American Public Health Association. 2012. Climate change: Mastering the public health role. http://www.apha.org/advocacy/reports/webinars/webinars_2012.html
6. <http://sciencepolicy.colorado.edu/zine/archives/1-29/26/correspond.html>
7. http://www.nws.noaa.gov/om/csd/graphics/content/outreach/brochures/Weather&Climate_General_Public.pdf
8. WeatherZine, Number 26, NCAR. 2001. <http://sciencepolicy.colorado.edu/zine/archives/1-29/26/correspond.html>
9. <http://www.esrl.noaa.gov/psd/data/reanalysis/reanalysis.shtml>

Chapter 3

Climate Change: Overview of Data Sources, Observed and Predicted Temperature Changes, and Impacts on Public and Environmental Health

David H. Levinson and Christopher J. Fettig

Abstract This chapter addresses the societal and the environmental impacts of climate change related to increasing surface temperatures on air quality and forest health. Increasing temperatures at and near the earth's surface, due to both a warming climate and urban heat island effects, have been shown to increase ground-level ozone concentrations in cities across the U.S. In terms of forest health, elevated surface air temperatures and increased water stress are raising the possibility that forests world-wide are increasingly responding to warming climate conditions, which may lead to widespread tree mortality. The importance of climate datasets is also addressed, specifically as it relates to understanding the observed and predicted changes in surface temperatures at the global, regional and local scale.

Keywords Anthropogenic-induced changes • Forest health • Change in surface temperature • Changing distribution of conifers • Phytophagous insects • Climate-induced forest mortality

Anthropogenic-induced changes to the earth's climate are among the most complex and difficult issues to be addressed by modern science and the scientific community. Small changes in the concentrations of atmospheric gases, such as carbon dioxide (CO₂) and methane (CH₄), have large impacts on society, ecosystems, and the hydrologic cycle [1]. Given the magnitude of observed and potential impacts,

D.H. Levinson, M.S., Ph.D. (✉)
Watershed, Fish, Wildlife, Air and Rare Plants, USDA Forest Service,
2150A Centre Avenue, Fort Collins, CO 80526, USA
e-mail: dlevinson@fs.fed.us

C.J. Fettig, M.S., Ph.D.
Pacific Southwest Research Station, USDA Forest Service,
1731 Research Park Drive, Davis, CA 95618, USA
e-mail: cfettig@fs.fed.us

numerous interagency and international efforts have been initiated over the past 3 decades to attempt to analyze every aspect of the earth's climate and to address the adaptation and mitigation options that have the potential to aid in addressing this important challenge. The most well known of these efforts is the Intergovernmental Panel on Climate Change (IPCC), formed by the United Nations in 1988 to help address the scientific, economic, and policy aspects of global climate change.

In this chapter, several areas of research will be elucidated that address both the societal and the environmental impacts of climate change. Specifically, these are the impacts related to increasing surface temperatures on air quality and forest health. The importance of quality climate datasets is also addressed as it relates to understanding the observed and predicted changes in surface temperatures at the global, regional, and local scale.

Observations of Changes in Surface Temperature

Previous studies, including both international assessments and independently published peer-reviewed articles, have demonstrated that surface temperatures have increased globally by approximately 0.7 °C per century since 1900 and 0.16 °C per decade since 1970 [2, 3] (Fig. 3.1). The slight differences in the estimates of annual means, rankings, and trends in global surface temperatures are the result of differences in the methods used to construct each of the three primary independent datasets that determine surface temperatures spatially across global ocean and land areas [4]. These three global datasets are those developed and maintained by NASA-GISS [5], HadCRUT3 [6], and NOAA-NCDC [7]. Despite the observed differences that result in variations in annual rankings of global surface temperature, each of these datasets is in close agreement, and all three have identified 2010 as tied for the warmest year or ranked as second warmest in the historical record since 1880 (Table 3.1).

Increased occurrences of public health and environmental impacts due to changes in climate over the past several decades have been attributed to rising surface and lower tropospheric temperatures, and these impacts include heat stress and increased occurrence of heat waves, respiratory stress due to degraded air quality conditions, impacts on food safety and water quality, increasing aeroallergens and pollen sources, and the spread of vector-borne diseases [8]. However, in most cases the impacts are primarily related to increasing extreme temperatures, specifically increases in the daily maximum temperature, rising nocturnal temperature, or both [3, 9, 10]. Figure 3.2 shows the global trend in the maximum and minimum temperature, along with the diurnal temperature range (DTR) ($T_{\max} - T_{\min}$) over the period 1950–2004 [9]. The observed decrease in the DTR is primarily a result of larger increases in the minimum temperature at land-based observing sites. Since the heat-related mortality is correlated primarily by nocturnal temperatures, the increases in minimum temperatures are of widespread concern [11–13].

For most applications related to analyzing regional or local public health and the environmental impacts of climate change, it is important to use those data sources that provide the highest quality information and rigorous quality assurance and

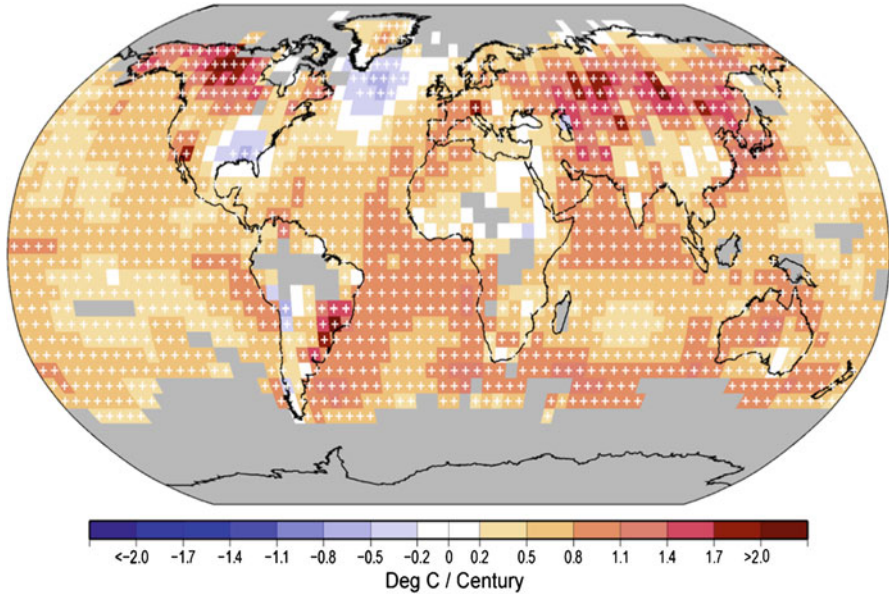


Fig. 3.1 Observed trend in annual average surface air temperature (°C per century) over the period 1901–2005 using the NOAA-NCDC Global Blended Dataset [71] and adapted from Trenberth et al. [2] (Fig. 3.9). Trends significant at the $\alpha=5\%$ level are indicated by white “plus” signs, and grey areas have insufficient data to determine statistically reliable trends. Requirements for inclusion were a minimum of 66 years needed to calculate a trend value and 10 valid monthly temperature anomaly values needed for inclusion of an individual year (adapted from Trenberth KE, Jones PD, Ambenje P, Bojariu R, Easterling D, Klein Tank A, Parker D, Rahimzadeh F, Renwick JA, Rusticucci M, Soden B, Zhai P (2007) Observations: Surface and Atmospheric Climate Change. 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 [Solomon S, Qin D, Manning M, Chen Z, Marquis M, Averyt KB, Tignor M, Miller HL (eds.)]. Cambridge University Press, Cambridge, United Kingdom and New York, NY, USA, with permission)

Table 3.1 The observed differences in annual global temperature anomaly for 2010 and its rank relative to the entire historical record since 1880 for the three primary datasets used to determine global average temperatures

	2010 Global anomaly relative to the 1961–1990 annual mean	Rank of 2010 to all years since 1880
HadCRUT3	0.50 °C	Second warmest after 1998
NASA-GISS	0.56 °C	Tied warmest with 2005
NOAA-NCDC	0.52 °C	Tied warmest with 2005

From Sanchez-Lugo A, Kennedy JJ, Berrisford P (2011) Surface temperatures. In “State of the Climate 2010,” *Bull Amer Meteor Soc* 92:6:S36-S37, with permission

quality control (QA/QC) methods. For surface temperature, that is the NOAA Global Historical Climate Network (GHCN) dataset [14] and its subset the US Historical Climate Network (USHCN) dataset. NOAA’s GHCN Monthly data (versions 2 and 3) can be accessed at <http://www.ncdc.noaa.gov/ghcnm/>, the routinely

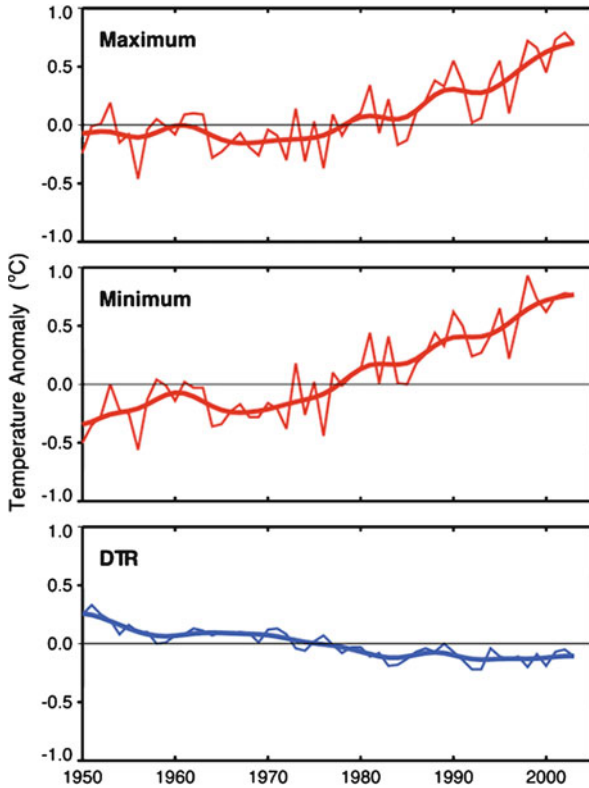


Fig. 3.2 Global annual anomalies of surface maximum (*top*), minimum (*middle*), and diurnal temperature range (DTR, *bottom*) in °C, over the period 1950–2004, with the *thinner line* the annual values and the *thicker line* showing the smoothed, decadal variations. Anomalies were determined relative to the 1961–1990 mean and averaged over the 71 % of land areas where data were available during the period of record (adapted from Vose RS, Easterling DR, Gleason B (2005) Maximum and minimum temperature trends for the globe: An update through 2004. *Geophys Res Lett* 32:L23822, with permission)

updated GHCN-Daily dataset can be found online at <http://www.ncdc.noaa.gov/oa/climate/gHCN-daily/>, and the USHCN dataset is available at <http://cdiac.ornl.gov/epubs/ndp/ushcn/ushcn.html>. These high-quality, integrated climate datasets provide the researcher the requisite information regarding the construction, maintenance, and historical provenance of data sources that are needed for reliable analysis of observed changes in temperatures at the global, regional, or local scale. To illustrate the variability of climate changes related to surface temperature, Fig. 3.1 shows the spatial variation in surface temperature trends covering the period from 1901 to 2005; the vast majority of the earth’s surface has warmed since the start of the twentieth century, with the largest increases observed at continental mid- to high-latitudes in the Northern Hemisphere. Only a few areas have shown a decreasing trend in

surface temperatures, and the vast majority of the surface temperature trends (both positive and negative) are statistically significant at the 95 % confidence level.

Predictions of Changes in Surface Temperature

Despite the well-documented uncertainty in the simulations of future climate conditions, associated with different emissions scenarios [15], it is clear that global temperatures will continue to rise due to the increasing radiative effects of greenhouse gases, primarily a result of increases in CO₂ but also increases in other greenhouse gases such as methane (CH₄), nitrous oxide (N₂O), and halocarbons. Based on simulations realized from multiple ensembles of global circulation models (GCMs), surface temperatures are predicted to continue to rise over the remainder of the twenty-first century. Predictions from the most recent IPCC report (AR4) include the following statement regarding the magnitude of the change expected: “Continued greenhouse gas emissions at or above current rates will cause further warming, and induce many changes in the global climate system during the twenty-first century that would *very likely* be larger than those observed during the twentieth century” [15].

In terms of the impacts of these warming temperatures, predicted increases in global temperatures due to a warming climate in the twenty-first century will result in an increase in heat waves, often measured as the number of days that maximum temperatures exceed 100 °F (37.78 °C), which are predicted to increase significantly for the USA. Figure 3.3 shows the occurrence of days exceeding 100 °F over the USA during a recent period in the past (1961–1979), compared with two different scenarios for the end of the twenty-first century based on a low and a high emissions scenario. In both cases, the number of days that are predicted to exceed 100 °F will increase, but as expected the increase is more dramatic with the higher emissions scenario. In both scenarios, large areas of the continental USA will experience a dramatic increase in heat waves (Fig. 3.3).

The rise in extreme temperatures and their potential impacts are of growing concern, given that the increasing temperatures across the USA are expected to accelerate between the middle (by 2050, using a 2041–2059 average) and the end of the twenty-first century (by 2090, using a 2081–2099 average) (Fig. 3.4). As shown in Fig. 3.5, the precise rise in temperature will depend largely on the eventual increase in greenhouse gas concentrations, which will depend on the future path of global emissions of CO₂, CH₄, and other greenhouse gases. Lower emissions will result in a smaller rise in surface temperatures, while larger increases in emissions of greenhouse gases will lead to more significant rises in surface temperatures. Either way, it is imperative to improve the scientific understanding of the observed and potential impacts of climate change, given the widespread potential for significant impacts to society and the environment. To address this issue, the following sections present the observed and potential future impacts on air quality and forest health, two areas of extensive research over the past several decades.

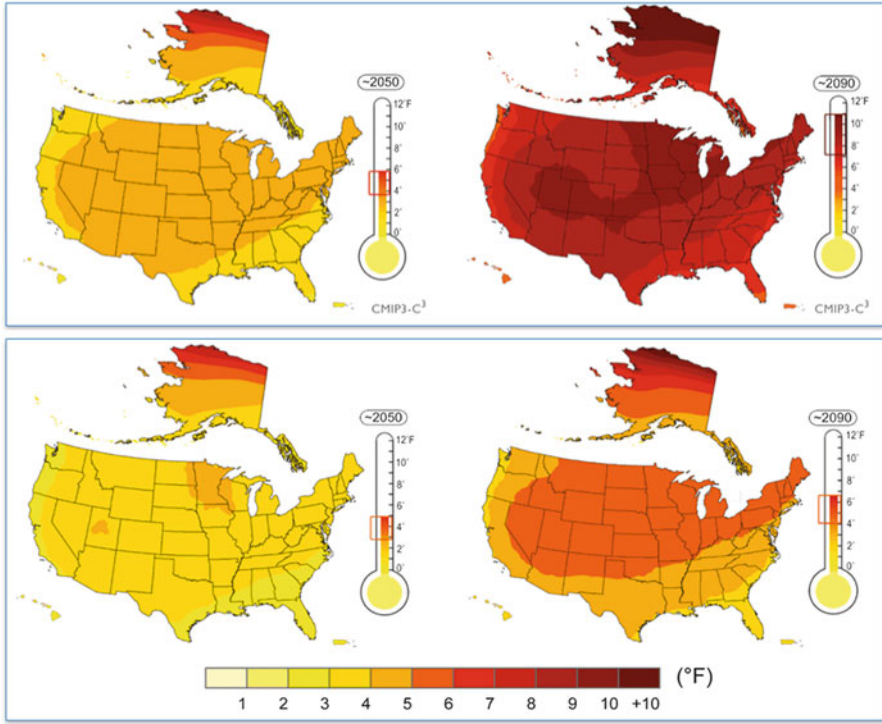


Fig. 3.3 Projected changes of the US (including Alaska and Hawaii, inset) surface air temperature (in °F) relative to the 1961–1979 base period for two different emissions scenarios: higher (*top*) and lower (*bottom*). Emissions scenarios are based on projections of future temperature by 16 of the Coupled Model Intercomparison Project Three (CMIP3) climate models using two emissions scenarios from the IPCC *Special Report on Emissions Scenarios* (Nakićenović N, Swart R (eds.) (2000) *Special Report on Emissions Scenarios*. A special report of Working Group III of the Intergovernmental Panel on Climate Change (IPCC), Cambridge University Press, Cambridge, UK, and New York, NY, USA (http://www.grida.no/publications/other/ipcc_sr/?src=/climate/ipcc/emission/)). The “lower” scenario is B1, while the “higher” is the A2 scenario. The *brackets* on the thermometers represent the likely range of model projections, though lower or higher outcomes are possible (adapted from Karl TR, Melillo JM, Peterson TC (2009) *Global Climate Change Impacts in the United States*, (eds.) Cambridge University Press, with permission)

Climate Change and Air Quality

Climate and weather conditions directly impact air pollutants, specifically their formation, transport, dispersion, and deposition (both wet and dry). Stagnant weather patterns (i.e., light winds due to the influence of surface high-pressure systems and boundary layer inversions) are conducive to the trapping and production of certain atmospheric pollutants that may lead to elevated concentrations of some pollutants, especially ozone (O₃) and particulate matter (PM). Increasing temperatures, due to both a warming climate and urban heat island (UHI) effects, have

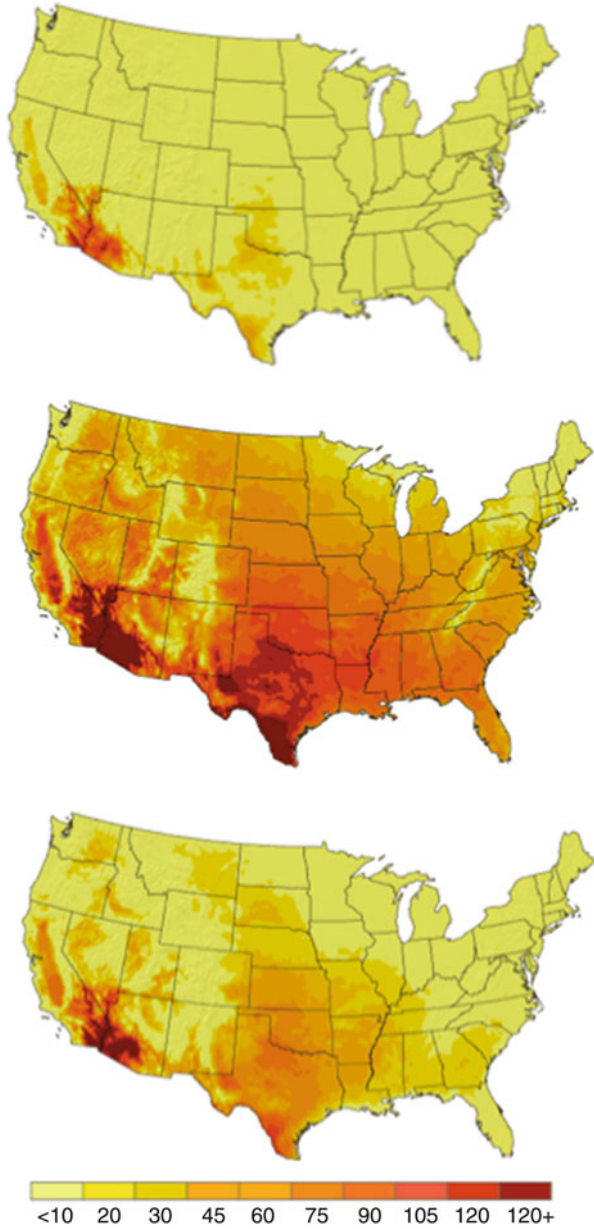


Fig. 3.4 Observed and projected increase in the annual number of days with temperature over 100 °F. The recent past 1961–1979 (*top*) shows significantly less days exceeding 100 °F when compared to the end-of-century (2080–2099) period under both the lower and the higher IPCC emissions scenarios (adapted from Karl TR, Melillo JM, Peterson TC (2009) *Global Climate Change Impacts in the United States*, (eds.) Cambridge University Press, with permission)

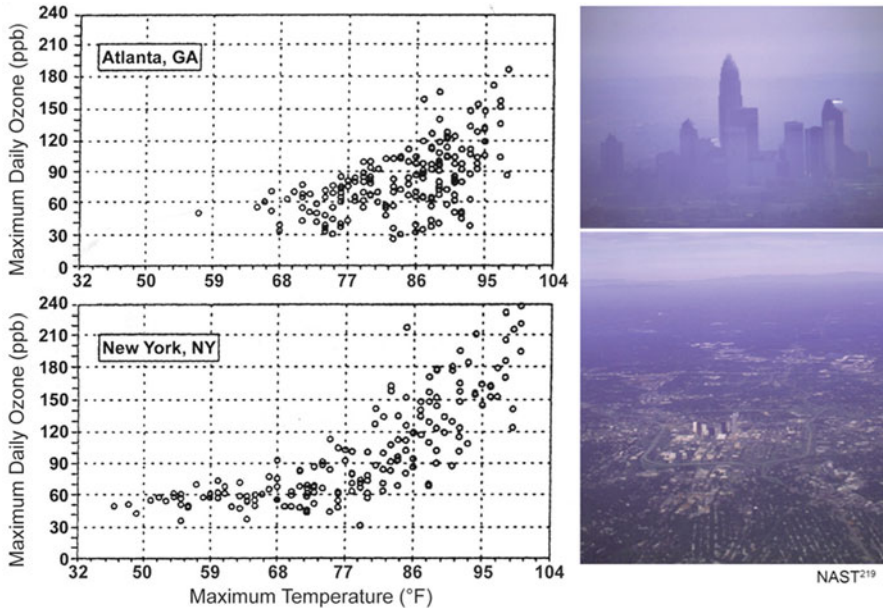


Fig. 3.5 Observed relationship between daily maximum ground-level ozone concentration (in parts per billion, ppb) and maximum surface temperature ($^{\circ}\text{F}$) based on measurements from (*top*) Atlanta, GA, and (*bottom*) New York, NY. Data are for the warm season (May to October) covering the period 1988–1990 at each location. The projected higher temperatures across the USA in the twenty-first century are likely to increase the occurrence of high ozone concentrations, although this will also depend on emissions of ozone precursors and meteorological factors that can enhance or suppress ozone formation in the lower troposphere (<http://www.usgcrp.gov/usgcrp/Library/nationalassessment/>)

been shown to increase concentrations of ground-level ozone [16], since it is both naturally occurring and a secondary pollutant formed through photochemical reactions of sunlight (solar radiation) with nitrogen oxides (N_2O) and volatile organic compounds (VOCs). Previous studies have clearly identified that ozone formation is positively correlated with temperature, but formation is primarily related to incoming shortwave solar radiation, since concentrations are typically highest during the summer months. However, concentrations are not seasonally dependent in all cities with above normal concentrations of ozone, as exceptions have been noted [17].

To illustrate the relationship between ground-level ozone concentrations and surface temperatures, Fig. 3.6 shows data from New York, New York, and Atlanta, Georgia. In terms of the overall relationship, measurements from both cities show that most high ozone level days occur when maximum daily temperatures (T_{max}) are higher and concentrations systematically decrease with lower temperatures. Both cities have the potential for high concentrations, but the data show that the potential for extremely high ozone concentrations (above 200 ppb) is greater in New York, where ground-level concentrations reached 220 ppb when T_{max} was only 85°F (29.5°C). Therefore, the variability of observed daily peak ground-level O_3 concentrations is higher in New York, but the potential for higher concentrations

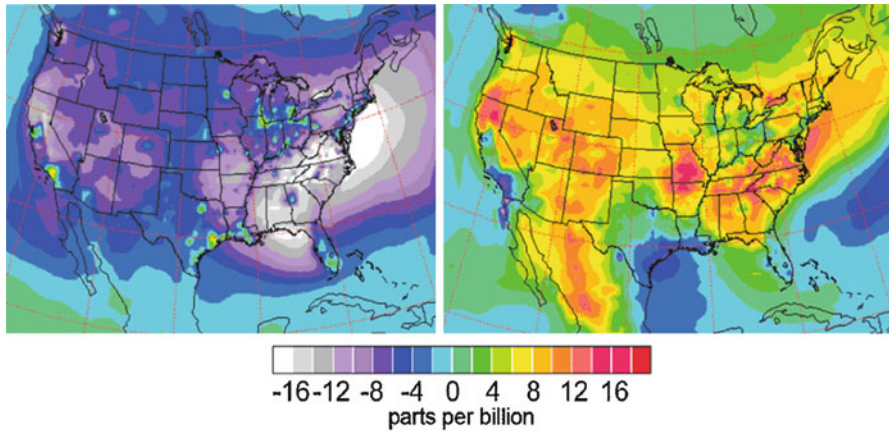


Fig. 3.6 Projected changes in ground-level ozone for the 2090s, averaged over the summer months (June–August) and relative to 1996–2000 under lower and higher emissions scenarios. The scenarios include both greenhouse gases and other emissions that lead to ozone formation, some of which decrease under the lower emissions scenario. By themselves, higher temperatures and other projected climate changes would increase ozone levels under both scenarios. However, future projections of ozone depend heavily on emissions, with the higher emissions scenario increasing ozone by large amounts, while the lower emissions scenario results in an overall decrease in ground-level ozone by the end of the century (adapted from Karl TR, Melillo JM, Peterson TC (2009) *Global Climate Change Impacts in the United States*, (eds.) Cambridge University Press, with permission)

at lower temperatures is more likely in Atlanta. Similar relationships have been found for Los Angeles, California, Phoenix, Arizona, and other cities with well-documented ground-level ozone concentration issues.

The impact of climate change has also been observed on aeroallergens and their sources, as the observed warming has caused an earlier onset of pollens in the spring in the Northern Hemisphere [18–20]. This is due to both the earlier initiation of pollen production in spring and also the introduction and spread of invasive plant species with highly allergenic pollen, such as ragweed (*Ambrosia artemisiifolia*), which is spreading in several areas of the world [21–24]. Laboratory studies have confirmed that increasing CO₂ concentrations and surface temperatures increase the production of ragweed pollen and lengthen the pollen season [25–29]. Therefore, the issue of increased length of the pollen season and the production of pollen from a variety of sources is expected to increase concentrations of aeroallergens in the twenty-first century as the climate continues to warm [8].

Climate Change and Forest Health

Forests cover ~42 million km² (~30 %) of the earth’s surface and are found in all regions at elevations and latitudes capable of sustaining tree growth, except where disturbances, whether natural or human-induced, are too frequent and/or too severe

to enable establishment. Forests provide immeasurable ecological, economic, and social goods and services to both natural systems and humankind. These include, among others, purification of the air that we breathe; regulation of edaphic formation and control of runoff and soil erosion; provision of fish and wildlife habitat; provision of food, medicine, shelter, and water; provision of wood and other forest products; provision of aesthetics, outdoor recreation, and spiritual renewal; and regulation of climate through carbon storage and complex physical, chemical, and biological processes that affect planetary energetics [30]. In short, forests represent one of the earth's most important ecosystems and are critical to the health, welfare, and survival of human societies.

Large amounts of CO₂ are released when forests are burned, defoliated, or deforested and converted to structures that have relatively small carbon pools. In these cases, forests that were once carbon sinks may become carbon sources [31–33]. Alternatively, healthy forests have the potential to assimilate, accumulate, and sequester large amounts of carbon from the atmosphere, thus reducing one of the primary drivers of climate change. We use “forest health” in the context of ecosystems functioning within their natural range of historic variability. The effects of climate change on forest health include both positive (e.g., increased growth through elevated water use efficiency and longer growing seasons) and negative impacts (e.g., increased frequency and severity of disturbances). Forest disturbances (storms, wildfire, herbivory, etc.) are relatively discrete events that affect the structure, composition, and function of forest ecosystems through alterations of the physical environment [34]. They release growing space, alter nutrient cycling, and affect other key processes essential to the proper functioning of ecosystems [35].

Schelhass et al. [36] provided a quantitative overview of the role of natural disturbances in European forests, which they suggested was useful as a basis for modeling the future impacts of climate change by establishing a baseline. They reported storms were responsible for 53 % of the net volume affected over a 40-year period, while biotic factors (e.g., bark beetle outbreaks) contributed 16 %. In the intensively managed forests of Europe and elsewhere (e.g., portions of the USA), natural disturbance cycles have been altered by active management aimed at reducing forest susceptibility to certain types of disturbances. In some cases, human interference in these natural disturbance cycles has later exacerbated their effects. For example, dry forests in portions of the western USA were once dominated by open and parklike stands of widely dispersed trees prior to Euro-American settlement. Frequent thinning of small-diameter and fire-intolerant tree species by low-intensity surface fires and competitive exclusion of tree seedlings by understory grasses are believed to have maintained such conditions. Many of these forests are now denser, have more small trees and fewer large trees, and are dominated by more shade-tolerant and fire-intolerant tree species, primarily as a result of fire suppression activities and harvesting practices implemented in the twentieth century. These changes have led to heavy accumulations of forest fuels [37] that feed severe wildfires when natural- or human-induced ignitions occur. Today, thinning and prescribed fire are commonly used to increase the resiliency of forests to wildfires (Fig. 3.7), which is important given increased wildfire activity is expected as a



Fig. 3.7 Current conditions of many seasonally dry forests in the western USA, especially those that once experienced low-to-moderate intensity fire regimes, leave them uncharacteristically susceptible to high-severity wildfire. Creating more fire-resilient stands generally requires treatment of surface and ladder fuels, reductions in crown density, and maintenance of large-diameter trees. A combination of thinning and prescribed burning is commonly used and highly effective when applied within prescription. Most evidence suggests that these treatments are typically accomplished with few unintended consequences as most ecosystem components (e.g., carbon sequestration, soils, wildlife) exhibit very subtle impacts or no measurable impacts. Since increased wildfire activity is expected as a result of climate change and desired treatment effects are transient, forest managers need to be persistent and repeat the application of fuel reduction treatments over time (photo credits: left, C.J. Fettig, and right, S.R. McKelvey, USFS Pacific Southwest Research Station)

consequence of climate change. In particular, a combination of thinning and prescribed fire has been shown to be highly effective for reducing the severity of wildfires [38] and will increase the resiliency of forests to other disturbances imposed on them by climate change [39].

Climate has always shaped the world's forests [40] and minor climatic shifts may have significant effects on community compositions [41]. Even under conservative scenarios, future climatic changes are likely to include further increases in temperature with significant drying in some regions and increases in the frequency and severity of extreme weather events [42]. These changes are predicted to further increase the frequency and severity of many other disturbances that shape forest ecosystems. A recent global assessment of forest health reported 88 unique episodes of tree mortality over the last 30 years [43]. Since then, several additional episodes have been identified [44]. The common implicated causal factor in these examples is elevated temperatures and/or water stress, raising the possibility that the world's forests are increasingly responding to ongoing warming and drying attributed to climate change [43]. While these episodes are well documented, the underlying causes are complex and uncertain and likely involve numerous predisposing, inciting, and contributing factors [45]. Reports of climate-induced forest mortality

are now common in both the popular press and scientific journals but are by no means a new phenomenon [43].

Across North America, temperature increases are projected to exceed global mean increases and more frequent extreme weather events are expected [42]. Associated changes in precipitation patterns may result in earlier and longer dry seasons across the western USA, with a greater frequency and duration of drought [46]. It is thought that these changes will significantly affect the condition, composition, distribution, and productivity of multiple ecosystems [47]. Since temperature increases are expected to be greatest at higher elevations and latitudes, conifers (the predominate vegetation of forests in these areas) are expected to be significantly affected.

The current distribution of coniferous vegetation across western North America resulted from climatic shifts dating back millions of years [48], in addition to more recent recolonization of deglaciated lands [49]. These historical patterns perhaps foreshadow changes to current coniferous vegetation as climate change accelerates. For example, based on the best existing data for 130 tree species in North America and associated climate information, McKenney et al. [50] predicted that on average the geographic range for a given tree species will decrease by 12 % and shift northward 700 km during the twenty-first century. Under a scenario where survival only occurs in areas where anticipated climatic conditions overlap with current climatic conditions, niches for tree survival decrease by 58 % and shift northward 330 km. In terms of tree species, there will be winners (e.g., ponderosa pine) and losers (e.g., Engelmann spruce, *Picea engelmannii*) [51]. By the end of the twenty-first century, others predict that ~48 % of the western USA landscape will experience climate profiles with no contemporary analog for the current coniferous vegetation [51]. The fate of any tree species will depend on genetic variation, phenotypic variation, fecundity and dispersal mechanisms, and their resilience to a multitude of disturbances. We consider three major disturbances (i.e., phytophagous insects, forest pathogens, and wildfire) that will serve as catalysts for much of this change.

Phytophagous insects are major components of forest ecosystems, representing most of the biological diversity and affecting virtually all forest processes and uses. Insects influence forest ecosystem structure and function by regulating certain aspects of primary production, nutrient cycling, ecological succession, and the size, distribution, and abundance of forest trees [52–54]. Elevated insect activity reduces tree growth and hastens decline, mortality, and subsequent replacement by other tree species and plant associations. Such effects are often amplified by other natural disturbances. The nature and extent of impacts are dependent upon the resource of concern, type of insect activity, size and distribution of the insect population, and metric used for evaluation [55]. Climate change is generally thought to increase levels of tree mortality attributed to insects, for example, bark beetles [56] and defoliators [57], but there are exceptions to this trend, for example, larch budworm (*Zeiraphera diniana*) [58].

In specific, bark beetles are commonly recognized as a primary disturbance agent in coniferous forests. Of the hundreds of native species in western North America, few species (<1 %) attack and reproduce in live trees. Frequently referred

to as “aggressive” bark beetles, these species can kill healthy trees and have the capacity to cause landscape-scale tree mortality. The last decade has seen elevated levels of tree mortality attributed to bark beetle outbreaks in spruce forests of south-central Alaska and the Rocky Mountains, lodgepole pine (*P. contorta*) forests of western Canada and the Rocky Mountains, pinyon-juniper woodlands of the southwestern USA, and ponderosa pine forests of Arizona, California, and South Dakota [59]. Because bark beetles, like many insects, are highly sensitive to thermal conditions conducive to population survival and growth, and water stress can influence host tree vigor, outbreaks have been correlated with shifts in temperature [60] and precipitation [61]. The life histories and ecological roles of the majority of bark beetle associates are not well understood, hampering full comprehension of the consequences of climate change on bark beetle population dynamics. However, Bentz et al. [56] predicted increases in thermal regimes conducive to population success for two economically important species, spruce beetle (*Dendroctonus rufipennis*) and mountain pine beetle (*D. ponderosae*), although there was considerable spatial and temporal variability in their predictions. These suggested a northward and upward in elevation movement of temperature suitability and identification of regions with a high potential for bark beetle outbreaks and associated levels of tree mortality in the twenty-first century. Evangelista et al. [62] predicted that suitable habitats for the mountain pine beetle and pine engraver (*Ips pini*) will stabilize or decrease under future climate conditions, while habitats for the western pine beetle (*D. brevicomis*) will increase (Fig. 3.8). Their work represents an estimate of potential distribution and not specific impacts to forest health.

As with phytophagous insects, outbreaks of forest diseases caused by native and introduced forest pathogens are generally predicted to become more frequent and severe as a result of climate change [63]. However, diseases caused by pathogens directly affected by climate (e.g., needle blights) are predicted to have a reduced impact under warmer and drier conditions. These groups of pathogens may cause disease in healthy hosts if the pathogen’s environmental requirements are met, many of which require moist conditions [64]. Forest diseases caused by pathogens indirectly affected by climate (e.g., root diseases) are generally predicted to have an increased impact [63]. While the ability of these pathogens to spread and infect new hosts is affected by moisture, factors associated with climate change that stress their hosts are generally considered to be more important to host invasion. Models frequently predict a reduction in the potential geographic distribution of forest diseases as a result of climate change [63, 65].

Increased wildfire activity is also expected as a result of climate change. In the western USA, increases in wildfire frequency have been well documented since the mid-1980s and concentrated between 1680 and 2590 m in elevation [66]. Wildfires at these elevations have been episodic, occurring during warm years and strongly associated with changes in spring snowmelt timing, which in turn is sensitive to changes in temperature [66] and precipitation. As a result, concerns regarding air quality (as discussed earlier), human safety, and protection of critical infrastructure are important, especially in the wild land urban interface where the presence of housing developments increases the cost and complexity of implementing fuel



Fig. 3.8 The western pine beetle (*Dendroctonus brevicomis*) is a primary disturbance agent in ponderosa pine (*Pinus ponderosa*) forests. Unlike many other bark beetles, western pine beetle is unique in that it has a very narrow host range. The only other common host is Coulter pine (*P. coulteri*), a species indigenous to the mountains of southern California, USA, and northern Baja California, Mexico. In the early 2000s, the mountain ranges of southern California started to experience elevated levels of tree mortality. Most experts attributed this mortality to drought (i.e., precipitation was the lowest in recorded history during 2001–2002) and elevated populations of bark beetles, specifically western pine beetle. Mortality was dispersed across >259,000 ha by 2004 and concentrated in several tree species, most notably ponderosa and Coulter pines. Significant mortality occurred in other plant associations as well. The resultant western pine beetle outbreak that occurred during 2001–2004 is considered by many experts to be the largest in recorded history for this species of bark beetle. In some areas, tree mortality was >80%. Climate change is generally thought to increase levels of tree mortality attributed to insects. Western pine beetle is unique in that the range of its primary host is expected to increase as a result of climate change. The species is likely to become a more important disturbance agent in the future (photo credits: C.J. Fettig, USFS Pacific Southwest Research Station)

reduction treatments to reduce fire risk (Fig. 3.7). Increases in wildfire activity are likely to magnify other threats to forest health. While in many cases it is recognized that bark beetle outbreaks and wildfire will serve as the catalyst for much of the ecological change to be associated with climate change in coniferous forests, few studies have thoroughly examined the interactions between these disturbances until recently [67]. There is evidence that bark beetle outbreaks and associated levels of tree mortality affect subsequent fire risk and severity in some forest types.

Rapid and broad-scale tree mortality events can have long-term impacts to both forest health [43] and human health [68] with feedbacks that further influence climate and land use [33, 69]. Complex interactions must be considered at numerous scales (e.g., from tree to forest to global scales) and on various aspects of the life histories of the numerous species that comprise these ecosystems. For example, the recent loss of whitebark pine (*P. albicaulis*) stands due to mountain pine beetle underscores the need for a greater understanding of climate change effects on complex interactions important to ecosystem resiliency and stability (Fig. 3.9). Characterizing thresholds for systems beyond which such changes are irreversible



Fig. 3.9 In western North America, recent outbreaks of mountain pine beetle (*Dendroctonus ponderosae*) have been severe, long lasting, and well documented. Since 2001, >25 million ha of lodgepole pine (*Pinus contorta*) forest have been impacted. The species ranges throughout British Columbia and Alberta, Canada, most of the western USA, into northern Mexico, and colonizes several pine species, most notably, lodgepole pine, ponderosa pine (*Pinus ponderosa*), sugar pine (*P. lambertiana*), limber pine (*P. flexilis*), western white pine (*P. monticola*), and whitebark pine (*P. albicaulis*). Episodic outbreaks are a common occurrence, but the magnitude of recent outbreaks have exceeded the range of historic variability and have occurred in areas where mountain pine beetle outbreaks were once rare and of limited scale (e.g., whitebark pine forests) or previously unrecorded (e.g., jack pine forests (*P. banksiana*) in Canada). Several scientists speculate that under continued warming the loss of whitebark pine, and the unique ecological services that this species provides, is imminent in many areas. The US Fish and Wildlife Service announced in 2011 that it determined whitebark pine warranted protection under the Endangered Species Act but that adding the species to the Federal List of Endangered and Threatened Wildlife and Plants was precluded by the need to address other listing actions of higher priority (photo credits: left, C.J. Fettig, and right, C.J. Hayes, USFS Pacific Southwest Research Station)

is important. There are tools available to restore forest health and to increase the resiliency of forests to disturbances [39, 54]. Resource managers can intervene and mitigate some of the effects of climate change [70]. Uncertainty is inherent, but it is clear that healthy forests have a vital role to play in combating climate change.

References

1. Rosenzweig C, Casassa G, Imeson A, Karoly DJ, Liu C, Menzel A, Rawlins S, Root TL, Seguin B, Tryjanowski P. Assessment of observed changes and responses in natural and managed systems. In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE, editors. Climate change 2007: impacts, adaptation and vulnerability contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press; 2007. p. 79–131.
2. Trenberth KE, Jones PD, Ambenje P, Bojariu R, Easterling D, Klein Tank A, Parker D, Rahimzadeh F, Renwick JA, Rusticucci M, Soden B, Zhai P. Observations: surface and atmospheric climate change. In: Solomon S, Qin D, Manning M, Chen Z, Marquis M, Averyt KB, Tignor M, Miller HL, editors. Climate change 2007: the physical science basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press; 2007.

3. Karl TR, Melillo JM, Peterson TC, editors. Global climate change impacts in the United States. New York: Cambridge University Press; 2009. p. 188.
4. Sanchez-Lugo A, Kennedy JJ, Berrisford P. Surface temperatures. In: State of the climate 2010. Bull Amer Meteor Soc. 2011;92:6:S36–7.
5. Hansen J, Ruedy R, Sato M, Lo K. Global surface temperature change. Rev Geophys. 2010;48, RG4004.
6. Brohan P, Kennedy JJ, Harris I, Tett SFB, Jones PD. Uncertainty estimates in regional and global observed temperature changes: a new data set from 1850. J Geophys Res. 2006;111, D12106.
7. Smith TM, Peterson TC, Lawrimore J. Improvements to NOAA's historical merged land-ocean surface temperature analyses (1880–2006). J Climate. 2008;21:2283–96.
8. Confalonieri U, Menne B, Akhtar R, Ebi KL, Hauengue M, Kovats RS, Revich B, Woodward A. Human health. In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE, editors. Climate change 2007: impacts, adaptation and vulnerability. Contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press; 2007. p. 391–431.
9. Vose RS, Easterling DR, Gleason B. Maximum and minimum temperature trends for the globe: an update through 2004. Geophys Res Lett. 2005;32, L23822.
10. Kunkel KE, Bromirski PD, Brooks HE, Cavazos T, Douglas AV, Easterling DR, Emanuel KA, Groisman PYa, Holland GJ, Knutson TR, Kossin JP, Komar PD, Levinson DH, Smith RL. Observed changes in weather and climate extremes. In: Karl TR, Meehl GA, Miller CD, Hassol SJ, Waple AM, Murray WL, editors. Weather and climate extremes in a changing climate. Regions of focus: North America, Hawaii, Caribbean, and U.S. Pacific Islands. A report by the US Climate Change Science Program and the Subcommittee on Global Change Research, Washington, DC; 2008.
11. Davis R, Knappenberger P, Novicoff W, Michaels P. Decadal changes in heat related human mortality in the eastern United States. Climate Res. 2002;22:175–84.
12. Davis R, Knappenberger P, Michaels P, Novicoff W. Changing heat related mortality in the United States. Environ Health Perspect. 2003;111:1712–8.
13. Davis R, Knappenberger P, Michaels P, Novicoff W. Seasonality of climate-human mortality relationships in US cities and impacts of climate change. Climate Res. 2004;26:61–76.
14. Menne MJ, Williams CN, Vose RS. The U.S. Historical Climatology Network monthly temperature data, version 2. Bull Amer Meteor Soc. 2009;90:993–1007.
15. Meehl GA, Stocker TF, Collins WD, Friedlingstein P, Gaye AT, Gregory JM, Kitoh A, Knutti R, Murphy JM, Noda A, Raper SCB, Watterson IG, Weaver AJ, Zhao Z-C. Global climate projections. In: Solomon S, Qin D, Manning M, Chen Z, Marquis M, Averyt KB, Tignor M, Miller HL, editors. Climate change 2007: the physical science basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press; 2007.
16. Morris CJG, Simmonds I. Associations between varying magnitudes of the urban heat island and the synoptic climatology in Melbourne, Australia. Int J Climatol. 2000;20:1931–54.
17. Bates DV. Ambient ozone and mortality. Epidemiology. 2005;16:427–9.
18. D'Amato G, Liccardi G, D'Amato M, Cazzola M. Outdoor air pollution, climatic changes and allergic bronchial asthma. Eur Respir J. 2002;20:763–76.
19. Weber RW. Mother nature strikes back: global warming, homeostasis, and implications for allergy. Ann Allergy Asthma Immunol. 2002;88:251–2.
20. Beggs PJ. Impacts of climate change on aeroallergens: past and future. Clin Exp Allergy. 2004;34:1507–13.
21. Rybnicek O, Jaeger S. Ambrosia (ragweed) in Europe. ACI International. 2001;13:60–6.
22. Huynen M, Menne B. Phenology and human health: allergic disorders. Report of a WHO meeting in Rome, Italy, 16–17 Jan 2003. Health and Global Environmental Series EUR/03/5036791. Copenhagen: World Health Organization; 2003. p. 64.
23. Taramarcaz P, Lambelet B, Clot B, Keimer C, Hauser C. Ragweed (Ambrosia) progression and its health risks: will Switzerland resist this invasion? Swiss Med Wkly. 2005;135:538–48.

24. Cecchi L, Morabito M, Domeneghetti P, Crisci MA, Onorari M, Orlandini S. Long distance transport of ragweed pollen as a potential cause of allergy in central Italy. *Ann Allergy Asthma Immunol.* 2006;96:86–91.
25. Wan SQ, Yuan T, Bowdish S, Wallace L, Russell SD, Luo YQ. Response of an allergenic species *Ambrosia psilostachya* (Asteraceae), to experimental warming and clipping: implications for public health. *Am J Bot.* 2002;89:1843–6.
26. 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;88:279–82.
27. 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;32:667–70.
28. Ziska LH, Emche SD, Johnson EL, George K, Reed DR, Sicher RC. Alterations in the production and concentration of selected alkaloids as a function of rising atmospheric carbon dioxide and air temperature: implications for ethno-pharmacology. *Glob Chang Biol.* 2005;11:1798–807.
29. Rogers C, Wayne P, Macklin E, Muilenberg M, Wagner C, Epstein P, Bazzaz F. Interaction of the onset of spring and elevated atmospheric CO₂ on ragweed (*Ambrosia artemisiifolia* L.) pollen production. *Environ Health Perspect.* 2006;114:865–9.
30. Bonan GD. Forests and climate change: forcings, feedbacks, and the climate benefits of forests. *Science.* 2009;320:1444–9.
31. Kurz WA, Apps MJ, Stocks BJ, Volney WJ. Global climate change: disturbance regimes and biospheric feedbacks of temperate and boreal forests. In: Woodwell GM, Mackenzie FT, editors. *Biotic feedbacks in the global climatic system: will the warming feed the warming?* Oxford: Oxford University Press; 1995.
32. Stocks BJ, Lee BS, Martell DL. Some potential carbon budget implications of fire management in the boreal forest. In: Apps MJ, Price DT, editors. *Forest ecosystems, forest management and the global carbon cycle.* Berlin: Springer; 1996.
33. Kurz WA, Dymond CC, Stinson G, Rampley GJ, Neilson ET, Carroll AL, Ebata T, Safranyik L. Mountain pine beetle and forest carbon feedback to climate change. *Nature.* 2008;452:987–90.
34. White PS, Pickett STA. *The ecology of natural disturbance and patch dynamics.* Orlando: Academic; 1985.
35. Folke C, Carpenter S, Walker B, Scheffer M, Elmqvist T, Gunderson L, Holling CS. Regime shifts, resilience, and biodiversity in ecosystem management. *Ann Rev Ecol Evol Syst.* 2004;35:557–81.
36. Schelhass MJ, Nabuurs GJ, Schuck A. Natural disturbances in the European forests in the 19th and 20th centuries. *Glob Chang Biol.* 2003;9:1620–33.
37. Youngblood A, Max T, Coe K. Stand structure in eastside old-growth ponderosa pine forests of Oregon and northern California. *For Ecol Manage.* 2004;199:191–217.
38. Ritchie MW, Skinner CN, Hamilton TA. Probability of tree survival after wildfire in an interior pine forest of northern California: effects of thinning and prescribed fire. *For Ecol Manage.* 2007;247:200–8.
39. Stephens SL, McIver JD, Boerner REJ, Fettig CJ, Fontaine JB, Hartsough BR, Kennedy P, Schwilk DW. Effects of forest fuel reduction treatments in the United States. *Bioscience.* 2012;62:549–60.
40. Bhatti JS, Lal R, Apps MJ, Price MA. *Climate change and managed ecosystems.* Boca Raton, FL: CRC; 2006.
41. Shugart HH. *A theory of forest dynamics: the ecological implications of forest succession models.* New York, NY: Springer; 2003.
42. IPCC. *Climate change 2007: the physical science basis.* In: Solomon S, Qin D, Manning M, Chen Z, Marquis M, Averyt KB, Tignor M, Miller HL, editors. *Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change.* Cambridge: Cambridge University Press; 2007.

43. Allen CD, Macalady AK, Chenchouni H, Bachelet D, McDowell N, Vennetier M, Kitzberger T, Rigling A, Breshears DD, Hogg EH, Gonzalez P, Fensham R, Zhangm Z, Castro J, Demidova N, Lim JH, Allard G, Running SW, Semerci A, Cobb N. A global overview of drought and heat-induced tree mortality reveals emerging climate change risks for forests. *For Ecol Manage.* 2010;259:660–84.
44. Martinez-Vilata J, Lloret F, Breshears DD. Drought-induced forest decline: causes, scope and implications. *Biol Lett.* 2012;8:689–91.
45. Manion PD. *Tree disease concepts.* Englewood Cliffs, NJ: Prentice-Hall; 1981.
46. Seager R, Ting M, Held I, Kushnir Y, Lu J, Vecchi G, Huang HP, Harnik N, Leetmaa A, Lau NC, Li C, Velez J, Naik N. Model projections of an imminent transition to a more arid climate in southwestern North America. *Science.* 2007;316:1181–4.
47. Easterling DR, Meehl GA, Parmesan C, Changnon SA, Karl TR, Mearns LO. Climate extremes: observations, modeling, and impacts. *Science.* 2000;289:2068–74.
48. Brunsfeld SJ, Sullivan J, Soltis DE, Soltis PS. Comparative phylogeography of northwestern North America: a synthesis. In: Antonovics J, Silvertown J, editors. *Integrating ecology and evolution in a spatial context.* Wiliston, VT: Blackwell; 2001.
49. Godbout J, Fazekas A, Newton CH, Yeh FC. Glacial vicariance in the Pacific Northwest: evidence from a lodgepole pine mitochondrial DNA minisatellite for multiple genetically distinct and widely separated refugia. *Mol Ecol.* 2008;17:2463–75.
50. McKenney DW, Pedlar JH, Lawrence K, Campbell K, Hutchinson MF. Potential impacts of climate change on the distribution of North American trees. *Bioscience.* 2007;57:939–48.
51. Rehfeldt GE, Crookston NL, Warwell MV, Evans JS. Empirical analyses of plant-climate relationships for the western United States. *Int J Plant Sci.* 2006;167:1123–50.
52. Mattson Jr WJ, Addy ND. Phytophagous insects as regulators of forest primary production. *Science.* 1975;90:515–22.
53. Schowalter TD. Insect herbivore relationship to the state of the host plant: biotic regulation of ecosystem nutrient cycling through ecological succession. *Oikos.* 1981;37:126–30.
54. Fettig CJ, Klepzig KD, Billings RF, Munson AS, Nebeker TE, Negrón JF, Nowak JT. The effectiveness of vegetation management practices for prevention and control of bark beetle infestations in coniferous forests of the western and southern United States. *For Ecol Manage.* 2007;238:24–53.
55. Coulson RN, Stephen FM. Impacts of insects in forest landscapes: implications for forest health management. In: Payne TD, editor. *Invasive forest insects, introduced forest trees, and altered ecosystems: ecological pest management in global forests of a changing world.* New York: Springer; 2006.
56. Bentz BJ, Régnière J, Fettig CJ, Hansen EM, Hayes JL, Hicke JA, Kelsey RG, Lundquist J, Negrón JF, Seybold SJ. Climate change and bark beetles of the western United States and Canada: direct and indirect effects. *Bioscience.* 2010;60:602–13.
57. Carnicera J, Colla M, Ninyerolac M, Pons X, Sánchez G, Peñuelasa J. Widespread crown condition decline, food web disruption, and amplified tree mortality with increased climate change-type drought. *Proc Natl Acad Sci USA.* 2011;108:1474–8.
58. Büntgen U, Frank D, Liebhold A, Johnson D, Carrer M, Urbinati C, Grabner M, Nicolussi K, Levanić T, Esper J. Three centuries of insect outbreaks across the European Alps. *New Phytol.* 2009;182:929–41.
59. Bentz BJ, Allen CD, Ayres M, Berg E, Carroll A, Hansen M, Hicke J, Joyce L, Logan J, MacFarlane W, MacMahon J, Munson AS, Negrón JF, Paine TD, Powell J, Raffa KF, Régnière J, Reid M, Romme W, Seybold SJ, Six DL, Tomback D, Vandygriff J, Veblen T, White M, Witcosky J, Wood DL. *Bark beetle outbreaks in western North America: causes and consequences.* Salt Lake City: University of Utah Press; 2009.
60. Powell JA, Logan JA. Insect seasonality-circle map analysis of temperature-driven life cycles. *Theor Popul Biol.* 2005;67:161–79.
61. Berg EE, Henry JD, Fastie CL, De Volder AD, Matsuoka SM. Spruce beetle outbreaks on the Kenai Peninsula, Alaska, and Kluane National Park and Reserve, Yukon Territory: relationship

- to summer temperatures and regional differences in disturbance regimes. For *Ecol Manage.* 2006;227:219–32.
62. Evangelista PH, Kumar S, Stohlgren TJ, Young NE. Assessing forest vulnerability and the potential distribution of pine beetles under current and future climate scenarios in the Interior West of the US. For *Ecol Manage.* 2011;262:307–16.
 63. Sturrock RN, Frankel SJ, Brown AV, Hennon PE, Kliejunas JT, Lewis KJ, Worrall JJ, Woods AJ. Climate change and forest diseases. *Plant Pathol.* 2011;60:133–49.
 64. Sinclair WA, Lyon HH, Johnson WT. *Diseases of trees and shrubs.* Ithaca: Cornell University Press; 1987.
 65. Venette RC. Implication of global climate change on the distribution and activity of *Phytophthora ramorum*. In: McManus KA, Gottschalk KW, editors. Proceedings of the 20th U.S. Department of Agriculture Interagency Research Forum on Invasive Species 2009. NRS-P-51, 58–9. Newtown Square, PA: U.S. Department of Agriculture, Forest Service, Northern Research Station; 2009.
 66. Westerling AL, Hidalgo HG, Cayan DR, Swetnam TW. Warming and earlier spring increase western U.S. forest wildfire activity. *Science.* 2006;313:940–3.
 67. Jenkins MJ, Hebertson E, Page W, Jorgensen CA. Bark beetles, fuels, fires and implications for forest management in the Intermountain West. For *Ecol Manage.* 2008;254:16–34.
 68. Allan BF, Keesing F, Ostfeld R. Effect of forest fragmentation on Lyme disease risk. *Conserv Biol.* 2003;17:267–72.
 69. McDowell NG, Pockman WT, Allen C, Breshears DD, Cobb N, Kolb T, Plaut J, Sperry J, West A, Williams D, Yepez EA. Mechanisms of plant survival and mortality during drought: why do some plants survive while others succumb to drought? *New Phytol.* 2008;178:719–39.
 70. Peterson DL, Millar CI, Joyce LA, Furniss MJ, Haolosky JE, Neilson RP, Morelli TL. Responding to climate change on national forests: a guidebook for developing adaptation options. PNW-GTR-855. Portland, OR: US Department of Agriculture, Forest Service, Pacific Northwest Research Station; 2011.
 71. Smith TM, Reynolds RW. A global merged land–air–sea surface temperature reconstruction based on historical observations (1880–1997). *Journal of Climate,* 2005; 18: 2021–36 (doi: <http://dx.doi.org/10.1175/JCLI3362.1>).

Chapter 4

Eyewitness to Global Warming

Will Steger and Nicole Rom

Abstract The United Nations Framework Convention on Climate Change (UNFCCC) is an international environmental treaty that was agreed upon in 1992 at the United Nations Conference on Environment and Development, known as the “Earth Summit,” in Rio de Janeiro, Brazil. The Parties to this treaty (i.e., the countries that have formally endorsed it) have been holding annual meetings since 1995. There are now 194 Parties that have formally endorsed the UNFCCC—which is nearly all of the world’s 203 sovereign states. Although the UNFCCC is technically considered a “treaty,” it’s most accurate to think of it as an “agreement to agree” to take action steps that prevent the worst impacts of climate change. Nothing in the UNFCCC itself requires countries to take such action steps. That’s why there have been 17 UNFCCC conferences since 1995: the world still has a lot to do before it will have a global agreement that stands a chance of dealing adequately with the threat of climate change.

Keywords Global warming • Polar exploration and global warming • Steger eyewitness account in polar explorations • North Pole global warming changes • United Nations Framework Convention on Climate Change

Eyewitness Account: Will Steger

Global warming is a reality. It threatens both our society and life as we know it on earth. The overwhelming consensus of the scientific community for the past 2 decades has been that the planetary warming we are now experiencing, and the

W. Steger, B.S., M.A. • N. Rom, M.S. (✉)
Will Steger Foundation, 2801 21st Avenue South Suite 110,
Minneapolis, MN 55407, USA
e-mail: nicole@willstegerfoundation.org



Fig. 4.1 1986 North Pole Expedition team uses sheets of ice as a raft to cross open leads (© Will Steger)

resulting climate change is largely a human-induced phenomenon. Global warming is brought on mainly by the release of carbon dioxide through the burning of fossil fuels, which blankets our atmosphere raising the earth's surface temperature.

The amount of carbon dioxide that is in the atmosphere today is the *minimum* level we're going to have to live with for the indefinite future. Once carbon dioxide is in the stratosphere above us, it will stay there for hundreds and hundreds of years. It's as though you gained the most weight in your life, and knew you'd never weigh even a single pound less, ever. Carbon dioxide does eventually get pulled back out of the atmosphere by natural processes, but that happens very slowly. Climate scientists like to compare the atmosphere to a bathtub half-full of water, with a very slow drain and a slowly trickling faucet. If the drain and the trickle are balanced, the water level never changes—just as the trickle of natural carbon dioxide into the atmosphere and the drainage into trees, carbonate rocks and other places have been in balance for at least 2,000 years, and probably more. Atmospheric carbon dioxide hovered at around 270–290 ppm that whole time, and the climate stayed more or less stable. Carbon dioxide levels in the Arctic have now reached 400 ppm and climate scientists consider 350 ppm to be the safe level to avoid catastrophic changes.

I am probably most known for my major expeditions. In 1986, I led the first confirmed unsupported dogsled trip to the North Pole (Fig. 4.1). With seven teammates and 49 dogs, I traveled 500 miles in 56 days. In 1989–1990, together with five other men from six countries, we crossed Antarctica—3,741 miles, farther than from New York to Los Angeles (Figs. 4.2 and 4.3). In a series of expeditions in the mid-1990s, I crossed the Arctic Ocean from Russia to Canada's Ellesmere Island on



Fig. 4.2 Crossing Antarctica involved dogsledding over dangerous crevasses. 1989–1990 International Trans-Antarctica Expedition (© Will Steger)



Fig. 4.3 Lunch is no picnic in Antarctica: windblown snow pelts the men’s faces, coating beards and eyelashes with ice crystals and denying them even the modest comfort of rest. 1989–1990 International Trans-Antarctica Expedition (© Will Steger)

some of the most dynamic and moving surfaces on Earth. I returned to the Canadian Arctic in 2004, 2007, and 2008 with a new focus. I was no longer setting out to break a record on my expeditions. I wanted to draw worldwide attention to the biggest threat of our time—global warming—and its impacts on the Arctic regions.

Between 1990 and 2007, dramatic changes happened in my remote home. To survive in these regions, I have become intimately familiar with their vast lands, wildlife, and climates. My expeditions have given me firsthand observations about how the Earth's surface has changed, including vanishing glaciers, shattered ice shelves, melting permafrost, and displaced communities of people and animals. The resulting changes from global warming deeply affect me in a way neither a scientific study nor a satellite image could. Every ice shelf I have crossed has disintegrated into the ocean as a result of global warming.

When my expedition team first flew over Antarctica's Weddell Sea, near the Antarctic Peninsula, I reached for my cassette recorder: "July 26th 1989: And it's Antarctica that we are looking at that is going to be the main player in the destiny of the human race. It's this snow and ice here. If the atmosphere warms up, this ice right in this area is going to break off into the ocean." At the time however, it didn't seem possible that an ice mass this large could actually break up. It seemed that the Larsen, a long ice shelf jutting into the Weddell Sea, was as permanent as the Antarctic continent itself. But on March 2nd 2002, I was thumbing through the Minneapolis-based Star Tribune newspaper and on page nine in bold print was the caption "Larsen B Ice Shelf Disintegrates." It seemed at first this was science fiction, and it took days before I could grasp the extent of this global environmental catastrophe. There is no way to comprehend the massiveness of the disintegration of the Larsen ice shelf unless you ski and walk every step of the way. It took us 31 days, from July 27th to August 26th to cross the full length of this ice shelf. Every day, camp after camp, through storm, whiteouts and clear weather, we skied and pushed our sleds. We became intimately familiar with the ice shelf that treated us for the most part with safe surface conditions.

While crossing the Larsen, the ice shelf felt very stable to my team. Scientists at Queen's University estimate the shelf could have been stable for as long as 12,000 years—that many years ago there were still mastodons, mammoths, and saber-toothed cats roaming the earth. Over the course of 1 month in 2002, however, a chunk of ice, the size of the New England state of Rhode Island, broke free from the Larsen B ice shelf. The speed of the collapse surprised even the scientists who were monitoring the shelf. Scientists link the collapse with global warming.

As of 2010, both the Larsen A and B ice shelves have disintegrated, along with the Wilkins ice shelf. Scientists are watching the continent closely, paying particular attention to melting, calving, and complete disintegration of the Larsen C, the Ross (about the size of France), and Ronne (about the size of Spain) ice shelves.

After Antarctica, Greenland's ice cap contains the second largest mass of frozen fresh water in the world. At 7,000 ft in July 2008, my kite-ski expedition team came across something very unusual on Greenland: running water. Every year in the summer on the coast of the ice cap, the temperature warms enough to melt out systems of rivers and lakes. Since 1992, the thawing levels during the summer season on

Greenland have increased in elevation. Data from NASA's Gravity Recovery and Climate Experiment show Greenland lost 150–250 cubic kilometers (36–60 cubic miles) of ice per year between 2002 and 2006. In 2008, when we literally ran into running water, we were at 7,000 ft, the highest point on Greenland I'd ever thought I would see rivers of water.

Unlike Antarctica, which sits at the bottom, the Arctic sits at the top of the world. Although they are both very cold and covered with ice, they are very different. Antarctica is a large continent covered with a sheet of ice two miles thick. The Arctic is an ocean two miles deep, surrounded by the land of eight nations. The Arctic Ocean is covered with a layer of ice 8–12 ft thick. It is like a bucket of water with a thin layer of dust on the surface—the bucket represents the Arctic Ocean, the layer of dust, the ice. In the spring and summer the ice breaks up and the ice is in constant motion, moved by wind currents and the ocean's movements.

Sea ice is frozen seawater that floats on the ocean surface. Blanketing millions of square kilometers, sea ice forms and melts with the polar seasons, affecting both humans and wildlife. In the Arctic, some sea ice persists year after year, whereas almost all Southern Ocean or Antarctic sea ice is “seasonal ice,” meaning it melts away and reforms annually. Sea ice in the Arctic plays a unique role in regulating the Earth's climate because of its role in regulating global temperature.

Following my successful Antarctica expedition, I knew that I wanted to continue to bring the Polar Regions into classrooms around the world. Computers were beginning to enter into the classroom environment so now my expeditions had new relevancy—I could bring the stories from the trail into schools. I began organizing an expedition that would cross the top of the world—leaving from Russia's Siberia, going over the North Pole, and finishing at Canada's Ellesmere Island. Along the way, we learned to be prepared for the unexpected. The biggest shock on that particular expedition was the large amounts of open water.

We had just left the North Pole a few days after Earth Day in 1995 when I noticed the ice beneath our skis was dark, almost black (Fig. 4.4). This is a sign of thin ice. Just ahead of me, it was too late for the sled and ten dogs that had broken through the ice and tipped onto its side, half in the water, half on thin ice. It took several hours to get the sled back on sturdier ice. Over the course of the expedition, we had to review our route, which changed daily because of varying ice conditions and shifting ice. It became clear that this expedition was behind schedule; not because of poor planning, but because of an unusual year in the Arctic. We had no idea at the time this would be the new normal for the Arctic. There was a lot of snow, and while the weather was cold, it was already seeing warmer than normal temperatures for the region, which means ice is thinner and there is more open water.

Water can soak up a lot of heat. When the oceans get warmer, sea ice begins to melt in the Arctic and around Greenland. NASA's Earth satellites show us that every summer some Arctic ice melts and shrinks, getting smallest by September. Then, when winter comes, the ice grows again. But, since 1979, the September ice has been getting smaller and smaller and thinner and thinner.

As a result of this change, the Arctic Ocean is also turning from a once-reflective surface to an absorptive surface. Traditionally, the Arctic Ocean's layer of thick ice



Fig. 4.4 On the twenty-fifth Anniversary of Earth Day, the 1995 International Arctic Project expedition team reaches the North Pole (© Gordon Wiltsie)

has reflected 90 % of the sun's energy (the same amount of energy that hits the tropical regions near the equator) back into space, helping to keep the planet cool. Now that the ice is smaller and thinner, it melts more ice, creating a positive feedback loop, and at the same time, revealing darker ocean surfaces, which absorbs the once reflected energy into the ocean, melting even more ice.

On a large scale, what we are witnessing around the world are feedback loops that spur large and rapid changes to our environment. The Arctic sea ice is a great example of those changes. The Arctic sea ice has lost half of its thickness and area in the last 2 decades. Its once-reflective surface is now exposing the darker ocean surfaces; because darker surfaces absorb more light and energy than lighter surfaces, warmth is accelerated and leads to more melting of ice. As a result, without any additional greenhouse gases, the Arctic will soon be ice-free during the summer. If the summer sea ice disappears, animals like the polar bear and walrus will face probable extinction.

On two different expeditions to Canada's Baffin Island, home of the Inuit, we set out to document how climate change was affecting the region, to meet with Inuit elders and students, to explore traditional ecological knowledge in the remote communities visited on the trail, and to put a human face and cultural voice on this complex issue.

Nowhere on earth is the climate changing more rapidly or more dramatically than in the Arctic's Baffin Island. My 2007 Global Warming 101 Expedition team



Fig. 4.5 Global Warming 101 Expedition dogsledding across Clyde fjord on Baffin Island, Nunavut (© 2007 Will Steger Foundation, Elizabeth Andre)

witnessed firsthand some impacts of global warming as we traveled by dog-team from Iqaluit to Pangnirtung (Fig. 4.5).

Warmer-than-normal temperatures made it difficult to simply walk from the land out on to the sea ice in Frobisher Bay. The tidal overflow along the shore was not refreezing. Instead the water remained liquid or slushy. We had to pick our way across the more solidly frozen sections. Even so, however, our feet sank into the slush, which soaked our moose-hide mukluk boots. During pre-trip planning, we assumed only the American members would sleep in tents. The Inuit members planned to make an igloo every night. Different from normal snow conditions, however, made igloo-making impossible. In many places there was simply not enough snow. In other places the snow had weak and soft layers that made blocks cut from it collapse instead of stand up. Living conditions are much warmer inside an igloo than inside a tent, so it was a disappointment to the Inuit members to not be able to build igloos.

On the Hall Peninsula as the dog-teams made their way overland from Iqaluit, the team crossed a small flowing creek that was completely open, unfrozen water (Fig. 4.6). Theo Ikummaq, the Inuit team leader, said this time of year that creek should be frozen solid. The temperatures on South Baffin Island had been, however, as much as 40° above normal during the weeks before the expedition's departure.

The 60-mile-wide Cumberland Sound stretches between the Hall Peninsula and Pangnirtung, the expedition's second village. Inuit elders recall a time when they would dogsled and snowmobile straight across Cumberland Sound to ice-fish for



Fig. 4.6 Inuit team member, Simon Qamanirq, dogsleds using the traditional fan hitch approach on the Global Warming 101 Expedition (© 2007 Will Steger Foundation, Abby Fenton)

turbot and to reach camps for seal hunting on the other side. In 2007, however, my team heard reports of the worst ice conditions ever; even seal pups were reported to be falling through the ice. When we reached the sound, our fears were confirmed; open water stretched all the way to the top of the sound. We added 70 miles to our trip to skirt around the open water. In some places large polynias, or open sections of water, separated us from the shore. Ikummaq said many of these polynias were larger than normal or in places where there had traditionally been only solid ice. We arrived safely in Pangnirtung on March 10. The next day, however, the ice over which we had traveled broke up.

Numerous glaciers carve their way down from the Penny Ice Cap and surrounding peaks in Auyuittuq National Park. Ironically, the name of the park translates to “the land that never melts,” but the glaciers are now receding rapidly. Fifty years ago the Fork Beard glacier reached all the way to the valley floor. It has now receded over 1,000 vertical feet and is no longer even visible from the valley floor.

The unusual ice and snow conditions make travel difficult for my expeditions. The conditions do, however, make it possible for us to achieve our goal of providing an eyewitness account of global warming and its impact.

On these expeditions, we came to listen to the voice of the Inuit people. Of course, as in any culture, there are a million voices, each one with its own unique perspective on the world. Despite differing perspectives, however, we did hear common threads. We heard over and over again, in each community, a concern for global warming and the changing Arctic environment. We heard much evidence of



Fig. 4.7 Young explorers travel across the ruins of the Arctic Ocean summer ice melt from the 2008 Ellesmere Island Expedition (© 2008 Will Steger Foundation, Sam Branson)

this change, of new species migrating north, of warming oceans and melting sea ice, and of the impact this has on the delicately balanced Arctic ecosystem. Most of all, we learned about the Inuit spirit of resiliency and adaptation. When we asked about Inuit cultural survival in the face of global warming, we heard the same reply time and time again: that the Inuit will continue to adapt as they always have. The question many Inuit asked us in return, can the rest of us adapt?

A year later, traveling even further north than Baffin Island, on Canada's Ellesmere Island, my team and I bore witness to an inimitable firsthand account of the effects of global warming. We traveled 700 miles across the sounds and straights of Ellesmere Island in the spring of 2008, a year after 2007's dramatic Arctic ice melt (Fig. 4.7). We were unable to reach our original goal—to visit the last remaining ice shelves on northern Ellesmere—because we were stopped by thick rubbles of ice. Ice, we later learned, that had been part of the Arctic Ocean, 500 miles away.

In 45 years of Arctic exploration, I have never witnessed ice conditions like what I experienced on this expedition. As confirmed by the US National Snow and Ice Data Center upon our return, we traveled through the ruins of the Arctic Ocean, encountering the melt of multiyear ice from the top of the globe. As an eyewitness to the changing topography of the Arctic, I was stunned to see the rapid repercussions of global warming for the region, its wildlife habitat and indigenous cultures. Swift loss of sea ice will considerably alter the landscape of the Polar Regions as we know it.

Addressing Climate Change at the International Level

The United Nations Framework Convention on Climate Change

The United Nations Framework Convention on Climate Change (UNFCCC) is an international environmental treaty that was agreed upon in 1992 at the United Nations Conference on Environment and Development, known as the “Earth Summit,” in Rio de Janeiro, Brazil. The Parties to this treaty (i.e., the countries that have formally endorsed it) have been holding annual meetings since 1995. There are now 194 Parties that have formally endorsed the UNFCCC—which is nearly all of the world’s 203 sovereign states. Although the UNFCCC is technically considered a “treaty,” it’s most accurate to think of it as an “agreement to agree” to take action steps that prevent the worst impacts of climate change. Nothing in the UNFCCC itself requires countries to take such action steps. That’s why there have been 17 UNFCCC conferences since 1995: the world still has a lot to do before it will have a global agreement that stands a chance of dealing adequately with the threat of climate change.

The Kyoto Protocol

The Kyoto Protocol, forged in 1997 in Kyoto, Japan, is the world’s first and so far only attempt at a global agreement to address climate change. It calls for mandatory cuts in greenhouse gas emissions from developed countries, but exempts developing countries—including China and India, which are the world’s first and fifth largest emitters of greenhouse gases despite their status as developing countries. Because of this, the United States has never ratified the Kyoto Protocol, which means that the binding emissions cuts accepted by every other developed country (except Australia, until it ratified the Protocol in 2007) do not apply here, although the United States is the world’s second largest emitter of greenhouse gases after China. The fact that three of the world’s five largest emitters of greenhouse gases are not bound to cut their emissions under the Kyoto Protocol has made this agreement inadequate to deal with climate change—though it has led to meaningful emissions cuts in most developed countries that ratified it (with some important exceptions).

The Bali Road Map

In December 2007, at the 13th Conference of Parties to the UNFCCC in Bali, Indonesia, the countries of the world agreed on a plan for producing a new agreement that would work alongside and eventually replace the Kyoto Protocol.

In particular, this “Bali Road Map” called on Parties to develop strategies to deal with five challenges:

- Finding consensus on an overall “shared vision” for a post-Kyoto agreement
- Cutting greenhouse gas emissions, including those resulting from deforestation
- Adapting to those climate change impacts that are already guaranteed to occur as a result of past emissions
- Developing clean energy technologies, and transferring knowledge of these technologies to underdeveloped countries
- Forging financial agreements between countries to pay for the efforts above

Under the Bali Road Map, it was hoped that countries would agree on plans for “enhanced action” on these issues in 2008 and 2009, in time to roll the action plans together into a new international climate agreement by the end of the 15th conference in Copenhagen. Between 2007 and 2009, negotiators from around the world worked steadily to address the issues above, in hopes that their work would culminate with an agreement in Copenhagen. This hoped-for agreement was laden with expectations as a result of the failures of the Kyoto Protocol. This is why the COP 15 conference in Copenhagen received so much attention before it began, while it was going on, and after it ended.

The Copenhagen Accord

The outcome of the conference was a three-page, non-binding “Copenhagen Accord” that, while not perfect, provides the beginnings of an agreement to tackle climate change. The Accord was agreed to in the final 48 h of the conference by heads of state from the United States, China, India, Brazil, and South Africa. The other countries assembled at the conference agreed to “take note of” the Accord. What “taking note” means is open to interpretation; it was an indication that many of the other countries at the conference were unwilling to endorse a non-binding climate agreement, but were supportive of this agreement insofar as it leads to a binding agreement later on.

Emissions Cuts in the Copenhagen Accord

The Copenhagen Accord is built on commitments to cut overall emissions by the United States and other developed countries and commitments to cut emissions intensity (emissions per unit of economic output) by India, China, and other developing countries. The emissions targets offered by the United States, China, and India are as follows:

The United States

- The United States will cut its overall greenhouse gas emissions 17 % by 2020, from a 2005 baseline.
 - This amounts to about a 4 % cut by 2020 from a 1990 baseline. The Intergovernmental Panel on Climate Change, the world’s leading authority on the science of global climate change, uses 1990 baselines in all its emissions recommendations and called on developed countries like the United States to cut their emissions 25–40 % by 2020 from a 1990 baseline in its 2007 report.
- The United States will cut overall greenhouse gas emissions 83 % by 2050, from a 2005 baseline.
 - This amounts to about an 80 % cut by 2050 from a 1990 baseline. The Intergovernmental Panel on Climate Change called on developing countries like the United States to cut their emissions 80–95 % by 2050 from a 1990 baseline in its 2007 report.

China

- China will cut its emissions intensity 40–45 % by 2020, from a 2005 baseline.
 - This does not mean China’s overall emissions will fall between now and 2020, since emissions intensity is a measure of emissions per unit of economic output. It only means that China will emit less per dollar of gross domestic product (GDP) that it adds to its economy.

India

- India will cut its emissions intensity 20 % by 2020, from a 2005 baseline.

Other developed and developing countries choosing to endorse the Copenhagen Accord had until January 31, 2010 to add their own emission reduction commitments to an annex to the agreement.

Financing Climate Change Response Measures Under the Copenhagen Accord

To finance emissions cuts, adaptation to climate impacts, and clean technology development and transfer in developing countries, the United States committed to “jointly mobilizing \$100 billion a year by 2020,” in collaboration with other developed countries. It is likely that the United States will cover 20–25 % of this amount, which will also include private funding. These funds will be managed and disbursed by a new “Copenhagen Green Climate Fund.”

Technology in the Copenhagen Accord

The Copenhagen Accord contains a pledge to “establish a Technology Mechanism to accelerate technology development and transfer” to countries that lack the capacity to develop clean energy technologies on their own. The specific functions of this Technology Mechanism have not yet been agreed upon, but it is very likely that the Mechanism will, at the least, oversee three new “Climate Technology Centers” around the world. These Centers will facilitate collaborative research and development between countries on new clean energy and other technologies.

Other climate technology development activities will take place outside the umbrella of the United Nations, through the Major Economies Forum on Energy and Climate. This Forum, which was launched in March 2009, is a collaborative effort between 17 of the world’s largest economies (Australia, Brazil, Canada, China, the European Union, France, Germany, India, Indonesia, Italy, Japan, Korea, Mexico, Russia, South Africa, the United Kingdom, and the United States). The Major Economies Forum’s “Global Partnership” on climate technology development began in July 2009 at the G8 Summit in L’Aquila, Italy. In Copenhagen, the Forum released 11 Technology Action Plans designed to accelerate the development and deployment of key clean energy and energy efficiency technologies, including wind, solar, bioenergy, advanced vehicles, and carbon capture and storage (CCS).

Post COP15

Politically, it will be virtually impossible for the United States to make a binding commitment to any international agreement before the United States. Senate passes energy and climate legislation, which will provide specific guidance about the size of the US emissions cuts and until the United States makes such a binding commitment, China and India are unlikely to do so either. In 2010, after the US House passed climate legislation, the US Senate failed to do so before the Cancun, Mexico COP16 Summit. This inaction meant that a binding post-Kyoto international climate agreement would be even less likely in 2010 and 2011.

COP17 Reflection, Durban Platform for Enhanced Action

COP17 had three key objectives: to reach agreement on the Kyoto Protocol’s future; to agree to a pathway to strengthen the overall global climate regime and lay the foundations for a new treaty; and to operationalize the various institutions and processes that had been established at the COP16 meeting in Cancun, particularly with respect to finance.

Parties managed to adopt decisions on all three issues, but few if any delegations were happy with the overall Durban package or are even sure where the process will

lead. While, on the one hand, governments have agreed that there will be a new legal agreement with mitigation obligations for all countries for the first time, on the other, many key issues remain unresolved, and the level of ambition is still too low to limit global warming temperature rise to 2 °C.

The key decisions reached in Durban are:

- Agreement to launch a new negotiating process that will develop a new “protocol, legal instrument or agreed outcome” by 2015 with implementation by 2020 and covering all countries.
- Agreement to establish a second commitment period under the Kyoto Protocol beginning in January 2013 and ending in either 2017 or 2020 (to be determined by COP18).
- Agreement to operationalize the new Green Climate Fund.

The new negotiating process—known as the Durban Platform for Enhanced Action—is effectively a continuation of the current “Convention” track negotiation, which was established 5 years ago at Bali’s COP13 (Bali Road Map), and should have concluded in Copenhagen with its own “agreed outcome” covering all major emitters including both the United States and China. In other words, Parties have committed, albeit in slightly stronger language, to an outcome in 2015 that they should have delivered in 2009. Nevertheless, this new agreement will cover all countries and all emissions, something as yet not achieved in the more than 20 years of climate negotiations.

Agreeing to this so-called pathway to a new global deal was essential for any deal on the future of the Kyoto Protocol—the number one priority for developing countries attending COP17 in Durban. The European Union’s willingness to sign up to further Kyoto targets after 2013 was conditional on securing a clear pathway to a new global regime for all countries. This keeps the Protocol—and its crucial rules and market mechanisms—alive, but it will be a “lite” version, with Canada, Japan, and Russia all confirming in the decision text that they will not be taking on any targets. Canada has since announced it will withdraw formerly from the Kyoto Protocol. While Australia, New Zealand, Norway, and Switzerland are all likely to join the EU (providing certain conditions are met), the Protocol after 2013 will cover at most 15–16 % of global emissions.

The third piece in the puzzle, the Green Climate Fund, was the second must-have for developing countries. The decision reached in Durban establishes the process for establishing the governing Board, selecting a host country and an independent secretariat. This should mean all practical operational elements of the fund, including key staff will be in place in the next 12 months. What remains missing is the actual funding, which is supposed to be scaled up to \$100 billion per year by 2020. A new work program on long-term finance will look at this issue in 2012.

In addition to these three core outcomes, the Durban package also delivered progress on other key elements of the Cancun Agreements. This included guidelines for monitoring, reporting, and verification of mitigation efforts; establishment of the Climate Technology Center and Network; modalities for the Review Mechanism to assess global mitigation progress; and agreement on membership of the Adaptation Committee.

Despite the negotiations slow pace though, companies, governments, and communities displayed great awareness of the threats and opportunities of climate change. Delegates reported there was heartening evidence of the progress being made across the globe: work on green growth plans and low carbon development strategies; a wide range of exciting new technologies; and evidence of how low carbon strategies can create jobs and new business opportunities. These actions will be critical in the next 9 years, both in keeping emissions at safe levels and by showing governments that a clean energy economy is possible.

Reflection on Participation in COP15: Engaging a New Generation of Leadership

The Will Steger Foundation, a Minnesota-based nonprofit organization launched Expedition Copenhagen, a Midwest youth delegation to the international climate negotiations at the UNFCCC in Copenhagen, Denmark, December 5–19, 2009, led by internationally renowned polar explorer Will Steger. Delegates assumed a leadership role in a regional climate campaign to pass strong climate legislation leading up to the international negotiations and also supported the Will Steger Foundation Citizen Climate curriculum in schools across the United States. The expedition goals were to build the US awareness of climate policy and investment in strong participation in the conference; highlight the unique role of the Midwest region since the Midwest is a key player in driving national climate policy, public opinion, and the renewable energy revolution; and bring the US youth voice to the negotiations whose future is at stake.

While in Copenhagen our delegation worked on a range of issues: interpreting high level policy discussions on adaptation, finance, climate justice, and technology transfer; participating in creative actions with youth from across the globe; and sharing their experiences with media and their peers in Copenhagen as well as back home in the Midwest. In addition to hosting a series of presentations and briefings over the course of the summit, Expedition Copenhagen team members met with government delegates and civil society from around the world.

At the conference, youth brought a moral voice to the negotiations. They told stories about how climate change is affecting their home communities and shared examples of how we can create a better world. Before and during the conference, youth joined together from around the world to network, sharing information and important skills while creating a stronger movement for solutions to climate change.

Not surprisingly, youth were able to parlay their size and coordination into results. On the night before the negotiations ended, as government delegates prepared for the possibility of leaving Copenhagen without any form of international agreement in place, 150 young people demonstrated outside the conference center. For hours they fought the bitter cold to call loudly for an agreement strong enough to prevent the worst impacts of climate change. At 2:30 am, they received a text message rehashing a statement just made inside by the UK Climate Minister, Ed

Miliband: “It’s youth and connected mobilization that put the pressure to get anything, especially the 130 leaders here. Stay strong.”

These and many, many other grassroots youth actions have expressed to policy-makers everywhere that young people are unwilling to see their futures destroyed by inaction, or to elect inactive candidates. Youth are the primary stakeholders in the struggle to solve climate change which threatens both the present and the distant future, and their efforts are now producing a groundswell of support. By meeting with senior leaders in the middle with youthful action from the bottom, youth helped create the political environment in which President Obama and other world leaders had no choice but to salvage a deal in Copenhagen.

Youth have strengths that they bring to these negotiations, but nothing is stronger than the moral voice and clarity they bring to the often intentionally complicated policy discussions that occur at the UN. Young people across the globe will bear the brunt of global warming consequences throughout our lifetime. Without key policy measures to encourage clean energy solutions, youth will inherit a more turbulent and expensive future as a result of unchecked global warming.

Youth also have the potential to move, organize, and act quickly. Young people represent more than the nongovernmental sector and have government delegates, media representatives, youth union reps, and more. They also are willing to call for bold action, develop innovative strategies for advocacy, and have a passion that is palpable to anyone that has spent any time in their presence. Yvo de Boer, former Executive Secretary of the UNFCCC, in an intergenerational inquiry on the role of youth at these negotiations, was asked what role young people should play in these talks. He said that too many NGOs have bureaucratized and dropped their banners to put on suits. He said young people must raise the profile of this issue in their home countries, until their governments are forced to listen, if they hope to influence the outcome. For a UN diplomat, it was quite a statement—acknowledging that governments need to be pressured publicly and NGOs were failing to act and remained myopically focused on research, policy expertise, and lobbying meetings.

In Copenhagen, and subsequent climate change conferences, the power of the youth voice continues to have an impact. Through media outreach, social networking, peaceful and creative actions, youth not only provide a call to action that is hard to ignore, but they also provide a sense of hope and inspire engagement in solutions.

To read dispatches and blogs from Expedition Copenhagen visit: <http://www.willstegerfoundation.org/expedition-copenhagen-2009/expedition-blog?start=5>. (There and back again, written by Jamie Racine—Expedition Copenhagen Delegate).

Many voices sounded in the streets across Copenhagen this past December, and they came together late in the evening on the 18th of December as COP15 came to a close. On that night, I sat in a small Danish apartment with a few of my fellow delegates and listened to President Obama give his final words on the accord. For the first time in nearly two and a half weeks, the world seemed quiet: Shock, exhaustion, disappointment, confusion. His seemingly empty words hung in the air. All I could hear was the breathing of my comrades and his political statement. Not a statement of conviction, passion, progression, change, but a statement of politics. I felt deceived, brokenhearted, emotional, and worn out. What had we worked so

hard for? What does this mean for us? For humanity? What does this mean? I felt blindsided by the auditory wrecking ball delivered by my President that thrust a gaping hole into my relentless hope for the impossible.

The world was quiet and still.

I took some time to talk with my friends and put this new reality to the back of my head. Just for a few hours, then I returned to the quiet, the quiet that had settled over Copenhagen and over the youth movement.

For the first time in 2 weeks, I did not have 350 emails to check by the end of the day. The world slowed back down. My psyche reverted to the corner of my mind with the oversized sofa and low-light lamp where I go to reflect when I don't know what to think. This is where I stayed for the next few days and my long travels home.

A few weeks later and back at home, a renewed passion has reignited in my heart. I have returned home to the thought and aspiration that initially inspired me to apply for Expedition Copenhagen: local, sustainable communities.

I met hundreds of young people and thousands of people of all ages from all over the world; each of their home communities has different ways to meet the same goal as communities all over the world: local and sustainable. We need to take care of each other here, at home. We can address this global issue of climate change through local solutions. The Midwest specifically has phenomenal opportunity to become a leader domestically and internationally through clean energy development.

The years ahead must be years of action. We must continue to hold our leaders at the top to the promises they campaign on, but we cannot go to the top alone. Our action, as we know, needs to happen at all levels of government and in the home of our community members.

Education for Action

Action begins with education. Because we are dealing with an immediate threat, we must launch a public education campaign to engage everyone in understanding the climate change issue and solutions to climate change. Congregations, environmental groups, youth organizations, campuses, and clubs of all kinds will play a pivotal role informing and engaging their members and moving them towards action. We must expect that our leaders in government, industry, congregations, and schools are well informed about climate change and its consequences.

In 2006, I decided to establish the Will Steger Foundation to address climate change through education and advocacy. The Will Steger Foundation's mission is to educate, inspire, and empower people to engage in solutions to climate change (Fig. 4.8). Our education program implements this mission through the support of educators, students, and the public with science-based interdisciplinary educational resources on climate change, its implications and solutions to achieve climate literacy.

There is virtually unanimous scientific agreement about climate change. Yet due to both the inherent complexity of the topic and the social controversies surrounding it, confusion and doubt often persist. If the nation is to address climate change, it



Fig. 4.8 Will Steger Foundation host annual educator conferences on climate change education with keynote speakers, including Dr. Naomi Oreskes (featured). Photo L to R: Carolyn Breedlove (formerly, National Education Association), Dr. Naomi Oreskes (author *Merchants of Doubt*), Nicole Rom (Executive Director, Will Steger Foundation), and Will Steger (President, Will Steger Foundation) (© 2010, Will Steger Foundation)

must begin with a public that is climate-literate. Starting with our educational system is critical [1–3]. Teaching and understanding climate change is a process involving scientific inquiry and educational pedagogy; it is not about politics or partisanship. Just recently, the National Center for Science Education, an organization responsible for defending the teaching of evolution in schools, began addressing the backlash that school districts are facing when climate deniers threaten the ability of educators to teach climate in their classrooms [4]. NCSE’s goal is to support educators who face pushback that’s based upon inaccuracies or misunderstanding of the science. Educators we work with are reporting their lack of knowledge about climate change. Broadening their understanding through professional development and curriculum resources strengthens their ability to teach the topic and answer colleagues, students, and parents who often do not know the facts of climate change.

Climate education means being able to understand the basics of Earth’s climate system, to know how to assess scientifically credible information about climate, to communicate about climate change in a meaningful way, and most importantly to be able to make informed and responsible decision regarding our actions that affect the climate. For example, we should know the reason for the seasons, the basic dynamics of the greenhouse effect and the carbon cycle, and the differences between weather and climate.

Standard curriculum and textbook cycles are often slow and subject to state and local review and debate, leaving them disconnected from new findings in climate science. For example, observed sea ice melt and changes in ice sheets are occurring faster than models had predicted. This disconnect quickly leads to outdated educational resources.

Climate change education must be based in peer-reviewed, consensus-based science. For this reason, our materials are aligned to the Climate and Energy Literacy principles. “Climate Literacy: The Essential Principles of Climate Science” and the Energy Literacy Principles are a product of the US Global Change Research Program and were compiled by an interagency group, led by NOAA.

We recognize the need for quality environmental education materials and educator support focused on climate change for a number of reasons. First, climate change is currently not included in most education curriculum. Teachers cite a lack of comfort, lack of time, and in some cases opposition from parents, administrators, and even students [5]. Second, there is a movement at a policy level to make it difficult for teachers to include climate change in their classroom, despite an overwhelming consensus among scientists of the reality of climate change and of humans as the main driver [6]. Recent legislation passed in Tennessee gives teachers the ability to challenge climate change in their classroom without fear of sanction [7]. Finally, as NASA scientist Dr. James Hansen pointed out in a TED talk, “climate change is like a giant asteroid that’s on course to hit earth.” Hansen’s comments illustrate how important it is for us to prepare educators to teach about climate change science *and* solutions and to develop students with the twenty-first century skills to mitigate and adapt to the impacts of climate change.

We also recognize an opportunity for climate change education on a number of levels. The newly released *Next Generation Science Standards* [8] explicitly include climate change. Science educators will need to have access to quality, science-based materials, and professional development as climate change becomes a core subject to include in the science classroom. We also see an opportunity to use climate change education as a means for addressing what Richard Louv has called, “Nature Deficit Disorder” [9]. Connecting educators and students with the natural world is a powerful tool for not only making climate change relevant, but for engaging students in solutions. This can occur through outdoor mitigation projects and through students own contributions to observations of change in their backyard.

While education is critical, action is also needed in our energy system. We need to explore diverse energy sources, continue our search for increased fuel efficiency, and increase our domestic production of transportation fuels. Significantly increasing the use of domestic-produced biofuels offers both immediate and potential long-term solutions to national security, economic competitiveness of the United States, and price and supply vulnerabilities for families and businesses. Domestically produced energy also benefit the United States by creating jobs, keeping dollars in the country, and lowering the environmental impacts associated with fossil fuel production and use. We can reduce global warming pollution through conservation, existing technologies that make power plants and factories more efficient, and cleaner technologies.

Global warming, an environmental and moral issue, is also a unifying issue. It affects all of us; therefore, the solution requires all of us. Individual action leads to collective action. But individual action alone will not solve the problem. We need to demand that our elected officials act to create solutions to climate change. State and local initiatives are proving that answers exist. To reinforce and expand these efforts, we need federal action that triggers solutions on a national scale. The US businesses can and should lead the world in developing new energy technologies, but many of these businesses will not lead without the guidance of mandatory limits.

The effects of global warming are pervasive. We cannot delay in slowing and reversing this trend. Our health, economy, national security, and the environment demand it.

References

1. Barry J, editor. The Belgrade Charter: A global framework for environmental education. Connect-UNESCO-UNEP Environmental Education Newsletter. 1976;1:1–9. <http://unesdoc.unesco.org/images/0015/001533/153391eb.pdf>
2. National Research Council. Climate change education goals, audiences, and strategies: a workshop summary. Washington, DC: National Academies Press; 2011.
3. USGCRP. Essential principles of climate science: a guide for individuals and communities. Washington, DC: USGCRP; 2009.
4. National Center for Science Education: Climate. 2012. <http://ncse.com/climate>. Accessed May 2012.
5. Board on Science Education, NAS. A framework for K-12 science education: practices, cross-cutting concepts, and core ideas. Washington, DC: National Academies Press; 2011.
6. Simmons B. Climate change education in the K-12 setting: lessons learned from environmental education. Washington, DC: National Academies Press; 2011.
7. Banerjee N. L.A. Times. 2012. <http://articles.latimes.com/2012/apr/11/nation/la-na-tennessee-climate-law-20120411>. Accessed May 2012.
8. Next Generation Science Standards. 2012. <http://www.nextgenscience.org/>
9. Louv R. Last child in the woods: saving our children from nature deficit disorder. Chapel Hill, NC: Algonquin Books; 2005.

Chapter 5

California and Climate Changes

Rupa Basu

Abstract Epidemiologic studies of temperature and adverse health outcomes in California are their incipient stage, as the majority of the research has been conducted in the past 5 years. Exposure has been defined primarily as apparent temperature, a combination of temperature and humidity, a measure that has been calculated from meteorologic monitors supplied by the California Irrigation Management System and the United States Environmental Protection Agency. The various outcomes that have been studied include mortality and morbidity, such as hospitalizations, emergency room visits, and in one study, preterm delivery. Air pollutants have often been examined as potential confounders or effect modifiers. The results have shown a positive association between temperature and various health outcomes and have identified increased risk for infants, young children, the elderly, and Blacks and for some specific cardiovascular and respiratory diseases. Identifying vulnerable subgroups for local regions will be essential to decreasing heat-related mortality and morbidity.

Keywords Temperature and mortality studies • Temperature and hospitalizations • Increased mortality • Morbidity • Vulnerable subgroups • Mortality displacement • California

According to the Intergovernmental Panel on Climate Change [1], global warming impacts are likely to result in increased deaths, cardiorespiratory diseases, and injury due to heat waves, among other public health impacts. This chapter summarizes

Disclaimer The opinions expressed in this article are solely those of the author and do not represent the policy or position of the State of California or the California Environmental Protection Agency.

R. Basu, Ph.D., M.P.H. (✉)

Office of Environmental Health Hazard Assessment, Air Pollution Epidemiology Section,
Cal EPA, 1515 Clay Street, 16th floor, Oakland, CA 94612, USA
e-mail: Rupa.Basu@oehha.ca.gov

epidemiologic studies of temperature and adverse health outcomes, focusing primarily on California. The health outcomes discussed include mortality and morbidity, such as hospital visits, emergency room visits, and preterm delivery.

Summary of Epidemiologic Studies of Temperature and Mortality

Temperature and Mortality Studies in the United States Including California

Previous studies of heat waves or elevated temperature and mortality have been documented worldwide and summarized in two recent epidemiologic review articles [2, 3].

A few investigators examining temperature and mortality in the United States have included cities or counties in California as a part of their analyses.

Among the first studies of temperature and cardiorespiratory mortality was conducted by Basu et al. [4] using National Morbidity and Mortality Air Pollution Study (NMMAPS) data from 20 metropolitan areas in the United States. The investigators reported a positive association between temperature and mortality in the summer for all regions and mostly null or negative associations during all other seasons. The Southwest region consisting of Phoenix, AZ; San Diego, CA; Santa Ana, CA; Los Angeles, CA; and San Bernardino, CA, had the highest regional effect with an odds ratio (OR) of 1.15 (95 % confidence interval (CI): 1.07, 1.24) per 10° Fahrenheit (°F) increase in mean daily temperature, adjusted for dew point temperature to account for humidity. In this study, the time-stratified case-crossover approach using logistic regression models (Fig. 5.1) and the time-series analysis using Poisson regression models produced virtually identical results. Since this study was based on 1 year of data in 1992, more studies of multiple areas over a longer time period are warranted.

Recently, other investigators have expanded the NMMAPS data to include more metropolitan areas throughout the United States [5, 6]. Barnett [5] included 107 cities in their analysis to compare findings between the summers of 1987 and 2000. He reported an elevated risk in 1987 for temperature and cardiovascular mortality that was no longer observed in 2000. Similar to the Basu et al. [4] study, regional analyses showed that southern California had among the greatest effects in 1987 but also had the largest decline in 2000. The author attributes the diminished effect partially to the increased availability of air conditioning (AC). However, racial disparities have been reported for access to AC in the United States [7], and thus, AC use is not a viable solution to mitigate heat-related health impacts equally for everyone that may be affected. Furthermore, prolonged and widespread AC use can lead to power brownouts and blackouts. In another investigation of 95 NMMAPS cities from 1987 to 2000, the potential effect modification by ozone on the

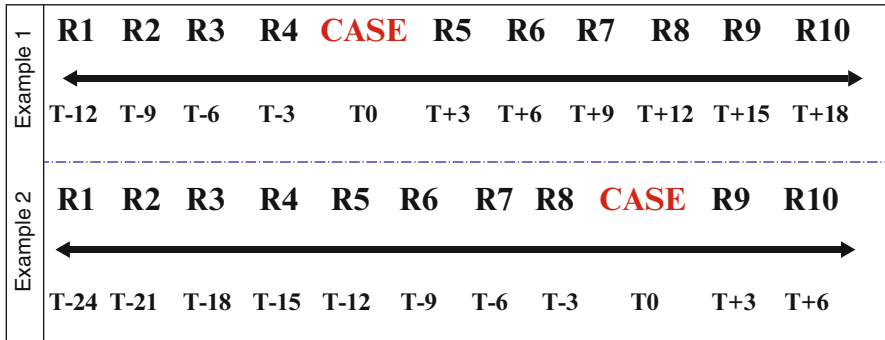


Fig. 5.1 Schematic diagram of time-stratified case-crossover design. CASE, case period; R1-R10, referent periods 1–10 every third day in the same month and year; T0, time that case occurred (death date); T–24 ... T+18, time that referent periods occurred

temperature and cardiovascular mortality association was assessed during the summer months [6]. A synergistic effect between ozone and temperature in most regions, including Southern California, was observed.

In another case-crossover study of temperature and mortality in 50 US cities using data from 1989 to 2000, investigators explored extreme temperatures, using various cutoff values for temperature [8]. In their analysis of over six million observations, mortality was found to increase with extreme heat (5.74 %, 95 % CI: 3.38, 8.15). Although no estimates were provided for California specifically, Los Angeles and San Diego were included in the overall analysis. The largest effects were generally observed in cities with milder summers, less AC, and higher population density. In another case-only study using the same data, Medina-Ramón et al. [9] found that older subjects, diabetics, Blacks, and those dying outside a hospital were more susceptible to the effects of extreme heat.

Temperature and Mortality Studies in California

California is unique since temperature and humidity tend to be relatively mild, while pollutant levels are generally high with distinct sources and patterns of exposure. Furthermore, people spend more time outdoors throughout the year in California, lending them the potential for more exposure to heat, air pollution, smoke, as well as vector-borne diseases. AC use is not a surrogate for socioeconomic status, as it may be in other parts of the country. Many homes in coastal areas do not have AC installed because predominantly cool temperatures minimize the need, although coastal homes tend to be more expensive and, thus, consist of a wealthier population. Thus, people living in coastal areas may be more impacted by a heat wave since they do not have air conditioning in their homes and are not acclimatized to high ambient temperatures.

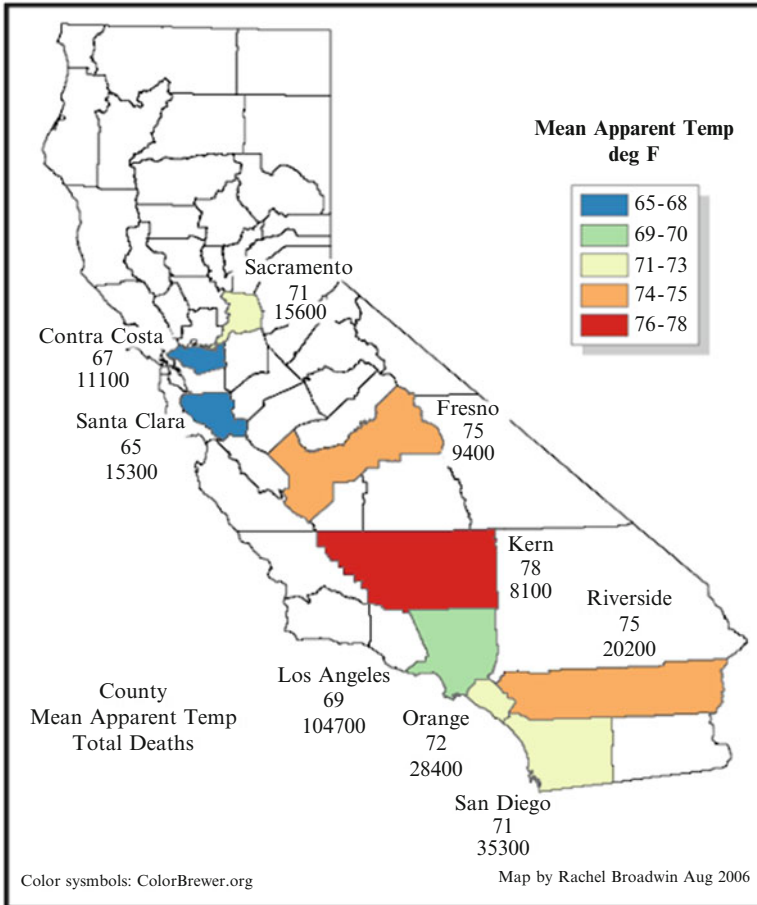


Fig. 5.2 Mean apparent temperature for nine California counties, May to September 1999–2003 (from Rupa Basu, PhD, MPH, Oral presentation entitled “An Epidemiologic Study of Temperature and Mortality in California: Implications for Climate Change.” California Energy Commission, Sacramento, September 15, 2006 with permission)

Investigators have estimated the impact of temperature on mortality in California [10–12]. Temperature and mortality data from nine counties in California were analyzed including Contra Costa, Fresno, Kern, Los Angeles, Orange, Riverside, Sacramento, San Diego, and Santa Clara, which comprise approximately 65 % of the State’s population, and include regions in northern and southern California and inland and coastal regions (Fig. 5.2). To focus on heat effects, data were limited to the warm season from May 1 to September 30, 1999–2003. Air pollutants were accounted for in the analyses as potential confounders or effect modifiers. County-specific estimates were obtained followed by an overall combined estimate using the random effects model in meta-analyses [13].

In the first epidemiologic study of temperature and mortality in California, the primary goal was to establish methods to examine the association independent from air pollutants [11]. A total of 248,019 deaths were included. Same-day lag was found to have the best data fit and also the highest risk estimates, demonstrating the acute effect of temperature on mortality. Each 10 °F increase in same-day mean apparent temperature corresponded to a 2.3 % increase in mortality (95 % CI: 1.0, 3.6) in the time-stratified case-crossover analysis for all nine counties combined, with similar results produced in the time-series analysis. No air pollutant examined was found to be a significant confounder or effect modifier. Regional differences within California were found between coastal and inland areas, and thus, region-specific policies are warranted. An association between temperature and California was observed in a relatively mild climate without focusing on extremes in apparent temperature or heat waves. The findings from this study are comparable to temperature and mortality in other regions in the United States using the same methods [14].

Vulnerable Subgroups

In a second time-stratified case-crossover study examining temperature and mortality in California, vulnerable subgroups were identified [12]. A total of 231,676 non-accidental deaths were included to evaluate several disease categories and subgroups including cardiovascular, respiratory, cerebrovascular, and diabetes. Effect modification by race/ethnic group, age, sex, and education level was also considered. Each 10 °F increase in mean daily apparent temperature corresponded to a 2.6 % (95 % CI: 1.3, 3.9) increase in cardiovascular disease mortality, with elevated risk especially found for ischemic heart disease. Acute myocardial infarction (MI) and congestive heart failure also had elevated risks, although respiratory disease mortality did not. High risks were also found for persons at least 65 years of age (2.2 %, 95 % CI: 0.04, 4.0), infants 1 year of age and under (4.9 %, 95 % CI: -1.8, 11.6), and Black non-Hispanic racial/ethnic group (4.9 %, 95 % CI: 2.0, 7.9). No differences were found by gender or education level. Thus, persons at risk for cardiovascular disease, the elderly, infants, and Blacks among others should be targeted to prevent mortality associated with high apparent temperature.

Mortality Displacement

In a time-series study, the potential effect of mortality displacement in the relationship between apparent temperature and mortality was explored [10]. Mortality displacement, also known as harvesting, refers to the phenomenon in which a specific exposure, such as temperature, impacts already frail individuals whose deaths may have only been brought forward by a few days. Significant associations were observed for the same day (excess risk, 4.3 % per 10 °F increase in apparent temperature, 95 % CI: 3.4, 5.2) continuing up to a maximum of 4 days following apparent temperature exposure for non-accidental mortality (Fig. 5.3). Similar patterns of

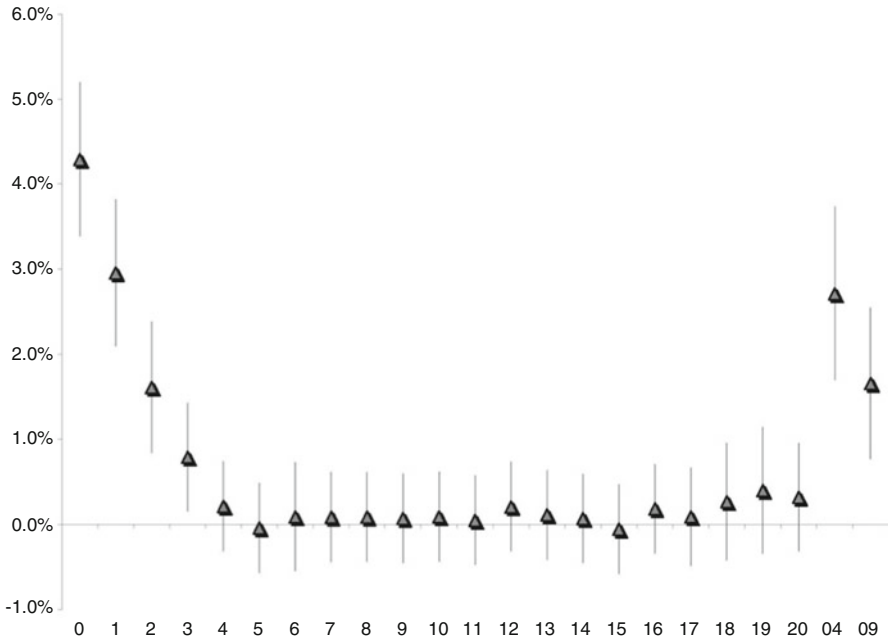


Fig. 5.3 Estimated percent change (95 % CI) associated with a 10 °F increase in mean apparent temperature and non-accidental mortality in 13 counties in California, May to September, 1999–2005

risk were found for mortality from cardiovascular diseases and respiratory diseases among children 0–18 years of age and among those 50 years and older. Since no significantly negative effects were observed in the following single or cumulative days, evidence of mortality displacement was not found. Thus, the effect of temperature on mortality in California appears to be an event that occurs within 3 days following exposure, with the most significant impact occurring on the same day, and appears to have a broad impact on the general population.

Heat Waves and Mortality

Two studies were recently conducted focusing on the Phoenix metropolitan area in Arizona [15, 16]. One study focused on heat-related deaths occurring from June to September 2000 to 2005 [15]. Per °F, a 6 % (1.00, 1.13) increase in mortality risk was observed. Most deaths occurring outdoors affected children under 5 years of age, while the majority of indoor deaths occurred among the elderly at least 65 years of age. Other investigators examined heat-related medical dispatches in Phoenix from 2001 through 2006 and found that maximum daytime temperature and elevated comfort indices, a measurement of temperature and relative humidity, were associated with the greatest risk [16].

The estimates provided in the previous section discussing studies conducted in California were based on background apparent temperature, including both heat wave and non-heat wave periods. Thus, they do not capture the worst-case scenario, as would be observed during heat wave periods only. Ostro et al. [17] investigated the July 2006 heat wave in California from July 14 to August 1, 2006. County coroners reported that high ambient temperatures caused 142 deaths. However, heat wave deaths are likely to be underreported due to a lack of a clear case definition and the multifactorial nature of mortality [2]. Furthermore, no systematic definition for heat-related deaths currently exists in the United States or California specifically. Daily data were collected for mortality, relative humidity, ambient temperature, and ozone in seven California counties known to be impacted by the July 2006 heat wave. The combined meta-analytic results suggested a 9 % (95 % CI: 1.6, 16.3) increase in daily mortality per 10 °F change in apparent temperature, which is more than 3 times larger than the effect estimated for the full warm season and corresponds to a number of deaths 2 or 3 times greater than the coroner estimates. The studies summarized provide a quantification of heat-related effects in California using epidemiologic methods. The first three studies described methodology, vulnerable subgroups, and potential mortality displacement of the apparent temperature-mortality association, while the last study focused on mortality during the 2006 heat wave.

Summary of Epidemiologic Studies of Temperature and Morbidity

Temperature and Hospitalizations/Emergency Room Visits

Using the same nine counties as the mortality analyses, temperature and hospitalizations from various causes were evaluated [18]. The study population consisted of 597,735 individuals who were admitted to a hospital with selected diagnoses and lived within 10 km of a temperature monitor in an effort to refine exposure assessment. A 10 °F increase in mean apparent temperature was associated with a 3.5 % (95 % CI: 1.5, 5.6) increase in several disease-specific outcomes, such as ischemic stroke, all respiratory diseases (2.0 %, 95 % CI: 0.7, 3.2), pneumonia (3.7 %, 95 % CI: 1.7, 3.7), dehydration (10.8 %, 95 % CI: 8.3, 13.6), diabetes (3.1 %, 95 % CI: 0.4, 5.9), and acute renal failure (7.4 %, 95 % CI: 4.0, 10.9). There was little evidence that the temperature effects found were due to confounding by either PM_{2.5} or ozone. In a follow-up study using data provided by a housing survey [19], the impact of AC use was investigated on the apparent temperature-hospitalization associations. [20] Although ownership and usage of ACs significantly reduced the effects of temperature on several health outcomes, even after controlling for potential confounding by family income and other socioeconomic factors, the associations between temperature and the health outcomes remained robust.

In a recently published time-stratified case-crossover study of over 1.2 million ER visits in 16 climate zones [19] in California, the study population consisted of cases who resided within 10 km of a temperature monitor in the same climate zone [21]. Significant positive associations for same-day apparent temperature and ischemic heart disease (% excess risk per 10 °F: 1.7; 95 % CI: 0.2, 3.3), ischemic stroke (2.8; 0.9, 4.7), cardiac dysrhythmia (2.8; 0.9, 4.9), hypotension (12.7; 8.3, 17.4), diabetes (4.3; 2.8, 5.9), intestinal infection (6.1; 3.3, 9.0), dehydration (25.6; 21.9, 29.4), acute renal failure (15.9; 12.7, 19.3), and heat illness (393.3; 331.2, 464.5). Statistically significant negative associations were found for aneurysm, hemorrhagic stroke, and hypertension. These estimates all remained relatively unchanged after adjusting for air pollutants, with the exception of pneumonia and all respiratory diseases, which were confounded by nitrogen dioxide and carbon monoxide. Risks often varied by age or racial/ethnic group. Thus, risk prevention strategies for morbidity during heat exposure require an immediate response and should consider those who are at greatest risk for cardiovascular disease, as well as the elderly, children, and minority race/ethnic groups.

Heat Waves and Morbidity

In a study examining the effects of the 2006 California heat wave on morbidity, Knowlton et al. [22] aggregated county-level hospitalizations and emergency department (ED) visits for all causes and for some specific causes for six geographic regions of California. Excess morbidity and rate ratios (RRs) during the heat wave (July 15 to August 1, 2006) were calculated and compared to a referent period (July 8–14 and August 12–22, 2006). During the heat wave, 16,166 excess ED visits and 1,182 excess hospitalizations occurred. ED visits for heat-related causes were found to be increased (RR 6.30, 95 % CI: 5.67, 7.01). The greatest risk was found in the Central Coast, children (0–4 years) and the elderly (≥ 65 years of age). Acute renal failure, cardiovascular diseases, diabetes, electrolyte imbalance, and nephritis also had significantly increased risk. Some regions with relatively mild temperatures were found to be at increased risk, suggesting the influential roles of population acclimatization and biological adaptation.

Temperature and Adverse Birth Outcomes

In the first large-scaled study of temperature and preterm delivery in the United States, Basu et al. [23] examined approximately 60,000 births spanning 16 counties in California from May through September 1999–2006. The investigators identified cases of preterm delivery from a state registry of births, which were combined

with temperature and air pollution monitoring data based on residential zip code. Apparent temperature was significantly associated with preterm birth for all mothers, regardless of maternal race/ethnic group, age, education, or infant sex. Per 10 °F increase in weekly average (lag06) apparent temperature, an 8.6 % (95 % CI: 6.0, 11.3) increase in preterm delivery was found. Greater associations were observed for younger mothers, African-Americans, and Asians. These associations were found to be independent of air pollutants. Since this study was the first to report positive associations between temperature during the warm season and preterm delivery, more large-scaled studies of temperature and other adverse birth outcomes are warranted to establish associations in various locales.

El Niño Events

El Niño refers to a temporary change in the climate of the Pacific Ocean, in the region around the equator. The changes in weather are observed in both the ocean and the atmosphere, generally in the Northern Hemisphere during the winter. Typically, the ocean surface warms up by a few degrees Celsius, causing thunderstorms to move eastward, as well as other marked effects on the world's climate.

Investigators have examined existing trends in weather and hospitalizations for several cardiovascular outcomes (MI, angina pectoris, congestive heart failure) and stroke during both normal weather patterns and during El Niño events in three regions of California: Los Angeles, Sacramento, and San Francisco from 1983 to 1998 [24]. Although they found minimal changes in hospitalizations due to weather in Los Angeles, a 5 °F decrease in maximum temperature or a 5 °F increase in minimum temperature was associated with significant increases (6–13 %) in hospitalizations for all outcomes studied among those 70 years of age and older in San Francisco. Similar patterns were observed for men 70 years of age and older in Sacramento: 6–11 % increase for MI and 10–18 % increase for stroke. El Niño events were found to be significantly associated with increased hospitalizations particularly for angina pectoris in San Francisco and Sacramento, but not in Los Angeles.

The same investigators also studied women over the same time period to examine the association between weather and viral pneumonia [25]. A 5 °F decrease in minimum temperature resulted in significant increases (30–50 %) in hospitalizations in San Francisco and Los Angeles, whereas a 5 °F decrease in maximum temperature difference produced significant increases (25–40 %) in hospitalizations in Sacramento. The associations were found to be independent of season. El Niño events were associated with hospitalizations only in Sacramento, with significant decreases for girls and increases for women.

An understanding of the changing patterns of hospital admissions during periods of weather changes is beneficial for evaluating population vulnerability and developing public health response.

Projections for Climate and Mortality

A few investigators have projected the effects of climate change and mortality, specifically for California. Using various climate models, greater increases in summer temperatures compared to winter temperatures are predicted. Based on the higher A1 emission scenarios, heat waves and extreme heat in Los Angeles are expected to be 6–8 times more frequent, with heat-related excess mortality increasing 5–8 times by the year 2100 [26]. The projections were slightly lower for the lower B2 emission scenarios. Other investigators also predicted a significant increase in heat events with longer duration and greater frequency over the twenty-first century, particularly for coastal areas of California [27]. By the 2090s, annual mortality could rise to a total of 4,684–8,757 deaths per year in California depending upon the scenario used from the General Circulation Model. The elderly over 65 years and urban centers are likely to face the greatest impact. A similar prediction was made in another study, with the central estimate of annual mortality ranging from 2,100 to 4,300 for the year 2025 and from 6,700 to 11,300 for 2050 [28]. Estimates using the low B1 emissions scenario are roughly half of these values. A 10 % and 20 % increase in AC use would generate reductions of 16 % and 33 % in the years 2025 and 2050, respectively. A national US estimate of annual incidence of heat-related mortality was found to be 3,700–3,800 from all causes, 3,500 from cardiovascular disease, and 21,000–27,000 from non-accidental deaths from May through September 2048 to 2052 relative to 1999–2003 using the A1 emissions scenario [29].

Biologic Mechanisms

Since heat-related mortality and morbidity have multiple etiologies, a clear biologic mechanism or cause is unknown. Susceptible individuals may not be able to thermoregulate efficiently. When body temperatures rise, the body generally shifts blood flow from the vital organs to the skin's surface in an effort to cool down [30]. Thus, thermoregulation may be inadequate when too much blood is diverted from the vital organs [31]. Increased blood viscosity, elevated cholesterol levels associated with higher temperatures, and a higher sweating threshold have also been reported in susceptible subgroups [32]. Another possible explanation for preterm delivery may be increased dehydration with heat exposure, which could decrease uterine blood flow and increase pituitary secretion of antidiuretic hormone and oxytocin to induce labor [33].

Conclusions

Public health impacts of climate change in California are expected to be broad, including direct impacts from increased temperature and extreme weather events. Most of the epidemiologic studies of temperature and mortality or morbidity that

have been published have been conducted over the past decade. Prior to that, most research had focused on case reports following heat waves, rather than using background apparent temperature as a measure of exposure. However, the topic is still in its nascent phase, and relatively very little research has focused on the Southwest or on California specifically.

Several important research questions remain regarding the relationship between temperature, heat waves, and subsequent human morbidity and mortality. More information from public health research is needed to provide the National Weather Service the best measure of heat warning (e.g., heat index) that is predictive of morbidity and mortality. Recommendations need to be developed based on the characteristics that comprise the most effective heat warning systems in the United States and abroad and how to develop such systems locally. Although individuals may know about heat warning systems, they may not be aware of what actions need to be taken or perceive themselves as being at increased risk [34]. Identifying comorbidities in vulnerable subgroups such as the elderly and children, as well as communicating to them precautionary efforts that can be taken, is crucial. Expansion of personal heat exposure assessment studies, using methods described previously by Basu and Samet [35], would be informative for identifying individual high-risk characteristics, as well as for understanding the biological mechanism between heat exposure and associated morbidity and mortality. Furthermore, no research has been conducted analyzing the characteristics of air masses (humidity, stagnation, period of occurrence, length) in relation to morbidity and mortality. Thus, the associations between temperature and adverse health outcomes need to be further investigated across all temperature exposure levels. Since heat waves are expected to occur more frequently with longer duration, the focus of epidemiologic studies should be on the higher end of temperature exposure, as they are expected to have the greatest public health impact in the future [1].

References

1. Intergovernmental Panel on Climate Change. Climate change 2007: synthesis report. In: Intergovernmental Panel on Climate Change fourth assessment report; 2007.
2. Basu R, Samet JM. Relation between elevated ambient temperature and mortality: a review of the epidemiologic evidence. *Epidemiol Rev.* 2002;24:190–202.
3. Basu R. High ambient temperature and mortality: a review of epidemiologic studies from 2001 to 2008. *Environ Health.* 2009;8:40.
4. Basu R, Dominici F, Samet JM. Temperature and mortality among the elderly in the United States: a comparison of epidemiologic methods. *Epidemiology.* 2005;16:58–66.
5. Barnett AG. Temperature and cardiovascular deaths in the US elderly: changes over time. *Epidemiology.* 2007;18:369–72.
6. Ren C, Williams GM, Morawska L, Mengersen K, Tong S. Ozone modifies associations between temperature and cardiovascular mortality: analysis of the NMMAPS data. *Occup Environ Med.* 2008;65:255–60.
7. O’Neill MS, Zanobetti A, Schwartz J. Disparities by race in heat-related mortality in four US cities: the role of air conditioning prevalence. *J Urban Health.* 2005;82:191–7.

8. Medina-Ramón M, Schwartz J. Temperature, temperature extremes, and mortality: a study of acclimatization and effect modification in 50 United States cities. *Occup Environ Med.* 2007;64(12):827–33.
9. 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;114:1331.
10. Basu R, Malig B. High ambient temperature and mortality in California: exploring the roles of age, disease, and mortality displacement. *Environ Res.* 2011;111(8):1286–92.
11. Basu R, Feng WY, Ostro BD. Characterizing temperature and mortality in nine California counties. *Epidemiology.* 2008;19:138–45.
12. Basu R, Ostro BD. A multicounty analysis identifying the populations vulnerable to mortality associated with high ambient temperature in California. *Am J Epidemiol.* 2008;168:632–7.
13. DerSimonian R, Laird N. Meta-analysis in clinical trials. *Control Clin Trials.* 1986;7:177–88.
14. Zanobetti A, Schwartz J. Temperature and mortality in nine US cities. *Epidemiology.* 2008;19:563–70.
15. Yip FY, Flanders WD, Wolkin A, et al. The impact of excess heat events in Maricopa County, Arizona: 2000–2005. *Int J Biometeorol.* 2008;52:765–72.
16. Golden JS, Hartz D, Brazel A, Lubber G, Phelan P. A biometeorology study of climate and heat-related morbidity in Phoenix from 2001 to 2006. *Int J Biometeorol.* 2008;52:471–80.
17. Ostro BD, Roth LA, Green RS, Basu R. Estimating the mortality effect of the July 2006 California heat wave. *Environ Res.* 2009;109:614–9.
18. Green RS, Basu R, Malig B, Broadwin R, Kim JJ, Ostro B. The effect of temperature on hospital admissions in nine California counties. *Int J Public Health.* 2010;55:113–21.
19. California Energy Commission (CEC). California Statewide Residential Appliance Saturation Study: final report. 400-04-009; 2004.
20. Ostro B, Rauch S, Green R, Malig B, Basu R. The effects of temperature and use of air conditioning on hospitalizations. *Am J Epidemiol.* 2010;172:1053–61.
21. Basu R, Pearson D, Malig B, Broadwin R, Green S. The effect of elevated ambient temperature on emergency room visits. *Epidemiol.* 2012;23(6):813–20.
22. Knowlton K, Rotkin-Ellman M, King G, Margolis HG, Smith D, Solomon G, et al. The 2006 California heat wave: impacts on hospitalizations and emergency department visits. *Environ Health Perspect.* 2009;117:61–7.
23. Basu R, Malig B, Ostro B. High ambient temperature and the risk of preterm delivery. *Am J Epidemiol.* 2010;172:1108–17.
24. Ebi KL, Exuzides KA, Lau E, Kelsh M, Barnston A. Weather changes associated with hospitalizations for cardiovascular diseases and stroke in California, 1983–1998. *Int J Biometeorol.* 2004;49:48–58.
25. Ebi KL, Exuzides KA, Lau E, Kelsh M, Barnston A. Association of normal weather periods and El Niño events with hospitalization for viral pneumonia in females: California, 1983–1998. *Am J Public Health.* 2001;91:1200–8.
26. Hayhoe K, Cayan D, Field CB, et al. Emissions pathways, climate change, and impacts on California. *Proc Natl Acad Sci USA.* 2004;101:12422–7.
27. Sheridan S, Kalkstein, L. A spatial synoptic classification approach to projected heat vulnerability in California under future climate changes scenarios. Prepared for the California Air Resources Board and California Environmental Protection Agency; 2011. p. 1–144.
28. Ostro B, Rauch S, Green S. Quantifying the health impacts of future changes in California. *Environ Res.* 2011;111:1258–64.
29. Voorhees AS, Fann N, Fulcher C, et al. Climate change-related temperature impacts on warm season heat mortality: a proof-of-Concept methodology using BenMAP. *Environ Sci Technol.* 2011;45:1450–7.
30. Charles L, Schauf DM, Stacie M. Human physiology: foundations and frontiers. 2nd ed. Oxford: William C Brown; 1993.
31. Bouchama A, Knochel JP. Heat stroke. *N Engl J Med.* 2002;346:1978–88.

32. Astrand P-O, Rodahl K, Dahl HA, Stromme SB. Textbook of work physiology: physiological bases of exercise. 4th ed. Canada: McGraw-Hill; 2003.
33. Stan C, Boulvain M, Hirsbrunner-Amagbaly P, Pfister R. Hydration for treatment of preterm labour. *Cochrane Database Syst Rev.* 2002; CD003096.
34. Kalkstein AJ, Sheridan SC. The social impacts of the heat-health watch/warning system in Phoenix, Arizona: assessing the perceived risk and response of the public. *Int J Biometeorol.* 2007;52:43–55.
35. Basu R, Samet JM. An exposure assessment study of ambient heat exposure in an elderly population in Baltimore, Maryland. *Environ Health Perspect.* 2002;110:1219–24.

Chapter 6

Heat Waves and Rising Temperatures: Human Health Impacts and the Determinants of Vulnerability

Helene G. Margolis

Abstract Globally, heat waves account for dramatic increases in mortality and morbidity; however, there is increasing awareness that day-to-day increases in temperature contribute to a significant risk of heat-related morbidity and mortality (HRMM) that over one or more warm seasons may exceed the public health burden of heat waves. Climate change has already and will continue to increase both average ambient temperatures and the frequency and intensity of excursions above those averages (i.e., heat waves or extreme heat events) and will thereby lead directly and indirectly to amplification of the risk of HRMM. This chapter provides a brief synopsis of our current knowledge about thermoregulation, thermotolerance and the pathophysiology of heat stroke, and the multiple determinants of health and illness that influence the risk of HRMM and that collectively define vulnerability. A particular focus is on two vulnerable populations, older adults and children. An Environmental Health Multiple-Determinant Model of Vulnerability is presented as a conceptual framework to integrate that knowledge, with the intent of providing a tool that can facilitate compilation and translation of the information to interventions and adaptation strategies relevant at the individual level and/or subpopulation and population levels and at one or more geopolitical scales in developing and/or developed nations. Three overarching strategies for HRMM risk reduction are discussed, including Extreme Heat Event and Warm Season Heat Preparedness and Response Action Plans, Promote Good Health and Access to Quality Healthcare (reduces risk and increases resiliency), and Reduce/Manage Potential Exposure(s) (individual, community) to Ambient Heat and Other Physical Environmental Stressors. A key focus of this chapter is on integration and translation of knowledge.

H.G. Margolis, Ph.D., M.A. (✉)

Department of Internal Medicine: General Medicine

Center for Healthcare Policy & Research, School of Medicine, University of California,

Davis, 4150 V Street, Suite #2400, Sacramento, CA 95817, USA

e-mail: helene.margolis@ucdmc.ucdavis.edu

Keywords Heat waves and climate change • Climate change and heat waves • Rising temperatures in climate change • Heat-related morbidity and mortality • Public health burden of heat waves • Heat stroke

Over evolutionary time scales, humans have evolved to tolerate ambient heat across a fairly wide range of environmental conditions; that ability is enabled by behavioral and complex biological/physiological thermoregulatory adaptations that serve to maintain an average core body temperature within a narrow life-sustaining range around 37 °C (98.6 °F) [1] regardless where they live or where their ancestors evolved [2]. Under past and present climatic conditions, human populations around the globe have been and continue to be exposed to periods of *extreme* high temperatures that pose a risk of adverse health impacts, which include but are not limited to a suite of mild-to-severe conditions within the rubric of “heat-related illness (HRI),” and acute exacerbations of prevalent chronic diseases [3, 4], as well as death that may or may not be attributed as a direct or indirect consequence of heat exposure or a combination of heat and comorbidity. Climate change has already and will continue to increase both average ambient temperatures and the frequency and intensity of excursions above those averages [5] and will thereby lead directly and indirectly to amplification of the risk of heat-related morbidity and mortality (HRMM) [6]. (Key terms used in this chapter are defined in Table 6.1).

Modern societies, especially politically and economically stable nations, have social systems that include mechanisms designed to protect the stability of the society by reducing the health risks and/or increase the resilience of the overall population during natural disasters, including heat waves or more generally “extreme heat events” (EHE) (see Table 6.1). Thus one would expect, at least in developed nations, for there to be sufficient experience and knowledge, guidance, policies, and infrastructure to adequately protect the population’s health during EHE. This expectation was proven wrong in 2003, when an intense and extended heat wave and exceptionally hot summer in Europe claimed about 70,000 lives [7]—with about 15,000 deaths occurring in France alone [8]. Extreme heat exposure remains the leading cause of weather-related deaths in the United States [9]. Although the death toll paled in comparison to the 2003 European heat wave, the summer 2006 California heat wave, which affected most of the State and was of unprecedented intensity (with both extreme high daytime maximum temperatures and high nighttime minimum temperatures) and duration (about 17 days) [10, 11], had a very significant public health burden. That event is estimated to have resulted in over 600 excess deaths [12, 13] and about 1,200 excess hospitalizations and 16,000 excess emergency department contacts for a variety of causes [14]. The economic cost of the health impacts (mortality and morbidity) of that event has been estimated to have been \$5.4 billion [15].

Importantly, although less dramatic than a heat wave-related sudden upsurge in deaths and illnesses, there are significant health risks associated with day-to-day excursions in temperature above local warm season means that might not meet a

Table 6.1 Glossary of terms

Heat-related morbidity and mortality (HRMM):	this term is used here to reflect the full-spectrum of causes of illness or death, including heat-related illness (HRI; a clinically defined spectrum of conditions associated with excessive heat stress). The abbreviation HRI is used when explicitly referring to one or more conditions within the spectrum of heat-related illnesses
Heat wave (extreme heat event):	there is no universally accepted definition of “heat wave”; however, commonly applied criteria include the occurrence of temperatures, or a temperature plus humidity metric (e.g., Heat Index or Humidex) above a threshold level that persists over 2 or 3 consecutive days. The term extreme (or excessive) heat event (EHE) is generally used synonymously with “heat wave”; for the purposes of this chapter, the term is used to represent any extreme excursion above usual average temperature conditions that may pose a health risk, regardless of whether it meets criteria for designation as a heat wave
Vulnerability:	the definition applied in this chapter (see text) has a public health orientation and differs from the definition used by the IPCC (Climate Change 2007: Synthesis Report), which states: <i>Vulnerability is the degree to which a system is susceptible to, and unable to cope with, adverse effects of climate change, including climate variability and extremes. Vulnerability is a function of the character, magnitude, and rate of climate change and variation to which a system is exposed, its sensitivity, and its adaptive capacity</i>
Heat stress:	<i>heat stress</i> is defined as the total heat load on the body from metabolic heat production plus external environmental factors; and <i>Heat Strain</i> is the total physiological stresses resulting from heat stress. An alternate common <i>heat stress</i> definition combines heat load and its consequences: <i>Heat Stress is any combination of work, airflow, humidity, air temperature, thermal radiation, or internal body condition that strains the body as it tries to regulate its temperature. When the strain to regulate body temperature exceeds the body’s capability to adjust, heat stress has become excessive</i> (US Navy definition)
Heat acclimatization:	the terms <i>heat acclimatization</i> and <i>heat acclimation</i> are often used interchangeably; however, acclimatization refers to adaptations that develop as a result of challenges in the natural environment (e.g., physical training in a hot country), and acclimation refers to similar adaptations acquired from experimental exposure to artificial conditions
Climate change mitigation strategies (CCMS):	actions to limit further climate change by reducing the production of greenhouse gases (GHG)
Climate change adaptation strategies (CCAS):	actions to lessen the adverse impacts by preparing for inevitable changes in climate and climate variability

definition of “extreme” heat and that might not be perceived by the overall population and specific at-risk subpopulations as hazardous [16–21]. In a meta-regression analysis using published results from multiple cities around the world, it was estimated that in nearly half of those locations, the risk of all-cause (all-age) mortality increased by one to three percent (1–3 %) per 1 °C increase above the city-specific threshold (i.e., the temperature at which the mortality/morbidity indicator is lowest or the temperature where there is a sharp increase in a nonlinear exposure-response function) with the effect estimate (i.e., slope of linear-response function) varying by different city-specific characteristics and a general trend for the thresholds to be higher in locations closer to the equator [17]. Geographic patterns in effects have been reported in a number of studies, for example, heat-related mortality in the United States tends to be greater in communities in cooler climates than in warmer climates; the smaller effect in warmer areas has been attributed to adaptation through physiological, behavioral, technological means [22–27].

A few studies have evaluated the added heat wave effect above the overall warm season increase in mortality. For example, in a meta-analysis of seven California counties, the July 2006 heat wave was associated with a 9 % (95 % CI: 1.6, 16.3) increase in all-cause daily mortality per 10 °F (5.6 °C approximately) change in apparent temperature or about threefold the effect estimated over the entire warm season (May–September) or July only in 1999–2005 [13]. That magnitude of added heat wave effect is consistent with those observed for some European cities [16]. Over one or multiple warm seasons and over large geographic areas with exposed populations, the increased risks associated with non-extreme temperatures, reflected in increases in numbers of deaths and emergency department visits or hospitalizations, are a major contributor to the cumulative public health and healthcare burden of ambient heat, potentially greater than heat wave periods (which are relatively rare) [16, 17, 28].

Organizations charged with protecting public health during natural disasters are becoming more aware of the potential for health effects (mortality or morbidity) to occur not only during EHE but also at less-than-extreme temperatures common over a warm season. However, most if not all of those organizations continue to use extreme heat alert systems and HRMM risk-reduction strategies that are formulated from an “emergency response” perspective and involve implementation of public health protection protocols that are triggered by forecasted or observed temperatures (or other biometeorological measures) that meet criteria for “extreme” heat conditions. Furthermore, to date, those criteria are always based on exposure-response functions derived from mortality studies, in part because there are overall and for specific locations far fewer studies of ambient heat impacts on morbidity than on mortality. Given that even under current climatologic conditions, ambient heat continues to lead to significant morbidity and mortality, despite the fact that HRI is potentially preventable [3, 9, 29] as is most of the excess HRMM observed in epidemiologic studies makes it clear that improved approaches for prevention of HRMM need to be developed and implemented in the near term. It will be essential to augment the emergency response approach and add a broad suite of strategies that aim to diminish individual and population risk under the full range of ambient heat conditions, not just extremes. To that end, it is necessary to identify the populations, subpopulations, and individuals at elevated risk and to define and understand the independent and joint influence of determinants that contribute to greater (or diminished) *vulnerability* (see Table 6.1 and next section). Furthermore, while epidemiologic observations and research conducted at the population level is critically important and has been invaluable in guiding current strategies for reducing HRMM, the existent burden of HRMM and the amplified challenges to public health posed by climate change and other global changes, such as migration to urban areas or increased prevalence of chronic diseases, that are adversely affecting population health and resilience make it essential that the science upon which risk-reduction strategies are based is broadened. Major advances in our understanding of the pathophysiology of HRI and how it may be related to underlying health status, in particular the role of the immune system (innate and adaptive) and systemic inflammation and oxidative stress [1, 3, 30–32], can provide critical insights to which

individuals and populations are most susceptible to HRMM and can guide identification of efficacious and cost-effective interventions.

This chapter provides a brief synopsis of our current knowledge about the multiple determinants of health and illness that influence the risk of HRMM and that collectively define vulnerability. A conceptual framework to integrate that knowledge is presented, with the intent of providing a tool that can facilitate compilation and translation of the information to interventions and adaptation strategies relevant at the individual level and/or subpopulation and population levels and at one or more geopolitical scales in developing and/or developed nations. The scope of this chapter does not allow a comprehensive exposition of the determinants of risk for all vulnerable populations; however, recent advances in knowledge about thermoregulation and risk factors in older adults and children are briefly discussed. Strategies for HRMM prevention are identified.

Vulnerable Populations: Multiple Determinants of Ambient Heat Health Impacts

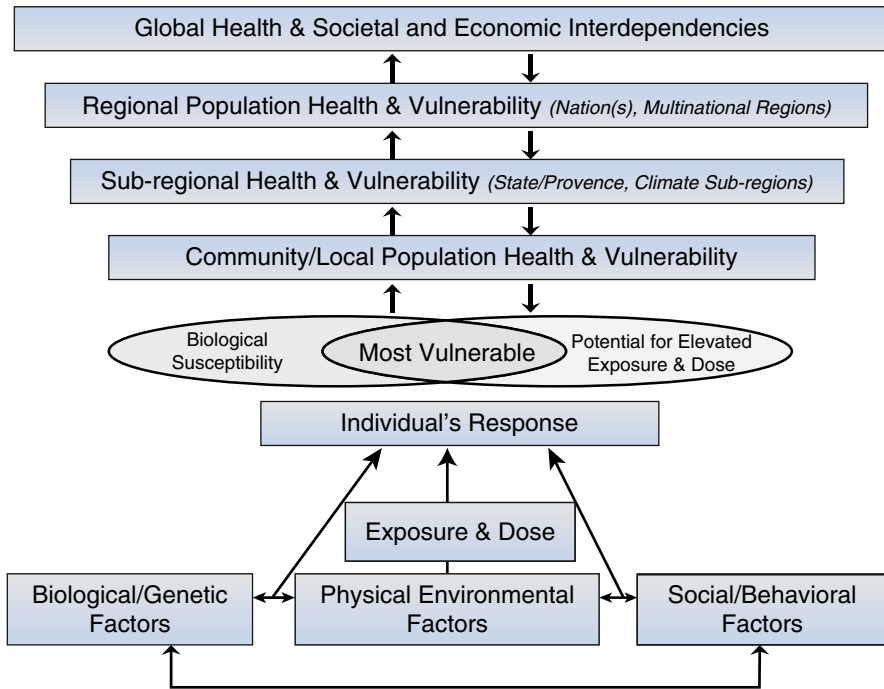
Populations and Subgroups at Elevated Risk: Insights from Epidemiology

Identification of vulnerable populations for the purposes of developing public health approaches to prevention of HRI and HRMM is primarily based on epidemiologic studies that utilize routinely collected administrative data (death certificates, hospital admissions, and emergency department contacts). A number of mortality and morbidity studies (case-control, case cross-over, time-series, and case-series) have evaluated the impacts of ambient heat on specific subpopulations defined by diagnosis group (i.e., to identify cases for specific-cause analyses), age, sex, race/ethnicity, or activity if the data are available (e.g., occupational workers, athletes) and/or evaluated the influence of population-specific or location-specific factors, such as socioeconomic indicators or co-exposure to air pollution either as potential confounders or as effect modifiers. Direct comparison of individual epidemiologic study results is challenging due to differences in study populations, locations, and designs, in particular the use of different temperature indicators and/or different definitions of a heat wave, and whether potential confounding or modifying factors have been considered [18, 33]. Importantly, the commonly used epidemiologic data and study designs preclude detailed examination of individual-level factors, such as obesity or comorbidity and treatment, or location-time-activity patterns that can modify exposures and that may account for the enhanced risk observed at the population level; thus, clear attribution of the elevated risk to just biological susceptibility or another factor is not possible. (For recent reviews of the epidemiologic literature on temperature effects on all-cause or specific causes of mortality, see Hajat and colleagues (2010) [17] or morbidity see Ye and colleagues (2012) [18]. In addition, see Smith and colleagues (2012) for a discussion of heat wave definitions [33]).

Among the different studies, there is heterogeneity in the results for some key factors, i.e., whether there is an effect or association and the direction and magnitude of the association, with some of the differences likely a function of whether the study is examining mortality or morbidity and the specific diagnoses being examined [17, 18, 34]. Age, specifically older adults (usually defined as ≥ 60 years of age) and the very young (infants, children < 5 years of age), is among the strongest and most consistent predictors of elevated risk for HRMM [14, 17, 18, 34]. There are mixed results for sex, with some studies indicating no influence, and others suggesting women or men are at greater risk (often dependent on the health outcome) [17, 18, 34]. Predisposing chronic diseases (e.g., psychiatric illness and neurological disorders, cardiovascular and pulmonary diseases, and diabetes) are also consistently implicated in elevated risk for HRMM [17, 18, 34, 35]. Other factors prognostic of increased risk of HRMM include: being confined to bed, not leaving home daily, and being unable to care for oneself [36]; various general indicators of being socially isolated (e.g., living alone, presence of or frequency of social contacts, or linguistically isolated) [36–40]; and persons who are socioeconomically disadvantaged [36–40]. Interestingly, some studies have indicated the higher risk associated with socioeconomic factors exists for American but not European cities [41], although in France during the 2003 heat wave, for older adults income was associated with greater risk of mortality [42]. Dehydration in general and dehydration associated with medications (neurological and non-neurological) that impair thermoregulation or thirst regulation were also significantly associated with elevated risk of mortality during the 2003 heat wave in France [43]. Factors associated with lower risk include air conditioning (as indicated by air conditioning saturation in a community or evidence of functional/used home air conditioning), visiting cool environments, and increasing social contacts [36, 44].

Environmental Health Multiple-Determinant Model of Vulnerability

Multiple (or Multi-) Determinant Models (MDM) are increasingly being used (qualitatively and quantitatively) to evaluate complex multifactorial chronic disease processes and incorporate consideration of a broad range of risk factors, especially host factors and social determinants of health. This approach is consistent with a paradigm shift by major public health organizations (e.g., WHO, US NIH, and CDC) from a model that just focuses on the determinants of health and disease at the individual level to a holistic model that considers the individual and populations within the context of their physical, societal/cultural, and economic environments across the lifespan [45, 46]. As is the case for complex diseases, complex environmental problems require a holistic approach. Figure 6.1 presents a schematic of the *Environmental Health Multiple-Determinant Model of Vulnerability (MDM_v)*, which is proposed here as a conceptual framework to evaluate the global health impacts of climate change in general, and for the purposes of this chapter ambient



In a lifetime everyone passes through stages of vulnerability.

Fig. 6.1 Schematic of the *Environmental Health Multiple-Determinant Model of Vulnerability*. The premise of the model is that, as for most public health issues, there are disparities in how and the extent to which physical environmental factors (e.g., heat, air pollution, water quality/access) impact different populations and subgroups. Further, the health impacts of environmental factors on populations, begins with impacts on individuals, and in a lifetime everyone passes through stages and degrees of vulnerability, with potential lifetime cumulative influences (positive and negative) affecting risk. Vulnerability is greatest among individuals (or subpopulations) who are most biologically susceptible and who have the largest exposure to one or more environmental hazards (depicted by Venn diagram). Vulnerability for development and severity of heat stress/heat strain and subsequent risk of illness or death (whether considering individual risk or population risk), is a function of complex interrelationships among *biologic factors*, including those that confer innate biologic sensitivity and/or resilience to an environmental insult (e.g., sex, race/ethnicity, oxidative stress, nutritional status, comorbidities and related treatments, and genetics/epigenetics); *physical environment and exposure characteristics* (e.g., physical/chemical nature of the exposure, duration and dose, coincident environmental stressors (such as water and/or food scarcity, air pollution)); and the *social, behavioral, and economic factors* that may influence (or be associated with) both biologic response and exposure (e.g., access to healthcare, social isolation, location-time-activity patterns, disparate neighborhood exposure levels)

heat in particular. Vulnerability factors and their relative importance may differ at the individual and population levels and at different geographical scales or geopolitical domains, and there can be cross-scale interactions among factors. Furthermore, the presence and importance of a given factor or factors can change over time,

Table 6.2 Determinants of heat-related morbidity and mortality

<p>Susceptibility : Biological/Physiological/Clinical Factors: Age (< 5 years, teens, ≥ 60 years) Sex/Gender Race/Ethnicity Genetics/Epigenetics Health Status Dehydration/hypohydration Nutrition Physical Fitness Obesity/Overweight Oxidative Stress & Inflammation Communicable Diseases: Water- & food borne diseases (Diarrheal) Influenza & other acute viral infections Chronic Diseases: CVD, respiratory (asthma, COPD), diabetes, renal insufficiency, immunologic disorders, neurologic disorders, mental illness Medications & Pharmacologic Agents Clinical management of chronic disease Access, adequacy, quality of care</p>	<p>Physical Environmental Factors Temperature, Humidity ↑ Long-term Average Temperature ↑ Freq. Hot Days/Nights ↑ Freq. Heat Waves/Extreme Heat Events (EHE) ↑ Intensity, duration, geographic extent EHE ↓ Freq. Cold Days/Nights <i>Sub-regional/local scale influences on meteorology</i> Topography Coastal (e.g., cloud cover) & sea surface temp. Land surface characteristics (e.g., soil moisture, irrigation, vegetation) Built environment (e.g., impervious surfaces) Coincident Challenges Air Pollution (additive, synergistic): ozone, MVE Water Quantity/Quality ↑ Heavy Rainfall Events (without ↑ in total annual precipitation) ↓ Snowfall & Snow pack ↓ Mountain Glaciers ↑ Drought (Areas, Freq. & Duration) ↓ Soil Moisture (Met. Feedbacks) ↑ Extreme High Sea Level (Storm surges)</p>	<p>Social/Cultural/Behavioral/Economic Factors Demographic Age Gender/Sex Race/Ethnicity Education Economic Built Environment Location-Time-Activity Patterns Building Age, type, condition, heating/ cooling systems (presence/usage) Time-outdoors (work, leisure activities) Community-level factors (e.g., design, assets such as parks) Disparities (and consequences of disparities) in quality of indoor environments: residences, schools, work Social/cultural influences (clothing, climate-influenced behaviors) Lifestyle Factors Physical activity (daily-living activities; exercise (recreational)) Water/diet/nutrition: quality, quantity, subsistence cultures' food sources Psychosocial support Living conditions (e.g., alone & isolated, crowding) Psychosocial stress Community infrastructure</p>
<p>Factors that Modify Exposures Location-Time-Activity Patterns Age-related differences: Children, older adults Time(s) outdoors & indoors Physical or cognitive development or impairment Built Environment – Outdoor & Indoor Factors Impervious surfaces Community Design (Trees/vegetation, land-use) Building Age, type, condition, heating/ cooling systems (presence/usage), indoor air quality & ventilation</p>		

affecting one or more scales differently. Table 6.2 lists observed and putative determinants of vulnerability for HRI and HRMM; selected factors are discussed further above.

Biological Adaptations to Heat Stress and Susceptibility and Pathophysiology of Heat Illness

To facilitate the understanding of the potential source of biological susceptibility, this section provides an overview of the normal physiologic responses involved in maintenance of thermal homeostasis (thermoregulation and acclimatization) and cellular adaptations (thermotolerance), and the pathophysiological consequences when the body’s heat load exceeds its cooling capacity. It is beyond the scope of this chapter to provide detailed information on the prevention, diagnosis, and treatment of HRI (or of other heat-related morbidity) in the general or vulnerable populations; in addition to authoritative medical texts, that information is available from other sources, including for the general population [47], and for older adults [48–50], infants and children [39, 51–53], athletes [39, 54], the occupationally exposed [55–58], persons with alcohol, drug and mental health disorders [59], and those taking medications (neurologic and non-neurological) [43].

Thermoregulation, Acclimatization, and Thermotolerance

Thermoregulation is a collective of mechanisms, behavioral and physiological, by which humans (and other homeotherms) maintain thermal homeostasis, and avoid development of, or minimize the adverse consequences of *heat stress* (see Table 6.1). *Behavioral Thermoregulation* ultimately aims to reduce exposure by modifying the microclimate (e.g., through clothes, buildings (residence and work), air conditioning) and by modifying location-time-activity patterns. The focus here is on *Physiological Thermoregulation*, which involves integrated biological processes that serve to balance the body's heat gain (from internal heat generated via mechanical work (i.e., physical activity) and basal metabolic processes, and/or gained from environmental heat exposure) and heat dissipation to the environment so as to maintain the core body temperature (T_c). The T_c is the operating temperature of vital organs in the head or trunk and must be maintained in a narrow range 35–40 °C (95–104° F) with an usual target temperature of 37 °C (98.6° F) at rest [1, 2, 4]. For healthy subjects at rest there can be between- and within-subject variation of T_c of up to about 1 °C due to a number of factors, e.g., diurnal fluctuations, menstrual cycle phase, acclimatization to heat, exercise-related fitness level, and age-related differences [2, 4, 60]. For most healthy (unclothed) humans at rest, ambient temperatures of 24–29 °C (75.2–84.2° F) are thermonutral, i.e., there is no heat transfer between the body and the environment and basal metabolic processes generate sufficient heat to maintain T_c at the target temperature [2, 4]. The summertime ambient temperature range for thermal comfort (i.e., when an individual expresses satisfaction with their thermal environment) is 23–27 °C (73.4–80.6° F) [61].

Heat balance (i.e., where heat gain equals heat dissipation) requires the continuous transfer of energy, most of which is in the form of heat, across tissues within the body, and between the body and the environment; the transfer of heat follows basic laws of thermodynamics and has been well characterized and quantified in terms of the heat balance equation [4, 61]. A simple form of the equation is shown here:

$$S = M_{(b+w)} \pm K \pm C \pm R - E$$

where S =net heat storage (in tissues); M =Metabolic heat production (basal metabolism (b)+mechanical work (w)); K =Conduction; C =Convection; R =Radiation; and E =Evaporation.

There is continuous heat exchange between the body and the environment that can be described and quantified by the Heat Balance Equation. Storage (S) of heat is a function of metabolic heat (M) produced by basal metabolic processes (b) and heat generated by physical activity (i.e., mechanical work (w) of which only a portion of the energy generated is expended on the work itself), the gain or loss of heat through conduction (K), convection (C), and radiation (R), and heat dissipation through evaporation (E). (For an in-depth discussion of the quantitative aspects of heat balance, see Wenger 2002 [4].) The flow of heat is from warmer to cooler media. Within the body, the tissues store the heat, with tissue average temperatures

and capacity to store and transfer heat varying by tissue type. For example, adipose tissue (i.e., fat) has lower heat capacity [62, 63], and its conductivity is about one-third that of muscle, with the rate of heat flow substantially slower (14 kcal/h for fat and 40 kcal/h for muscle) [4]. Convective heat transfer is involved in the flow of heat via the blood from working muscles to the core and from the core to the surface tissues [2, 4]. Conductive heat transfer occurs between tissues that are in direct contact, with the net heat flow from the core to the surface [2, 4]. Heat exchange between the body and the environment is primarily through radiation, convection, and evaporation (most important for dissipation of heat in warm environments) with all three processes occurring at the skin, but only convection and evaporation occurring in the respiratory tract (i.e., air is usually cooler and dryer than exhaled air) [4]. Notably, for a person at rest, radiation (in the form of infrared rays) is the primary pathway by which the body loses heat to the environment; however, the temperature gradient between the skin and the environment influences whether there is heat loss or gain via radiation. Heat gain from solar radiant energy or from solid objects such as paved surfaces can be a significant contributor to heat stress. Conduction usually plays a negligible role in body-to-environment heat transfer; however, it has an important role in treatment of extreme hyperthermia if the patient is immersed in a cool water bath (or shower) to facilitate rapid cooling (with careful monitoring of patient T_c to prevent overcooling) [64]. Clothing can significantly affect heat gain and heat loss (by impeding evaporation and heat transfer) and can be a major contributor to uncompensable heat stress, for example, in occupational workers wearing heavy impermeable clothing [4, 62].

Within a 1 °C rise in blood temperature, afferent heat receptors in the body core and skin transmit signals to the central nervous system's (CNS) primary thermoregulatory centers in the preoptic and anterior hypothalamus, where thermodetectors sensitive to increases in their own temperature trigger an efferent response. That response includes a suite of physiologic processes that ensure adequate energy and oxygen while increasing flow of the heated blood from the core and working muscles to the surface of the body from where the heat can be dissipated to the environment, primarily by an increase in sweating (rate and the number of eccrine sweat glands activated) [4, 65]. (Temperature receptors in other CNS sites (e.g., medulla) also play a role, and there are thermal receptors outside the CNS, (e.g., in heart, and pulmonary vessels) the role of which is not known [4].) Blood flow to the skin is the result of active sympathetic cutaneous vasodilatation. Increased heart rate, cardiac output, and minute ventilation rate facilitate the shift in blood to the body surface [3, 65]. Efficiency of cooling by evaporation of sweat depends on the air velocity and the water vapor pressure gradient between the skin and the air surrounding the body. The greater the water saturation of air the less cooling can occur. For the thermoregulatory response to be sustained, there must be adequate water intake and electrolyte supplementation to offset the losses [3, 4, 65].

Heat Acclimatization and Thermotolerance

Repeated exposure to either passive-heat or exercise-heat stress with attendant increases in T_c leads to physiological adaptations, referred to as *heat acclimatization* (see Table 6.1) that enhance perception of thermal comfort, increase work/athletic performance, and ultimately mitigate risk of heat-related morbidity [1, 66, 67]. There are various definitions of *Thermotolerance* (aka thermal or heat tolerance) in the literature; however, as defined by Moseley [67] it is “a cellular adaptation caused by a single severe but nonlethal heat exposure that allows the organism to survive a subsequent and otherwise lethal heat stress.” Thermotolerance is associated with the presence (and upregulated gene expression) of families of heat shock proteins (HSP), which protect cells and tissues from initial damage and accelerate repair if damage occurs as a result of heat stress, as well as a variety of other insults [1, 67]. The HSP have different cellular locations and functions that include binding to and processing of denatured proteins, management of protein fragments, maintenance of structural proteins, and chaperone of proteins across cell membranes [1, 67]. Acclimatization and thermotolerance are usually considered separately, however, there is evidence they are related through a shared dependence on the Heat Shock Response [67, 68] or more broadly the Stress Response [67, 69]. In that context, acclimatization can be viewed as a whole organism adaptation, of which thermotolerance—a cellular adaptation—is one part. After exposure to repeated heat-exercise stress, there is a reduction in gastrointestinal barrier permeability (discussed further in section on HRI pathophysiology), and there is an increase in cytoprotective HSP70 along with a decrease in plasma levels of tumor necrosis factor-alpha (TNF- α) and the pro-inflammatory interleukins (IL) IL6 and IL10, leading to lower levels of cellular and systemic markers of heat strain [68]. It should be noted that the complex array of cytokines involved in the systemic inflammatory response syndrome (SIRS) have both a role in promoting and resolving the SIRS [31].

Most of the information on acclimatization in humans is derived from sports physiology or military medicine research on acclimation among young healthy study subjects, usually males, that examined the immediate and/or adaptive physiologic responses from short-term exposures to heat-exercise stress under experimental (i.e., controlled) conditions. Short-term acclimation and acclimatization reflect similar physiologic adaptations that develop (or decay in the absence of heat-stress exposure) over a period of days to weeks [66]. There are very few published studies of long-term acclimatization (or habituation), which occurs over a period of years and reflects both the short-term physiologic adaptations and other usually poorly characterized physiological, behavioral, and technological adaptations by populations and individuals. There is also little published research on acclimation/acclimatization in the general population or vulnerable subgroups, such as the elderly, children, or those with chronic medical conditions.

When acclimatized, an individual's metabolic rate and T_c are lower at rest, accompanied by a lower heart rate, and under conditions of heat stress, there is an

increase in stroke volume and blood/plasma volume, a reduced loss of electrolytes in sweat/urine, and increased thermal tolerance (i.e., cellular stress protein adaptations) [1, 65, 66, 68]. Among the physiologic adjustments that underlie those changes are a lower T_c threshold required for sweating to be initiated and the sweat rate is greater per degree rise in T_c , which enhances evaporative heat loss and the ability to lower skin and core temperatures [4, 66]. Also, skin vasodilatation and core-to-skin heat transfer is initiated at lower T_c and skin blood flow is higher for a given T_c [66]. The physiologic systems involved in acclimatization adapt at different rates, with changes in heart rate and plasma volume occurring first, then the reduction in resting T_c , and finally changes to sweat and sweat rate [66].

The rate of induction of heat acclimatization is exponential with 75 % of the adaptations occurring within about the first 4–6 days of heat-exercise stress exposure and almost complete adaptation present after about 7–10 days [66]. One recommended protocol to achieve acclimatization is a single-daily exposure of about 100 min, with a work rate sufficient to increase T_c to 38.5 °C (101.3° F) [66]. Moseley [67] has noted that passive heat exposure-induced hyperthermia is usually associated with only partial acclimatization. Once heat acclimatized, unless there is repeated heat-exercise or passive heat exposure(s), there is a decay in acclimatization that can occur in as little as a week, with the decline in the different physiologic systems' adaptations occurring in reverse order of induction [66]. Depending on the interval without exposure to heat stress, re-acclimation is more rapid than initial acclimation. There is far less research on the time course of acclimatization decay and re-acclimatization or the determinants of those rates. One rule of thumb has been that for every 2 days without to heat stress exposure, there is 1 day of acclimatization lost; however, more recent research suggests that decay occurs more slowly and that at least for healthy young adults they can safely return to work or athletic competition after as long as a month away from heat stress conditions [66].

Adaptations associated with thermotolerance, i.e., the HSP response, are evident within several hours of heat stress exposure (messenger RNA levels peak within the first hour) and increase for several days [1, 67]; however, the duration of the adaptations is only for 2–7 days (in contrast to acclimatization which is indefinite as long as a person has periodic mild elevations in T_c) [67]. After the initial exposure, HSP synthesis is a function of the intensity, duration, and cumulative effects of subsequent heat-stress exposures [1]. Importantly, although passive heat exposure and physical exercise can independently trigger HSP synthesis, there is a greater HSP response when those two stressors are combined as compared to either one alone [1].

It is important to emphasize that, although there is a paucity of data for the general or vulnerable populations, it is known that the time required to acclimatize or to see significant decay in acclimatization and to re-acclimatization can vary substantially depending on an individual's age, health status (especially by physical fitness, obesity (adiposity), or cardiopulmonary diseases), and the type of exposure (i.e., passive heat or heat-exercise exposure).

Heat Stress-Related Morbidity and Pathophysiology of Severe Heat-Related Illness

Any individual, regardless of age, sex, or health status, can develop heat stress if engaged in intense physical activity and/or exposed to environmental heat (dry or humid), especially if they are not acclimatized. If heat stress exceeds the physiologic capacity to cool and T_c rises, then a range of heat-related symptoms and conditions can develop. The medical conditions that result from heat stress/heat strain and fall within the formal classification of *Heat-Related Illness* (HRI) represent a spectrum that starts with relatively mild and easily treated illness (heat cramps, heat edema, and heat syncope) and progresses in severity to heat exhaustion and then to heat stroke, an extreme medical emergency. While the mild conditions may not be life threatening, to prevent progression to more serious HRI, they should be treated appropriately and taken as warning signs to immediately remove an affected individual from the exposure situation. Table 6.3 provides an overview of the milder forms of HRI; the focus below is on the most severe condition—heat stroke.

Table 6.3 Heat-related illness: heat cramps, heat edema, heat syncope, and heat exhaustion^a

Heat cramps: severe painful cramping of muscles in the legs or abdomen are the hallmarks of heat cramps, which result from electrolyte disturbance, most notably when plasma sodium levels fall significantly below normal. Heat cramps are commonly caused by exertion, with profuse sweating, and often occur during cool down after activity has stopped. Stopping intense activity and consumption of drinks with electrolytes (e.g., some sports drinks) to replenish fluid volume and electrolytes is usually sufficient treatment

Heat edema: swelling in the legs due to accumulation of fluids in the tissues; results from prolonged dilatation of the small arteries in the legs, especially after prolonged standing or sitting still in the heat. Treatment is to increase circulation (venous return) by alternating between elevating the legs and gently moving them

Heat syncope: sudden loss of consciousness (fainting), usually preceded by light-headedness or weakness, can result from orthostatic hypotension related to peripheral blood pooling. Loss of consciousness can be prevented by sitting or lying down at the initial signs of illness (dizziness, weakness)

Heat exhaustion: extreme depletion of blood plasma volume, which may be coincident with low plasma levels of electrolytes, as well as peripheral blood pooling, can lead to heat exhaustion. Core temperature may be in the normal range or slightly elevated but less than 40 °C. Symptoms can include generalized malaise, weakness, nausea, vomiting, headache, tachycardia, and hypotension. Although there can also be mild disorientation, the absence of clear neurologic complications distinguishes heat exhaustion from heat stroke

If heat exhaustion is suspected, the recommended course of action is to immediately move the affected individual to a cool environment and give them fluids supplemented with electrolytes. It may be necessary to actively cool the person by loosening clothing, increasing air flow across the skin, for example with a fan while misting or wiping them down with cool water, or placing ice packs on their extremities. Massage of extremities to mitigate vasoconstriction associated with use of cold water or ice is usually recommended

^aHeat stroke is the most extreme form of HRI and is discussed in main text

Heat Stroke

Heat exhaustion may be the early stage of heat stroke [54], and within a 24-h period if untreated, it can progress to heat stroke; thus, to prevent heat stroke and improve patient outcome, treatment should begin at the first signs of heat exhaustion. Heat exhaustion does not necessarily present with definitive symptoms, therefore it is frequently misdiagnosed, commonly as acute viral infection, leading to delayed treatment. Importantly, acute viral or bacterial infections coincident with heat stress are implicated in increased risk of heat stroke [31], as well as sudden infant death syndrome (SIDS) in infants who were also more heavily wrapped in clothing [70]. Heat stroke is typically divided into two types: “Exertional Heat Stroke” as the name implies involves strenuous physical activity usually under high temperature conditions to which the person was not acclimatized and usually affects healthy older teens and young adults, such as athletes, occupational workers, and soldiers. “Classic heat stroke”, by definition, does not involve exertion and usually affects biologically susceptible individuals, such as infants and young children, the elderly, persons with chronic illness and/or taking medications (prescribed or over-the-counter), as well as persons with alcohol or drug dependencies and with mental illness or neurologic conditions [43, 59]. It is imperative that measures be taken to prevent and/or aggressively treat heat stroke, which, even if treated, can have a crude mortality rate as high as 50 %, and a large proportion of heat stroke survivors suffer permanent neurologic damage [3, 71]. Among 58 survivors of near-fatal classic heat stroke that occurred during the 1995 Chicago heat wave, 33 % had substantial functional impairment at discharge from the hospital and had not improved at 1-year follow-up [71]. The sequelae of heat stroke-related multiorgan system dysfunction/failure (discussed below) can persist months or years after the initial treatment thereby increasing the risk of mortality over the long term [31].

For both types of heat stroke, the clinical definition is when a person’s body core temperature rises above 40 °C (104° F) and there are CNS neurologic complications (e.g., initially headache, dizziness, and weakness followed by hallucinations, combative behavior, coma, and seizures) [3, 31]. The more quickly the patient receives treatment to bring down their T_c to 39 °C (102° F) or below (ideally within 30 min of presentation [71]), and supportive therapies such as replacement of blood volume and electrolytes are administered, the less likely are severe complications and the better the prognosis [3, 31]. Although the clinical criteria and overall treatment of both types of heat stroke are essentially the same, a number of differences in patient characteristics, including signs and symptoms have been noted [65] that reflect the population subgroups commonly affected and that may require medical interventions specific to their unique physiology and medical status. For example, in classic heat stroke sweating is usually absent, respiratory alkalosis is a dominant feature, coagulopathies (i.e., disseminated intravascular coagulation (DIC)) is mild, and if present rhabdomyolysis is rarely severe, whereas in exertional heat stroke sweating is often present, respiratory alkalosis is mild, DIC is marked, and rhabdomyolysis is severe [65].

Heat-Related Illness: Pathophysiology

Over the past 2 decades, research has led to critical insights to the pathophysiology of heat stroke [3, 31, 65]; based on that information, Bouchama and Knochel (2002) proposed that heat stroke be defined as *a form of hyperthermia associated with a systemic inflammatory response leading to a syndrome of multiorgan dysfunction in which encephalopathy predominates* [3]. It has long been known that heat stroke is associated with an overload of the thermoregulatory response, including reduced capacity to increase cardiac output due to water and electrolyte depletion, cardiovascular disease, or medications or alcohol and illicit drugs, that affect cardiovascular, respiratory, or neurologic function [3, 43, 59]. As the T_c rises above 40 °C (104° F), there is tissue injury, with the extent of injury a function of the level and duration of heating [3]; the acute injury triggers the acute phase response (APR). It is now recognized that an upregulated APR and oxidative stress (likely both a precipitant and a downstream consequence of the APR) and possible altered expression of cytoprotective HSP are central to the pathophysiology of heat stroke [1, 3]. The cytotoxic effects of heat and the APR-associated inflammatory and coagulation responses of the affected individual contribute to the multiorgan injury [31]. As noted above, as part of the normal thermoregulatory process in response to hyperthermia, i.e., increased T_c , the circulation of blood is shifted to the skin and working muscles and away from vital organs, including the gastrointestinal tract; this can lead to ischemia of the gut and intestinal hyperpermeability. An emerging body of evidence, primarily from animal models, indicates that endotoxemia resulting from intestinal hyperpermeability and leakage into the circulation may contribute to the progression from heat stress to heat stroke [1, 3, 31, 65].

Within the scope of this chapter, it is not possible to review the literature on this critical line of investigation linking heat stroke and the heat-stress response, oxidative stress and systemic inflammation, and the complex interplay between the innate and adaptive immune systems' responses (see Leon and Helwig for an overview [31]). However, it is important to note that over the past 2 decades a robust body of evidence has linked systemic and/or organ-/tissue-specific inflammation and oxidative stress pathways to: aging [72]; to the pathophysiology underlying a number of chronic diseases and related conditions (e.g., atherosclerosis and cardiovascular disease) [73, 74], chronic respiratory disease (e.g., asthma and chronic obstructive pulmonary disease (COPD)) [73, 75, 76], diabetes and obesity [77, 78]; and as potential mechanisms by which ambient air pollution increases the risk of acute exacerbations of those chronic diseases/conditions and/or contributes to their development and severity [79–84]. Furthermore, oxidative stress may impair the protective heat shock response [30], potentially reducing thermotolerance and increasing risk and severity of heat stroke. The implication of these observations is that individuals with chronic health conditions/diseases who already have high levels of oxidative stress and chronic inflammation are at elevated risk of HRI [31], and that this is an important underlying mechanism that contributes to the excess acute

cardiovascular, respiratory, and diabetes cases associated with ambient heat. This will be an important area of further delineation and research, as it also opens the door to many more clinical and public health intervention options.

Vulnerable Populations: Determinants of Thermoregulatory Capacity

The strongest and most consistent observations in epidemiologic studies have been an elevated risk for HRMM among older adults, children, and people with chronic diseases regardless of age. There are physiologic attributes specific to older adults and children that affect thermoregulation (described below); however, recent literature suggests age per se is not in of itself necessarily the major driver of risk, but rather it is the common (often interrelated) correlates of age specific to these age groups that contribute greater risk. Some of these factors are shared determinants of risk (SDR), i.e., factors that impact these and other population subgroups.

Older Adults

Under resting thermoneutral conditions, older men and women have been reported to have lower T_c than younger adults; however, after accounting for factors such as nutrition, comorbidity, and medication effects, the differences in T_c related to age essentially disappear [49]. The number of sweat glands and sweat gland function, in particular the amount of sweat produced per gland, diminishes with aging [49]. Sweating rate of older adults has been reported to be diminished under passive heat exposure; this appears to be a function of maximal oxygen uptake (VO_{2max} ; a measure of aerobic capacity) rather than chronological age [49]. Chronological age-related reductions in skin blood flow do occur (attributed to reduced superficial microvasculature), accompanied by lower cardiac outputs and less redistribution of blood flow from the splanchnic and renal circulations [48, 49], with some yet to be understood sex differences in the central cardiovascular changes observed under conditions of heat stress [49]. Overall with age there is potential for greater heat gain and a diminished capacity for heat dissipation, especially via evaporation, as a result of the changes to sweating capacity and cardiovascular function noted above and an increase in body mass (and associated increase in adiposity). The greater the body mass, the more heat is generated for a given activity level [48], and the smaller the surface area to body mass ratio so cooling capacity is diminished. In addition to adipose tissue acting as insulation and impeding heat exchange, there are less heat-activated sweat glands found in skin covering adipose tissue [48]. Importantly, with aging peripheral and central thermosensor neurons are less sensitive and respond less effectively to temperature changes, with the result that elderly have a decreased perception of heat along with less effective heat dissipation mechanisms [48], which together has important implications for HRMM risk and prevention.

A number of chronic medical conditions disproportionately affect older adults and predispose them to heat illness. (48) Cardiovascular disease is the most important, with direct effects on thermoregulatory mechanisms and capacity, e.g., heart failure and myocardial infarction affect cardiac output and potentially cutaneous vasodilatation. Atherosclerosis, hypertension, and type II diabetes mellitus reduce vascular compliance and can directly affect thermoregulatory capacity [48]. Chronic respiratory diseases, such as COPD and asthma, can impair thermoregulatory capacity (due to diminished ability to provide sufficient oxygen to support increased energy demands) and contribute to hypoxemia that amplifies tissue damage and the risk and severity of heat stroke. Reduced fluid and electrolyte retention and dehydration are associated with aging-related renal insufficiency and with diabetes (type II diabetes mellitus, diabetes insipidus)-related renal damage and impaired renal function. Hypohydration and dehydration are common among older adults, who in addition to changes in renal function also experience a decreased sense of thirst, or to manage bladder control problems they (or their caregivers) may limit their fluid intake [50]. Obesity and/or lower lean body mass are common among the elderly, and as described above can directly affect thermoregulation and risk of HRI. And as noted above, cardiovascular and respiratory diseases, diabetes, and obesity/overweight are associated with elevated oxidative stress and chronic inflammation, which can contribute to pathophysiology and risk of heat stroke. Hyperthyroidism (via increased metabolic heat production or hyperpyrexia), and extensive skin damage or disease, can also directly affect thermoregulatory mechanisms [48]. Neurologic and psychiatric disorders that disproportionately affect older adults may directly impact CNS thermoregulatory centers and efferent responses and/or contribute to behaviors (e.g., wearing excess clothing or not removing themselves from excessive heat exposure) and social conditions (e.g., being socially isolated) that increase the risk of HRI [48]. A point of concern for the elderly, and an area that has not received much consideration in the context of direct or indirect influence on HRI, is nutritional deficiencies, such as inadequate intake of antioxidant-rich foods. Many of the above conditions occur concurrently, with complex physiologic and clinical interrelationships, including treatment and disease management that further complicates delineating a clear path to HRI risk prevention strategies. For example, recommendations to increase fluid intake to prevent hypohydration/dehydration may be contraindicated for a person with heart failure or with renal failure on hemodialysis. Medications may play a critical role in altering risk for HRI [43, 59]. While the literature focuses on increased risks of HRI and HRMM associated with commonly prescribed or over-the-counter medications, there may also be protective effects afforded by medications, such as anti-inflammatory agents.

Infants and Children

A number of studies point to increased risk of HRMM among children, especially those less than 5 years of age [14, 41, 85] and adolescent athletes [86]. Heat stroke is the third leading cause of death among high school athletes in the United States [86].

Most of the information on heat stress and HRI in children is in the context of exercise and physical activity, which by default focuses on school-aged children (e.g., ≥ 5 years of age). Despite the epidemiological evidence pointing to infants and very young children being at especially high risk, there is a paucity of literature that discusses thermoregulation or risk factors (other than extreme exposures such as being left in a car) for HRI in this age group, especially infants. There is a rich literature on hypothermia in neonates and on SIDS. From birth through age 3 months, an infant's metabolic rate increases, the ratio of body mass to surface area increases, and at 3 months there is a thicker layer of subcutaneous fat which together shifts thermal balance towards heat conservation [87]. Some research on SIDS has pointed to a combination of ambient heat and concurrent viral infection in conjunction with excess covering (e.g., blankets or clothing), especially of the head where 40 % of heat production and 85 % of heat loss occurs in an infant in bed (elevated head/brain temperature could affect thermoregulation and respiratory control); the risk of SIDS was greater in infants older than 2–3 months as compared to those younger [70, 87]. It was suggested that an increase in metabolic rate associated with viral infection in the older infants reflected an acute phase response, which would not be as well developed in some younger infants [70].

There are physiological differences between children and adults, including morphologic, metabolic, cardiovascular, and sweating capacity that traditionally have been viewed as conferring less thermotolerance and greater risk of heat stress and HRI among children [52, 88]. Children (past early infancy) have a higher body surface to mass ratio which can increase heat gain from the environment (when ambient temperature is greater than skin temperature), and depending on the water vapor pressure of the air (or humidity) evaporative cooling by sweating may not be sufficient to compensate for that gain. Younger children are less metabolically efficient when walking or running such that their oxygen consumption and heat production is greater than that of adults engaged in a similar level of activity, thus potentially increasing heat strain. (This is less of a factor for non-weight-bearing exercises such as cycling or rowing [52]). When children are exercising in heat, heat convection to the body surface (and cooling) may be compromised (relative to similar heat loads in an adult) as a result of the combined cardiac output demands of working muscles and of moving blood to their larger body surface area. Under similar conditions of ambient heat children have a higher skin blood flow (and peripheral vasodilatation), which compromises venous return and in turn cardiac output and potentially thermoregulation and/or exercise performance. The greatest difference between children and adults is their sweating rates (absolute, relative to body surface, and per gland), and there are apparent sex differences, with lower sweat rates more pronounced in boys compared to men, than in girls compared to women [52]. Children also take longer to acclimatize than do adults [53].

Based on recent research, it has been suggested that due to compensatory mechanisms children's thermoregulatory capacity may be more similar to adults than traditionally accepted, at least under less extreme environmental conditions [52, 89]. This position has been adopted in the 2011 revised American Academy of Pediatrics Council on Sports Medicine and Fitness and Council on School Health Policy

statement-Climatic Heat Stress and Exercising Children and Adolescents [89]. A number of risk factors for exertional HRI (heat exhaustion and heat stroke) other than age-specific differences in thermoregulation were identified, including: current or recent illnesses that alter hydration status or thermoregulation (e.g., gastrointestinal illness and/or fever); chronic clinical conditions (diabetes insipidus, type II diabetes mellitus, obesity, juvenile hyperthyroidism (Graves disease), and cystic fibrosis); medications (e.g., dopamine-reuptake inhibitor to treat attention deficit/hyperactivity disorder or enhance performance, or diuretics); any other acute or chronic medical condition or an injury that affects water-electrolyte balance, thermoregulation or exercise-heat tolerance; and lastly Sickle cell trait, which can contribute to risk and severity or complications of HRI [89].

Chronic respiratory diseases (allergic airways diseases and asthma), and obesity and associated with it type II diabetes mellitus have reached epidemic proportions among children, especially in developed nations. (In developing nations obesity is also epidemic; however, there are complex interrelationships between malnutrition in children and obesity in adults [90]). The pathways by which these conditions can amplify risk of HRI or HRMM in children are for the most part the same as noted above for the general population and older adults and will not be revisited here. However, in the context of climate change and the projected increases in ground level ozone (a potent oxidant), it is also important to note that children are especially vulnerable for developing chronic respiratory disease. They are biologically more susceptible due to their developing respiratory tracts and immune system, and they have potential for greater exposures and doses of air pollution as their breathing rates relative to body size are greater than adults, and they spend more time outdoors. In a cohort of children in southern California, participation in three or more team sports (an indicator of intense physical activity outdoors) in communities with high ozone was associated with a threefold higher risk of developing new onset asthma, as compared with children playing no sports. No effect of sports was observed in low ozone communities [91]. In another study of children with asthma, anti-inflammatory medication was observed to modify (diminish) the effect of air pollution on asthma symptoms [92]. There is also accumulating evidence that dietary intake of antioxidants (e.g., vitamin C), and specific genetic polymorphisms that are associated with antioxidant capacity, independently and/or jointly can modify the effects of ozone on children's lung function and growth [93, 94].

Determinants of Thermoregulatory Capacity: Additional Population Subgroups

Sex/Gender

Epidemiologic studies have yielded heterogeneous results when sex/gender is considered as a risk factor for HRI or HRMM. Most past research on thermoregulation has been in young healthy men and has not explicitly examined

thermoregulation in women or sex-related differences in men and women. A review by Kaciuba-Uscilko and Grucza [60] concluded that *despite a smaller sweating response to heat load in women than in men, there are no substantial sex differences in the effectiveness of thermoregulation, except those that resulted from differences in body size and composition and physical working capacity*. They noted there were sex-hormone-related fluctuations in body temperature and some thermoregulatory processes during the menstrual cycle and in menopause; however, the mechanisms by which sex hormones affect thermoregulation require further study. To the extent there is differential distribution of predisposing chronic conditions/diseases or that lifestyle factors and location-time-activity patterns differ among men/boys and women/girls, the impacts of ambient heat and risk of HRMM would be expected to differ.

Race/Ethnicity

A review of temperature regulation and ethnicity by Lambert and colleagues (2008) [95] provides insights to variation in physiological traits across human populations that developed over the long term as a function of different climatic conditions. They noted the evidence suggests the differences reflect phenotypic rather than genotypic variation [95]. As in the case of sex-related differences in risk, differential distribution of predisposing chronic conditions/diseases across race/ethnicities also would affect the impacts of ambient heat. Disentangling the complex relations between physiological and morphological characteristics (and potentially the underlying genetics) that affect thermoregulatory capacity in warm/hot climatic conditions, from the social, behavioral, economic, and environmental determinants of health that affect overall health (resiliency) and risk of HRI and/or HRMM poses significant challenges. There are both challenges and research opportunities afforded by the increasing ethnic diversity of many nations resulting from modern migrations facilitated by population mobility.

Genetics/Epigenetics

Research on genetic polymorphisms and epigenetic processes that modulate (increase/diminish) susceptibility to physiological heat stress, oxidative stress, and/or the heat shock response associated with environmental challenges (e.g., heat, air pollution, toxins) or specific diseases/conditions and subsequent risk and severity of heat illness are areas of intense investigation [96, 97]. This research offers future promise of identifying the most at-risk individuals and subpopulations to target interventions for prevention. It may also provide more definitive insights to a biological basis for observed variation in risk of HRMM among different race/ethnic groups or between females and males.

Global Environmental and Societal Challenges Affecting Population Vulnerability

Global warming, in addition to increasing land surface average temperatures and frequency of EHE that are of greater intensity and duration [5], will also lead to other concurrent environmental changes, such as increased occurrence of droughts and extreme precipitation events, to sea level rise and higher storm surges, and to higher levels of air pollution, most notably ozone [6], the independent and joint effects of which will significantly affect the ability of ecosystems and human populations to cope with changes in temperatures. From a global health perspective, the most important coincident challenge will be hydrological system perturbations and downstream consequences on water and food security, and energy production and distribution (e.g., due to infrastructure damage), which have direct and indirect impacts on individuals', populations', and societal adaptive capacity. Of critical importance is that not only will there be coincident challenges to health within a given region, there is mounting scientific evidence that synoptic climatic processes are leading to coupled extreme weather events in distant regions. For example, EHE and extended droughts in Russia have been climatically tied to extreme precipitation events in Pakistan [98]. Among the effects these extreme weather events have locally are impacts on water availability and quality, and on crop production. A related concern is there is high confidence that many semiarid areas (e.g., the Mediterranean Basin, western United States, southern Africa, and northeastern Brazil) will experience decreased water resources [6]; many of these areas are among the most productive agricultural regions globally. Thus, not only is water and food security impacted within each affected region, the overall capacity for the international community to provide aid to any one region is diminished due to multiple regions being affected and potentially needing aid at the same time.

While global warming discussions usually note average *global* increases in temperature (land and ocean), at the local and subregional scales (e.g., subcommunity, community), there exist large variations in land surface temperatures—averages and excursions above averages (variability), and with climate change the degree to which temperature will increase in a given location will also vary and not always predictably. For example, climate models predict that year-round average temperatures throughout California will keep increasing with warming more pronounced in the summer than in the winter season, and depending on the general circulation model (GCM) and greenhouse gas (GHG) emissions scenario, the summer (July–September) increases range from 1.5 to 6 °C (2.7–10.8 °F) [99]. Also predicted is greater warming in inland areas, as compared with coastal locations (within ~50 km of the coast) with the increase as much as 4 °C (7.2 °F) higher in the interior land areas as compared to the coast [99]. As elsewhere, the frequency, intensity, duration, and geographic extent of EHE are predicted to increase in California; a trend already evident in the past decade along with the emergence of EHE characterized by higher humidity and higher minimum (overnight) temperatures [10]. Urbanization/suburbanization accounts for areas with the largest increases; however, there are also

many rural areas that have experienced substantial temperature increases [99, 100]. That noted, the urban heat island effect can contribute to ambient temperatures being more than 10 °C higher than neighboring rural areas. Among the factors that contribute to this phenomenon is greater heat generation from local sources such as vehicles and other machinery; dark surfaces with low albedo (i.e., reflectivity) that absorb and reradiate heat; low vegetation density and commensurate reduction in capacity to cool through evapotranspiration; and layout and design of buildings and other structures (e.g., urban canyons, height) that result in heat retention [101, 102]. Interestingly, independent of climate zone, metropolitan population size or rate of metropolitan population growth, over the last half century the rate of increase in the annual number of EHE was reported to be greater in metropolitan regions characterized by greater urban sprawl compared with more compact metropolitan regions [101]. The primary mechanism attributed to this observation was the rate of deforestation in more sprawling areas and the associated loss of regional vegetative land cover [101].

Human populations are not just facing unprecedented environmental changes but also global societal and demographic shifts. Key among the societal changes is the migration from rural communities to densely populated urban locations where in addition to higher temperatures there are other challenges to health [103]. In developing nations, migrants tend to be poor and frequently end up in “irregular settlements” where there is little or no health protective infrastructure such as sewer systems and reliable potable water sources [29, 104]. In these settlements, as well as many other urban and rural communities in developing nations, water- and food-borne diseases, especially diarrheal diseases among infants and children under 5 years of age, remain a leading cause of illness and premature preventable deaths, despite the eradication and improved management of many communicable diseases that have been achieved globally [105]. Even in developed nations, populations that are economically disadvantaged (and/or medically underserved) or displaced (e.g., due to natural disasters) are also at elevated risk of communicable diseases, as was seen in the aftermath of Hurricane Katrina in the United States [106]. Diarrheal and other communicable diseases, including intercurrent infections, can predispose affected individuals to heat stress and HRMM [3, 71, 89]. Wherever populations reside, work, or recreate, insufficient access to potable water increases the risk of hypohydration and dehydration and in turn to increased risk of heat stress and HRMM in general and HRI in particular.

Strategies to Reduce Vulnerability and Incidence of Heat-Related Morbidity/Mortality

As noted at the beginning of this chapter, the existent and projected large public health and healthcare burden associated with ambient heat requires that the emergency response approach to EHE be augmented with strategies that reduce individual and population risk of HRMM over the full range of ambient heat

conditions. Effective policies and interventions require knowledge, not assumptions about who is at risk, the drivers of that risk, and where and when those determinants of risk are greatest, as well as the efficacy of risk-reduction strategies. Within the framework of an Environmental Health Multiple-Determinant Model of Vulnerability (Fig. 6.1; Table 6.2) that incorporates knowledge from different disciplines, it is possible to identify the factors that independently or jointly confer increased (or diminished) risk of HRMM within the general population and within or across specific subpopulations already identified as *vulnerable*. In addition to developing/implementing evidence-based *Extreme Heat Event and Warm Season Heat Preparedness and Response Action Plans*, two other overarching and interrelated strategies are self-evident: *Promote Good Health & Access to Quality Healthcare (reduces risk and increases resiliency)* and *Reduce/Manage Potential Exposure(s) (individual, community) to Ambient Heat and Other Physical Environmental Stressors*. To be efficacious and resource-efficient, all three strategies require a coordinated “top-down” and “bottom-up” approach involving governments, nongovernmental organizations, communities, and strong partnerships with diverse stakeholders (e.g., public health officials, healthcare and social service providers, educators, athletic coaches, and other private sector participants such as faith-based organizations). The translation of those broad strategies to specific actions is where careful integrative considerations of the multiple determinants of risk becomes most critical, and the implementation is most challenging, especially in light of climate change-related environmental shifts. The discussion below primarily focuses on examples of translation and integration in the context of the two overarching strategies and heat-health action plans.

Promote Good Health and Access to Quality Healthcare

The above overview of normal thermoregulatory processes, pathophysiology of severe HRI (heat stroke), and the characteristics of older adults and children that affect their risk for HRMM highlighted key points of knowledge. Most notably, the recurrent theme for both age groups (with special considerations for infants) and applicable to other age groups is that individuals (females and males) who are more physically fit, have greater percent lean body mass, are adequately hydrated, and are not afflicted with a chronic disease (especially cardiovascular, respiratory, neurological, renal, or diabetes), and do not have an acute intercurrent infection, are less biologically susceptible to HRI and HRMM because they have the physiological reserves to experience moderate-to-extreme heat stress and heat strain and still maintain thermal homeostasis, with less cell and tissue damage, and low risk of acute cardiopulmonary events or other complications of heat strain. In addition, physiological acclimatization can further reduce susceptibility and enhance resilience to heat stress/heat strain. Although far from being fully elucidated in the context of the sequelae from heat stress to heat exhaustion and heat stroke, a biological mechanism that unifies these observations in the healthy heat acclimatized

phenotype is a lower level of oxidative stress and less chronic low-grade inflammation and potentially modulation of the acute phase response and stress response (e.g., downregulation of pro-inflammatory cytokines and upregulation of HSP response) that together confer greater thermotolerance. Beyond thermotolerance there may be important co-benefits of enhancing the HSP response. HSP have the potential to alter obesity-induced insulin resistance (via preventing inflammatory disruption of insulin signaling), and lower HSP expression has been observed in human diabetes patients [78]; thus maintenance of HSP expression may be a pathway by which insulin resistance and diabetes are or could be improved with exercise [78] (and potentially exercise-heat acclimatization protocols).

Thus, the broadest recommendation to diminish HRMM across an entire population over the long term, with near-term benefits, is to invest in and capitalize on public health programs and interventions that aim to improve health and prevent/manage common chronic diseases, especially through improved nutrition and increased physical activity, as well as prevent/manage communicable diseases with specific consideration of the impacts (e.g., via dehydration, fever) on risk of HRMM. Integral to achieving that overall aim is to ensure access to healthcare (especially preventive medicine), and ensure clinicians and other healthcare service providers or points of patient contact (e.g., pharmacists) are informed about the HRMM risk factors relevant to their patients and measures that can be taken to manage that risk. This approach can contribute significantly to reducing the pressures on the public health infrastructure created by the global demographic trend towards older populations, and the global increase in prevalence of chronic diseases and obesity, as well as climate change.

Reduce/Manage Potential Exposures to Ambient Heat and Other Physical Environmental Stressors

Achieving “good health” and reducing HRMM, especially as the climate changes, will require concurrently addressing physical environmental stressors. In addition to advocating for and investment in pollution prevention programs at all geopolitical scales, specific actions need to be developed/implemented to reduce potential exposures (to heat, chemical and/or infectious agents) experienced by populations and individuals at the local scale. For example, when making the recommendation to increase physical activity (e.g., to manage weight), assuming the majority of the population does not have options to exercise in indoor locations (with healthful environmental conditions), there also has to be guidance on minimizing exposure to ambient air pollution, which can vary substantially temporally (e.g., diurnally and seasonally) and spatially at the local scale (e.g., neighborhood-to-neighborhood, proximity to a roadway), as well as provide advice to avoid the hottest time of the day (which usually is also coincident with the highest ozone levels). If the individual has compromised health, even if an apparently relatively benign condition such as

being overweight (but not obese and with no other health problems), or if they are taking medications that predispose them to heat stress/heat strain, they need to be alerted to their potentially heightened susceptibility to heat strain and risk of HRI or HRMM. Warnings to acclimatize before engaging in outdoor physical activities need to be accompanied by specific guidance on how to acclimatize. Such guidance is available for athletes (e.g., see Bergeron [89]); however, few if any of the documents that recommend acclimatization specifically address the issue of co-exposure to air pollution or aeroallergens. Currently there is little or no published quantitative information that specifically outlines or provides the basis for acclimatization protocols (that consider both exercise-heat exposure and passive heat exposure) for the general healthy population or subgroups defined by age and/or specific health conditions. This is an area of investigation that should be a priority.

Access to an air-conditioned cooler environment has consistently been associated with lower risk of HRMM over usual summertime and extreme heat conditions [22, 27, 71, 107, 108]. And during EHE, recommendations to use air conditioning or move to an air-conditioned location, including public access cooling centers, have become a cornerstone of HRMM prevention strategies. There are however a number of potential pitfalls to this strategy. Even in developed nations, the energy generation and distribution infrastructure may not be able to support energy demands during EHE of long duration and large geographic extent, especially if there is increased penetration of AC into homes and businesses. During the 2006 California heat wave that also affected other western states (that can share energy resources with California), there were near failures of the power supply, with some areas experiencing brownouts. If there are coincident extreme weather events such as hurricanes or storm surges, the energy infrastructure, including power plants, is at risk. In consideration of climate change and the need to reduce GHG emissions, unless sufficient (truly non-polluting) “green energy” is available, reliance on air conditioning may be counterproductive for health in the near and longer term. Public gathering places, such as older schools or workplaces, and eldercare residential facilities often do not have air conditioning, even in developed nations. Many populations (e.g., in irregular settlements) or individuals within populations (e.g., urban or rural poor in older residences) do not have nor is it feasible for them to have and/or use an air conditioner. A related concern is that the recommendation to avoid heat exposure by going indoors is not universally protective due to highly variable indoor heat and air quality conditions. Furthermore, by avoiding any heat exposure, the opportunity for acclimatization is diminished.

With respect to recommendations to minimize heat exposure, a critical caution regarding the use of fans is warranted. It is not recommended to use fans to prevent an individual from becoming overheated under certain climatic conditions of high humidity (greater than about 33 % relative humidity) and high temperatures (i.e., temperature is ≥ 32.3 °C (90° F)); when temperatures are above 37.8 °C (100° F), fans may actually contribute to heat stress and subsequent illness (37). However, the use of a fan in conjunction with wetting down the skin of a person showing signs of heat stress or illness can facilitate evaporation and the cooling process.

Clearly, completely abandoning air conditioning as a solution for HRMM prevention is not recommended or feasible. However, more sustainable strategies that focus on reducing heat exposure by modifying the built environment to minimize heat gain (inside buildings and outside) and maximize heat loss and transfer from inhabited areas can reduce the need for air conditioning. Increasingly national and provincial municipal governments are developing/implementing sustainability policies and plans that include improved community design and land-use planning (e.g., increase green space, and rerouting of traffic to decrease vehicle miles traveled), retrofitting existing buildings (e.g., with green roofs, energy efficient windows), and replacing pavement with pervious surfaces. In addition to reducing temperatures (and potentially air pollution exposures), many of these strategies also promote increased physical activity and positively enhance the psychosocial environment and livability of a neighborhood and community and ultimately improve overall health [109].

Extreme Heat Event and Warm Season Heat Preparedness and Response Action Plans

Formal EHE emergency response plans developed and implemented by government organizations at the national, regional, and local levels can significantly reduce HRMM. Comprehensive guidelines and considerations for designing and implementing heat-health action plans focused on emergency response to EHE have been developed by the WHO (Europe) [110]; the guidelines include principles and core elements (summarized in Table 6.4) of a potentially optimum system to prevent EHE-related HRMM that can be adapted to different geopolitical scales and infrastructures. Rather than reiterating recommendations contained in that document, the focus here will be on some of the issues related to enhancing HRMM risk-reduction plans to improve their efficacy during EHE, as well as potentially extending their application to an entire warm season.

The particular issues were identified after the 2006 California heat wave, when the State's Contingency Plan for Excessive Heat Emergencies was reviewed by officials and scientists from public health and emergency response organizations and the US National Oceanic and Atmospheric Administration's National Weather Service (NWS) with the aims to improve heat alert system(s) and emergency preparedness and response, including medical resource planning, and the public health messages and interventions especially those targeted to vulnerable populations. Key gaps in information and limitations in prior studies upon which those systems are based were identified. Among the major issues raised during the evaluation was the need for local scale (i.e., subcommunity such as neighborhood or US census tract) environmental and population data, and two key questions regarding criteria for issuance of heat alerts, including: (1) *Should the definition of a heat wave and heat alert criteria be based on morbidity rather than mortality-response studies as*

Table 6.4 Principles and core elements of heat-health action plans as delineated by the World Health Organization^a

Principles

- Use existing systems and link to general emergency response arrangements
- Adopt a long-term approach
- Be broad (i.e., emergency response requires multiagency and multi-sector participation)
- Communicate effectively
- Ensure that responses to heat waves do not exacerbate the problem of climate change
- Evaluate (a key public health principle—evaluate efficacy of an intervention or strategy)

Core elements for implementation of an heat-health action plan

- Establish agreement on a lead organization
- Accurate and timely alert systems (i.e., heat-health warning systems to trigger weather-related warnings, determine the threshold for action, and communicate risks)
- A heat-related health information plan (what to communicate, to whom, and when)
- A reduction in indoor heat exposure (medium- and short-term strategies)
- Particular care for vulnerable population groups
- Preparedness of the health and social care system
- Long-term urban planning
- Real-time surveillance and evaluation

^aWorld Health Organization: Europe. Heat-health action plans: guidance. 2008. Copenhagen, Denmark. http://www.euro.who.int/_data/assets/pdf_file/0006/95919/E91347.pdf

currently done? and (2) *Should the temperature indicator thresholds be lowered to account for the HRMM that occurs during less than extreme conditions?* Subsequent considerations highlighted issue related to risk communication and engaging the public. A discussion of these issues follows.

Local-scale population and environmental information (in urban, suburban and rural areas) is required to identify high-risk locations and vulnerable populations and individuals, as well as establish mechanisms to contact those individuals in order for local government agencies (and nongovernmental organizations (NGOs)) to target public health and individual clinical or exposure mitigation interventions and allocate resources to *prevent* HRMM. An example of why local-scale information across the urban-to-rural gradient (i.e., not just urban areas) is necessary lies in the fact that while only 6 % of California's population lives in areas designated as rural, the rural populations tend to be older, with about 20 % of Californians ≥ 65 years of age living in a rural area [111, 112]. The older adults residing in rural areas tend to be less healthy, with higher rates of overweight/obesity, physical inactivity and food insecurity, and less access to medical resources, than older adults living in suburban areas; for a number of measures, rural older adults are more similar to their urban counterparts than to those in suburban areas [111]. Prior epidemiologic evidence of spatial heterogeneity in HRMM indicates that exposure-response relations derived from one community may not be applicable in another location [27], which combined with differential distribution of vulnerable populations reinforces the need for location-specific data at the finest spatial resolution possible. Community vulnerability mapping, facilitated by the use of geographic information systems (GIS) and advances in geospatial analysis, including methods of protecting

confidentiality of individuals [28] is an important tool to identify at-risk populations, determinants of risk, and evaluate efficacy of interventions through ongoing surveillance.

The need for local-scale information partly informs the answer to the first question. (*Should the definition of a heat wave and heat alert criteria be based on morbidity rather than mortality-response studies?*) In general, administrative morbidity data (e.g., emergency department contacts, hospitalizations) are less readily available (especially for research) and there can be wide variation in quality and content. However, when they are available, the benefits are that there are many more observations representing a broader cross-section of the population, and heat-related morbidity outcomes occur more frequently than deaths, providing significantly larger sample sizes, which usually provides greater spatial coverage and density at finer spatial resolution (e.g., patient residence Zip Code [postal code]). These attributes facilitate evaluation of HRMM risk and vulnerability factors at a fine spatial scale and the provision of local information. There are also good reasons for reliance on mortality as an endpoint. Vital statistics death data are almost always available and are collected with some degree of consistency, their use generally generates less concern with issues of confidentiality, and there are long records across many years lending them to time-series analyses and application of similar heat-mortality modeling strategies in diverse locations. However, use of mortality data has the implicit assumption that deaths represent the most extreme endpoint of a fixed chain of events, i.e., people are exposed to heat, get sick, and then die, and those deaths can (always) be used as a marker of a relevant population exposure and of a predictable risk. Evidence suggests this is not necessarily the case, as mortality may strike quickly prior to the notice of emergency responders and affects elderly, socially isolated, and nonmobile populations [113, 114]. Thus, to the extent the spatial distribution of vulnerable subgroups more likely to die does not track with subgroups who are more likely to contact an emergency department, mortality-based analyses, and heat alert criteria derived from those analyses from one location would not necessarily provide the best information to reduce risk of morbidity or mortality in another location.

An analysis of hospitalizations and emergency department visits (ED) for all-causes and selected causes during the 2006 California heat wave revealed an intriguing and important observation related to spatial variation in different health outcomes [14]. In that analysis, the State was divided into six geographic regions, based approximately on climate zones, each comprised of multiple counties. Risk ratios (RR) that compared rates during the heat wave and during a referent period (each period = 17 days) in the same summer were computed. Unexpectedly, while the highest risk of HRI ED visits (RR = 23.1, 95 % CI: 15.1, 37.1) occurred in the usually cooler region of central coast counties (including San Francisco), there were too few hospitalizations to calculate a risk estimate (due to small cell sizes and required data suppression) for that region (and two other regions). In contrast, in the Central Valley (a much warmer region), the HRI ED-visit risk was substantially lower, but risk of hospitalization for HRI was very high (RR = 17.1, 95 % CI: 9.8, 36.3). That observation is of particular interest because when the

~140 coroner-reported deaths attributed to hyperthermia (126 of the cases were classic heat stroke) during the heat wave were evaluated the majority occurred in the Central Valley, which is a more rural agricultural region and an area with many socioeconomic-driven health disparities [35]. Taken together, the findings indicate the importance of examining/comparing different measures of health impacts—ED, hospitalizations, and deaths—for which the spatial heterogeneity may reflect a variety of determinants of risk that could influence/inform intervention and adaptation strategies. Thus, when possible, heat alert criteria would ideally reflect the composite information.

With respect to the second question, there are practical reasons for continuing to use extreme temperature thresholds (usually the 95th or 99th percentile of daily maximum temperature or temperature-humidity index) to trigger emergency response protocols and to develop supplemental strategies to diminish the health risks associated with usual warm season elevated temperatures. The primary reason being in many locations lower thresholds would be met repeatedly (if not almost continuously), especially during the hottest months. For example, in a Zip Code-level analysis of emergency department visits in California in the warm seasons (May–September) of 2005–2008, significant increases in patients diagnosed with electrolyte imbalance were observed when deviation of the daily maximum temperature from the Zip Code-specific seasonal mean daily maximum temperature was +6 °C (about the 88th percentile for most locations) [28]. Thus, redefining the threshold criteria for issuance of heat alerts based on this relatively low threshold would not likely be the optimum strategy to reduce public health risk. Not only is it impractical and a resource burden to keep the emergency response and public health infrastructures for EHE risk mitigation in a near-constant state of activation, the communities and populations would likely become desensitized to public health messages about the potential health risks of heat exposure and not take requisite precautions even when a severe EHE is forecast.

There must be a careful balance between informing and overwhelming (and desensitizing) the public with information on risk and prevention of HRMM across the full range of ambient heat exposures. This becomes even more of an issue when trying to share information about joint hazards (e.g., heat and air pollution), while also trying to promote health-protective measures such as exercise. Thus, one of the most critical elements of any heat-health action plan, whether aimed at just EHE or also considering less-than-extreme temperatures, is an evidence-based well-designed communication and education-outreach plan (e.g., the heat-related health information plan suggested by WHO). An essential part of the plan is ensuring the public health messages and recommended actions are correct and that they are effective, and if they are not effective, the reasons and how to remedy the deficits. A prime example of an action that could be effective but is not always is the recommendation, usually targeted to older adults or those with chronic health conditions, to use home air conditioning or go to an air-conditioned location such as a “cooling center.” Experience in California and elsewhere indicates cooling centers are often underutilized, including by older adults, which has led some municipalities to consider not opening centers to save the expense of their operation. Among the

recognized ancillary actions required to increase use of centers (cooling or for other emergencies) is to identify persons needing transportation to the center and then provide that service. In addition, emergency plans must consider care of companion animals as many people will not evacuate if they have to leave their pets behind.

It is well established that public health messaging can be a powerful tool for health promotion and protection, and obtaining such information from multiple sources (top-down (e.g., government issued health warnings) and bottom-up (e.g., healthcare provider)) can enhance the public's awareness and adoption of health-protective measures (to improve overall health or in emergencies). However, the implications of the observations about perception of individual risk among vulnerable populations strongly point to the need for innovative approaches and testing the efficacy of those approaches, as well as additional research. That said, the reasons vulnerable populations may not take health-protective measures (even when they are aware of a heat alert and heard public health warnings), such as using a home air conditioner, are complex and may reflect their knowledge, attitudes, and beliefs about the level of personal risk related to their age or chronic illness [115]. For example, as noted by Richard et al. [115], many older adults do not see themselves as old or at risk, and the individuals who believe limitations in their lives are related to aging are less likely to adopt preventive or adaptive behaviors. Socioeconomic deterrents to air conditioning use may be less of a factor than perception of risk [115]. In addition, the source of information about their vulnerability, including from their physicians, may not influence their perception of risk or adoption of protective measures [115]. Direct one-on-one contact and provision of education and assistance is one solution when individuals cannot due to mental or physical limitations, or who do not of their own accord, take preventive measures.

In general, and to enhance the efficacy of direct contacts, there is an urgent need to engage and educate a wider range of stakeholders, especially social service and healthcare providers, and persons in direct contact with vulnerable populations than are currently knowledgeable and proactive about reducing risk of HRMM among the populations with which they interact. In addition to older adults, the chronically ill and socially isolated, this is especially important for reducing risk of HRMM among infants and children. Children's physical and emotional development and their location-time-activity patterns clearly can contribute to differences in ambient heat exposures, exercise-related heat loads, and ultimately to risk of heat stress and HRI. Infants do not have the motor skills to remove blankets or remove themselves from hot environments [51], young children may continue to play outside even when overheating (past their thermal comfort zone) and often do not know/or sense the need to drink fluids [86], and young athletes may push themselves well past thermal comfort levels that are signaling heat stress and illness onset [86]. It thus becomes imperative that adults (parents and other caregivers, teachers, sports coaches, and observers) be cognizant of the risks and remedies and ensure all precautions and necessary actions be implemented to guarantee the safety of children. Specific guidance for each group needs to be built into the heat plan communication and education element.

A key to reducing HRMM is to have a full heat-health action plan with all the elements outlined (Table 6.4); if the requisite resources (including data on where vulnerable individuals/populations reside and the optimum mode for directly contacting them) are not available at the outset, then the plan should include specific contingencies to fill resource gaps, and timelines and steps to build the infrastructure. Unfortunately, even in developed nations EHE emergency response plans are often not available or of inconsistent quality, as was found to be the case in a survey of selected municipal heat wave response plans from cities in the United States that had a history of or were at risk for heat-related mortality [113]. Adding elements to plans to address HRMM that occur at less-than-extreme temperatures will add a layer of complexity; however, with climate-change-related rising temperatures and increased variability superimposed on the existent risks, this is an essential task. Regardless of the apparent completeness of the plan, once developed it will need to be regularly evaluated for its efficacy and updated to reflect lessons learned.

Conclusions

The rapid convergence of all of the climatologic and anthropologic changes in the present and over the very near term (next 2 or 3 decades) and throughout the twenty-first century exceed the current adaptive capacity of many if not most human social systems around the globe to cope with rising temperatures and increasing frequency and magnitudes of EHE. At all levels—from global to local—there needs to be proactive development of a broad range of strategies to reduce the societal, public health, and healthcare burden of HRMM, especially through primary and secondary prevention of chronic and communicable diseases. This will require an integrated multidisciplinary approach to evaluate and define the problem, including the determinants of individual and population vulnerability for HRMM, and develop the solutions in consideration of those vulnerabilities reflecting both morbidity and mortality. The conceptual framework of the Environmental Health Multiple-Determinant Model of Vulnerability provides a tool that allows quantitative and qualitative consideration of factors that independently or jointly confer increased (or diminished) risk of HRMM and identification of strategies to reduce that risk, including those that might not be evident when the problem is viewed less holistically. Furthermore, it fosters multidimensional thinking when developing/applying solutions, including revealing opportunities to integrate climate change mitigation and adaptation strategies that can realize co-benefits for public health and environmental welfare, and/or identify potential adverse unintended consequences of strategies.

Fortunately, through strategic development and implementation of “top-down” and “bottom-up” HRMM risk mitigation policies and actions that are coordinated with and leverage existing global, regional, national, and local public health and healthcare services programs targeting the root causes of poor health, as well as programs aimed at pollution (including GHG) and exposure prevention, significant

progress can be made towards reducing HRMM efficaciously and cost effectively. The global interconnectedness of economies and of the health and welfare of populations creates an imperative for nations to work together to prevent and/or respond to all of those challenges.

References

1. Sawka MN, et al. Human adaptations to heat and cold stress. In: RTO HFM symposium on "Blowing hot and cold: protecting against climatic extremes". Dresden, Germany: RTO-MP; 2001. p. KN4-1–15.
2. Hanna JM, Brown DE. Human heat tolerance: an anthropological perspective. *Annu Rev Anthropol.* 1983;12:259–84.
3. Bouchama A, Knochel JP. Heat stroke. *N Engl J Med.* 2002;346(25):1978–88.
4. Wenger, C.B. (2002) *Human Adaptation to Hot Environments*. In: Pandolf, K.B. and Burr, RE (Eds) *Medical Aspects of Harsh Environments*. Washington, DC: Office of The Surgeon General Department of the Army, United States of America. p. 51–86.
5. Meehl G, Tebaldi C. More intense, more frequent, and longer lasting heat waves in the 21st century. *Science.* 2004;305:994–7.
6. IPCC. Intergovernmental panel on climate change, climate change 2007: synthesis report. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. 2007. p. 103.
7. Robine JM, et al. Death toll exceeded 70,000 in Europe during the summer of 2003. *C R Biol.* 2008;331(2):171–8.
8. Fouillet A, et al. Excess mortality related to the August 2003 heat wave in France. *Int Arch Occup Environ Health.* 2006;80(1):16–24.
9. Luber G, McGeehin M. Climate change and extreme heat events. *Am J Prev Med.* 2008;35(5):429–35.
10. Gershunov A, Cayan D, Iacobellis S. The great 2006 California heat wave: signal of an increasing trend. *J Climate.* 2009;22:6181–203.
11. Kozlowski DR, Edwards LM. An analysis and summary of the July 2006 record-breaking heat wave across the state of California. NOAA Western Regional Tech Attach, No. 07–05 (Feb 27, 2007). Salt Lake City, UT: NOAA; 2007.
12. Hoshiko S, et al. A simple method for estimating excess mortality due to heat waves, as applied to the 2006 California heat wave. *Int J Public Health.* 2010;55(2):133–7.
13. Ostro BD, et al. Estimating the mortality effect of the July 2006 California heat wave. *Environ Res.* 2009;109(5):614–9.
14. Knowlton K, et al. The 2006 California heat wave: impacts on hospitalizations and emergency department visits. *Environ Health Perspect.* 2009;117(1):61–7.
15. Knowlton K, et al. Six climate change-related events in the United States accounted for about \$14 billion in lost lives and health costs. *Health Aff (Millwood).* 2011;30(11):2167–76.
16. Hajat S, et al. Impact of high temperatures on mortality: is there an added heat wave effect? *Epidemiology.* 2006;17(6):632–8.
17. Hajat S, Kosatky T. Heat-related mortality: a review and exploration of heterogeneity. *J Epidemiol Community Health.* 2010;64(9):753–60.
18. Ye X, et al. Ambient temperature and morbidity: a review of epidemiological evidence. *Environ Health Perspect.* 2012;120(1):19–28.
19. Zanobetti A, et al. Summer temperature variability and long-term survival among elderly people with chronic disease. *Proc Natl Acad Sci USA.* 2012;109(17):6608–13.
20. McMichael AJ, et al. International study of temperature, heat and urban mortality: the 'ISOTHURM' project. *Int J Epidemiol.* 2008;37(5):1121–31.

21. McMichael AJ, Woodruff RE, Hales S. Climate change and human health: present and future risks. *Lancet*. 2006;367(9513):859–69.
22. Chestnut LG, et al. Analysis of differences in hot-weather-related mortality across 44 US metropolitan areas. *Environ Sci Technol*. 1998;1:59–70.
23. Haines A, et al. Climate change and human health: impacts, vulnerability and public health. *Public Health*. 2006;120(7):585–96.
24. Haines A, Patz JA. Health effects of climate change. *JAMA*. 2004;291(1):99–103.
25. Kalkstein LS, Greene JS. An evaluation of climate/mortality relationships in large U.S. cities and the possible impacts of a climate change. *Environ Health Perspect*. 1997;105(1):84–93.
26. Ebi KL, et al. Weather changes associated with hospitalizations for cardiovascular diseases and stroke in California, 1983–1998. *Int J Biometeorol*. 2004;49(1):48–58.
27. Anderson BG, Bell ML. Weather-related mortality: how heat, cold, and heat waves affect mortality in the United States. *Epidemiology*. 2009;20(2):205–13.
28. Roberts EM, et al. Personal communication. 2012.
29. Durand-Lasserre A, Clerc V. Regularization and integration of irregular settlements: lessons from experience (Working Paper No. 6) (Urban Management and Land); 1996.
30. Adachi M, et al. Oxidative stress impairs the heat stress response and delays unfolded protein recovery. *PLoS One*. 2009;4(11):e7719.
31. Leon LR, Helwig BG. Heat stroke: role of the systemic inflammatory response. *J Appl Physiol*. 2010;109(6):1980–8.
32. Horowitz M, Robinson SD. Heat shock proteins and the heat shock response during hyperthermia and its modulation by altered physiological conditions. *Prog Brain Res*. 2007;162:433–46.
33. Smith T, Zaitchik B, Gohlke J. Heat waves in the United States: definitions, patterns and trends. *Clim Change*. 2013;118:811–25.
34. Basu R. High ambient temperature and mortality: a review of epidemiologic studies from 2001 to 2008. *Environ Health*. 2009;8:40.
35. Trent RB, et al. Review of July 2006 heat wave related fatalities in California. Sacramento, CA: California Department of Public Health; 2008. <http://www.cdph.ca.gov/HealthInfo/injviosa/ Documents/HeatPlanAssessment-EPIC.pdf>.
36. Bouchama A, et al. Prognostic factors in heat wave related deaths: a meta-analysis. *Arch Intern Med*. 2007;167(20):2170–6.
37. CDC. Heat-related deaths—Los Angeles County, California, 1999–2000, and United States, 1979–1998. *MMWR Morb Mortal Wkly Rep*. 2001;50(29):623–6.
38. Medina-Ramon M, et al. 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):1331–6. doi:10.1289/ehp.9074 (<http://dx.doi.org/>).
39. Naughton GA, Carlson JS. Reducing the risk of heat-related decrements to physical activity in young people. *J Sci Med Sport*. 2008;11(1):58–65.
40. Naughton MP, et al. Heat-related mortality during a 1999 heat wave in Chicago. *Am J Prev Med*. 2002;22(4):221–7.
41. Kovats RS, Hajat S. Heat stress and public health: a critical review. *Annu Rev Public Health*. 2008;29:41–55.
42. Belmin J, et al. Level of dependency: a simple marker associated with mortality during the 2003 heatwave among French dependent elderly people living in the community or in institutions. *Age Ageing*. 2007;36(3):298–303.
43. Stollberger C, Lutz W, Finsterer J. Heat-related side-effects of neurological and non-neurological medication may increase heatwave fatalities. *Eur J Neurol*. 2009;16(7):879–82.
44. Fouillet A, et al. Has the impact of heat waves on mortality changed in France since the European heat wave of summer 2003? A study of the 2006 heat wave. *Int J Epidemiol*. 2008;37(2):309–17.
45. Gehlert S, et al. Targeting health disparities: a model linking upstream determinants to downstream interventions: knowing about the interactions of societal factors and disease

- can enable targeted interventions to reduce health disparities. *Health Aff (Millwood)*. 2008;27(2):339–49.
46. Liburd LC, Sniezek JE. Changing times: new possibilities for community health and well-being. *Prev Chronic Dis*. 2007. http://www.cdc.gov/pcd/issues/2007/jul/07_0048.htm. Accessed July 2007.
 47. Blum LN, Bresolin LB, Williams MA. From the AMA Council on Scientific Affairs Heat-related illness during extreme weather emergencies. *JAMA*. 1998;279(19):1514.
 48. Allen AJ, Segal-Gidan F. Heat-related illness in the elderly. *Clin Geriatr*. 2007;15(7):37–45.
 49. Kenney WL, Munce TA. Invited review: aging and human temperature regulation. *J Appl Physiol*. 2003;95(6):2598–603.
 50. Worfolk JB. Heat waves: their impact on the health of elders. *Geriatr Nurs*. 2000; 21(2):70–7.
 51. Jardine DS. Heat illness and heat stroke. *Pediatr Rev*. 2007;28(7):249–58.
 52. Falk B, Dotan R. Children's thermoregulation during exercise in the heat: a revisit. *Appl Physiol Nutr Metab*. 2008;33(2):420–7.
 53. Bytomski JR, Squire DL. Heat illness in children. *Curr Sports Med Rep*. 2003;2(6):320–4.
 54. Howe AS, Boden BP. Heat-related illness in athletes. *Am J Sports Med*. 2007;35(8):1384–95.
 55. Bates G, Gazey C, Cena K. Factors affecting heat illness when working in conditions of thermal stress. *J Hum Ergol (Tokyo)*. 1996;25(1):13–20.
 56. Jay O, Kenny GP. Heat exposure in the Canadian workplace. *Am J Ind Med*. 2010; 53(8):842–53.
 57. Rodahl K. Occupational health conditions in extreme environments. *Ann Occup Hyg*. 2003;47(3):241–52.
 58. Parsons KC. International standards for the assessment of the risk of thermal strain on clothed workers in hot environments. *Ann Occup Hyg*. 1999;43(5):297–308.
 59. Cusack L, de Crespigny C, Athanasos P. Heatwaves and their impact on people with alcohol, drug and mental health conditions: a discussion paper on clinical practice considerations. *J Adv Nurs*. 2011;67(4):915–22.
 60. Kaciuba-Uscilko H, Gruzca R. Gender differences in thermoregulation. *Curr Opin Clin Nutr Metab Care*. 2001;4(6):533–6.
 61. Epstein Y, Moran DS. Thermal comfort and the heat stress indices. *Ind Health*. 2006;44(3): 388–98.
 62. McLellan TM. The importance of aerobic fitness in determining tolerance to uncompensable heat stress. *Comp Biochem Physiol A Mol Integr Physiol*. 2001;128(4):691–700.
 63. Selkirk GA, McLellan TM. Influence of aerobic fitness and body fatness on tolerance to uncompensable heat stress. *J Appl Physiol*. 2001;91(5):2055–63.
 64. Gagnon D, et al. Cold-water immersion and the treatment of hyperthermia: using 38.6 degrees C as a safe rectal temperature cooling limit. *J Athl Train*. 2010;45(5):439–44.
 65. Hales JRS, Hubbard RW, Gaffin SL. Limitation of heat tolerance, In: Fregly MJ, Blatteis CM, editors. *Handbook of physiology, environmental physiology*, Suppl 14. New York: Wiley; 1996.
 66. Weller AS, et al. Quantification of the decay and re-induction of heat acclimation in dry-heat following 12 and 26 days without exposure to heat stress. *Eur J Appl Physiol*. 2007; 102(1):57–66.
 67. Moseley PL. Heat shock proteins and heat adaptation of the whole organism. *J Appl Physiol*. 1997;83(5):1413–7.
 68. Kuennen M, et al. Thermotolerance and heat acclimation may share a common mechanism in humans. *Am J Physiol Regul Integr Comp Physiol*. 2011;301(2):R524–33.
 69. Moseley P. Stress proteins and the immune response. *Immunopharmacology*. 2000; 48(3):299–302.
 70. Fleming PJ, et al. Thermal balance and metabolic rate during upper respiratory tract infection in infants. *Arch Dis Child*. 1994;70(3):187–91.
 71. Dematte JE, et al. Near-fatal heat stroke during the 1995 heat wave in Chicago. *Ann Intern Med*. 1998;129(3):173–81.

72. Finkel T, Holbrook NJ. Oxidants, oxidative stress and the biology of ageing. *Nature*. 2000;408(6809):239–47.
73. Lee IT, Yang CM. Role of NADPH oxidase/ROS in pro-inflammatory mediators-induced airway and pulmonary diseases. *Biochem Pharmacol*. 2012;84(5):581–90.
74. Libby P. Inflammation in atherosclerosis. *Nature*. 2002;420(6917):868–74.
75. Agarwal SK, et al. Airflow obstruction, lung function, and risk of incident heart failure: the Atherosclerosis Risk in Communities (ARIC) study. *Eur J Heart Fail*. 2012;14(4):414–22.
76. Park HS, Kim SR, Lee YC. Impact of oxidative stress on lung diseases. *Respirology*. 2009;14(1):27–38.
77. Emanuela F, et al. Inflammation as a link between obesity and metabolic syndrome. *J Nutr Metab*. 2012;2012:476380.
78. Whitham M, Febbraio MA. HSP and diabetes. In: Asea AAA, Pederson BK, editors. *Heat shock proteins and whole body physiology*. New York: Springer; 2010. p. 3–18.
79. Plummer LE, Smiley-Jewell S, Pinkerton KE. Impact of air pollution on lung inflammation and the roll of toll-like receptors. *Int J Inference Cytokine Mediator Res*. 2012;4:43–57.
80. Breton CV, et al. Genetic variation in the glutathione synthesis pathway, air pollution, and children's lung function growth. *Am J Respir Crit Care Med*. 2011;183(2):243–8.
81. Islam T, et al. Glutathione-S-transferase (GST) P1, GSTM1, exercise, ozone and asthma incidence in school children. *Thorax*. 2009;64(3):197–202.
82. Gilliland FD, et al. A theoretical basis for investigating ambient air pollution and children's respiratory health. *Environ Health Perspect*. 1999;107 Suppl 3:403–7.
83. Franchini M, et al. Air pollution, vascular disease and thrombosis: linking clinical data and pathogenic mechanisms. *J Thromb Haemost*. 2012;10(12):2438–51.
84. Miller MR, Shaw CA, Langrish JP. From particles to patients: oxidative stress and the cardiovascular effects of air pollution. *Future Cardiol*. 2012;8(4):577–602.
85. Basu R, Ostro BD. A multicounty analysis identifying the populations vulnerable to mortality associated with high ambient temperature in California. *Am J Epidemiol*. 2008;168(6):632–7.
86. Coris EE, Ramirez AM, Van Durme DJ. Heat illness in athletes: the dangerous combination of heat, humidity and exercise. *Sports Med*. 2004;34(1):9–16.
87. Fleming PJ, Azaz Y, Wigfield R. Development of thermoregulation in infancy: possible implications for SIDS. *J Clin Pathol*. 1992;45(11 Suppl):17–9.
88. Falk B. Effects of thermal stress during rest and exercise in the paediatric population. *Sports Med*. 1998;25(4):221–40.
89. Bergeron MF, Devore C, Rice SG. Policy statement—climatic heat stress and exercising children and adolescents. *Pediatrics*. 2011;128(3):e741–7.
90. Prentice AM. The emerging epidemic of obesity in developing countries. *Int J Epidemiol*. 2006;35(1):93–9.
91. McConnell R, et al. Asthma in exercising children exposed to ozone: a cohort study. *Lancet*. 2002;359(9304):386–91.
92. Delfino RJ, et al. Association of asthma symptoms with peak particulate air pollution and effect modification by anti-inflammatory medication use. *Environ Health Perspect*. 2002;110(10):A607–17.
93. Moreno-Macias H, et al. Ozone exposure, vitamin C intake, and genetic susceptibility of asthmatic children in Mexico City: a cohort study. *Respir Res*. 2013;14(1):14.
94. Romieu I, et al. Genetic polymorphism of GSTM1 and antioxidant supplementation influence lung function in relation to ozone exposure in asthmatic children in Mexico City. *Thorax*. 2004;59(1):8–10.
95. Lambert MI, Mann T, Dugas JP. Ethnicity and temperature regulation. *Med Sport Sci*. 2008;53:104–20.
96. Horowitz M. Heat acclimation and cross-tolerance against novel stressors: genomic-physiological linkage. *Prog Brain Res*. 2007;162:373–92.
97. Horowitz M, Kodesh E. Molecular signals that shape the integrative responses of the heat-acclimated phenotype. *Med Sci Sports Exerc*. 2010;42(12):2164–72.

98. Lau WKM, Kim K-M. The 2010 Pakistan flood and Russian heat wave: teleconnections of hydrometeorological extremes. *J Hydrometeorol.* 2012;13:392–403.
99. Cayan D, et al. *Climate scenarios for California*. Sacramento, CA: California Energy Commission, California Climate Change Center; 2006. p. 52.
100. CDPH. Heat-related illness and mortality: information for the public health network in California. In: *Public health impacts of climate change in California: community vulnerability assessments and adaptation strategies*. Sacramento, CA: California Department of Public Health, Public Health Institute; 2007. http://www.ehib.org/papers/Heat_Vulnerability_2007.pdf.
101. Stone B, Hess JJ, Frumkin H. Urban form and extreme heat events: are sprawling cities more vulnerable to climate change than compact cities? *Environ Health Perspect.* 2010; 118(10):1425–8.
102. Oke T. The energetic basis of the urban heat island. *Q J Roy Meteorol Soc.* 1982; 108(455):1–24.
103. United Nations. *World urbanization prospects: the 2007 revision*. New York: United Nations; 2008.
104. UNHabitat. (2011). *Cities and climate change global report on human settlements 2011*. United Nations Human Settlements Programme. London, UK: Earthscan Ltd; Washington, DC: Earthscan LLC.
105. WHO. *World health statistics 2012*. Geneva: World Health Organization; 2012.
106. CDC. Infectious disease and dermatologic conditions in evacuees and rescue workers after hurricane Katrina—multiple States, August–September, 2005. *MMWR Morb Mortal Wkly Rep.* 2005;54(38):961–4.
107. Keatinge WR. Death in heat waves. *BMJ.* 2003;327(7414):512–3.
108. Semenza JC, et al. Excess hospital admissions during the July 1995 heat wave in Chicago. *Am J Prev Med.* 1999;16(4):269–77.
109. Dannenberg AL, Frumkin H, Jackson R, editors. *Making healthy places: designing and building for health, well-being, and sustainability*. Washington, DC: Island Press; 2011.
110. WHO. *Heat-health action plans: guidance*. Europe, Copenhagen, Denmark: World Health Organization; 2008.
111. Durazo EM, et al. The health status and unique health challenges of rural older adults in California. Los Angeles, CA: University of California, Los Angeles (UCLA) Center for Health Policy research; 2011.
112. CHHSA. *Strategic plan for an aging California population: Getting California ready for the “Baby Boomers”*. Sacramento, CA: California Health and Human Services Agency, Editor; 2003. p. 258.
113. Bernard SM, McGeehin MA. Municipal heat wave response plans. *Am J Public Health.* 2004;94(9):1520–2.
114. Diaz J, Linares C, Tobias A. A critical comment on heat wave response plans. *Eur J Public Health.* 2006;16(6):600.
115. Richard L, Kosatsky T, Renouf A. Correlates of hot day air-conditioning use among middle-aged and older adults with chronic heart and lung diseases: the role of health beliefs and cues to action. *Health Educ Res.* 2011;26(1):77–88.

Chapter 7

Climate, Air Quality, and Allergy: Emerging Methods for Detecting Linkages

Patrick L. Kinney, Perry E. Sheffield, and Kate R. Weinberger

Abstract Both anthropogenic and naturally occurring air contaminants can be influenced by climate variability and change and in turn may have important implications for human health. Anthropogenic ozone (O₃) is a pollutant that poses serious health concerns and whose formation in the lower atmosphere depends on temperature and sunlight as well as other meteorologic parameters. Airborne pollens released by trees, grasses, and weeds are responsible for considerable respiratory morbidity and are also influenced by climate factors, as well as by changing carbon dioxide concentrations. Here we report recent findings from a research team in New York City (NYC) that has been investigating interactions between climate, air quality, and human health. The first case study we cover made projections of future O₃ and temperature levels at the county level in the NYC metro area under alternative climate change scenarios, and then translation of these changes into mortality impacts using exposure–response equations derived from historical data in NYC. Findings suggested that heat-related mortality could grow in importance over future

P.L. Kinney, Sc.D., M.S. (✉)

Department of Environmental Health Sciences, Mailman School of Public Health, Columbia University, 722 West 168th Street, New York, NY 10032, USA

Columbia Climate and Health Program, Mailman School of Public Health, Columbia University, 722 West 168th Street, New York, NY 10032, USA
e-mail: plk3@columbia.edu

P.E. Sheffield, M.D., M.P.H.

Department of Preventive Medicine and Pediatrics, Mount Sinai School of Medicine, 1 Gustave Levy Place, Box 1057, New York, NY 10029, USA
e-mail: perry.sheffield@mssm.edu

K.R. Weinberger, M.A.

Department of Environmental Health Sciences, Mailman School of Public Health, Columbia University, 722 West 168th Street, New York, NY 10032, USA
e-mail: krw2114@columbia.edu

decades as compared with O₃-related mortality, and that health effects could be reduced by policies that limit greenhouse gas emissions. In the second case study, we analyzed the effects of spring tree pollen peaks on sales of over-the-counter allergy medications in NYC over a 6-year period. We found a significant effect which had a maximum at lag 2, indicating that tree pollen peaks precede spikes in medication sales by approximately 2 days. Both sets of findings highlight the value of climate and health research as a tool for policy makers concerned with anticipating and preventing adverse health impacts related to climate change.

Keywords Aeroallergens • Allergic rhinitis • Syndromic surveillance • Pollen forecasting • Allergy medication • Ozone • Temperature • Mortality • Climate change • Projection

Climate factors like temperature, wind, and precipitation play important roles in determining patterns and concentrations of air pollution over multiple scales in time and space [1, 2]. These may operate through changes in air pollution emissions, transport, dilution, chemical transformation, and eventual deposition of air pollutants, especially for secondary pollutants like ozone (O₃). Naturally occurring air contaminants of relevance to human health, including airborne pollens, also may be influenced by climate. Thus, there are a range of air contaminants, both anthropogenic and natural, for which climate change impacts are of potential importance.

O₃ is a serious health concern and is formed in the lower atmosphere by reactions involving precursor air pollutants in the presence of sunlight. The key precursor pollutants for O₃ formation are nitrogen oxides (emitted mainly by burning of fuels) and volatile organic compounds (VOCs) (emitted both by burning of fuels and evaporation from vegetation and stored fuels). Because O₃ formation increases with greater sunlight and higher temperatures, it reaches unhealthy levels primarily during the warm half of the year. It has been firmly established that breathing O₃ can cause inflammation in the deep lung as well as short-term, reversible decreases in lung function. In addition, epidemiology studies have demonstrated that O₃ can increase the risk of asthma-related hospital visits and premature mortality [3–6].

Airborne allergens are substances present in the air that stimulate an allergic response in sensitized individuals upon inhalation. Outdoor pollens are one important class of airborne allergens. Pollens are released by plants at specific times of the year that depend to varying degrees on temperature, sunlight, and moisture. Thus, airborne pollen concentrations are sensitive to climate variability and change.

The influence of climate on air quality is substantial and well established [1, 2], giving rise to the expectation that changes in climate are likely to alter patterns of air pollution concentrations. Higher temperatures hasten the chemical reactions that lead to O₃ formation. Higher temperatures, and perhaps elevated carbon dioxide (CO₂) concentrations, also lead to increased emissions of O₃-relevant VOC precursors by vegetation [7]. Weather patterns influence the movement and dispersion of all pollutants in the atmosphere through the action of winds, vertical mixing, and rainfall. Air pollution episodes can occur with atmospheric conditions that limit

both vertical and horizontal dispersion. Emissions from power plants increase substantially during heat waves when air-conditioning use peaks. Finally, the production and distribution of airborne allergens such as pollens and molds are highly influenced by weather phenomena and also have been shown to be sensitive to atmospheric CO₂ levels [8]. For example, the timing of phenologic events such as flowering and pollen release is closely linked with temperature.

Human-induced climate change is likely to alter the distributions over both time and space of all of the meteorologic factors discussed above, which could in turn lead to changes in air contaminants. One concern is that multiple interacting exposures could be affected simultaneously by climate change, leading to enhanced adverse health impacts. For example, the severe heat wave in 2003 in France was associated with elevated levels of ozone [9].

Research into the potential effects of climate change on air quality and human health is challenging, due in part to the highly interdisciplinary nature of the underlying science. Expertise is needed across a range of disciplines that have not often been linked in the past, including climate data acquisition and processing, climate modeling, air quality modeling, exposure assessment, epidemiology, and clinical science. After teams are formed, they need to learn to communicate effectively so that the research can proceed productively, which can take considerable time and effort. An important technical challenge is the need to take the broad scale predictions generated by global climate models and make them relevant and meaningful for impact assessments at fine geographic scales. Outputs from global models typically are resolved at a scale of hundreds of kilometers. Development and integration of research on the human dimensions of global environmental change require down-scaling these projections to the regional metropolitan scale (10 miles/kilometers or finer). Data at these finer scales facilitate planning for mitigation and adaptation strategies.

In the remainder of this chapter, we present two case studies investigating health impacts of climate change, one involving mortality effects of heat and air quality and the other examining the influence of pollen on allergic responses, to illustrate and illuminate the challenges and potentials for climate, air quality, and health research.

Case Study 1: Climate, O₃, and Heat in the NYC Metropolitan Region

Episodes of heat and/or O₃ are current risk factors for adverse health effects in many urban areas around the world, and New York City (NYC) is no exception. Much of the NYC metropolitan region remains out of compliance with the O₃ air quality standard, and heat waves are on the increase, a trend that is likely to continue for several decades. Future O₃ concentrations and resulting health effects will depend both on precursor emissions and on climate conditions. Here, we focus on the climate effect, holding O₃ precursor emissions constant.

The New York Climate and Health Project (or NYCHP) was designed to project future health impacts of climate-related changes in temperatures and ground-level O₃ concentrations [10]. We compared acute summertime heat- and O₃-related mortality from the past (using data from the 1990s) to several future decades (modeled for the 2020s, 2050s, and 2080s). We used a three-part methodology to assess these health impacts. First, we estimated coefficients describing mortality effects of temperature and O₃ using historical (1990–1999) death, weather, and air quality data for the study area. Next, we developed an integrated modeling system to project future environmental conditions under two scenarios of climate change, including modules for global climate, regional climate, and regional air quality. Third, the exposure–response coefficients were combined with the projections of future temperature and O₃ to estimate mortality in future decades under a changing climate. This is a good example of a project requiring multidisciplinary expertise, including climate and air quality modelers, public health scientists, and others.

Epidemiologic Analysis of Historical Data

Mortality data were obtained from the US National Center for Health Statistics (NCHS) for 1990–1999. Daily death counts were computed for each of 31 counties comprising the NYC metropolitan area for all internal causes (ICD-9 codes 0–799.9 for 1990–1998 and ICD-10 codes A00-R99 for 1999), excluding accidental causes and those among nonresidents. Air quality data were obtained for all O₃ monitoring stations within the study area. Of 39 stations that reported summer season data, those with fewer than 80 % non-missing days were removed from further analyses, leaving 16 stations. Daily mean temperature (T_{ave}) (°F) data were obtained from the National Oceanic and Atmospheric Administration (NOAA) National Climatic Data Center (NCDC) data inventory. Stations within the study area with at least 80 % non-missing T_{ave} data included 16 meteorological stations (not the same as those where O₃ monitoring took place).

Coefficients representing the effects of O₃ and temperature on daily mortality were estimated using a Poisson generalized additive regression model with log daily death counts as the outcome variable. Analysis was restricted to the period between June 1 and August 30 for the years 1990–1999, to be consistent with the future projections (see below). Based on prior studies, we used T_{ave} at lag 0 and the 2-day average of the 1-h daily maximum O₃ from lags 0 and 1. O₃ was treated as a linear term in the model, whereas temperature was modeled as a 3rd polynomial in order to capture nonlinear effects at high temperatures. We examined possible confounding effects of particulate matter with aerodynamic diameter less than 10 μm (PM₁₀) on the relationship between O₃ and mortality and found no such evidence in our dataset, consistent with previous work [6, 11, 12].

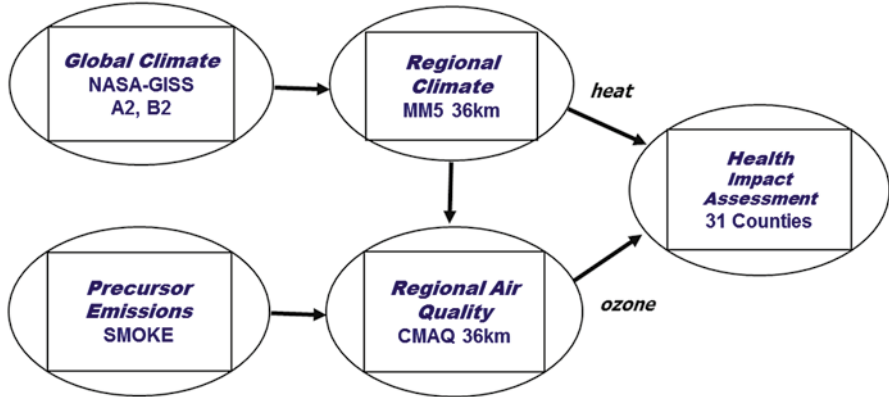


Fig. 7.1 Goddard Institute for Space Studies (GISS) coupled global ocean/atmosphere model

Future Projections of Temperature and O₃

As described previously [13, 14], we use the Goddard Institute for Space Studies (GISS) coupled global ocean/atmosphere model (driven by two different greenhouse gas (GHG) scenarios of the Intergovernmental Panel on Climate Change (IPCC)). The A2 scenario assumes relatively high and the B2 relatively low emissions of GHGs over the century. The GISS global model was linked via initial and boundary conditions to the Penn State-NCAR Mesoscale Model 5 (MM5) regional climate model. MM5 was run on two nested domains of 108 and 36 km over the USA. To simulate O₃ concentrations, the community mesoscale air quality (CMAQ) model was run at 36 km and took its initial conditions from the GISS–MM5 simulations [14]. For CMAQ the 1996 US Environmental Protection Agency (EPA) National Emission Trends (NET) database was processed by the Sparse Matrix Operator Kernel Emissions Modeling System (SMOKE). The simulation periods were June 1993–August 1997, June 2023–August 2027, June 2053–August 2057, and June 2083–August 2087. In the work presented here, O₃ precursor emissions were held constant in the baseline and future simulations, in order to isolate the climate effect. The MM5 model simulated T_{ave} and the CMAQ model simulated 1-h daily maximum O₃ concentrations across the model domain in summers for these four future decades. Gridded temperatures and O₃ concentrations were interpolated to county geographic centroids using inverse distance weighting (IDW). Because of model biases for temperature, the modeled temperatures were converted to anomalies (i.e., monthly difference between future decadal estimate and baseline estimate), and these were used to adjust observed temperatures from the baseline period (1990s) to future decades. Further details are given in Knowlton et al. [15]. The modeling system is shown schematically in Fig. 7.1.

Health Impact Assessment

The daily model simulations of temperature and O₃ at 36 km were combined with the exposure–response functions developed above to compute mortality risks in the baseline and future time periods. In order to isolate the impacts of climate changes on future regional mortality, we held population constant at the Census 2000 county totals. We also held mortality rates constant at county-specific mean 1990s reference rates for the same reason. Preliminary analysis of the mortality and temperature data suggested that days with mean temperatures below 63.6 °F were not associated with excess mortality; thus, we only estimated mortality above this threshold temperature.

Results

Statistically significant coefficients of both temperature and O₃ on mortality were observed in the epidemiologic analysis of data from the 1990s, with results as follows:

$$\begin{aligned} \text{Heat-related mortality} = & (\text{Population} / 100,000) \times (\text{County daily mortality rate}) \\ & \times [\exp((\text{Temp} \times 0.29193) + (\text{Temp}^2 \times -.00434) \\ & + (\text{Temp}^3 \times .00002152)) - 1] \end{aligned}$$

$$\begin{aligned} \text{O}_3\text{-related mortality} = & (\text{Population} / 100,000) \times (\text{Daily mortality rate}) \\ & \times [\exp(\text{max O}_3 \times 0.00045738) - 1] \end{aligned}$$

Total temperature- or O₃-related deaths in the June–August period, averaged over each decade, were computed and compared to that in the 1990s. Figure 7.2 shows the regional distribution of percentage changes in heat-related mortality by the 2050s under the A2 GHG emissions scenario, and Fig. 7.3 shows the O₃ effects for the same conditions. While highly populated counties showed greater absolute numbers of heat-related deaths, higher percentage increases occurred in nonurban counties on the perimeter of the study area. For O₃, higher concentrations by the 2050s spread beyond the urban core into nonurban counties along the SW–NE prevailing wind directional axis.

Table 7.1 and Fig. 7.4 show the projected evolution over time of heat vs. O₃ mortality impacts under the A2 scenario. In the 1990s, summer O₃-related mortality was on par with heat-related mortality in a typical summer, but by mid-century this could change with heat-related mortality approximately doubling as compared to the 1990s, while O₃ mortality increased by just 5 %; and by the 2080s, heat-related mortality could be over four times that from O₃.

Sensitivity analyses compared the 2050s B2 scenario mortality projections to that of the A2 scenario (Table 7.2) and found approximately 27 % fewer heat-related deaths with the B2 scenario. These represent potential health benefits of more aggressive GHG regulatory schemes. While larger O₃-related mortality was

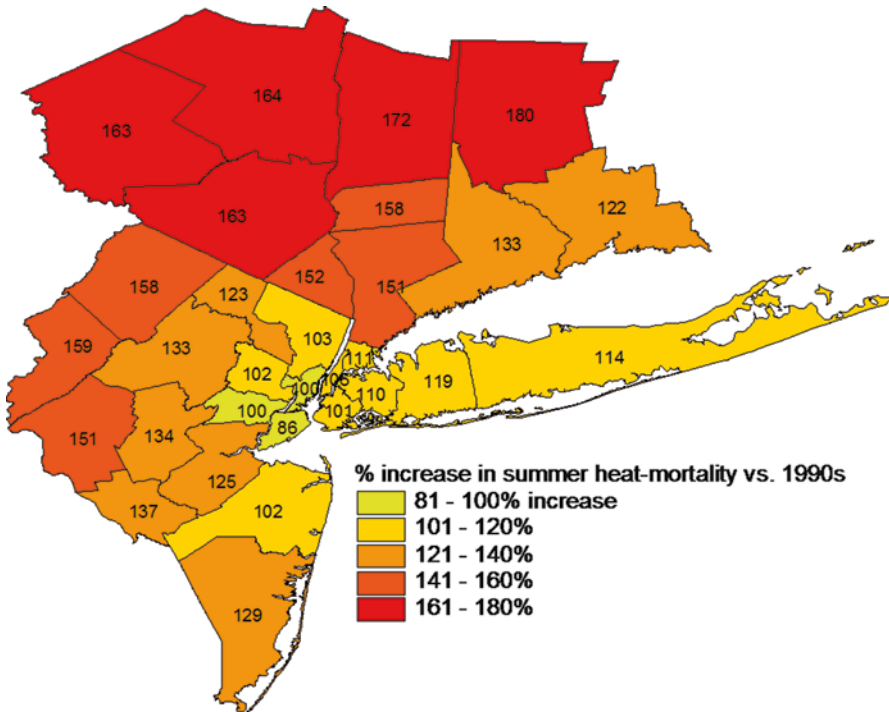


Fig. 7.2 The regional distribution of percentage changes in heat-related mortality by the 2050s under the A2 greenhouse gas emissions scenario

projected for the New York metro region under the B2 scenario assumptions, different patterns across the eastern USA were seen; domain-wide O₃ was projected to increase more under the 2050s A2 scenario than under the B2 scenario.

Discussion and Implications

This work illustrates an interdisciplinary study to develop local scale projections of some possible health impacts of climate change in the NYC metropolitan region. In the USA, health policy decisions (emergency planning, hospital surveillance, etc.) are often made by county health departments, so climate impact projections are likely to be most meaningful if framed at the county level. Further, in the absence of federal regulations, GHG emission control policies often begin at the local level. If in the future the potential health impacts of climate change are monetized and become part of cost-benefit regulatory schemes, then risk assessments such as this could provide information useful not only to public health care infrastructure planning but also to regulators and legislative policy makers. An important limitation of this work is that we did not account for possible acclimatization to heat effects over multiple years as warming trends continue. This is an area in need of future research.

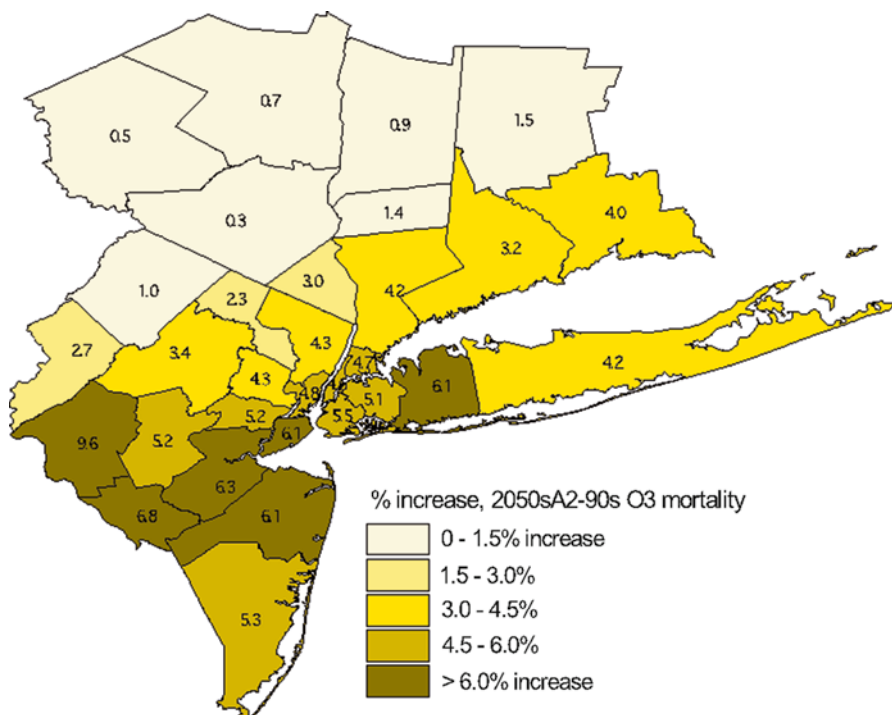


Fig. 7.3 The O₃ effects under the A2 greenhouse gas emissions scenario

Table 7.1 Evolution over decades of temperature- and O₃-related deaths, under the A2 greenhouse gas emission scenario

Decade	Regional summer heat-related mortality	Regional summer O ₃ -related mortality
1990s	1,116	1,059
2020s	1,542	1,174
	38 % increase vs. 1990s	11 % increase vs. 1990s
2050s	2,347	1,108
	110 % increase vs. 1990s	5 % increase vs. 1990s
2080s	5,533	1,266
	396 % increase vs. 1990s	20 % increase vs. 1990s

Case Study 2: Spring Pollen Peaks and Over-the-Counter Allergy Medication Sales

Studies of the onset and duration of pollen seasons have shown significant advances in seasonal onset that are consistent with warming trends [16–25]. What remains unknown is whether, and to what extent, recent trends in pollen seasons may be

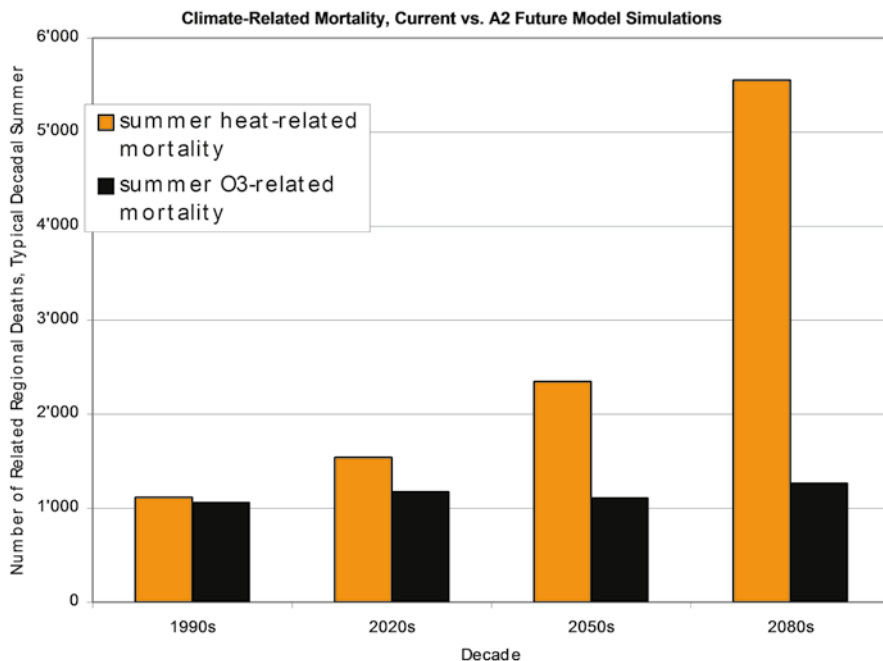


Fig. 7.4 The projected evolution over time of heat vs. O₃ mortality impacts under the A2 scenario

Table 7.2 Comparison of temperature- and O₃-related deaths in a typical summer for the 2050s vs. 1990s, under two different greenhouse gas emission scenarios

	1990s	2050s B2 (lower CO ₂ emissions)	2050s A2 (higher CO ₂ emissions)
Heat-related mortality	1,116	2013 80 % increase relative to 1990s	2347 110 % increase relative to 1990s
O ₃ -related mortality	1,059	1,139 7.6 % increase relative to 1990s	1,108 4.6 % increase relative to 1990s

linked with upward trends in allergic diseases like hay fever and asthma that have been seen in recent decades.

In addition to earlier onset of the pollen season and possibly enhanced seasonal pollen loads in response to higher temperatures and resulting longer growing seasons, there is evidence that CO₂ rise itself may cause increases in pollen levels. Experimental studies have shown that elevated CO₂ concentrations stimulate greater vigor, pollen production, and allergen potency in ragweed [8, 26, 27]. Ragweed is arguably the most important pollen in the USA with up to 75 % of hay fever sufferers sensitized [28]. Significant differences in allergenic pollen protein were observed

in comparing plants grown under historical CO₂ concentrations of 280 (ppm) and recent concentrations of 370 ppm, with potential future concentrations of 600 ppm [27]. Interestingly, significant differences in ragweed productivity were observed in outdoor plots situated in urban, suburban, and rural locales where measurable gradients were observed in both CO₂ concentrations and temperatures. Cities are not only heat islands but also CO₂ islands and thus represent, to some extent, proxies for a future warmer, high-CO₂ world [8]. With warming over the longer term, changing patterns of plant habitat and species density are likely, with gradual movement northward of cool-climate species like maple, birch, and northern spruce [29].

Concentrations of various pollens have been associated with rates of allergic sensitization [30, 31], tendency towards increased asthma episodes [32], higher numbers of asthma-related emergency department (ED) visits [33–36], and asthma-related hospital admissions [37, 38] as well as higher numbers of allergic rhinitis ED visits [39] and allergic rhinitis physician visits [40]. Allergic rhinitis, a type of allergic airway disease that is a risk factor for increased asthma severity [41], decreases the quality of life of a substantial proportion of the US population (10–30 % of adults and up to 40 % of children) and imposes large costs on our health care system [28, 42–44].

Symptomatic relief of allergic rhinitis primarily involves ambulatory care and self-administration of medications. Thus, studies that look at more severe health outcomes like ED visits and hospitalizations only capture a small fraction of the population affected. Also, other factors that influence asthma morbidity, such as respiratory infection, air pollution, and weather, complicate the attribution of illness to pollen exposure.

To examine whether pollen concentrations are temporally linked to allergic responses, we analyzed the association of daily tree pollen peaks and over-the-counter (OTC) allergy medication sales over a 6-year period in the NYC metropolitan area [45].

Data and Methods

Airborne pollen was collected with a Burkard volumetric spore trap (Burkard Manufacturing Co., Rickmansworth, UK) located on the rooftop of Calder Hall at Fordham University's Louis Calder Biological Station in Armonk, NY, about 30 miles north of midtown Manhattan. This station is the closest long-term, nearly continuous pollen record for the NYC region. Trained counters carried out microscopic analysis of pollen slides for 6 years from 2003 to 2008. Peak dates for each pollen type in each year were identified.

We computed daily concentrations of three genera of tree pollen: maple (*Acer* spp.), birch (*Betula* spp.), and oak (*Quercus* spp.). These subtypes were selected because they are clinically relevant aeroallergens in the USA [46] during the early season (March–May) and have well-established sensitization patterns in populations from the northeast region of the USA [47, 48].

Daily temperature data from LaGuardia International Airport were downloaded from the NCDC. Data for $PM_{2.5}$ were obtained from US EPA's Air Quality System. The temporal variations of $PM_{2.5}$ across 21 sites were highly correlated ($r > 0.85$). Therefore, we computed the average of multiple sites, taking into consideration the difference in site-specific means and standard deviations [49].

Data on OTC pharmacy sales are reported electronically to the New York City Department of Health and Mental Hygiene on a daily basis from over 200 store locations, disproportionately in Manhattan but also from the other four NYC boroughs and nearby suburbs in New York State and New Jersey. The store locations in this database cover approximately 30 % of retail pharmacies in NYC [50]. For this analysis, the following brand name and generic products were classified as allergy medications: Alavert, Benadryl, Claritin, loratidine, Sudafed, and Tavist, as well as other oral and nasal spray medications that include the word "allergy" in their name. Eye drops and topical creams were not included.

We used an indicator variable (1 for peak dates; 0 otherwise) for the tree pollen peak dates between March and May each year for each genus. There was a total of 18 pollen peak dates over the 6-year study period. A regression model was used to estimate the impact of the tree pollen peak dates on the daily allergy medication sales, adjusting for potential confounding factors. We examined lags 0–6 days from the pollen peak dates (i.e., we compared today's allergy medication sales with today's tree pollen peak, today's allergy medication sales with yesterday's pollen peak). We first included individual lags of the tree pollen peak date indicator to determine the lag structure of associations and then included all of the 7-day lags to estimate the multiday effects (i.e., unconstrained distributed lag model). Covariates considered in the regression model included a day-of-week indicator variable, a year indicator variable, and air pollution and temperature variables to capture the effects of temperature on allergy symptoms [51] or on purchasing behaviors.

Results and Discussion

Figure 7.5 shows time series plots of OTC allergy medication sales for the entire city during the years 2003–2008, with tree pollen peak dates superimposed for maple, oak, and birch. The tree pollen peak dates appear to coincide with sharp peaks in the spring medication sales. A general upward trend in sales across years can partially be explained by the number of stores reporting, which increased from 206 in 2003 to 231 in 2008.

Figure 7.6 shows the estimated impacts of tree pollen peaks when all the lagged peak date indicators were included simultaneously in the regression model. The largest statistically significant impact occurred at lag 2 day (28.7 % [95%CI: 17.4, 41.2]), followed by lag 1 day. In the distributed lag model, the sum of the effects over the 7-day period was 141.1 % (95%CI: 79.4, 224.1). These results were not changed substantially in sensitivity analyses that tested alternative covariates and modeling methods.

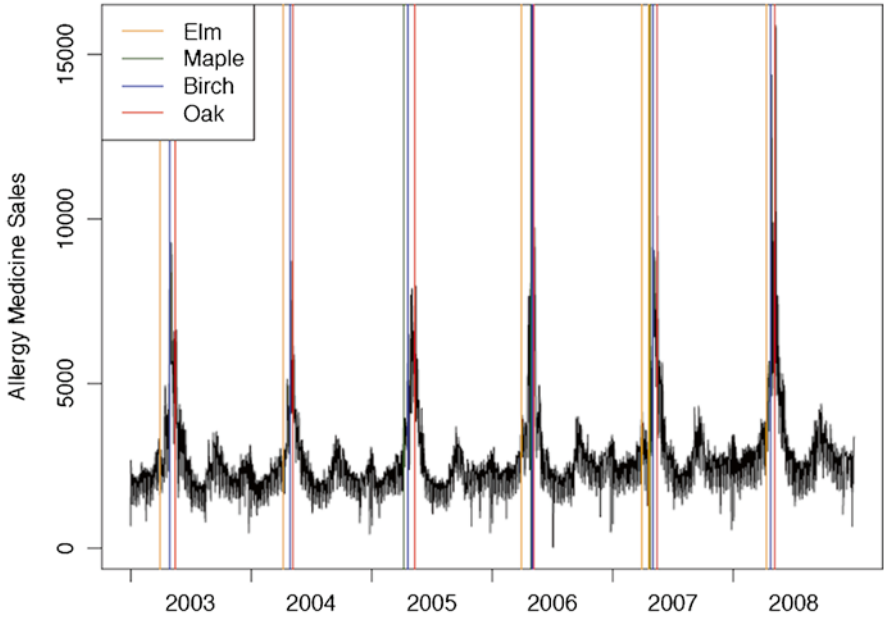


Fig. 7.5 Time series plots of OTC allergy medication sales for New York City during the years 2003–2008, with tree pollen peak dates superimposed for maple, oak, and birch

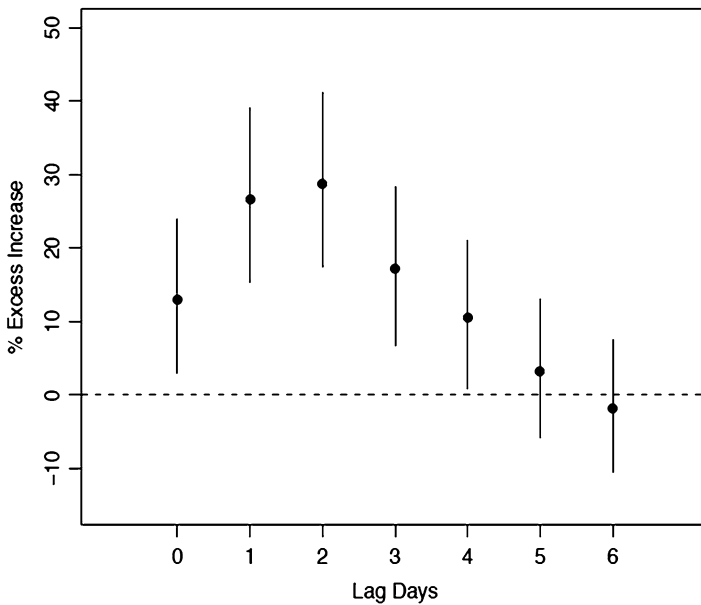


Fig. 7.6 The estimated impacts of tree pollen peaks when all the lagged peak date indicators were included simultaneously in the regression model

These findings suggest that monitoring OTC medication sales may be a useful method of population surveillance for allergic illness and the impact of pollen. Our findings are generally but not entirely consistent with other studies examining the relation of ambient pollen to minor allergic illness. In an urban area in France, insurance claims were used to show that daily purchases of prescription allergy medications were associated with same-day concentrations of some tree pollens and grass pollen while controlling for weather and air pollution [52]. A study in Ottawa, Canada, found no effect of tree pollen on ED visits for conjunctivitis and rhinitis, but ragweed and fungal spore concentrations appeared to be associated with same-day ED visits while controlling for weather and air pollution. The exploration of lagged effects was not described in detail by the authors [39]. In Toronto, Canada, physician visits among the elderly for allergic rhinitis were associated with 10-day average ragweed concentrations but not with air pollution; they did not analyze pollen types other than ragweed [40]. One strength of our study is that it includes a fuller examination of lags than these previous studies.

An advantage of using OTC medications is that this health outcome reflects minor illness, as many will not seek health care nor have claims filed for prescription medications. The observed associations support use of genus-specific tree pollen season charts in clinical allergy practice, which are not currently being used in allergy clinics in New York (personal correspondence, President of the NY Allergy Society, January 2010). However, limitations of this approach include the possibility that individuals may self-medicate using previously purchased OTC medications, that the single purchase of an OTC allergy medication could result in usage at multiple different times other than the day of purchase, and that the available in-home medications may vary within a calendar year. Thus, our analysis likely underestimates the overall contribution of pollen to use of OTC allergy medications. Furthermore, purchase of an OTC allergy medication does not describe frequency of use, severity of symptoms, nor the number of individuals using a particular medication.

While this study did not directly address the role of climate factors in variations in pollen exposure and allergic health responses, the interannual variations that we observed in tree pollen peaks and OTC medication sales are probably reflective of interannual variations in climate, such as winter and spring temperatures and precipitation. Efforts to better detail the role of weather and climate variables as drivers of pollen-related health impacts, the geospatial variation of pollen concentrations, and the timing and intensity of pollen season will contribute to work in this field. Future work should explore relationships between meteorologic variables and the timing and intensity of the pollen season, specifically temperature and precipitation over seasonal time scales.

Summary

The two case studies reviewed above demonstrate some of the methods that have recently been applied to study climate interactions with human health, mediated by temperature, air pollution, and/or airborne pollen. These examples demonstrate some of the characteristic features of emerging research in this area, including the formation of interdisciplinary teams, merging of health and climate data, the role of

geographical downscaling, and the tools of health impact assessment. These approaches and others will be needed to further examine linkages between climate variations and human health impacts across a range of disease outcomes.

Acknowledgments This work was supported by the US Environmental Protection Agency under Science To Achieve Results (STAR) grant R828733. Additional support was provided by the National Institute of Environmental Health Sciences Center grant ES09089 and from the National Aeronautics and Space Administration/Goddard Institute for Space Studies Climate Impacts Group Drs. Kim Knowlton, Christian Hogrefe, Cynthia Rosenzweig, Tom Matte, Kax Ito, and Guy Robinson. Although the research described in this article has been funded wholly or in part by the US EPA, it has not been subjected to the Agency's required peer and policy review and therefore does not necessarily reflect the views of the Agency and no official endorsement should be inferred.

References

1. Kinney PL. Climate change, air quality, and human health. *Am J Prev Med.* 2008;35(5): 459–67. <http://www.ncbi.nlm.nih.gov/pubmed/18929972>. Accessed 19 June 2011.
2. Jacob DJ, Winner DA. Effect of climate change on air quality. *Atmos Environ.* 2009;43(1): 51–63. <http://linkinghub.elsevier.com/retrieve/pii/S1352231008008571>. Accessed 30 July 2012.
3. Peel JL, Tolbert PE, Klein M, et al. Ambient air pollution and respiratory emergency department visits. *Epidemiology.* 2005;16(2):164–74. <http://content.wkhealth.com/linkback/openurl?sid=WKPTLP:landingpage&an=00001648-200503000-00004>. Accessed 25 August 2012.
4. Peel JL, Metzger KB, Klein M, et al. Ambient air pollution and cardiovascular emergency department visits in potentially sensitive groups. *Am J Epidemiol.* 2006;165(6):625–33. <http://www.ncbi.nlm.nih.gov/pubmed/17194748>. Accessed 4 September 2012.
5. Kinney PL, Ozkaynak H. Associations of daily mortality and air pollution in Los Angeles County. *Environ Res.* 1991;54(2):99–120. <http://www.ncbi.nlm.nih.gov/pubmed/2029880>.
6. Levy JI, Chemerynski SM, Sarnat J. Ozone exposure and mortality: an empiric bayes metaregression analysis. *Epidemiology.* 2005;16(4):458–68. <http://content.wkhealth.com/linkback/openurl?sid=WKPTLP:landingpage&an=00001648-200507000-00006>. Accessed 18 Aug 2012.
7. Hogrefe C, Leung LR, Mickle L, Hunt SW, Winner D. Considering climate change in U.S. air quality management. *J Air Waste Manag Assoc.* 2005;55:35–40.
8. Ziska LH, Gebhard DE, Frenz D, et al. Cities as harbingers of climate change: Common ragweed, urbanization, and public health. *J Allergy Clin Immunol.* 2003;111(2):290–5. <http://linkinghub.elsevier.com/retrieve/pii/S009167490300959X>. Accessed 19 July 2012.
9. Filleul L, Cassadou S, Médina S, et al. The relation between temperature, ozone, and mortality in nine French cities during the heat wave of 2003. *Environ Health Perspect.* 2006;114(9):1344–7. <http://www.ehponline.org/ambra-doi-resolver/10.1289/ehp.8328>. Accessed 16 July 2012.
10. Knowlton K, Hogrefe C, Lynn B, et al. Impacts of heat and ozone on mortality risk in the New York City metropolitan region under a changing climate. In: Thomson MC, editor. *Seasonal forecasts, climatic change and human health*. New York: Springer Science + Business Media B.V; 2008. p. 143–60.
11. Ito K, De Leon SF, Lippmann M. Associations between ozone and daily mortality. *Epidemiology.* 2005;16(4):446–57. <http://content.wkhealth.com/linkback/openurl?sid=WKPTLP:landingpage&an=00001648-200507000-00005>. Accessed 14 July 2012.
12. Bell ML, Dominici F, Samet JM. A meta-analysis of time-series studies of ozone and mortality with comparison to the national morbidity, mortality, and air pollution study. *Epidemiology.* 2005;16(4):436–45. <http://content.wkhealth.com/linkback/openurl?sid=WKPTLP:landingpage&an=00001648-200507000-00004>. Accessed 8 August 2012.
13. Hogrefe C. Simulating changes in regional air pollution over the eastern United States due to changes in global and regional climate and emissions. *J Geophys Res.* 2004;109(D22301): 1–13. <http://www.agu.org/pubs/crossref/2004/2004JD004690.shtml>. Accessed 18 August 2012.

14. Hogrefe C. Simulating regional-scale ozone climatology over the eastern United States: model evaluation results. *Atmos Environ.* 2004;38(17):2627–38. <http://linkinghub.elsevier.com/retrieve/pii/S1352231004001748>. Accessed 3 May 2011.
15. Knowlton K, Rosenthal JE, Hogrefe C, et al. Assessing ozone-related health impacts under a changing climate. *Environ Health Perspect.* 2004;112(15):1557–63. <http://www.ehponline.org/ambra-doi-resolver/10.1289/ehp.7163>. Accessed 15 Aug 2012.
16. Root TL, Price JT, Hall KR, Schneider SH. Fingerprints of global warming on wild animals and plants. *Nature.* 2003;421(6918):57–60.
17. Beggs PJ, Bambrick HJ. Is the global rise of asthma an early impact of anthropogenic climate change? *Environ Health Perspect.* 2005;113(8):745–52. <http://www.ehponline.org/ambra-doi-resolver/10.1289/ehp.7724>. Accessed 4 Sept 2012.
18. Beggs PJ. Impacts of climate change on aeroallergens: past and future. *Clin Exp Allergy.* 2004;34(10):1507–13. <http://www.ncbi.nlm.nih.gov/pubmed/15479264>. Accessed 4 Sept 2012.
19. Clot B. Trends in airborne pollen: An overview of 21 years of data in Neuchâtel (Switzerland). *Aerobiologia.* 2003;19(3/4):227–34. <http://www.springerlink.com/openurl.asp?id=doi:10.1023/B:AERO.0000006572.53105.17>.
20. Emberlin J, Detandt M, Gehrig R, et al. Responses in the start of *Betula* (birch) pollen seasons to recent changes in spring temperatures across Europe. *Int J Biometeorol.* 2002;46(4):159–70. <http://www.ncbi.nlm.nih.gov/pubmed/12242471>. Accessed 26 July 2012.
21. Galán C, García-Mozo H, Vázquez L, et al. Heat requirement for the onset of the *Olea europaea* L. pollen season in several sites in Andalusia and the effect of the expected future climate change. *Int J Biometeorol.* 2005;49(3):184–8. <http://www.ncbi.nlm.nih.gov/pubmed/15645246>. Accessed 4 Sept 2012.
22. Rasmussen A. The effects of climate change on the birch pollen season in Denmark. *Aerobiologia.* 2002;18(3):253–65.
23. Teranishi H, Kenda Y, Katoh T, et al. Possible role of climate change in the pollen scatter of Japanese cedar *Cryptomeria japonica* in Japan. *Climate Res.* 2000;14(1):65–70.
24. van Vliet AJH, Overeem A, De Groot RS, Jacobs AFG, Spijksma FTM. The influence of temperature and climate change on the timing of pollen release in the Netherlands. *Int J Climatol.* 2002;22(14):1757–67. <http://doi.wiley.com/10.1002/joc.820>. Accessed 4 Sept 2012.
25. Huynen M, Menne B, Behrendt H, et al. Phenology and health: allergic disorders. Rome: WHO; 2003.
26. Ziska LH, Caufield FA. Rising CO₂ and pollen production of common ragweed (*Ambrosia artemisiifolia*), a known allergy-inducing species: implications for public health. *Aust J Plant Physiol.* 2000;27:893–8.
27. Singer BD, Ziska LH, Frenz DA, Gebhard DE, Straka JG. Research note : 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.
28. American Academy of Allergy Asthma and Immunology (AAAAI). Allergy statistics. *Ann Allergy.* 2000;24(11). <http://www.aaaai.org/about-the-aaaai/newsroom/allergy-statistics.aspx>.
29. Rosenzweig C, Casassa G, Karoly D, et al. Assessment of observed changes and responses in natural and managed systems. In: *Climate change 2007: impacts, adaptation and vulnerability.* Cambridge: Cambridge University Press; 2007:79–131. <http://www.ipcc.ch/pdf/assessment-report/ar4/wg2/ar4-wg2-chapter1.pdf>.
30. Björkstén F, Suoniemi I. Time and intensity of first pollen contacts and risk of subsequent pollen allergies. *Acta Med Scand.* 1981;209(4):299–303. <http://www.ncbi.nlm.nih.gov/pubmed/7234505>.
31. Porsbjerg C, Linstow ML, Nepper-Christensen SC, et al. Allergen sensitization and allergen exposure in Greenland Inuit residing in Denmark and Greenland. *Respir Med.* 2002;96(9):736–44. <http://linkinghub.elsevier.com/retrieve/pii/S0954611102913417>. Accessed 4 Sept 2012.
32. Delfino RJ, Zeiger RS, Seltzer JM, Street DH, McLaren CE. Association of asthma symptoms with peak particulate air pollution and effect modification by anti-inflammatory medication use. *Environ Health Perspect.* 2002;110(10):A607–17. <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1241047&tool=pmcentrez&rendertype=abstract>.
33. Babin SM, Burkom HS, Holtry RS, et al. Pediatric patient asthma-related emergency department visits and admissions in Washington, DC, from 2001–2004, and associations with air

- quality, socio-economic status and age group. *Environ Health*. 2007;6:9. <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1845147&tool=pmcentrez&rendertype=abstract>. Accessed 13 July 2012.
34. Lierl MB, Hornung RW. Relationship of outdoor air quality to pediatric asthma exacerbations. *Ann Allergy Asthma Immunol*. 2003;90(1):28–33. <http://www.ncbi.nlm.nih.gov/pubmed/12546334>. Accessed 4 Sept 2012.
 35. Wang HC, Yousef E. Air quality and pediatric asthma-related emergencies. *J Asthma*. 2007;44(10):839–41. <http://www.ncbi.nlm.nih.gov/pubmed/18097860>. Accessed 4 Sept 2012.
 36. Zhong W, Levin L, Reponen T, et al. Analysis of short-term influences of ambient aeroallergens on pediatric asthma hospital visits. *Sci Total Environ*. 2006;370(2–3):330–6.
 37. Dales RE, Cakmak S, Judek S, Coates F. Tree pollen and hospitalization for asthma in urban Canada. *Int Arch Allergy Immunol*. 2008;146(3):241–7. <http://www.ncbi.nlm.nih.gov/pubmed/18270491>. Accessed 4 Sept 2012.
 38. Im W, Schneider D. Effect of weed pollen on children's hospital admissions for asthma during the fall season. *Int Arch Allergy Immunol*. 2006;60(5):257–66.
 39. Cakmak S, Dales RE, Burnett RT, et al. Effect of airborne allergens on emergency visits by children for conjunctivitis and rhinitis. *Lancet*. 2002;359(9310):947–8. <http://www.ncbi.nlm.nih.gov/pubmed/11918918>.
 40. Villeneuve PJ, Doiron M-S, Stieb D, et al. Is outdoor air pollution associated with physician visits for allergic rhinitis among the elderly in Toronto, Canada? *Allergy*. 2006;61(6):750–8. <http://www.ncbi.nlm.nih.gov/pubmed/16677246>. Accessed 14 Feb 2012.
 41. Thomas M. Allergic rhinitis: evidence for impact on asthma. *BMC Pulm Med*. 2006;6 Suppl 1:S4. <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1698497&tool=pmcentrez&rendertype=abstract>. Accessed 8 Aug 2012.
 42. Soni A. Allergic rhinitis: trends in use and expenditures, 2000 and 2005. 2008. http://meps.ahrq.gov/mepsweb/data_files/publications/st204/stat204.pdf.
 43. Anon. Top 200 generic drugs by retail dollars in 2007. 2008. <http://drugtopics.modernmedicine.com/drugtopics/data/articlestandard//drugtopics/102008/500218/article.pdf>.
 44. Nielsen-Wolter Kluwer Health. Consumer behavior and managed care impact of the Zyrtec Rx-to-OTC Switch. 2008. http://download.lww.com/wolterskluwer_vitalstream_com/PermaLink/Allergy-whitepaperFinal.pdf.
 45. Sheffield PE, Weinberger KR, Ito K, et al. The association of tree pollen concentration peaks and allergy medication sales in New York City: 2003–2008. *ISRN Allergy*. 2011;2011:1–7. <http://www.isrn.com/journals/allergy/2011/537194/>. Accessed 15 Aug 2012.
 46. U.S. EPA. A review of the impact of climate variability and change on aeroallergens and their associated effects. Washington, DC: U.S. EPA; 2008. <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=190306>.
 47. Lin RY, Clauss AE, Bennett ES. Hypersensitivity to common tree pollens in New York City patients. *N Engl Reg Allergy Proc*. 2002;23(4):253–8. <http://www.ncbi.nlm.nih.gov/pubmed/12221895>.
 48. White JF, Levin L, Villareal M, et al. Lack of correlation between regional pollen counts and percutaneous reactivity to tree pollen extracts in patients with seasonal allergic rhinitis. *Ann Allergy Asthma Immunol*. 2005;94(2):240–6. <http://linkinghub.elsevier.com/retrieve/pii/S1081120610613026>. Accessed 4 Sept 2012.
 49. Zanobetti A, Schwartz J, Dockery DW. Airborne particles are a risk factor for hospital admissions for heart and lung disease. *Environ Health Perspect*. 2000;108(11):1071–7. <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1240165&tool=pmcentrez&rendertype=abstract>.
 50. Das D, Metzger K, Heffernan R, et al. Monitoring over-the-counter medication sales for early detection of disease outbreaks—New York City. *MMWR Morb Mortal Wkly Rep*. 2005; 54(Suppl):41–6.
 51. Cruz AA, Togias A. Upper airways reactions to cold air. *Curr Allergy Asthma Rep*. 2008;8(2):111–7. <http://www.ncbi.nlm.nih.gov/pubmed/18417052>.
 52. Fuhrman C, Sarter H, Thibaudon M, et al. Short-term effect of pollen exposure on antiallergic drug consumption. *Ann Allergy Asthma Immunol*. 2007;99(3):225–31.

Chapter 8

The Human Health Co-benefits of Air Quality Improvements Associated with Climate Change Mitigation

George D. Thurston and Michelle L. Bell

Abstract Fossil fuel combustion processes that generate greenhouse gases (GHG) also emit and or cause the creation of other harmful air pollutants. Thus, while policies designed to avert the course of climate change would eventually result in direct human health benefits from lessened global temperature changes and associated impacts, they would also bring much more immediate ancillary human health co-benefits from the associated reduced ground-level air pollution in the short term. Several measures aimed at reducing GHG emissions, notably the reduced use of fossil fuels such as coal, can also improve local air quality, most notably particulate matter (PM) and ozone (O₃) air pollution. Further, whereas the benefits from climate change mitigation would materialize far in the future, these co-benefits, or ancillary benefits, would provide much more immediate “return on investment” in climate change mitigation. Thus, as detailed below, the near-term human health co-benefits of climate mitigation (e.g., fossil fuel emission reductions) may provide the most economically compelling justification for immediate action towards climate change mitigation. Here we discuss the health impacts of PM and ozone, two key air pollutants that have substantial impacts on human health and that are likely to be reduced by policies aimed at controlling GHG emissions.

Keywords Air quality and climate change • Climate change and air quality • Air pollution and health • Greenhouse gases and human health • Ozone exposures • Particulate matter time series studies

G.D. Thurston, A.B., Sc.B., S.M., Sc.D. (✉)
Department of Environmental Medicine, New York University School of Medicine,
Tuxedo, NY 10987, USA
e-mail: george.thurston@nyu.edu

M.L. Bell, Ph.D.
School of Forestry and Environmental Studies, Yale University, 195 Prospect Street,
New Haven, CT 06511, USA
e-mail: michelle.bell@yale.edu

Fossil-fuel combustion processes that generate greenhouse gases (GHG) also emit and or cause the creation of other harmful air pollutants. Thus, while policies designed to avert the course of climate change would eventually result in direct human health benefits from lessened global temperature changes and associated impacts, they would also bring much more immediate ancillary human health co-benefits from the associated reduced ground-level air pollution in the short term [1–6]. Several measures aimed at reducing GHG emissions, notably the reduced use of fossil fuels such as coal, can also improve local air quality, most notably particulate matter (PM) and ozone (O₃) air pollution. Further, whereas the benefits from climate change mitigation would materialize far in the future, these co-benefits, or ancillary benefits, would provide much more immediate “return on investment” in climate change mitigation. Thus, as detailed below, the near-term human health co-benefits of climate mitigation (e.g., fossil fuel emission reductions) may provide the most economically compelling justification for immediate action towards climate change mitigation. Here we discuss the health impacts of PM and ozone, two key air pollutants that have substantial impacts on human health and that are likely to be reduced by policies aimed at controlling GHG emissions.

Health Effects of Particulate Matter

Tropospheric aerosols that affect climate change also have significant human health implications. A wealth of scientific literature clearly links particulate matter with numerous adverse health effects. Indeed, a US Environmental Protection Agency (USEPA) assessment of human health effects benefits of the Clean Air Act attributed nearly 90 % of the estimated monetary valuation of the human health effects benefits to be derived from the act during 1990–2010 to reductions in PM [7].

Short-Term Exposure Effects of PM

Acute (short-term) exposure to particulate air pollution has been found to be associated with increases in the rates of daily asthma attacks, hospital admissions, and mortality. PM is associated with increased risk of respiratory hospital admissions in New York, NY, Buffalo, NY, and Toronto, ON [8], as well as with mortality in cities such as Chicago, IL, and Los Angeles, CA [9, 10]. These results have been confirmed by other researchers considering locales elsewhere in the USA, and in other cities throughout the world, including national multi-city studies [11–15].

In addition to lung damage, recent epidemiological and toxicological studies of PM air pollution have shown adverse effects on the heart, including an increased risk of heart attacks. For example, when PM stresses the lung (e.g., by inducing edema), it places extra burden on the heart, which can induce fatal complications for

persons with cardiac problems. Indeed, Peters et al. [16] found that elevated concentrations of fine particles ($PM \leq 2.5 \mu\text{m}$ in aerodynamic diameter, i.e., $PM_{2.5}$) in the air could elevate the risk of myocardial infarctions (MIs) within a few hours, and extending 1 day after $PM_{2.5}$ exposure. Others found that a 48 % increase in the risk of MI was associated with an increase of $25 \mu\text{g}/\text{m}^3$ $PM_{2.5}$ during a 2-h period before the onset of MI, and a 69 % increase in risk to be related to an increase of $20 \mu\text{g}/\text{m}^3$ $PM_{2.5}$ in the 24-h average 1 day before the MI onset [16].

Epidemiologic research conducted in the USA and elsewhere has indicated that acute exposure to PM air pollution is associated with increased risk of mortality. For example, a national multi-city time-series statistical analysis of mortality and $PM \leq 10 \mu\text{m}$ in aerodynamic diameter (PM_{10}) air pollution in 90 US cities indicates that an increase of $10 \mu\text{g}/\text{m}^3$ in daily PM_{10} is associated with an increase of approximately 0.3 % in the daily risk of death [17]. This result of a 0.3 % change in the daily mortality rate is tied to the increment of pollution; in other words, a pollution increase larger than $10 \mu\text{g}/\text{m}^3$ would be associated with a larger increase in risk of mortality. Further, such added risks apply to the entire population and accumulate for every day of exposure until they account for many deaths from air pollution globally each year.

Long-Term Exposure Effects of PM

In addition to the health effects associated with acute exposure to PM pollution, long-term chronic exposure to particles is also associated with increased lifetime risk of death and has been estimated to take years from the life expectancy of people living in the most polluted cities, relative to those living in cleaner cities. The first studies to show this association were cross-sectional studies that compared metropolitan area death rates in high and low PM cities, after adjusting for potentially confounding factors in the populations, such as age, sex, and race [18]. These results have since been confirmed by cohort studies that followed large groups of individuals in various cities over time that are able to control for potential confounding factors on an individual level. For example, in the Six-Cities Study, which was a key basis for the setting of the USEPA's original health-based regulation for a $PM_{2.5}$ annual standard in 1997, Dockery et al. [19] analyzed survival probabilities among 8,111 adults living in six cities in the central and eastern portions of the USA during the 1970s and 1980s. The cities were Portage, WI (P); Topeka, KS (T); a section of St. Louis, MO (L); Steubenville, OH (S); Watertown, MA (M); and Kingston-Harriman, TN (K). Air quality was averaged over the period of study in order to study long-term (chronic) effects. It was found that the long-term risk of death, relative to the cleanest city, increased with fine particle exposure, even after adjusting for potentially confounding factors such as age, sex, race, and smoking.

More recently, it is recognized that long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for

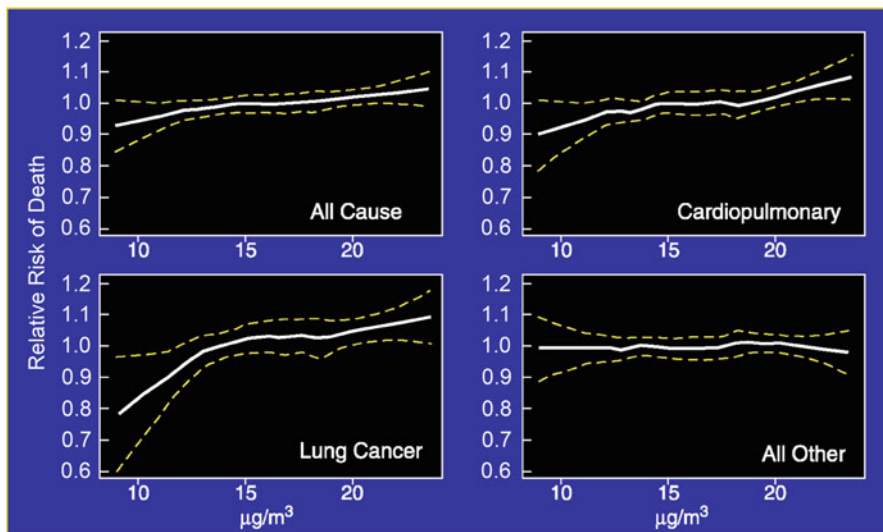


Fig. 8.1 The cardiac, lung, and cancer mortality risks of long-term fine PM exposure increase monotonically with exposure (Adapted from Pope, C.A. III, Burnett, R.T., Thun, M.J., Calle, E.E., Krewski, D., Ito, K., and Thurston, G.D. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *J. Am. Med. Assoc. (JAMA)* 287(9):1132–1141 (2002), with permission)

cardiopulmonary and lung cancer mortality (Fig. 8.1). Indeed, this study indicates that the increase in risk of lung cancer from long-term exposure to $PM_{2.5}$ was of roughly the same size as the increase in lung cancer risk of a non-smoker who breathes passive smoke while living with a smoker, or about a 20 % increase in lung cancer risk [20].

Other studies indicating health risk from chronic exposure to PM include a multi-city US study finding that a $10 \mu\text{g}/\text{m}^3$ increase in yearly $PM_{2.5}$ is associated with approximately a 11–21 % increase in mortality [21]. A systematic review of research on long-term PM exposure found that collectively, the studies indicate a 15–21 % increase in mortality per $10 \mu\text{g}/\text{m}^3$ $PM_{2.5}$ [22].

Health Effects of PM Constituents

Particulate matter is a complex mixture of a wide array of chemical constituents, and PM's chemical composition varies seasonally and regionally [23]. For example, some particles may have a larger contribution of sulfate whereas others may have more nitrate. The chemical structure of particles is related to the sources. While most past studies have investigated the effects of the PM *mass* concentration on human health effects, newer studies have begun to evaluate the mortality impacts of

PM by specific components or sources, including two key *aerosol components* that affect climate change: sulfates and elemental black carbon (BC) soot.

With regard to acute effects of PM components, Thurston et al. [24] found that coal burning-related sulfate containing aerosols were among those most associated with increases in daily mortality. Bell et al. [25] found that communities with higher PM_{2.5} content of nickel (Ni), vanadium (V), and elemental carbon (EC) and/or their related sources were found to have higher risk of hospitalizations associated with short-term exposure to PM_{2.5}. Lall et al. [26] similarly found that EC of traffic origins was associated with higher risk of cardiovascular disease (CVD) hospital admissions in New York, NY than PM_{2.5} mass in general. In a study of mortality in New York, NY, Ito et al. [27] have reported that coal combustion-related components (e.g., selenium (Se) and sulfur) were associated with CVD mortality in summer, whereas the traffic-related EC showed associations with CVD mortality throughout the year. Zhou et al. [28] investigated the PM_{2.5} components and gaseous pollutants associated with mortality in Detroit, MI and Seattle, WA. These authors similarly found that CVD and respiratory mortality were most associated with warm season secondary aerosols (e.g., sulfates) and traffic-markers (e.g., EC) in Detroit, while in Seattle, the component species most closely associated with mortality included those for cold season traffic and other combustion sources, such as residual oil and wood burning. In addition, recent evidence has implicated diesel traffic-derived EC as a factor in increased risk of acute asthma morbidity [29]. Overall, these studies of PM_{2.5} components and constituents largely indicate that both EC and sulfates (and their associated sources, including diesel traffic and coal burning) were among the most explanatory of the acute adverse health effects of PM_{2.5}.

With regard to the long-term effects of PM air pollution, Ozkaynak and Thurston [18] conducted the first source apportionment of PM_{2.5}-mortality effects, finding that sulfate-related particles, largely from coal burning, were most associated with the mortality impacts of long-term exposure to PM_{2.5}. More recently, Ostro et al. [30] examined daily data from 2000 to 2003 on mortality and PM_{2.5} mass and components, including elemental and organic carbon (EC and OC), nitrates, sulfates, and various metals. The authors examined associations of PM_{2.5} and its constituents with daily counts of several mortality categories: all-cause, cardiovascular, respiratory, and mortality age >65 years, finding the strongest associations between mortality and sulfates and several metals. Ostro et al. [30] used data from a prospective cohort of active and former female public school professionals to develop estimates of long-term exposures to PM_{2.5} and several of its constituents, including EC, OC, sulfates, nitrates, iron (Fe), potassium (K), silicon (Si), and zinc (Zn), finding increased risks of all-cause and cardiopulmonary mortality from exposure to constituents derived from combustion of fossil fuel (including diesel), as well as those of crustal origin. In addition, Smith et al. [6] undertook a meta-analysis of existing time-series studies, as well as an analysis of a cohort of 352,000 people in 66 US cities during 18 years of follow-up of the ACS cohort, finding total mortality effects from long-term exposure to both the elemental BC and sulfate components of PM_{2.5} aerosols.

Health Effects of Tropospheric Ozone

Tropospheric ozone is a highly reactive pollutant that is common in the urban environment, as it largely results from emissions from fossil fuel combustion. O_3 is a secondary pollutant, meaning it is not directly emitted, but rather is formed through complex nonlinear reactions from the precursor's volatile organic compounds (VOCs) and nitrogen oxides (NO_x) in the presence of sunlight. Sources of VOCs and NO_x include transportation, industry, and power plants. Both VOCs and NO_x have natural sources, of which vegetative emissions are key contributors of VOCs. Levels of O_3 are especially of concern in urban environments, and in fact in the USA more persons live in areas that exceed the health-based regulations for O_3 than for any other criteria pollutant. Ozone is a growing problem in developing regions of the world, with expanding transportation networks and industry. Thus, this pollutant is not only a global warming pollutant but also has significant global health impacts.

Short-Term Exposure Effects of O_3

The scientific evidence for the respiratory morbidity effects from acute exposure to O_3 is well documented. Animal toxicological studies have indicated that chronic O_3 exposure caused structural changes in the respiratory tract, and simulated seasonal exposure studies in animals have also suggested that such exposures might have cumulative impacts, providing evidence of a biological foundation for the associations observed in population-based studies [31]. Recent epidemiologic studies have also observed that reduced lung function growth in children is associated with seasonal exposure to O_3 [32–34]. Based on evidence from animal toxicological studies, short-term and sub-chronic exposures to O_3 can cause morphological changes in the respiratory systems of a number of species, including primates. Following chronic O_3 exposure, structural changes have been observed in the centriacinar region (CAR), the region typically affected in most chronic airway diseases of the human lung. In addition, a substantial number of human exposure studies have been published that have provided important information on lung inflammation and epithelial permeability. Mudway and Kelly [35], for example, examined O_3 -induced inflammatory responses and epithelial permeability with a meta-analysis of 21 controlled human exposure studies, finding that polymorphonuclear neutrophils (PMN) influx in healthy subjects is associated with total O_3 dose product of O_3 concentration, exposure duration, and minute ventilation. Overall, animal toxicological studies indicate that short-term and sub-chronic exposures to O_3 can cause morphological changes in the respiratory systems, particularly in the CAR [31]. Thus, there is strong supportive evidence from both acute epidemiological studies and toxicological studies of respiratory morbidity that ozone exposure can have serious respiratory morbidity health effects.

O_3 exposure has also been found to be associated with short-term increases in risk of mortality as a result of acute exposures. Indeed, robust associations have been

identified between various measures of daily O₃ concentrations and increased risk of mortality. As summarized by USEPA [31]: “most of the single-pollutant model estimates from single-city studies fall in the range between 0.5 and 5 % excess deaths per standardized increment (40 ppb for 1-h max O₃, 30 ppb for 8-h max O₃, and 20 ppb for 24-h avg. O₃).” In addition, several studies in recent years conducted meta-analyses of O₃-mortality associations [36–39]. Subsequent combined O₃ excess mortality risk estimates from the meta-analyses by Bell et al. [40], Ito et al. [41], and Levy et al. [36] were also very consistent. Associations have also been observed in other study designs including multi-city time-series of 95 US urban cities over a 14-year period finding a 0.52 % (95 % interval 0.27, 0.77 %) increase in mortality risk for a 10 ppb increase in daily ozone over the previous week [42]. O₃ effects were also observed for cardiorespiratory mortality. The Air Pollution and Health: A European Approach (APHEA2) project examined ozone and mortality for 23 European cities with at least 3 years of data [43]. A 10 µg/m³ increase in the 1-h max ozone was associated with a 0.33 % (0.17, 0.52 %) increase in mortality risk, with associations also observed for cardiovascular and respiratory deaths. A case-crossover study of 14 US cities found a 0.23 % (0.01, 0.44 %) increase in mortality risk for a 10 ppb increase in daily maximum ozone levels, with matching on days of similar temperature [44].

There is evidence that the association between ozone and mortality persists at low concentrations. A study of 98 US urban communities with 14 years of data used several modeling approaches to investigate the shape of the exposure-response curve. The first method assumed that any level of ozone could potentially be associated with mortality risk; this is the traditionally applied time-series approach. The second method examined the subset of data below specified values of 5–60 ppb, at 5 ppb increments for daily ozone. A threshold model was fit to assume no association for ozone levels below a specified threshold value and a traditional shape for higher ozone levels. The final approach used a nonlinear function of ozone levels to allow a flexible relationship between ozone and mortality. None of the alternative models found evidence of a threshold at policy-relevant concentrations. The study found that associations were significant at levels nearing natural background concentrations and levels below the USEPA’s National Ambient Air Quality Standard at the time of the study.

Several studies have examined whether associations between short-term exposure to ozone and risk of mortality are confounded by airborne particles, which have demonstrated links with mortality as discussed above. The most common approach, to include a variable for particles in the model, was found to result in little change to ozone effect estimates [37, 38, 40–44]. Figure 8.2 provides estimates of the association between ozone and mortality with and without adjustment for particulate matter for early single-city studies and combined estimates of those studies. Other approaches to exploring confounding have also provided evidence for the hypothesis that the ozone-mortality association is not confounded by particulate matter, including at low levels of ozone [23].

Some segments of the population may face a disproportionate burden from ozone pollution. Communities with higher unemployment had higher effect estimates for short-term ozone and mortality for 98 US urban communities [45]. A higher proportion of Black/African-American residents was also associated with higher effect

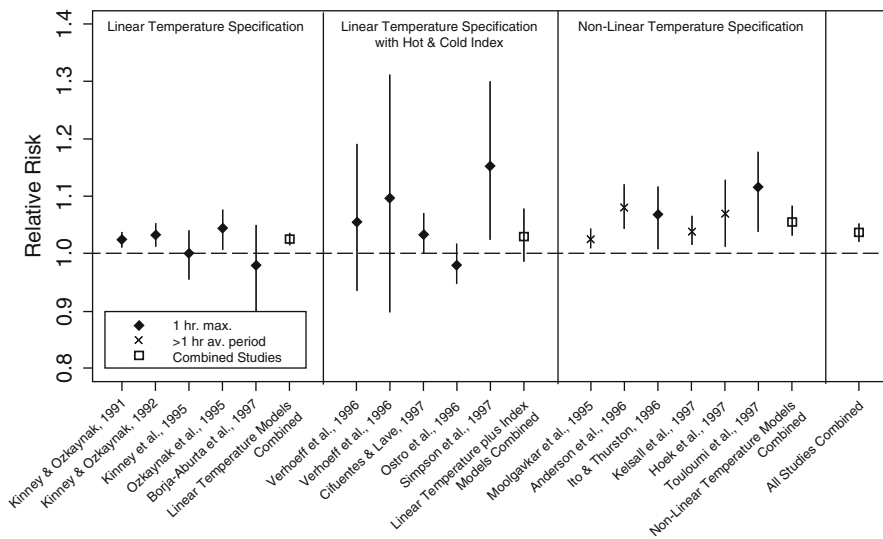


Fig. 8.2 Association between short-term ozone exposure and risk of mortality per 100 ppb increase in 1-h maximum ozone. Note: The *points* reflect central estimates of individual studies. The *open squares* reflect central estimates based on combining studies. The *vertical lines* reflect 95 % confidence intervals (From Thurston, G. D.; Ito, K. Epidemiological studies of acute ozone exposures and mortality. *J. Exposure Anal. Environ. Epidemiol.* 2001;11: 286–294, with permission)

estimates. These findings may relate to differences in baseline healthcare status, access to health care, or exposure patterns. However, the impact of population characteristics on ozone effect estimates is not fully understood. Findings on socioeconomic status and effect modification of short-term ozone associations are not consistent across the few studies that have investigated this issue. In Mexico City, socioeconomic status did not demonstrate clear patterns for ozone and mortality associations [46].

Overall, there is substantial and growing body evidence on acute adverse effects of O_3 , and it can be concluded that robust associations have been identified between various measures of daily O_3 concentrations and increased risk of mortality. Further, the scientific evidence covers a variety of study designs and locations, and studies have consistently demonstrated an acute mortality effect of ozone that is not confounded by particulate matter.

Long-Term Exposure Mortality Effects of O_3

A limited number of epidemiologic studies have assessed the relationship between long-term exposure to O_3 and mortality. While the 2006 O_3 AQCD concluded that an insufficient amount of evidence exists “to suggest a causal relationship between chronic O_3 exposure and increased risk for mortality in humans” [31], more recent evidence specifically points to a relationship between long-term ozone exposure and an increased risk of respiratory mortality.

In the Harvard Six Cities Study [19], adjusted mortality rate ratios were examined in relation to long-term mean O₃ concentrations in six cities: Topeka, KS; St. Louis, MO; Portage, WI; Harriman, TN; Steubenville, OH; and Watertown, MA. Mortality rate ratios were adjusted for age, sex, smoking, education, and body mass index. Mean O₃ concentrations from 1977 to 1985 ranged from 19.7 ppb in Watertown to 28.0 ppb in Portage. Long-term mean O₃ concentrations were not found to be associated with mortality in the six cities. However, the authors noted that “The small differences in ozone levels among the (six) cities limited the power of the study to detect associations between mortality and ozone levels.” In addition, while total and cardiopulmonary mortality were considered in this study, respiratory mortality was not specifically considered.

In a subsequent large prospective cohort study of approximately 500,000 US adults, Pope et al. [20] examined the effects of long-term exposure to air pollutants on mortality (American Cancer Society, Cancer Prevention Study II). All cause, cardiopulmonary, lung cancer, and all other cause mortality risk estimates for long-term O₃ exposure are shown in Fig. 8.1. While no consistently significant positive associations were observed between O₃ and mortality, the mortality risk estimates were larger when analyses considered more accurate exposure metrics, rising when the entire period was considered compared to analysis using just the start of the study period, and becoming marginally significant when the exposure estimates were restricted to the summer months (July–September), especially when considering cardiopulmonary deaths.

In the more recent extended follow-up analysis of the ACS cohort [47], cardiopulmonary deaths were subdivided into respiratory and cardiovascular, separately, as opposed to combined in the Pope et al. [20] work. This analysis utilized the ACS cohort with data from 1977 through 2000 (mean O₃ concentration ranged from 33.3 to 104.0 ppb). In two-pollutant models, PM_{2.5} was associated with the risk of death from cardiovascular causes, whereas ozone was associated with the risk of death from respiratory causes. Exposure to O₃ was positively associated with risk of death from respiratory causes. The relative risk of death from respiratory causes=1.040 (95 % confidence interval, 1.010–1.067) was found to be associated with an increment in ozone season (April 1–September 30) concentration of 10 ppb. The association of ozone with risk of death from respiratory causes was insensitive to adjustment for confounders and to the type of statistical model. Overall, this analysis strongly suggests that, while long-term exposure to PM_{2.5} increases risk of cardiac death, long-term exposure to O₃ is specifically associated with an increased risk of respiratory death.

Ancillary Health Benefits of Climate Change Mitigation

Framework of Climate Mitigation Co-benefits Assessment

Figure 8.3 describes the relationships among the health consequences of climate change and air quality policies and the general framework of how these responses can be assessed. Air quality policies are routinely evaluated in terms of the

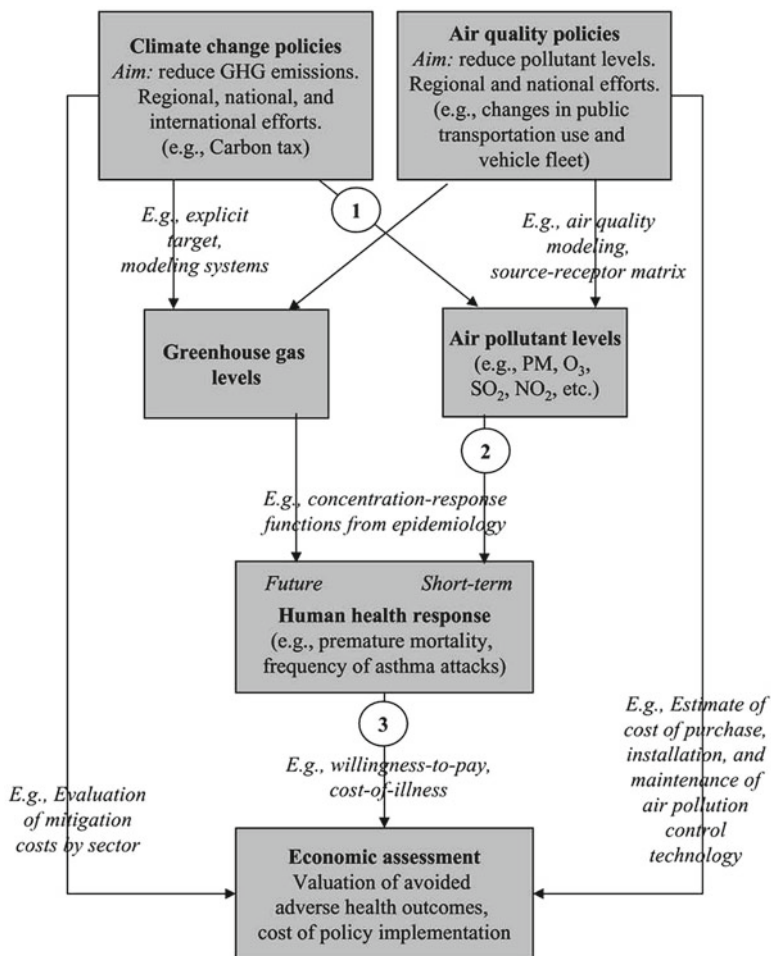


Fig. 8.3 Framework of air pollution co-benefit estimation (From Bell, M.L., Dominici, F. Effect modification by community characteristics on the short-term effects of ozone exposure and mortality in 98 US communities. *Am J Epidemiol* 2008;167:986–997, with permission)

estimated health outcomes avoided and their economic impact [7, 48]. However, assessment of the health impacts of GHG strategies often considers only consequences in the far future (i.e., left side of Fig. 8.3), without integration of the short-term benefits of related policies [49]. Well-informed public health and environmental strategies require full consideration of consequences, including co-benefits and potential ancillary harms.

A broad array of tools to evaluate the health-related ancillary costs and benefits of climate change is currently available, and some examples are provided in italics in Fig. 8.3. As described in detail in Bell et al. [50], the general structure for most assessments involves three key steps: (1) estimating changes in air pollutant

concentrations, comparing levels in response to GHG mitigation to concentrations under a baseline “business-as-usual” scenario; (2) estimating the adverse health impacts avoided from reduced air pollution; and (3) for some studies, estimating the monetary benefit from these averted health consequences, often with comparison to the cost of the climate change mitigation measure.

The first step in such a co-benefit analysis is often the development of emissions scenarios and information regarding how emissions translate into pollutant concentrations, such as with air quality modeling systems. The second step employs concentration-response functions from existing epidemiological studies on ambient air pollution and health. The third stage utilizes a variety of techniques to translate health benefits into monetary terms. Potential additional steps include sensitivity analysis, such as applying multiple climate change scenarios or concentration-response functions for health effects.

Studies of Health and Air Pollution Benefits and Costs of Climate Change Mitigation

A variety of studies have been conducted to estimate the health and air pollution ancillary benefits and costs from GHG reduction, with a wide range of methods and study areas. Energy scenarios, emission inventories, and global change and regional air quality modeling systems have been linked to estimate the short-term incremental changes in public health and the environment that could result from various GHG mitigation policies [51, 52].

There are now numerous analyses indicating substantial health co-benefits from reductions in PM pollution that can be induced by GHG mitigation measures that involve reductions in fossil fuel combustion emissions. As shown in Fig. 8.4, a study of New York, NY and three Latin American cities identified significant health benefits from reducing GHG, including about 64,000 cases of avoided premature mortality over a 20-year period [53]. Country-wide assessments of GHG mitigation policies on public health have been produced for Canada [54] and selected energy sectors in China [55, 56], under differing baseline assumptions. A synthesis of research on co-benefits and climate change policies in China concluded that China’s Clean Development Mechanism potentially could save 3,000–40,000 lives annually through co-benefits of improved air pollution [57]. Several studies investigated the links between regional air pollution and climate policy in Europe [58–60].

Monetary Valuations of Mitigation Co-benefits

To help decision-makers assess policies with a wide array of health consequences, outcomes are often converted into comparable formats. One used approach is to convert health outcomes into economic terms to allow direct comparison of costs

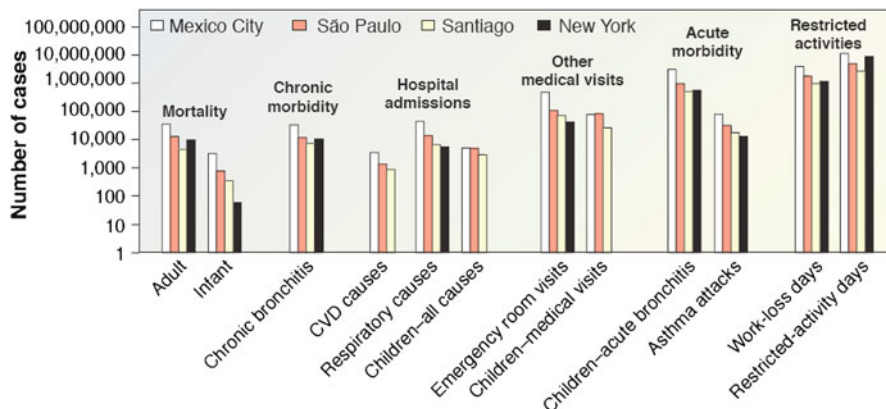


Fig. 8.4 Estimated potential human health benefits from reductions in air pollution associated with implementing GHG mitigation measures in four cities (2001–2020). (From Cifuentes., L, Borja-Aburto, V.H., Gouveia, N., Thurston, G.D., Davis, D.L. Climate change. Hidden health benefits of greenhouse gas mitigation. *Science*. 2001 Aug 17;293(5533):1257–9, with permission)

and benefits. There are several common approaches for economic valuation of averted health consequences (step 3 of Fig. 8.3): Cost of illness (COI); human capital; willingness to pay (WTP) methods; and quality-adjusted life year (QALY) approaches. The COI method totals medical and other out-of-pocket expenditures and has been used for acute and chronic health endpoints. For instance, separate models of cancer progression and respiratory disease were used to estimate medical costs from these diseases over one’s lifetime [61]. However, early attempts to value mortality risk reductions applied the human capital approach, which estimates the “value of life” as lost productivity. This method is generally recognized as problematic and not based on modern welfare economics, where preferences for reducing death risks are not captured. Another limitation is incorporation of racial- or gender-based discrimination in wages. This method assigns value based solely on income, without regard to social value, so unpaid positions such as homemaker and lower paid positions such as social worker receive lower values. Because data are often available for superior alternatives, this approach is rarely used in health benefit studies. WTP generates estimates of preferences for improved health that meet the theoretical requirements of neoclassical welfare economics, by aiming to measure the monetary amount persons would willingly sacrifice to avoid negative health outcomes. Complications arise in analysis and interpretation because changes in environmental quality or health often will themselves change the real income (utility) distribution of society. A valuation procedure that sums individual WTP does not capture individual preferences about changes in income distribution. Another complication is that the value of avoided health risk may differ by type of health event and age. The QALY approach attempts to account for the quality of life lost by adjusting for time “lost” from disease or death, but these estimates may be very insensitive to different severities and types of acute morbidity [62].

Estimating the ancillary public health consequences of GHG policies is a challenging task, drawing upon expertise in economics, emission inventories, air pollution modeling, and public health. However, most assessments to date have focused more heavily on one aspect of the framework (i.e., a portion of Fig. 8.3), whether it be estimation of changes in air pollutant concentrations, health response, or economic analysis.

Results from current ancillary benefits studies may be underestimates due to unquantified benefits, as only a subset of the health consequences from air pollution have adequate exposure-response relationships [14, 63, 64]. A USEPA evaluation of the clean air interstate rule (CAIR) noted numerous unquantified health impacts such as chronic respiratory damage for O₃, loss of pulmonary function for PM, and lung irritation for NO_x [14]. The nature of unquantified effects is continually evolving. Some pollution and health relationships considered unquantifiable by USEPA [7] have since been identified, such as PM air pollution's association with lung cancer [65, 66]. Furthermore, some endpoints may be included in one analysis, but regarded as too uncertain for another, perhaps due to a different study location or differences in researchers' judgment. One approach to addressing health endpoints with uncertain concentration-response functions is to include these effects qualitatively in discussion of unquantified benefits. Another is to incorporate these effects within a sensitivity analysis.

Valuations of mortality risk reductions associated with environmental policies are usually the largest category of benefits, both among health responses and compared to other attributes. For instance, a USEPA analysis of the Clean Air Act estimated a value of \$100 billion annually for reduced premature mortality out of \$120 billion in total benefits, compared to costs of approximately \$20 billion [7]. European and Canadian studies similarly found that mortality risk dominates analysis of pollution reductions [62, 67]. Next to mortality, reductions in the probability of developing a chronic respiratory disease have been estimated to have the highest monetary value, recognizing that values for other types of diseases are sparse.

Recently, the Stern Review [68] addressed a wide range of global benefits and costs associated with climate change, including air pollution co-benefits. Citing a study by the European Environmental Agency, the Review notes that limiting global mean temperature increase to 2 °C would lead to annual savings in the implementation of existing European air pollution control measures of 10€ billion and additional avoided annual health costs of 16–46€ billion. Even larger co-benefits are estimated in developing countries, including via the substitution of modern fuels for biomass. The Stern review also recognizes some of the trade-offs between climate change objectives and local air quality gains. For instance, switching from petrol to diesel reduces carbon dioxide (CO₂) emissions but increases PM₁₀ and NO_x emissions. Other GHG mitigating actions present fewer environmental trade-offs (e.g., reductions in aircraft weight can decrease CO₂ emissions and simultaneously improve local air quality).

Overall, though still a work in progress, the present techniques available for the analyses of the ancillary public health costs and benefits are adequate and appropriate for implementation by those comparing the relative merits and overall value of

various GHG mitigation policies. Estimates of considerable benefits that remain after a variety of sensitivity analyses can alleviate some concerns regarding limitations of individual methods or assumptions. The PM air pollution associated public health changes associated with GHG mitigation strategies should be considered as a key factor in the choice of GHG policies and noted as a potentially major local incentive for programs to reduce GHG emissions.

Implications

The anthropogenic contribution to the climate change pollutants is largely caused by the same activities that cause most air pollution health effects. This indicates that, if a city, state, or nation acts to reduce the combustion of fossil fuels and the air pollution caused by them, it will reap not only the climate change benefits but also the localized health benefits associated with that air pollution reduction. Thus, substantial near-term air pollution associated health benefits of climate control measures may go to the cities and countries that act most vigorously to control their combustion emissions of GHG. These local and near-term health “co-benefits” of reductions in the air pollution from fossil fuel combustion should be considered in the overall analysis, including economic consequences, for climate change mitigation measures.

References

1. Swart R, Amann M, Raes F, Tuinstra W. A good climate for clean air: linkages between climate change and air pollution: an editorial essay. *Clim Change*. 2004;66:263–9.
2. Thurston GD. Air pollution, human health, climate change and you. *Thorax*. 2007;62:748–9.
3. Walsh MP. Ancillary benefits for climate change mitigation and air pollution control in the world’s motor vehicle fleets. *Annu Rev Public Health*. 2008;29:1–9.
4. Haines A, Smith KR, Anderson D, et al. Policies for accelerating access to clean energy, improving health, advancing development, and mitigating climate change. *Lancet*. 2007;370:1264–81.
5. Smith KR, Haigler E. Co-benefits of climate mitigation and health protection in energy systems: scoping methods. *Annu Rev Public Health*. 2008;29:11–25.
6. Smith KR, Jerrett M, Anderson HR, et al. Public health benefits of strategies to reduce greenhouse-gas emissions: health implications of short-lived greenhouse pollutants. *Lancet*. 2009;374(9707):2091–103.
7. USEPA. The benefits and costs of the Clean Air Act 1990 to 1999. Washington, DC: USEPA; 2010. EPA-410-R-99-001.
8. Thurston GD, Ito K, Kinney PL, Lippmann M. A multi-year study of air pollution and respiratory hospital admissions in three New York State metropolitan areas: results for 1988 and 1989 summers. *J Expo Anal Environ Epidemiol*. 1992;2(4):429–50.
9. Kinney PL, Ito K, Thurston GD. A sensitivity analysis of mortality/PM10 associations in Los Angeles. *Inhal Toxicol*. 1995;7:59–69.
10. Thurston GD. A critical review of PM10-mortality time-series studies. *J Expo Anal Environ Epidemiol*. 1996;6:3–22.

11. Schwartz J. Health effects of air pollution from traffic: ozone and particulate matter. In: Fletcher T, McMichael AJ, editors. *Health at the crossroads: transport policy and urban health*. New York, NY: Wiley; 1997.
12. Dominici F, Peng RD, Bell ML, et al. Fine particulate air pollution and hospital admissions for cardiovascular and respiratory diseases. *J Am Med Assoc*. 2006;295(10):1127–34.
13. Samoli E, Peng R, Ramsay T, et al. Acute effects of ambient particulate matter on mortality in Europe and North America: results from the APHENA study. *Environ Health Perspect*. 2008;116(11):1480–6.
14. USEPA. *Regulatory impact analysis for the final clean air interstate rule*. Washington, DC: USEPA; 2005. EPA-452/R-05-002.
15. Bell ML, Dominici F, Samet JM. Time-series of particulate matter. *Annu Rev Public Health*. 2004;25:247–80.
16. Peters A, Dockery DW, Muller JE, Mittleman MA. Increased particulate air pollution and the triggering of myocardial infarction. *Circulation*. 2001;103(23):2810–5.
17. Dominici F, McDermott A, Daniels M, Zeger SL, Samet JM. Revised analyses of the National Morbidity, Mortality, and Air Pollution Study: mortality among residents of 90 cities. *J Toxicol Environ Health A*. 2005;68:1071–92.
18. Ozkaynak H, Thurston GD. Associations between 1980 U.S. mortality rates and alternative measures of airborne particle concentration. *Risk Anal*. 1987;7:449–60.
19. Dockery DW, Pope III CA, Xu X, et al. An association between air pollution and mortality in six U.S. cities. *N Engl J Med*. 1993;329(24):1753–9.
20. Pope III CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. Lung cancer, cardiopulmonary mortality and long-term exposure to fine particulate air pollution. *JAMA*. 2002;287(9):1132–41.
21. Eftim SE, Samet JM, Janes H, McDermott A, Dominici F. Fine particulate matter and mortality: a comparison of the six cities and American Cancer Society cohorts with a Medicare cohort. *Epidemiology*. 2008;19(2):209–16.
22. Chen H, Goldberg MS, Villeneuve PJ. A systematic review of the relation between long-term exposure to ambient air pollution and chronic diseases. *Rev Environ Health*. 2008;23(4):243–97.
23. Bell ML, Dominici F, Ebisu K, Zeger SL, Samet JM. Spatial and temporal variation in $PM_{2.5}$ chemical composition in the United States for health effects studies. *Environ Health Perspect*. 2007;115(7):989–95.
24. Thurston GD, Ito K, Mar T, et al. Workgroup report: workshop on source apportionment of particulate matter health effects—intercomparison of results and implications. *Environ Health Perspect*. 2005;113(12):1768–74.
25. Bell ML, Ebisu K, Peng RD, Samet JM, Dominici F. Hospital admissions and chemical composition of fine particle air pollution. *Am J Respir Crit Care Med*. 2009;179(12):1115–20. Epub 2009 Mar 19.
26. Lall R, Ito K, Thurston GD. Distributed lag analyses of daily hospital admissions and source-apportioned fine particle air pollution. *Environ Health Perspect*. 2010;119(4):455–60.
27. Ito K, Mathes R, Ross Z, Nádas A, Thurston G, Matte T. Fine particulate matter constituents associated with cardiovascular hospitalizations and mortality in New York City. *Environ Health Perspect*. 2011;119(4):467–73.
28. Zhou J, Ito K, Lall R, Lippmann M, Thurston GD. Time-series analysis of mortality effects of fine particulate matter components in Detroit and Seattle. *Environ Health Perspect*. 2011;119(4):461–6.
29. Spira-Cohen A, Chen LC, Kendall M, Lall R, Thurston GD. Personal exposures to traffic-related air pollution and acute respiratory health among Bronx school children with asthma. *Environ Health Perspect*. 2011;119(4):559–65.
30. Ostro B, Lipsett M, Reynolds P, et al. Long-term exposure to constituents of fine particulate air pollution and mortality: results from the California teachers study. *Environ Health Perspect*. 2010;118(3):363–9.

31. U.S. Environmental Protection Agency. Air quality criteria for ozone and related photochemical oxidants. Research Triangle Park, NC: Office of Research and Development; 2006. Report nos. EPA 600/R-05/004aF.
32. Gauderman WJ, McConnell R, Gilliland F, London S, Thomas D, Avol E, Vora H, Berhane K, Rappaport EB, Lurmann F, Margolis HG, Peters J. Association between air pollution and lung function growth in southern California children. *Am J Respir Crit Care Med.* 2000;162(4 Pt 1):1383–90.
33. Gauderman WJ, Gilliland GF, Vora H, Avol E, Stram D, McConnell R, Thomas D, Lurmann F, Margolis HG, Rappaport EB, Berhane K, Peters JM. Association between air pollution and lung function growth in southern California children: results from a second cohort. *Am J Respir Crit Care Med.* 2002;166(1):76–84.
34. Rojas-Martinez R, Perez-Padilla R, Olaiz-Fernandez G, Mendoza-Alvarado L, Moreno-Macias H, Fortoul T, McDonnell W, Loomis D, Romieu I. Lung function growth in children with long-term exposure to air pollutants in Mexico City. *Am J Respir Crit Care Med.* 2007;176:377–84.
35. Mudway IS, Kelly FJ. An investigation of inhaled ozone dose and the magnitude of airway inflammation in healthy adults. *Am J Respir Crit Care Med.* 2004;169:1089–95.
36. Levy JI, Carrothers TJ, Tuomisto JT, Hammitt JK, Evans JS. Assessing the public health benefits of reduced ozone concentrations. *Environ Health Perspect.* 2001;109:1215–26.
37. Stieb DM, Judek S, Burnett RT. Meta-analysis of time-series studies of air pollution and mortality: effects of gases and particles and the influence of cause of death, age, and season. *J Air Waste Manage Assoc.* 2002;52:470–84.
38. Thurston GD, Ito K. Epidemiological studies of acute ozone exposures and mortality. *J Expo Anal Environ Epidemiol.* 2001;11:286–94.
39. World Health Organization. Meta-analysis of time-series studies and panel studies of particulate matter (PM) and ozone (O₃): report of a WHO task group. Copenhagen, Denmark: WHO Regional Office for Europe. 2004. Document no. EUR/04/5042688. <http://www.euro.who.int/document/E82792.pdf>. Accessed 18 Nov 2004.
40. Bell ML, Dominici F, Samet JM. A meta-analysis of time-series studies of ozone and mortality with comparison to the national morbidity, mortality, and air pollution study. *Epidemiology.* 2005;16:436–45.
41. Ito K, De Leon SF, Lippmann M. Associations between ozone and daily mortality, analysis and meta-analysis. *Epidemiology.* 2005;16:446–57.
42. Bell ML, McDermott A, Zeger SL, Samet JM, Dominici F. Ozone and short-term mortality in 95 US urban communities, 1987–2000. *J Am Med Assoc.* 2004;292:2372–8.
43. Gryparis A, Forsberg B, Katsouyanni K, Analitis A, Touloumi G, Schwartz J, Samoli E, Medina S, Anderson HR, Niciu EM, Wichmann HE, Kriz B, Kosnik M, Skorkovsky J, Vonk JM, Dörnbudak Z. Acute effects of ozone on mortality from the “air pollution and health: a European approach” project. *Am J Respir Crit Care Med.* 2004;170:1080–7.
44. Schwartz J. How sensitive is the association between ozone and daily deaths to control for temperature? *Am J Respir Crit Care Med.* 2005;171:627–31.
45. Bell ML, Dominici F. Effect modification by community characteristics on the short-term effects of ozone exposure and mortality in 98 U.S. communities. *Am J Epidemiol.* 2008;167:986–97.
46. O’Neill MS, Loomis D, Borja-Aburto VH. Ozone, area social conditions, and mortality in Mexico City. *Environ Res.* 2004;94:234–42.
47. Jerrett M, Burnett RT, Pope III CA, Ito K, Thurston G, Krewski D, Shi Y, Calle J, Thun M. The contribution of long-term ozone exposure to mortality. *N Engl J Med.* 2009;360:108595.
48. USEPA. The benefits and costs of the Clean Air Act 1970 to 1990. Washington, DC: USEPA; 1997.
49. Ebi K, Mills DM, Smith JB, Grambsch A. Climate change and human health impacts in the United States: an update on the results of the U.S. national assessment. *Environ Health Perspect.* 2006;114:1318–24.

50. Bell ML, Davis DL, Cifuentes LA, Krupnick AJ, Morgenstern RD, Thurston GD. Ancillary human health benefits of improved air quality resulting from climate change mitigation. *Environ Health*. 2008;7:41.
51. Burtraw D, Toman M. The benefits of reduced air pollutants in the U.S. from greenhouse gas mitigation policies. Washington, DC: Resources for the Future; 1997. Discussion Paper 98-01-REV.
52. McCarthy JJ, Canziani OF, Leary NA, Dokken DJ, White KS, editors. Climate change 2001: impacts, adaptation & vulnerability. Contribution of working group II to the third assessment report of the intergovernmental panel on climate change. Cambridge: Cambridge University Press; 2001.
53. Cifuentes L, Borja-Aburto VH, Gouveia N, Thurston GD, Davis DL. Assessing the health benefits of urban air pollution reductions associated with climate change mitigation (2000–2020): Santiago, São Paulo, Mexico City, and New York City. *Environ Health Perspect*. 2000;2001(109):S419–25.
54. Last J, Trouton K, Pengelly D. Taking our breath away: the health effects of air pollution and climate change. Vancouver, Canada: David Suzuki Foundation; 1988.
55. Wang X, Smith KR. Near-term health benefits of greenhouse gas reductions: a proposed assessment method and application in two energy sectors of China. Geneva: World Health Organization; 1999. WHO/SDE/PHE/99.1.
56. Cao J, Ho MS, Jorgenson DW. “Co-benefits” of greenhouse gas mitigation policies in China. Washington, DC: Resources for the Future; 2008.
57. Vennemo H, Aunan K, Jinghua F, et al. Domestic environmental benefits of China’s energy-related CDM potential. *Clim Change*. 2006;75:215–39.
58. Alcamo J, Mayerhofer P, Gaudans R, et al. An integrated assessment of regional air pollution and climate change in Europe: findings of the IAR-CLIM project. *Environ Sci Pol*. 2002;5:257–72.
59. van Harmelen T, Bakker J, de Vries B, van Vuuren D, den Elzen J, Mayerhofer P. Long-term reductions in costs of controlling regional air pollution in Europe due to climate policy. *Environ Sci Pol*. 2002;5:349–65.
60. Working Group on Public Health and Fossil Fuel Combustion. Short-term improvements in public health from global-climate policies on fossil-fuel combustion: an interim report. *Lancet*. 1997;350:1341–9.
61. Hartunian NS, Smart CN, Thompson MS. The incidence and economic costs of major health impairments: a comparative analysis of cancer, motor vehicle injuries, coronary heart disease, and stroke. Lexington, MA: Lexington Books; 1981.
62. Miller W, Robinson LA, Lawrence RS, editors. Institute of Medicine (IOM) Committee to evaluate measures of health benefits for environmental, health, and safety regulation. Valuing stratus consulting: air quality valuation model documentation, for Health Canada. Boulder, CO: Stratus Consulting; 1999.
63. Committee on Estimating the Health-Risk-Reduction Benefits of Proposed Air Pollution Regulations, National Research Council. Estimating the public health benefits of proposed air pollution regulations. Washington, DC: National Academies Press; 2002.
64. Voorhees SA. Benefits analysis of particulate matter control programs – a case study of Tokyo. 2005;8(4):311–29.
65. Krewski D, Burnett R, Jerrett M, Pope C, Rainham D, Calle E, Thurston G, Thun M. Mortality and long-term exposure to ambient air pollution: ongoing analyses based on the American Cancer Society cohort. *J Toxicol Environ Health A*. 2005;68:1093–109.
66. Filleul L, Rondeau V, Vandentorren S, Le Moual N, Cantagrel A, Annesi-Maesano I, et al. Twenty five year mortality and air pollution: results from the French PAARC survey. *Occup Environ Med*. 2005;62:453–60.
67. Bickel P, Friedrich RE. ExternE, externalities of energy, methodology 2005 update. Luxembourg: European Commission; 2005.
68. Stern N. The economics of climate change: the stern review. New York, NY: Cambridge University Press; 2007.

Further Reading

- Ammann CM, Washington WM, Meehl GA, Buja L, Teng H. Climate engineering through artificial enhancement of natural forcings: magnitudes and implied consequences. *J Geophys Res.* 2010;115(22), D22109.
- Anderson GB, Bell ML. Does one size fit all? The suitability of standard ozone exposure metric conversion ratios and implications for epidemiology. *J Expo Sci Environ Epidemiol.* 2010;20(1):2–11.
- Ban-Weiss GA, Caldeira K. Geoengineering as an optimization problem. *Environ Res Lett.* 2010;5(3), 034009.
- Bell ML, Peng RD, Dominci F. The exposure-response curve for ozone and risk of mortality and the adequacy of current ozone regulations. *Environ Health Perspect.* 2006;114:532–6.
- Bell ML, Kim JY, Domincini F. Potential confounding of particulate matter on the short-term association between ozone and mortality in multisite time-series studies. *Environ Health Perspect.* 2007;115:1591–5.
- Chen LH, Knutsen SF, Shavlik D, et al. The association between fatal coronary heart disease and ambient particulate air pollution: are females at greater risk? *Environ Health Perspect.* 2005;113:1723–9.
- Cifuentes L, Borja-Aburto VH, Gouveia N, Thurston GD, Davis DL. Climate change. Hidden health benefits of greenhouse gas mitigation. *Science.* 2001;293(5533):1257–9.
- Goldstein B, Kobos PH, Brady PV. Unintended consequences of atmospheric injection of sulphate aerosol. SAND2010-7571. Albuquerque, NM: Sandia National Laboratories; 2010.
- Intergovernmental Panel on Climate Change (IPCC). Climate change 2007: the physical science basis. Summary for policymakers. Contribution of working group I to the fourth assessment report of the intergovernmental panel on climate change. Geneva: IPCC Secretariat; 2007.
- Kious WJ, Tilling RI. This dynamic earth: the story of plate tectonics. Washington, D.C.: USGS, USGPO; 1996.
- Krupnick A, Davis D, Thurston GD. The ancillary health benefits and costs of GHG mitigation: scope, scale, and credibility. In: Workshop on assessing the ancillary benefits and costs of greenhouse gas mitigation strategies. Washington, DC: Organization for Economic Cooperation and Development (OECD), Intergovernmental Panel on Climate Change (IPCC); 2000.
- Miller W, Robinson LA, Lawrence RS, editors. Valuing health for regulatory cost-effectiveness analysis. Washington, DC: National Academies Press; 2006.
- Moore JC, Jevrejeva S, Grinsted A. Efficacy of geoengineering to limit 21st century sea-level rise. *Proc Natl Acad Sci USA.* 2010;107(36):15699–703. Epub 2010 Aug 23.
- National Academy of Science (NAS). Radiative forcing of climate change: expanding the concept and addressing uncertainties. Washington, DC: National Research Council, Committee on Radiative Forcing Effects on Climate, Climate Research Committee; 2005.
- Ostro B, Feng WY, Broadwin R, Green S, Lipsett M. The effects of components of fine particulate air pollution on mortality in California: results from CALFINE. *Environ Health Perspect.* 2007;115(1):13–9.
- Rasch PJ, Tilmes S, Turco RP, et al. An overview of geoengineering of climate using stratospheric sulphate aerosols. *Philos Trans A Math Phys Eng Sci.* 2008;366:4007–37.
- Tuck AF, Donaldson DJ, Hitchman MH, et al. On geoengineering with sulphate aerosols in the tropical upper troposphere and lower stratosphere. *Clim Change.* 2008;90:315–31.
- Voorhees AS, Sakai R, Araki S, Sato H, Otsu A. Benefits analysis of nitrogen dioxide control programmes: a case-study of Chiyoda-ku, Tokyo. *J Environ Plann Manag.* 2001;44:149–65.

Chapter 9

Asthma, Hay Fever, Pollen, and Climate Change

Anthony M. Szema

Abstract 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). Hot weather generates more pollen from plants. Cold weather is associated with asthma emergency room visits in New York City. More pollen causes more disease, not only allergically induced, but also non-allergic, since, ragweed, for example, produces 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. Clean Air Act enforcement may reduce sources of anthropogenic heat.

Keywords Asthma and climate change • Hay fever and climate change • Pollen and climate change • Allergies and climate change • Climate change and asthma, hay fever, and pollen

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

A.M. Szema, M.D., F.C.C.P., F.A.C.P. (✉)
Department of Medicine and Surgery, Stony Brook University School of Medicine,
Three Village Allergy & Asthma, PLLC, 3771 Nesconset Highway, Suite 105,
South Setacket, NY 11790, USA

New York State Center for Biotechnology, Stony Brook, NY 11794, USA
e-mail: aszema22@hotmail.com

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 room visits in New York City [3]. More pollen causes more disease, not only allergically induced, but also non-allergic, since, ragweed, for example, produces 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].

Effect of Climate Change on Human Health

Does climate change have two 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 possibly, mortality.

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].

P.J. Beggs has reviewed work of other investigators, who have noted an association between increases in carbon dioxide (CO₂) concentration in ambient air and increases in pollen, even irrespective of temperature. Ziska and Caulfield determined that ragweed pollen (*Ambrosia artemisiifolia* L.) production increased from preindustrial times to the present. Wayne identified a twofold increase in atmospheric CO₂ concentration led to a significant increase in ragweed pollen production.

Ziska noted a CO₂ temperature gradient between rural and urban areas such that the higher CO₂ concentration and air temperature of the urban area resulted in ragweed in air at higher concentrations. In another study, *Ambrosia* taxa actually decreased, while concentrations of Juniper tree pollen (*Juniperus*), *Quercus*, *Carya*, and *Betula* (birch tree pollen) increased. Speksma studied *Betula* pollen in five European cities from 1961 to 1993 and found slightly rising trends over this time. Teranishi found that over a 15-year period from 1983 to 1998, Japanese cedar pollen (*Cryptomeria japonica*) significantly correlated between total pollen count in a year and temperature in July the previous year [7].

Not only do increased pollen counts provoke allergic disease, but also the *potency* or *allergenicity* of pollen is concerning. Birch pollen grown at two temperatures differing by 1.1 °C yielded significantly stronger allergenicity in pollen from trees grown at higher temperatures. Hjelmroos found that heterogeneity of

antigenic proteins was more diverse in pollen from the south side of trees, supporting the concept that higher temperature from the south side of trees may modulate this phenomenon.

Longer grass pollen seasons with earlier start dates have been associated with increases in cumulative temperatures over 5.5 °C during winter–early spring (January–March). Emberlin showed that start dates of the birch pollen season advance 6 days over 10 years, for birch pollen, based on changes in spring temperatures in four out of six sites in Europe.

In Italy, from 1981 to 2000, temperature warming was associated with an earlier initiation of the pollen season. In particular, a plant family called Urticaceae had prolongation of its pollen season—critically important, since this is clinically significant in that region. A World Health Organization report concluded that an earlier start and peak of the pollen season is more pronounced in species that start flowering earlier in the year—and the duration of the season is extended in some summer and late flowering species.

In North America, earlier start dates for juniper trees (*Juniperus*) and related taxa *Ulmus* and *Morus* have been studied. Actually, an earlier start time was associated with increasing winter temperatures. Other studies for the Japanese cedar (*C. japonica*) have noted the first date of the pollen season advanced from 1983 to 1998, from mid-March to late February, according to the mean February temperature. Ziska found that higher CO₂ concentrations and air temperature of the urban area resulted in earlier ragweed seasons, compared to rural areas.

Heat May Change Plant and Pollen Distribution at a Given Latitude

Predictions of extending the northern limit of birch by several hundred kilometers and increasing the altitudinal tree line have also been modeled with contraction of the distribution in the south. *Plantago lanceolata*, a common allergen producer benefited from more abundance after experimental studies of climate, soil, fertility, and disturbance, though other species declined or became extinct.

A recent report found that the duration of the ragweed (*Ambrosia* spp.) pollen season has been increasing in recent decades as a function of latitude in North America. These latitudinal effects leading to increasing season length were associated with a delay in first frost of the fall season and lengthening of the frost-free period. A significant increase in the length of the ragweed pollen season was found between 13 and 27 days at latitudes above 44°N since 1995.

These data support the Intergovernmental Panel on Climate Change Projections, which notes enhanced warming is a function of latitude. Greater exposure times to seasonal allergens may therefore occur with subsequent effects on human health. For example, 10 % of the US population is estimated to be ragweed-sensitive.

As an explanation for the increased prevalence of allergic disease worldwide over the past 30 years, ragweed, is an important factor, and climate change is a plausible etiologic agent.

Shoot Growth, Water Use Efficiency, and Phenological Phases (Leaf Unfolding, Needle Flush, Flowering) Potentially Affected by Warming

Increased CO₂ concentration near perennial ryegrass seedlings leads to increased shoot growth and increased biomass. In addition, Lindroth showed that carbon to nitrogen ratios, or C:N, as well as starch concentrations and condensed tannin, of paper birch, significantly increased in response to increased CO₂. Other variables that increase include below-ground mass, carbon, nitrogen, hexose sugar, gas exchange properties, water use efficiency, and total mass.

The growing season can start earlier with warmth. In Europe, *Betula pubescens* and *Quercus robur* have such phenological phases: (1) leaf unfolding, (2) needle flush, and (3) flowering spring events. These advanced by 6.3 days, while autumn events were delayed by 4.5 days, resulting in a longer growing season lengthening by 10.8 days since the 1960s. This has been called the “anthropogenic greenhouse effect.” Other studies have shown that elevated CO₂ concentration decreased seed weight, increased germination percentage and rate, and increased seedling size for the progeny of *P. lanceolata*. Both higher CO₂ concentration and air temperature of the urban area led to ragweed plants which grew quicker and generated more above-ground biomass than rural areas.

While this chapter has a focus on pollen, it is possible that other sources of aero-allergens such as cockroach (*Blattidae*) may proliferate in the presence of higher temperatures, since they would be able to survive passage between buildings, thwarting extermination efforts [7].

Worsening Asthma and Allergies

Our research group determined that for the latter question—cold weather and asthma—atmospheric temperature indeed impacts New York City asthma emergency room 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 μm-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 room visit data from NYC.GOV from 2000 to 2007. Relations among these data were determined based on correlation coefficients. There was a reverse relationship between asthma indicators and temperature, i.e., extremely low temperature was associated with higher asthma discharge rates in the Bronx [3].

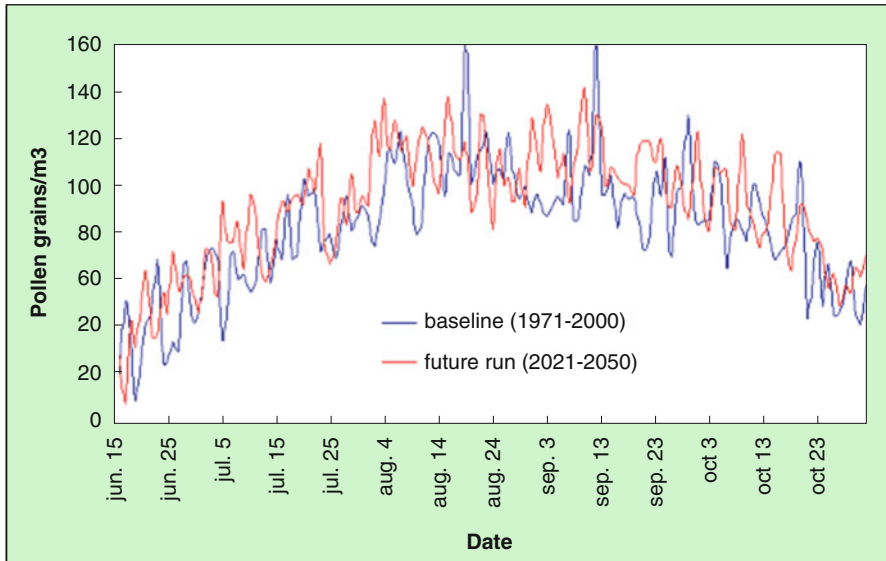


Fig. 9.1 Ragweed pollen counts over time in Europe. Ragweed pollen counts increase in season and are predicted to rise in Europe over time. The hotter summer months in August/September are associated with more release of pollen. From the Climate Change and Variability: Impact on Central and Eastern Europe website coordinated by the Max Planck Institute of Meteorology (From Pálvölgyi, T., Szabó, É. and Makra, L., 2009. Ragweed Impact Case Study (in: Evaluation and assessment of various impacts in the framework of CLAVIER Project—Climate Change and Variability: Impact on Central and Eastern Europe, www.clavier-eu.org). Scientific Report, Env-in-Cent Ltd., Budapest, <http://www.clavier-eu.org/?q=node/880>, with permission)

Does More Pollen Cause More Disease?

Prolonged pollen seasons may increase the duration of human exposure to aeroallergens and may increase the risk of allergic sensitization. In those persons with allergic disease, a longer pollen season may increase the duration of allergy symptoms. Higher concentrations of atmospheric pollen may also increase the severity of allergic symptoms [2].

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 due to 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. Figure 9.1 shows that ragweed pollen counts increase in season and are predicted to increase over time, supporting the possibility that more pollen will cause more disease in the future.

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 %). Chronic urticaria, though present in this series, likely was unrelated to seasonal pollen. 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 towards 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 [8].

In Japan, cypress and cedar plantations account for ¼ of the population suffering from hay fever in the spring. Kouji Murayama, quoted in *Nature* points to global warming as linking summer temperatures to the amount of pollen produced the following spring and that these data already provide the basis for pollen forecasts [9]. Tokyo's average yearly temperature has increased by 3 °C since 1890 and is predicted to rise up to 3.5 °C by the end of the century. If this is indeed the case, then it is possible that the number of hay fever sufferers will rise by 40 % by the year 2050. Thus, global warming has the potential to magnify an already entrenched, important health problem in Japan.

Global warming may be additive with higher levels of industrial carbon dioxide and diesel exhaust. However, even economic factors may intensify the problem, since unmaintained cedar and cypress plantations allow trees to mature to their prime pollen-producing age. A solution would be to replace these pollen-producing trees with pollen-free cypress and cedar, an approach which may take decades to implement.

Pollen types are temporally related seasonally. In the Northeast US, tree pollen sheds in the spring, grass pollen is released during summer, and weed typically is disbursed in late summer (classically taught as August 15, especially with ragweed).

These large pollen grains, about 5 µm in size or larger, are deemed too small to be respirable, and rather deposit in the ocular conjunctiva to cause allergic conjunctivitis—watery, itchy, red eyes, are sequellae. These pollen grains also contact the nasal mucosa and trigger allergic rhinitis or hay fever via an IgE-mediated mechanism, in those allergically sensitized.

For ragweed pollen, even in those not allergically sensitized, ROS are produced to incite inflammation. Runny nose, itchy nose, postnasal drip, repetitive sneezing, stuffiness/congestion, and dry cough are cardinal symptoms.

Physical exam signs related to histamine release include allergic shiners (dark, puffy eyes from histamine release), Dennie–Morgan lines (lines below the eyelid

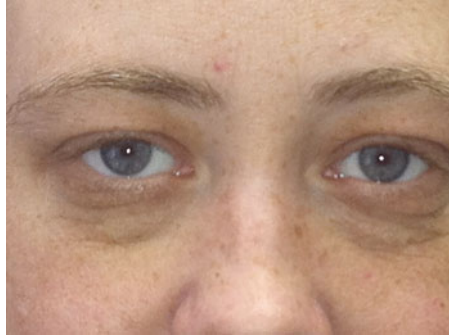


Fig. 9.2 Cardinal physical examination signs of atopic disease. Allergic shiners in a 34-year-old pregnant woman. The swollen dark eyelids are from histamine release. The patient appears tired despite many hours of sleep. The physical appearance makes her appear older than her stated age. Dennie–Morgan lines are *horizontal lines* across *eyelids*. A nasal crease is the horizontal band across the bridge of the nose. The nasal crease is caused by upward rubbing of the nose in itchy patients. The physical act of upward rubbing of the nose is called the “allergic salute,” which is responsible for the nasal crease. They should rub downward to prevent this permanent sign. This patient was admitted with throat closure and uvular swelling after inhaling hyacinth pollen at her house at the onset of the spring vernal equinox (Courtesy of Shauna McCleary, Stony Brook Allergy & Asthma, Stony Brook, NY)

from histamine release), nasal crease (from rubbing the nose in an upward fashion leading to bent cartilage in the nose), and the nasal salute (rubbing one’s nose with an upward movement of the hand) (Fig. 9.2). Since pollen grains are too large to be respirable, they do not directly reach the bronchi. However, pollen-induced asthma does occur and manifests late in the season and after it ends.

For grass, in particular, the English have noted “thunderstorm asthma” when respirable particles become airborne during gusts of wind. The reason for the lack of immediate asthma symptoms may be due to the location of allergens in pollen. Important allergens are on the outside of the cell membrane called the exine. They are actually not produced by the pollen cell itself but are “stuccoed” onto the exine by other cells of the male flower. Considerable amount of allergens remain behind for weeks after pollen is shed. Also, allergens extracted from pollen by raindrops may lead to airborne dust particles after drying. So, asthma symptoms may begin after hay fever symptoms and persist longer [10].

Figure 9.3 describes the cascade of pollen inhalation leading to allergic asthma. Pollen is inhaled and the protein antigens in pollen are engulfed by antigen-presenting cells such as the macrophage, which degrades protein into peptides. The peptides are shuttled to the macrophage surface and presented to an activated T cell in the context of major histocompatibility complex type II. The T cell, when activated, engages a B cell via CD40 ligand (also known as CD154) interactions with CD40, using accessory molecules CD80 on the T cell and CD86 on the B cell. Depending on the cytokine environment near these cells, for example, if IL-4 is

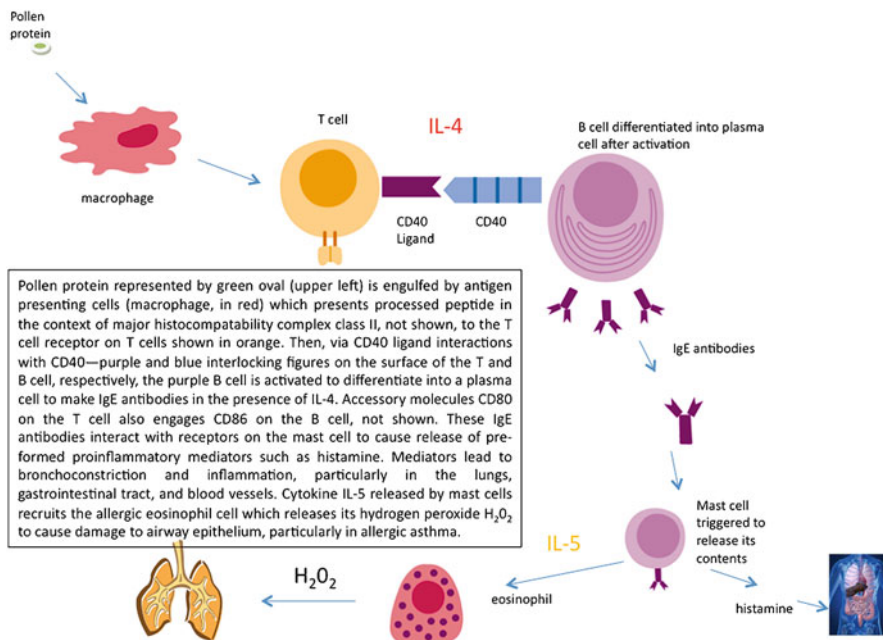


Fig. 9.3 The allergic cascade simplified from pollen inhalation to disease

present, then the B cell will differentiate into a plasma cell and class switch from IgM to IgE in order to make immunoglobulin molecules IgE, the allergic antibody. These IgE allergic antibodies bind to IgE receptors in mast cells, and when two IgE molecules are in close proximity, they dimerize and engage the mast cell to release its content of pro-inflammatory pre-formed mediators, such as histamine, which causes clinical airway constriction and gastrointestinal symptoms, and vascular inflammation. Generation of pro-inflammatory cytokines such as IL-5 will recruit the eosinophil allergic cell to release its hydrogen peroxide, which damages airway epithelium.

Is There an Additional Effect of Non-allergenic Air Pollution Acting in Concert with Aeroallergens?

Ragweed has physicochemical properties to release ROS to cause disease; a two-hit hypothesis may come into play for those allergic to ragweed. For those not sensitized, ROS may play a role solely; for those with allergies, ROS plus IgE-mediated disease would enhance the inflammation [11].

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 [12]. 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 asthmatics. 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 PM2.5, nitrogen dioxide, and ozone [5]. Diesel exhaust is particularly troublesome, since it increases the production of allergic IgE antibodies [13]. Ozone levels have been modeled to track asthma emergency room visits and are predicted to be associated with increased pediatric emergency room visits for asthma for the next decade. Changing levels of ozone could lead to a 7.3 % increase in asthma-related emergency room visits by children, ages 0–17.

This asthma and ozone study, led by Perry Sheffield, MD at Mount Sinai School of Medicine, used regional and atmospheric chemistry models. Regional climate and air quality information was linked to New York State Department of Health records of pediatric, asthma-related emergency room visits in 14 counties that are part of the New York City metropolitan area. They simulated ozone levels for June through August for 5 consecutive years in the 2020s, and compared them with 1990s levels. They then determined a median increase of 7.3 % in ozone-related asthma emergency department visits, with increases ranging from 5.2 to 10.2 % per county [14].

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 [15]. An example of melting of even glacial ice over time is seen in Figs. 9.4 and 9.5, which show Hubbard Glacier, Alaska in 1986, the last year ice reached “the gap” to land. In 2011, the gap is wide and ice floes are melting, shrinking the size of the glacier.

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 chapter, based on letter published in the January 2012 issue of the *Journal of Occupational and Environmental Medicine*, I reaffirm my position [5, 16].



Fig. 9.4 Hubbard Glacier Alaska circa 1986. In 1986, Hubbard Glacier, Alaska, squeezed the passage between Russell Fiord (background) and Disenchantment Bay (foreground) in this photo taken the last time Hubbard “galloped” and closed the passage (From the US Forest Service public website. http://www.fs.fed.us/r10/tongass/forest_facts/photogallery/hubbard_photos.html)



Fig. 9.5 Hubbard Glacier Alaska, July 2011. Hubbard Glacier photo taken by the author, July 2011, aboard the MS Westerdam, Holland America Line. Note the melting ice floes. There is a gap between the glacier and land to the *right*

References

1. Petoukhov V, Semenov VA. A link between reduced Barents-Kara sea ice and cold winter extremes over northern continents. *J Geophys Res.* 2010;115:D21111.
2. 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):4248–51.
3. Chen Y, Forsyth E, Pan K, Chen H, Szema A, Szema K, et al. Atmospheric temperature & pollen counts impact New York City asthma ER visits. *J Allergy Clin Immunol.* 2010;125(2 Suppl 1):Ab208.
4. 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.
5. Ziska LH. Rising atmospheric carbon dioxide and plant biology: the overlooked paradigm. *DNA Cell Biol.* 2008;27(4):165–72.
6. Kizilpinar I et al. Pollen counts and their relationship to meteorological factors in Ankara, Turkey during 2005–2008. *Int J Biometeorol.* 2011;55(4):623–31.
7. Beggs PJ. Impacts of climate change on aeroallergens: past and future. *Clin Exp Allergy.* 2004;34(10):1507–13.
8. 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):189–99.
9. Williams R. Climate change blamed for rise in hay fever. *Nature.* 2005;43:1059.
10. Peden DB, Bush RK. Advances in environmental and occupational respiratory disease in 2010. *J Allergy Clin Immunol.* 2011;127(3):696–700.
11. Dharajiya N et al. Role of pollen NAD(P)H oxidase in allergic inflammation. *Curr Opin Allergy Clin Immunol.* 2008;8(1):57–62.
12. Cecchi L et al. Projections of the effects of climate change on allergic asthma: the contribution of aerobiology. *Allergy.* 2010;65(9):1073–81.
13. Diaz-Sanchez D et al. Diesel exhaust particles induce local IgE production in vivo and alter the pattern of IgE messenger RNA isoforms. *J Clin Invest.* 1994;94(4):1417–25.
14. Sheffield PE et al. Modeling of regional climate change effects on ground-level ozone and childhood asthma. *Am J Prev Med.* 2011;41(3):251–7; quiz A3.
15. D'Amato G et al. 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.
16. Szema AM. Climate change, allergies, and asthma. *J Occup Environ Med.* 2011;53(12):1353–4.

Chapter 10

Dengue Fever and Climate Change

Lauren Cromar and Kevin Cromar

Abstract Dengue fever is a viral, tropical, and subtropical mosquito-borne disease. In recent history, transmission has increased drastically with incidence increasing 30-fold over the past 50 years. Today, an estimated 50–100 million infections occur annually and dengue fever is now ranked as the most important vector-borne viral disease in the world. Once localized to a few areas in the tropics, dengue fever is now endemic in over 100 countries. Population growth, unplanned and uncontrolled urbanization, and increased travel paired with ineffective vector control, disease surveillance, and inadequate public health infrastructure have been cited as drivers in the recent escalation of cases.

Keywords Dengue fever and climate change • Climate change and dengue fever • Dengue disease • Mosquito-borne disease • Urbanization and dengue fever

Dengue fever is a viral, tropical, and subtropical mosquito-borne disease. In recent history, transmission has increased drastically with incidence increasing 30-fold over the past 50 years [1]. Today, an estimated 50–100 million infections occur annually and dengue fever is now ranked as the most important vector-borne viral disease in the world. Once localized to a few areas in the tropics, dengue fever is now endemic in over 100 countries. Population growth, unplanned and uncontrolled urbanization, and increased travel paired with ineffective vector control, disease

L. Cromar, M.S.

Tahoe Consulting, LLC, 966 E. Eaglewood Loop, North Salt Lake, UT 84054, USA
e-mail: lauren.sobieski@gmail.com

K. Cromar, Ph.D. (✉)

Department of Environmental Medicine, New York University School of Medicine,
57 Old Forge Road, Tuxedo, NY 10987, USA
e-mail: kevin.cromar@nyumc.org

surveillance, and inadequate public health infrastructure have been cited as drivers in the recent escalation of cases [2].

A growing public health concern exists not only due to the increased magnitude of incidence but also to the escalating severity of its complications. A more severe and deadly form of the disease, dengue hemorrhagic fever (DHF), has also become more prominent in recent decades. Currently, an estimated 500,000 people are hospitalized due to DHF each year resulting in 22,000 deaths which occur mainly in children. DHF is now a leading cause of death in children in several countries in Southeast Asia where the virus has an established history of hyperendemicity [3]. In areas such as the Americas where hyperendemicity is a new and growing occurrence, DHF rates have increased dramatically and experts fear that a continuing escalation of DHF incidence and mortality is inevitable [4].

This chapter addresses climate-related factors that are associated with dengue disease today and provide insight into how this growing epidemic may be impacted by a continuing changing climate in the future. Special attention will be paid not only to direct climate impacts on the transmission of the disease but will also delve into how the socioeconomic and cultural changes that are likely to accompany climate change may impact the spread of the disease. The chapter also ends with a discussion of projected climate change associated impacts on dengue fever in several distinct geographic regions around the global.

Dengue Disease

The dengue virus is a member of the family *Flaviviridae* along with West Nile virus and yellow fever virus. The dengue virus has four distinct serotypes, DENV-1, DENV-2, DENV-3, and DENV-4. The genetic variations between serotypes result in variations in the transmissibility and severity of the disease.

Infection from one of the dengue serotypes can cause a range in disease severity from asymptomatic cases to severe and even fatal infections [5]. The most common manifestation of infection is dengue fever. Classical symptoms include a high fever with an abrupt onset accompanied by severe pain in the muscles and joints (thus earning it the name of “breakbone fever”), severe headaches, pain behind the eye, and a rash. Unfortunately, there is no specific drug treatment for dengue fever.

While the adaptive immune response from an initial infection proves to be protective from subsequent infection by the same serotype, it is not protective from infections from any of the three remaining serotypes. In fact, after a brief period of cross protection, antibodies from a previous infection of a different serotype are believed to be major factors in the development of DHF, the more severe form of the disease [6]. This occurs through a phenomenon known as antibody-dependent enhancement [7]. Due to antibody-dependent enhancement, even primary infections in infants can result in the development of DHF if maternal anti-dengue virus antibodies have been supplied by the mother [8]. Several other factors have been identified in triggering the development of DHF including varying degrees of

virulence of the infecting strain and differences in the susceptibility of the host due to age, immune status, race, and other genetic factors [9, 10].

In recent decades, greater movement of dengue serotypes between populations has been fueled in part by increased global travel. Only nine countries had reported DHF previous to 1970, but that number had increased fourfold by 1995 [1].

There is no current vaccine against dengue virus infection. As dengue fever can be caused by four separate serotypes of the virus, an effective vaccine must immunize against all four serotypes to be effective. Reinforcing this necessity is the fact that if a vaccine fails to provide immunity against one of the serotypes, an immunized individual is put at risk of developing DHF via antibody-dependent enhancement. The requirement that four dengue vaccines must be developed and combined in a single vaccine to preclude the development of DHF presents one of the largest challenges in vaccine development. Despite the challenges, many vaccine candidates have been developed and are at various stages of development. As the greatest hope in reducing future dengue fever incidence lies in vaccination, it is hoped that such development efforts may prove successful.

Vectors and Transmission of Dengue

Dengue is transmitted by two mosquito species: primarily by *Aedes aegypti*, and secondarily by *Aedes albopictus*. *A. aegypti* is distributed around the globe in many areas throughout the tropics and subtropics and often invades farther north and south during the warmer summertime months [11, 12]. It is currently at the widest global distribution in its history due in part to increased globalization, international trade, and travel.

A. aegypti exhibits a very high dengue virus infection rate, making it a very competent vector. It is this species that is primarily responsible for the high levels of endemic dengue fever in so many countries and for the explosive outbreaks that occur. *A. aegypti* are not vectors for dengue virus alone, but also carry the yellow fever virus which, despite the existence of an effective vaccine, still causes approximately 200,000 illnesses and 30,000 deaths per year.

A. aegypti has several characteristics and tendencies which make it especially adapted to domestic life and to being an especially effective disease vector. It has almost entirely adapted to urban life, preferring to breed in or around homes in artificial household or yard water containers [13]. Common breeding sites include water storage drums, discarded automobile tires, vases, buckets, flower pots with saucers for water collection, and general trash (such as plastic containers) which can collect rainwater [14].

Adult mosquitoes have a tendency to live within homes and buildings, often taking refuge in the rafters and on the walls at night and feeding on the human inhabitants during the daytime hours [15]. Females are strongly anthropophilic, vastly preferring to feed on humans than on non-human mammals [16]. They are also easily interrupted while feeding and tend to have multiple feedings per completion of each gonotrophic cycle thus allowing for disease transmission to multiple individuals [17].

Despite continued efforts to control *A. aegypti* in a multitude of areas around the globe, increasing dengue epidemics likewise bear witness to their shortcomings and, at times, outright failures. The large-scale control of *A. aegypti* has been stated as being one of the most conspicuous failures in the public health sector [18].

Aedes (Stegomyia) albopictus, a species commonly referred to as the “Asia tiger mosquito” is considered to be a secondary vector for the dengue virus. Like *A. aegypti*, *A. albopictus* has experienced a wide expansion in global range in recent history. The range of *A. albopictus* includes more northern and southern extremes than that of *A. aegypti* [19]. *A. albopictus* also utilizes a wider range of habitats and is more likely to be found in rural and suburban areas. The invasion of *A. albopictus* is theorized to be responsible for the displacement of *A. aegypti* in several locations such as the Southern United States and Brazil [20, 21].

A. albopictus are considered to be less competent dengue vectors for several reasons. They are much less domesticated than *A. aegypti*. The fact that they largely utilize natural breeding habitats and generally fail to become established in well-populated urban areas naturally results in their smaller role in precipitating outbreaks [22]. *A. albopictus* are much less likely to be found indoors. They are opportunistic feeders and do not display the marked preference for feeding on humans as do *A. aegypti* [19]. Evidence also exists that *A. albopictus* has a lower oral receptivity and infection rate for the dengue virus [23].

Climatic Effects on Entomological and Viral Parameters in the Dengue Transmission Cycle

An investigation into the relationship between climate and dengue fever reveals that climatic factors are strong determining factors on many of the biological and mechanical processes which drive dengue transmission. Many of the entomological variables which directly affect the severity of dengue epidemics are highly correlated with factors such as temperature, humidity, and rainfall. Not only do climatic factors primarily determine the range, density, and vector efficiency of *A. aegypti*, but they are also major factors in determining the rates of dengue virus multiplication and transmission. While the impacts of climatic factors on dengue transmission will be discussed specifically for *A. aegypti*, the general principles may be applicable to other mosquito species as well.

Climatic Factors and the A. aegypti Life Cycle

Temperature

Of all the climatic factors that affect the life cycle of *A. aegypti*, and consequently the transmission of dengue fever, the importance of temperature is perhaps the most apparent. The global range of *A. aegypti* is limited both longitudinally and

latitudinally by decreasing temperatures [15]. Beyond limiting *A. aegypti* distribution, temperature is a large factor in determining the population size within that range. Field data has demonstrated the link between temperature and *A. aegypti* density over space and time and it is understood that populations are generally favored with increasing temperatures.

The *A. aegypti* life cycle displays several minimum and maximum temperature survival thresholds. Long-term exposure to temperatures under 10 °C or over 40 °C is generally lethal to eggs [24]. Upon hatching, larval and pupae survival is generally highest between 16 and 36 °C, dropping off steeply at lower and higher temperatures [25]. Adults activity and survival is limited outside of the range of 15–36 °C [26]. In practice, however, *A. aegypti* populations have been found to survive despite extreme temperatures by taking refuge in or around buildings or breeding in large water storage tanks.

The time required to complete each life cycle and the vector population size are also highly temperature-dependent. In most of its range *A. aegypti* completes many life cycles per year, but near the border of its distribution that only 3–4 life cycles can be completed in a year [15]. This difference is explained by temperature impacts on multiple stages throughout the life cycle. For example, as temperatures decrease from 20 °C, females display a large delay in the time between blood meals and oviposition (the laying of eggs). Oviposition rate, number of eggs laid per female per day, is strongly correlated with temperature with females laying about twice as many eggs at 25 °C than at 20 °C and three times as many at 31 °C [15, 27]. The incubation time before eclosion (hatching) is brief at high temperatures, taking only 2 days at 31 °C and becoming longer as temperatures decreases, taking 20 days at 16 °C [24]. Upon eclosion, development through the immature stages is positively correlated with temperature with total time to development taking about 15 days at 20 °C, about 9 days at 25 °C, and about 6 or 7 days above 30 °C [27]. From these examples it is clear that the time required to complete one life cycle is much shorter at higher temperatures.

Relative Humidity

In addition to temperature impacts, the effects of relative humidity on the *A. aegypti* life cycle are also significant. Relative humidity can play a role in evaporative loss of water from smaller containers serving as habitats for immature forms, but the most notable effects are limited to events within the adult and egg stages. Laboratory experiments with varying humidity levels tend to show graduated effects over a wide range of relative humidity levels as opposed to having defined threshold effects.

Under low-humidity conditions females significantly delay oviposition and lay fewer eggs (e.g., an average of 10.6 eggs in 34 % relative humidity compared to 31.02 eggs in 84 % relative humidity over a 19-day period) [28]. Eggs remain viable for 2 months at 42 % humidity, but for twice as long at 88 % humidity [29].

Survival of adults is the most influential aspect of relative humidity in affecting dengue virus transmission rates. Because the latent period of the dengue virus within the mosquito can be fairly long, the lifespan of the mosquito is a critical

factor on whether or not an infected mosquito may be able to transmit the virus before her death. The relationship between humidity and mortality is near linear with large differences existing even between higher levels of humidity [28, 30].

Climatic Factors and Biting Behavior

The role of biting rates in the epidemiology of dengue fever is pivotal. An increase in the biting rate not only increases the probability of a mosquito becoming infected with the dengue virus but also her ability to transmit it. Whereas *A. aegypti* population size is linearly related to its vectoral capacity, biting rates are exponentially related [31]. It is clear that even small increases in the biting rate have the potential to result in relatively large increases in the incidence of dengue fever [32].

The quicker immature *A. aegypti* development time that results from higher temperatures yields smaller adult mosquitoes with lower energy reserves. As this occurs, females need to feed sooner and require more than one blood meal in order to complete a reproductive cycle and biting rates increase [13, 33, 34]. Experiments show a pattern of increasing biting rates with temperature until an optimal temperature around 30–35 °C is reached [35]. Humidity levels have also been found to promote increases in the general activity levels of the *A. aegypti* mosquito and contribute to increased biting rates [15].

Viral replication is also highly affected by temperatures. The length of the extrinsic incubation time, or the period after a mosquito feeds on an infectious host until it is able to transmit the virus, is a large factor in transmission risk. When female mosquitoes bite an infected host, most are not capable of passing on the virus due to the fact that they often die before the extrinsic incubation time has completed [11]. A clear temperature-dependent variation on the extrinsic incubation time of the dengue virus has been found. For example, in mosquitoes infected with DEN-2, the extrinsic incubation time was found to be 7 days at 32 and 35 °C and 12 days at 30 °C. No viral transmission was found at 26 °C [36]. Increased biting rates and shorter extrinsic incubation time may account for seasonal epidemics where a seasonally dynamic *A. aegypti* population has not been found and in epidemics in areas where *A. aegypti* density is below levels considered to be protective [37–39].

Climate-Based, Mechanistic Dengue Transmission Models

As many facets of the growth, survival, reproduction, and behavior of *A. aegypti* and replication of the dengue virus have demonstrated clear relationships with climatic variables, several attempts have been made to mathematically model *A. aegypti* populations and the risk of dengue transmission. Modeling based on several entomological temperature-dependent variables calculates the optimal temperature for maximum growth of *A. aegypti* populations to be 29.2 °C with a range of 27–30 °C

[26, 27]. Using only vector life cycle parameters, a continuation of this model suggests that 28 °C yields the greatest risk of dengue transmission. When modeling includes dynamic viral replication and biting rates, an increasing risk of transmission is found even as temperatures rise beyond the optimal range for *A. aegypti* [40].

Using this methodology, temperature-dependent transmission thresholds in terms of pupae per person have been calculated. These thresholds demonstrate a clear dependency on temperature and illustrate how hotter climates can make effective control efforts very difficult. For example, Bangkok, Thailand has an observed 1.69 pupae per person on average [41, 42]. With an average summer temperature of 29.2 °C, the protective threshold is calculated at 0.29 pupae per person (assuming 33 % seroprevalence), a value which would require control efforts to decrease *A. aegypti* prevalence by 83 %. Mayaguez, Puerto Rico, on the other hand, has a similar average of pupae per person, 1.73, but with a temperature of 26.6 °C, would need only a 40 % effective control effort in order to bring pupae count down to a protective threshold of 1.05 per person [40]. It is interesting that this model predicts the greatest decrease in the transmission threshold between 28 and 30 °C (approximately a fourfold decrease) as this range includes the “dengue season” temperatures of many dengue-endemic countries.

Other modeling based on multiple climate variables suggests that in different climates, *A. aegypti* density (and thus the risk of dengue fever) is most strongly correlated with differing climatic variables. For example, in moist and tropical region, rainfall is already sufficient to support larval survival and oviposition. It is therefore the increases in temperature, and to a smaller extent relative humidity, which cause the largest increases in *A. aegypti* populations. Drier regions, in contrast, exhibit stronger correlations with precipitation than with other factors as moisture is the limiting factor for mosquito survivorship and fecundity.

Lessons Learned from Observational Studies

Observational studies that analyze the relationship between climatic variations and dengue fever incidence in locations around the globe yield evidence to further reveal the relationship between climate and dengue in real-world settings. Dengue and climate have been linked by a multitude of studies across both temporal and spatial scales utilizing various tools and methods of analysis [57].

Because of the multiplicity of factors which contribute to dengue transmission within a community, climatic factors can only account for a portion of transmission-supporting conditions and so cannot explain all the variations in dengue rates. Suitable weather is necessary for sustained dengue transmission, but it is not sufficient. Still, strong evidence linking the two exists that can help to evaluate the effects of projected changes in climate.

Analyses which test the correlation of dengue rates and climatic conditions over time are most common. The climatic factor most commonly associated with dengue incidence and *A. aegypti* population size in time-series studies is rainfall. Rainfall is

highly seasonal in many areas that experience dengue fever. In such areas, *A. aegypti* populations and dengue infections become very low or nonexistent during the dry periods of the year when vector breeding becomes inhibited but increase sharply with the onset of the rainy season. While *A. aegypti* and dengue incidence are often strongly linked to the timing of rainfall, there is less evidence of a strong link to the magnitude of rainfall. In fact, an overabundance of rainfall may decrease the *A. aegypti* population by washing larvae from breeding containers [43].

One caveat in the importance of rainfall is the fact that dry periods can increase household storage of water creating ideal breeding sites in close proximity to humans. Incidentally, water storage containers are one of the most productive vector breeding sites [44, 45]. Thus the vector cycle and dengue fever epidemics may also be induced by a lack of rainfall in areas where the water supply is unreliable or not easily accessible [46]. Epidemics are also commonly linked to periods of moderate to severe drought. In such times, water storage becomes widespread and the emptying and cleaning of water containers is avoided [47–50].

While the relationship between dengue and rainfall is clear in many locations, a strong correlation with temperature is often not found. Some have cited these findings as evidence of a weak link between the two. However, many dengue endemic countries have little intra-annual temperature variation and rarely cool to levels which would inhibit mosquito activity. In such situations, temperature is sufficiently high during the period of highest rainfall, but a lack of rainfall inhibits the reproduction of the vector often during the period of the year when temperatures are the highest. In areas where temperatures seasonally cycle to a level nonconductive to mosquito and viral activities, temperature is likewise found to be highly correlated with dengue incidence [51–53]. Additionally, when the effects of rainfall are taken out of the equation, such as in areas where there is sufficient rainfall year-round for vector breeding or where breeding sites are human-filled, a strong association with temperature is often revealed [54–56].

Further evidence of the importance of temperature can be found in spatially based studies where a strong association with annual temperature values is found more often than with rainfall levels. Humidity levels are less likely to drop to inhibiting levels in dengue-endemic countries and as such, strong associations between relative humidity and dengue incidence are rarely found [57].

In areas with seasonal climates, it has been established that seasonal variations in temperature and rainfall drive the timing of “dengue seasons,” but an understanding of what drives the magnitude of seasonal epidemics year to year is more useful. Of particular use are studies which analyze deviations in climate factors from normal cyclic levels. In Puerto Rico, for example, it was found that while intra-annual fluctuations were driven by rainfall, year-to-year differences were temperature-driven [43, 58]. In Thailand, the timing of dengue was also linked to the timing of rainfall. Annual increases in dengue infection rates, however, were likewise found to be driven by increases in mean temperatures [59]. Similar conclusions have been found in other locations [56].

The El Niño Southern Oscillation (ENSO) phenomenon provides added data on dengue and climate and affords additional clues on the effects of long-term climate

change on dengue incidence and human health. ENSO-related deviations in climate mark a change from normal seasonal patterns allowing researchers to analyze dengue incidence under differing climatic conditions within the same geographical area. Prolonged dry conditions, altered rainfall patterns, and increases or decreases in temperature have been linked to El Niño and La Niña years as have changes in dengue incidence.

In many areas affected by the varying conditions of the ENSO cycle, dengue epidemics have been consistently linked to either El Niño or La Niña conditions. One of the most dramatic examples of ENSO-related epidemics occurred in conjunction with the 1997–1998 El Niño. This El Niño event proved to be the most severe in recorded history and was linked to catastrophic weather and profound widespread health effects, including severe dengue epidemics. In Asia, many countries and urban areas saw the highest rates of dengue-related morbidity and mortality on record [60–63]. Retrospective analyses linked many of these epidemics to the El Niño conditions. In Indonesia, for example, a severe epidemic was preceded by a 2-month delay of the rainy season and was accompanied by elevated temperatures. Analyses found that the high temperatures played a major role in precipitating the explosive outbreaks [64, 65]. The link between ENSO-related warmer temperatures and decreased precipitation with increased dengue rates in Indonesia has been confirmed by decades of weather and dengue data [66].

In Northern South America, a significant relationship was also found between ENSO and dengue epidemics with the increased temperatures and decreased rainfall in El Niño years being linked to epidemics in several countries [66, 67]. In these areas, epidemics begun during drought conditions, highlighting the promoting effect of drought conditions on vector breeding. Dengue and El Niño have also been linked in parts of the Caribbean where it is hypothesized that warmer temperatures allow disease transmission to continue into the next year resulting in earlier and larger epidemics [68]. In the South Pacific Islands, a strong association was found between the dengue epidemics and the Southern Oscillation Index (SOI) during La Niña years when both temperatures and rainfall levels are higher than normal [69, 70].

The strong associations observed with the ENSO clearly illustrate a causal relationship between climate, particularly warmer temperatures, and dengue and warn of the potential harmful effects of an altered and warming climate.

Populations at Risk

The future geographic spread and intensity of dengue risk have been estimated by researchers using general circulation models (GCMs). These projections are derived from the empirical observations of the distribution of dengue and on mathematical equations that model vectoral capacity [71–73]. Vectoral capacity, the rate at which subsequent inoculations arise from a currently infective case, is based on climate (primarily temperature)-dependent vector and viral parameters. Such models project a spread of risk to more temperate latitudes and higher altitudes. In regions that

are already climatically at risk of dengue transmission increased epidemic potential and longer transmission periods are generally projected. As can be expected, the models indicate that the largest expected increases in potential transmission intensity will occur in areas that already support vector mosquito populations, but where insufficient temperatures slow viral replication.

In 1990, 1.5 billion people, almost 30 % of the world's population, lived in at-risk areas. Based on projected population growth alone, this is calculated to increase to 3–5 billion people by the year 2085. If both population growth and climate change are factored in, an estimated 5–6 billion people, or 50–60 % of the projected global population, will be living in at-risk areas (based on a 1 % increase in CO₂ per year) [71].

Whether on the global or local scale, most climate-based models do not incorporate the multiplicity of factors which moderate the actual risk of dengue despite climatic suitability. Demographic, societal, and public health factors play a pivotal role in contributing to dengue transmission in some areas and eliminating it in others, even if a vector population is present. The availability of reliable piped water, which eliminates the need for water storage, has been cited as one of the largest protective factors. Factors which limit contact between vectors and hosts such as well-sealed homes with air conditioning or screens, widespread automobile use, and a population which spends most of its time indoors further prohibit transmission in wealthier areas. The hallmarks of an area with high risk of dengue include lower socioeconomic status, high population density, low-quality housing, lack of waste removal services, lack of health services, and poor vector control. In endemic countries, transmission intensity is also largely influenced by the immunological state of the population, a factor which will likely play an increasingly large role as dengue rates continue to rise.

Trends in Non-climatic Risk Factors

Two factors which will greatly affect the distribution and magnitude of dengue incidence across the globe are urbanization and population growth. The global urban population is projected to double by 2050 with much of that growth attributed to less developed nations which are already at high risk of dengue [74]. Dengue is primarily an urban disease. The conditions in poorer urban areas often result in ideal habitats for *A. aegypti* with the accompanying overcrowding and human density providing the means for high transmission rates [75]. Rapid urbanization often results in informal housing and slums, the conditions of which have been blamed for the epidemic conditions in many countries [76]. Such communities often lack health services, a reliable and accessible water supply, waste removal services, surface water drainage systems, and a multitude of additional services which cause them to become prime habitats for *A. aegypti* and ideal areas for dengue transmission [77]. Effective vector control programs have been deemed unattainable in such communities.

Other Indirect Effects of Climate Change on Dengue Risk

In addition to directly affecting vector populations and viral replication rates, climate change also has a tremendous potential to increase the risk of dengue indirectly. Dengue is strongly influenced by socioeconomic factors and major economic sectors such as agriculture and fishing in many at-risk countries are highly susceptible to variations in climate leaving their economies vulnerable. Shifts in optimum growing conditions, drought or flood-related crop failure, and reduction in water resources are projected to have large negative impacts on subsistence farmers and rural communities. As a result, population displacement and even greater rural-to-urban migrations will result. Thus, one result of climate change is that urban communities will experience an even greater influx of people putting further strain on public health, infrastructure systems, and water resources often already stressed by current population growth and urbanization trends. Additionally, climate change alterations in rainfall patterns, surface water availability, and sea level-related intrusion of saltwater into water tables can further exacerbate water shortages and storage practices thus providing habits for vector populations.

Refugee conditions, which also have been linked to dengue outbreaks, may also be climatically induced by increased ethnic conflict aggravated by climate-related economic failures. Any climatic event that acts to destabilize communities, displace populations, breakdown infrastructure, limit public health and services, or cause a lapse in coordinated vector control efforts has the potential to increase the risk of dengue.

Regional Implications

Asia

Dengue has a long history in the Asian region. Rapid urbanization following World War II led to epidemic conditions in Southeast Asia and the first major epidemics of DHF. Most dengue cases occur in the Southeast and South Central regions of Asia. Together with western Pacific region, Southeast Asia currently bears nearly 75 % of the current global dengue fever burden [78]. Travel between areas and the co-circulation of multiple dengue virus serotypes have resulted in a state of hyperendemicity. Thus, DHF incidence has since risen dramatically and has been a major cause of hospitalizations and death in children since the mid-1970s [14].

Looking forward, Southeast and South Asian countries face multiple obstacles in dealing with dengue. Most of the population currently lives in rural areas, but the region is undergoing a massive shift towards urbanization. In Southeast Asia, urbanization is projected to increase from 39 to 73 % by 2050 [74]. Rural to urban migrations paired with a likewise rapid growth in population size will greatly increase the number of people who will be living in slum conditions where dengue thrives.

Modeling of climatic suitability to dengue transmission confirms that many parts of Southeast Asia are at extremely high levels of risk [71, 79]. GCM-based modeling predicts an increase of the epidemic potential within these areas and a spread of high dengue-transmission risk to many parts of South Central Asia where warming is predicted to be even greater than the global mean. While much of India is currently modeled to be at a lower transmission risk level, future predictions indicate it as having the largest expansion of high transmission risk to new areas. Currently, the highest number of dengue cases in the region is reported in Indonesia where dengue is listed as the second worst health problem the country faces with an average of over 140,000 cases a year from 2006 to 2009 [78].

In comparison, India reports much smaller numbers, though weaknesses in surveillance and reporting are cited. As it has with other countries in the region, dengue and DHF rates have risen over the past decades. Currently, less than a third of India's population lives in urban areas. By 2050 this is projected to increase to over half, which in conjunction with population growth, is projected to result in a 230 % increase in the urban population [80]. The combination of urbanization, population growth, and a changing climate is anticipated to lead to a large increase in the number of dengue cases in India.

Climate modeling predicts both an increase in intense precipitation events and periods of precipitation shortfalls for South and Southeast Asia. Failure in agricultural sectors is considered likely and will likely further contribute to urban poverty by producing even larger rural-to-urban migrations [81]. Of all factors relating to climate change, expansion of areas under severe water shortage is expected to become one of the greatest environmental problems in South and Southeast Asia. A growing population combined with climate change-related water shortages is expected to result in a substantial increase in the number of people living under severe water stress. The necessity for water storage under such conditions can be expected to contribute to increased vector breeding.

Australia and New Zealand

In Australia, dengue is currently limited to the northern part of Queensland where outbreaks occur when the dengue virus is imported by travelers. While dengue is not endemic to Australia, such outbreaks have occurred with increasing frequency and magnitude over the past few decades [82]. The geographic distribution of *A. aegypti* is likewise currently limited to parts of Queensland, but in the past has extended far south along the eastern regions. This change in distribution was due to a loss of breeding sites and climate-based analyses confirm that much of the populous south-east region of Australia is still currently suitable for *A. aegypti* [83]. However, changing climate conditions have already been observed and drying conditions and water shortages in Southeast Australia have resulted in the installation of many government-subsidized and ad hoc water storage tanks in many cities and towns which are anticipated to be ideal breeding grounds for *A. aegypti*. The number of such containers is predicted to grow as the drying trend is projected to continue [84].

In addition to reestablishing their former range, *A. aegypti* are projected to spread to many new areas as temperatures warm. In conjunction with the expansion of domestic water tanks, climate change could lead to *A. aegypti* coexisting with over 95 % of Australia's population.

Models also predict an extended range of climatic suitability to viral transmission with the size of vulnerable populations doubling to tripling as soon as 2020 [72, 85]. It is theorized that the risk of dengue could extend from its northern distribution south along the coast into Brisbane and possibly as far as Sydney by the year 2100 [86]. However, actual risk of dengue transmission will continue to be moderated by housing, cultural, socioeconomic, and other factors.

In New Zealand there are currently no established populations of dengue vector mosquitoes. However, many nearby islands in the Asia-Pacific area are home to populations of *A. aegypti* and *A. aegypti* have been intercepted in New Zealand. Multilayer computer modeling suggests that under warming scenarios, the northern area of the North Island may become inhabitable by *A. aegypti* by 2050 with an even greater area (including Auckland City, New Zealand's largest city) inhabitable by 2100 [87]. Further modeling of projected epidemic potential has found that a climate change scenario could result in potential dengue fever transmission in New Zealand, though this hasn't been confirmed in all models [85].

Small Island Nations

Small islands account for a significant number of the total number of global dengue cases each year. Since 1970, pan-Pacific epidemics have occurred with many islands reporting very high incidence as well as the presence of DHF cases [69, 88]. Dengue epidemics also occur in the islands of the Indian Ocean. One such epidemic in Seychelles involved approximately 80 % of the population [89].

Much of the increase in dengue incidence in islands in recent years can be attributed to increasing international travel with concurrent epidemics often occurring in islands separated by great distances [90]. Rapid urbanization, poor public health practices, inadequate infrastructure, poor waste management practices, and water storage practices also account for the increase of dengue and other diseases in small island states [91].

Dengue epidemics have been strongly linked with ENSO events in the Pacific and Caribbean [68, 69]. As ENSO events have increased in recent history, corresponding peaks in dengue incidence have been observed as well.

Empirical modeling confirms that all Pacific island states are at currently suitable for dengue transmission. Future projections indicate the greatest increase in transmission risk will likely occur in Hawaii, New Caledonia, Fiji, and Vanuatu which currently have the least suitable climates in the Pacific for dengue transmission [85].

Temperature increases for small islands are projected to be generally less than the global mean increase due to the fact that the greatest warming is projected to be over large land masses [84]. As a result, the greatest climate change-associated

increase in dengue risk for islands will be from factors such as sea level changes, extreme weather events, and the indirect effects of climate change.

Islands have an intrinsically heightened vulnerability to climate and weather [91]. In most cases, they have low adaptive capacity and adaptation requires large economic resources. Islands also generally have small economies which are very sensitive to external shocks. Climate change is projected to have large impacts on main economic sectors such as agriculture and fishing. Tourism, which makes up a large portion of many island economies, is likely to be impacted as well due to factors such as beach erosion, terrestrial environmental degradation, and degradation of coral reefs.

Sea levels have already risen and are projected to raise an additional 0.35 m rise by the end of the century [92]. A rise in sea level could be catastrophic for small islands in several ways. Much of island states' industry, infrastructure, and housing are all vulnerable to rises in sea level. In addition, intrusion of salt water into the water table is expected as sea levels rise. Small islands have very limited freshwater sources and there is strong evidence that water resources will become seriously compromised due to climate change in small islands.

Small islands are also prone to natural disasters. Typhoons are expected to increase in intensity as a result of climate change. Floods and droughts may likewise become more intense. Effects from such natural disasters which are expected to increase as a result of climate change include impacts on economic sectors, damaged infrastructure, displaced populations, and exacerbated water stress. These environmental stressors on small islands will greatly contribute to the future risk of dengue associated with climate change.

The Middle East

Dengue has recently reemerged in the Middle East causing sporadic yet increasingly common outbreaks after half a century of its absence. Among the factors believed to be responsible for the recent reemergence of dengue are decreased use of DDT, rapid urbanization and the development of slums and shanty towns, long-lasting conflicts, deteriorating public health services, large numbers of displaced populations resulting in numerous refugee camps, and increasing viral introduction through migrations and travel [93]. Positive trends for many of these factors are expected to continue into the near future.

The scarcity of rainfall in this region results in climatic conditions that are not ideal for the dengue vectors, although disease vectors are still found in many countries in the region [94]. GCM-based modeling predicts future climatic conditions favoring large increases in the probability of dengue transmission in the southern Middle East region. This will be primarily due to anticipated severe water stress in the Middle East as a result of climate change. Besides aggravating economic and social disruptions which promote disease-favoring conditions, water stress may further fuel the increase in dengue incidence by causing a proliferation of water storage containers which are ideal for vector breeding.

Africa

The current burden of dengue in Africa is poorly understood [95]. The number of recorded cases has increased considerably since 1980 with most cases occurring in East Africa [96]. However, many fewer cases of dengue have been reported in Africa than in other areas along similar latitudes. There are several possible explanations for this. In Africa, most febrile illnesses are often assumed to be malaria and are treated as such without proper medical examination or laboratory diagnosis. This problem is exacerbated by a lack of funding for surveillance and research which is generally not available for study of dengue. However, in addition to low awareness of dengue fever among health workers, there is also laboratory evidence that African *Aedes* mosquito species may have lower susceptibility to dengue virus infection that may cause lower disease rates.

Despite the low reported numbers, it is very likely that the actual number of infections is much higher. Limited serologic surveys, existing outbreak reports, and the confirmed dengue cases of travelers returning from African nations suggest endemic dengue in all or many parts of Africa [97].

High levels of warming, exceeding the global mean, are projected in the coming decades for the African continent. Current climatic suitability for dengue transmission maps large portions of Middle and East Africa and areas along the western coast as being at high risk. Under a climate change scenario, projected increases in transmission risk predict an enhancement of risk within these areas and an expansion of risk into greater portions of East, Middle, and West Africa [71].

GCM-based modeling predicts a several month extension of the dengue transmission period for many urban areas throughout Africa [72]. In Kenya, where currently no period of transmission is modeled, a 7-week transmission period is projected under a 2 °C warming scenario. Under a 4 °C warming scenario, the transmission period is projected to increase to 9 months.

In addition to the other factors which may account for the lower reported numbers of dengue cases, the degree of urbanization and population density have likely been protective in inhibiting epidemics. The population in Africa is growing rapidly, however, and is expected to double by the year 2050 [98]. Moreover, whereas most of Africans currently live in rural areas, Africa is urbanizing quickly and the urban population in Sub-Saharan Africa is projected to grow by almost 400 % by 2050 [74]. As population density climbs in areas already modeled to be climatically at high risk of dengue, an increase in dengue-related morbidity and mortality rates in these areas is highly likely. Additionally, the areas which are projected to experience the greatest increases of population growth and urbanization in the coming decades are the same areas which are projected to become climatically at high risk for dengue transmission.

Current and projected socioeconomic and demographic factors make Africa particularly vulnerable to the effects of climate change and the risk of dengue. Africa has an extremely high poverty index with 50.8 % of the population living on less than a dollar a day [99]. Only 58 % of the Sub-Saharan population has access to improved water sources and in urban areas, only 42 % living in urban areas had

access to improved sanitation facilities [100]. The future outlook is not encouraging. Certain countries in sub-Saharan Africa are declining in overall wealth with the average person becoming poorer by a factor of 2 every 25 years. Climate change is likely to exacerbate nearly every stressor which currently plagues African nations [101]. Mass relocations and urban migration are likely to place new demands on already insufficient infrastructures. The combination of poverty, population density, and insufficient infrastructure and public services will likely create the crowded and unsanitary conditions ideal for dengue transmission.

The IPCC has identified Africa as “one of the most vulnerable continents to climate change and climate variability” [101]. While warming is expected to be considerable, it is the continent’s low adaptive capacity compounded by multiple stressors that make it especially at risk to the changing climate. Multiple, major economic sectors in Africa are vulnerable to climate change. In addition, endemic poverty, developmental challenges, and degradation of natural resources, among other factors, will significantly weaken its adaptive capacity.

Europe

Until very recently, natural transmission of dengue was not found to occur in European countries, although both *A. aegypti* and dengue had been found in Europe historically. Past dengue epidemics have occurred in Spain, the Canary Islands, and in many Mediterranean nations [102]. One of the worst epidemics on record occurred in refugee camps in Greece in 1927–1928 when an estimated 650,000 infections and 1,000 deaths occurred. Since the mid-twentieth century, *A. aegypti* largely disappeared from Europe due to increased hygiene, reliable and piped water supplies, and the use of insecticide [103].

While *A. aegypti* may play a role in future dengue risk in Europe, *A. albopictus*, is the vector of greatest concern for current and projected risk in the European area. Recently, *A. albopictus* was introduced into Italy where it has spread to most of the country within a relatively short time period. Since its introduction, the species has been identified in many European countries, but firmly established populations are found mainly around the coasts of the Mediterranean and Adriatic. Modeling past and current climatic suitability asserts that the trend of milder winters has resulted in an expansion of suitable range and that larger portions of Europe are already suitable. GCM-based modeling projects a northward shift in *A. albopictus* populations with climatic hotspots in Portugal, the Southern UK, western Germany, the Benelux, Slovakia, Cyprus, Bulgaria, Macedonia, Hungary, and Turkey [104]. *A. aegypti*’s has also recently become established in Italy and there is growing concern over its potential spread to additional parts of Europe [102, 105].

In a recent risk assessment of vector-borne diseases in Europe, the European Centre for Disease Prevention and Control lists dengue among the top ten vector-borne diseases with the greatest potential to affect European citizens [105]. The dengue virus is routinely introduced through infected travelers. Since 1999, 1,117 cases of dengue were reported in European travelers, though the actual number is

assumed to be much higher [106]. As dengue rates continue to rise around the globe, so too will introductions into non-endemic areas.

In August 2010, the first autochthonous transmission of dengue in continental Europe since 1928 occurred in France. That same year a dengue outbreak was discovered in Croatia. *A. albopictus* was deemed the vector responsible for both outbreaks.

Concern over possible future dengue transmission in the Mediterranean region is warranted as several characteristics of the region add to the danger of transmission. Cities are generally densely populated. Windows are generally left open during the summer months as air conditioners are rarely used. In addition, ideal contact with the vector can occur during activities and social gatherings which typically are held outdoors [104].

Mediterranean summers are projected to experience the greatest seasonal warming in Europe. It is anticipated that warmer temperatures will result in a higher transmission risk in the European region in addition to expected lengthening of transmission periods for more temperate areas [72]. Modeling based on viral factors predicts a geographic extension of the at-risk areas around the Mediterranean and into central Europe by the end of the century with the Southwest Iberian Peninsula being especially at risk [107]. The summer months of Athens are projected to see significant increases in the epidemic potential although other modeling suggests that the Mediterranean regions may also become less habitable to *A. albopictus* due to increased drying [73, 103].

Projected risks based on viral- and vector-based factors alone do not take into account other regulating factors such as socioeconomic status. The same factors which have inhibited outbreaks in the past decades can be expected to dampen the effects of a more transmission-prone climate in areas that provide an insufficient vector habitat and limit contact between the vectors and the human population. Additionally, the subprime status of *A. albopictus*, compared to *A. aegypti*, as a vector will result in less explosive outbreaks than have been seen in other parts of the world. It has been noted that nearly all the outbreaks in which *A. albopictus* has been implicated have been infrequent, mild, and limited despite very low herd immunity and numerous imported cases [108].

Latin America

Of all the regions in the world, the emergence of dengue and DHF in recent decades has been the most dramatic in the Americas. Historically, outbreaks of dengue occurred sporadically in the Americas for hundreds of years. In 1947, a coordinated hemisphere-wide effort by the Pan American Health Organization to eradicate *A. aegypti* proved hugely successful. By the early 1960s *A. aegypti* was eradicated from most of its previous territory with dengue infections largely disappearing as well. Unfortunately, control efforts soon lapsed and *A. aegypti* quickly disseminated to nearly every country in the Western Hemisphere. The increase in international travel and commerce spread both vector and virus which flourished in the conditions of rapidly urbanizing nations. By the 1980s major epidemics began again.

Over the last 3 decades, a 6.5-fold increase in reported dengue fever cases was observed in the Americas (roughly one million cases during the 1980s to 6.7 million during 2000–2009). During that time, DHF cases increased 12.4-fold (~13,400 in the 1980s to 172,000 DHF from 2000 to 2009). In 2010 alone, over 1.6 million cases of dengue were reported in the Americas, of which 49,000 cases were severe dengue [109, 110]. Increased international travel has increased viral introduction and outbreaks in the Americas have now been caused by all four serotypes. As co-circulation of multiple serotypes continues to become more widespread, the incidence of DHF will continue to rise.

The IPCC has classified dengue as being one of the main human-health climate change-related concerns in Latin America [111]. Warming trends are predicted to continue with larger than global mean increases of warming in most of Central and South America. Precipitation is likely to decrease in Central America with drier than usual springs and a high probability of droughts [84]. GCM-based modeling confirms that most of Latin America's climate currently yields very high risk of dengue transmission. An extension of at-risk areas is projected to extend into larger regions of Mexico, Brazil, Peru, and Ecuador [71]. Under a 2 °C warming scenario, projected increases in transmission periods are predicted for large urban areas such as Lima and Buenos Aires. In Caracas, Venezuela the current transmission period of about 7 months is modeled to become year-round. However, the high altitude cities such as Quito, Ecuador and Bogota, Colombia are not expected to develop suitable transmission periods even under a 4 °C warming scenario. Mexico City, which is also at a higher altitude, is not expected to develop a transmission period until a 4 °C increase [72].

An increase in temperature (about 1 °C in Mesoamerica and 0.5 °C in South America) and changes in rainfall patterns have already been recorded in Latin America [111]. Whether a changing climate is to blame for the increase of dengue in the region has been heavily debated topic. While climate is undeniably linked to dengue, it is not sufficient to explain the explosive rise in dengue rates. The *A. aegypti* eradication campaign coincided with a remarkable period of urbanization in Latin America in which it became the most urbanized region of the developing world. The 1980s, however, brought a serious debt crisis to the region resulting in a deterioration of economic and social conditions, large inequalities, unemployment, poverty, and failures in health systems. These conditions, paired with the reestablishment of the vector and increased mobility of people and virus within and between countries, have resulted in state prime for dengue transmission.

The inequality in Latin America's population in regard to income and opportunities is among the highest in the world [112]. Substantial disparities in access to water and health services also exist. Future projections indicate that accelerated urban growth under conditions of increasing poverty combined with low investment in water supplies will result in a high proportion of the urban population without access to sanitation services and a reliable water supply [111]. Such factors perpetuate high dengue transmission risk for large portions of the population despite any national economic gains and leave them highly vulnerable to the present and future conditions of climate change.

The United States

Dengue is largely absent in the continental United States, but it has not always been so. Pandemics that stretched through the Caribbean and Gulf region also struck the southern states up through the first half of the twentieth century with an epidemic occurring as far north as Philadelphia during an unusually hot summer in 1780 [34, 113].

A. aegypti have formerly ranged as far north as Boston in the east and Southern California in the west, but their current range is much more limited. The United States was one of the few places that failed to completely eradicate *A. aegypti* during the pan-American eradication campaign during the mid-twentieth century and served as a source for its reintroduction throughout the hemisphere. Over the last several decades, *A. albopictus* has become established in many southern and eastern states, replacing *A. aegypti* in many areas. However, *A. aegypti* is still common in urban settings in Southern Florida and in cities along the Gulf Coast of Texas and Louisiana and can still be found in several other states.

Mexican states bordering the United States have had repeated large epidemics of dengue. Beginning in 1980, after a long absence, small dengue outbreaks began to occur sporadically along the Texas–Mexico border. While these outbreaks highlight the potential for dengue transmission within the United States, they also underscore the conditions inherent to American society which are prohibitive to dengue. In a 2005 outbreak, a handful of people in Brownsville Texas were diagnosed with dengue fever, but over the border in Tamaulipas, Mexico, over 7,000 cases were reported [114]. Abundant *A. aegypti* mosquitoes were found breeding in both cities. The use of air conditioning, which limits vector-human contact, and larger lot sizes, which result in less dense urban environments, were found to be protective. These factors are among the many socioeconomic and societal factors that highly limit dengue transmission in the United States.

While it is known that climatic conditions in some parts of the United States already support dengue transmission, modeling of current climatic suitability suggests that risk level is relatively low. Both mechanistic and empirical modeling generally show low suitability outside of areas such as Southern Florida and the southernmost part of Texas, though mechanistic-based modeling predicts periods of potential transmission during the year in southern and eastern cities.

However, GCM-based modeling projects that a changing climate may significantly increase the risk of dengue transmission. An increase of risk is predicted along the southern states bordering the Gulf of Mexico accompanied by the opening up of new transmission areas. In Miami, the potential transmission period is projected to become year-round. Speaking of the country in general, a 2 °C increase in temperature is expected to raise the potential transmission intensity 2–3 times its current level [71–73, 79].

In 2009 and 2010, dengue fever gained attention in the United States when dozens of cases resulting from local transmission occurred in Key West, Florida and surrounding areas. A random serosurvey found that over 5 % of the population was likely infected [115]. Outside of the cases along the Texas border, this marked the first outbreak within the continental United States since 1946. Dengue has also

recently reemerged in Hawaii. In 2001, 122 cases of dengue were confirmed on three of the six islands after over half a century of its absence. Unlike in Florida, where *A. aegypti* served as vector for the outbreak, *A. albopictus* was responsible for the outbreak in Hawaii.

Whether the recent outbreaks of dengue in the United States mark the beginning of a larger scale reemergence has been a topic of debate. Dengue is now the leading cause of acute febrile illness in travelers returning from the Caribbean, South America, and Asia. As both dengue incidence and international travel rise across the globe, so too will the number of viral introductions to the United States. Modeling of viral introductions attributed to travel suggests that Florida and Texas are especially at risk [106]. Conversely, the trend of continued geographic expansion of *A. albopictus* and its replacement of *A. aegypti* might reduce the risk of epidemic dengue activity [108]. The fact that dengue has been largely absent in the United States paired with the substantial disparity of rates across the border is the most convincing argument that dengue outbreaks will remain a rarity. The societal factors which have proved so effective against transmission are unlikely to change.

It remains to be seen whether increased risk due to an altered climate will result in more frequent outbreaks of dengue in the United States. What is not in doubt is the need for greater awareness of dengue in the health sector. While the growing number of cases in returning travelers is tracked, it is considered far below the actual number as most cases likely go undiagnosed. Retrospective serological surveys in the local outbreaks in Florida and Texas found that most cases were misdiagnosed [116]. Better public health knowledge, along with intensified surveillance and enhanced vector control efforts will be needed to guard against the growing threat of dengue in the United States.

References

1. Dengue [Internet]. <http://www.who.int/topics/dengue/en/>. 2012. Accessed 19 July 2012.
2. Hales S, Edwards SJ, Kovats RS. Impacts on health of climate extremes. Geneva: World Health Organization; 2003.
3. Phillips ML. Dengue reborn: widespread resurgence of a resilient vector. *Environ Health Perspect*. 2008;116(9):A382–8.
4. Arias JR. Dengue: how are we doing? Washington, D.C.: PAHO/WHO; 2002.
5. Dengue [Internet]. <http://www.cdc.gov/Dengue/>. 2012. Accessed 19 July 2012.
6. Halstead SB, Yamarat C. Recent epidemics of hemorrhagic fever in Thailand. Observations related to pathogenesis of a “new” dengue disease. *Am J Public Health Nations Health*. 1965;55:1386–95.
7. Gubler DJ. Dengue and dengue hemorrhagic fever. *Clin Microbiol Rev*. 1998;11(3):480–96.
8. Kliks SC, Nimmanitya S, Nisalak A, Burke DS. Evidence that maternal dengue antibodies are important in the development of dengue hemorrhagic fever in infants. *Am J Trop Med Hyg*. 1988;38(2):411–9.
9. Halstead SB, Streit TG, Lafontant JG, Putvatana R, Russell K, Sun W, et al. Haiti: absence of dengue hemorrhagic fever despite hyperendemic dengue virus transmission. *Am J Trop Med Hyg*. 2001;65(3):180–3.

10. Stephens HA, Klaythong R, Sirikong M, Vaughn DW, Green S, Kalayanaroj S, et al. HLA-A and -B allele associations with secondary dengue virus infections correlate with disease severity and the infecting viral serotype in ethnic Thais. *Tissue Antigens*. 2002;60(4):309–18.
11. Halstead SB. Dengue virus-mosquito interactions. *Annu Rev Entomol*. 2008;53:273–91.
12. Hopp MJ, Foley JA. Global-scale relationships between climate and the dengue fever vector, *Aedes aegypti*. *Clim Change*. 2001;48(2–3):441–63.
13. MacDonald WW. *Aedes aegypti* in Malaya. II. Larval and adult biology. *Ann Trop Med Parasitol*. 1956;50(4):399–414.
14. Gubler DJ. Dengue/dengue haemorrhagic fever: history and current status. *Novartis Found Symp*. 2006;277:3–16. discussion 16–22, 71–3, 251–3.
15. Christophers SR. *Aedes aegypti* (L.) the yellow fever mosquito: its life history, bionomics and structure. London: Cambridge University Press; 1960.
16. Scott TW, Chow E, Strickman D, Kittayapong P, Wirtz RA, Lorenz LH, et al. Blood-feeding patterns of *Aedes aegypti* (Diptera: Culicidae) collected in a rural Thai village. *J Med Entomol*. 1993;30(5):922–7.
17. Yasuno M, Tonn RJ. A study of biting habits of *Aedes aegypti* in Bangkok, Thailand. *Bull World Health Organ*. 1970;43(2):319–25.
18. Halstead SB. Successes and failures in dengue control—global experience. *Dengue Bull*. 2000;24:66–70.
19. Paupy C, Delatte H, Bagny L, Corbel V, Fontenille D. *Aedes albopictus*, an arbovirus vector: from the darkness to the light. *Microbes Infect*. 2009;11:14–5.
20. Juliano SA, Lounibos LP, O’Meara GF. A field test for competitive effects of *Aedes albopictus* on *A. aegypti* in south Florida: differences between sites of coexistence and exclusion? *Oecologia*. 2004;139(4):583–93.
21. O’Meara GF, Evans Jr LF, Gettman AD, Cuda JP. Spread of *Aedes albopictus* and decline of *Ae. aegypti* (Diptera: Culicidae) in Florida. *J Med Entomol*. 1995;32(4):554–62.
22. Gratz NG. Critical review of the vector status of *Aedes albopictus*. *Med Vet Entomol*. 2004;18(3):215–27.
23. Moore PR, Johnson PH, Smith GA, Ritchie SA, Van Den Hurk AF. Infection and dissemination of dengue virus type 2 in *Aedes aegypti*, *Aedes albopictus*, and *Aedes scutellaris* from the Torres Strait, Australia. *J Am Mosq Control Assoc*. 2007;23(4):383–8.
24. Farnesi LC, Martins AJ, Valle D, Rezende GL. Embryonic development of *Aedes aegypti* (Diptera: Culicidae): influence of different constant temperatures. *Mem Inst Oswaldo Cruz*. 2009;104(1):124–6.
25. Chang LH, Hsu EL, Teng HJ, Ho CM. Differential survival of *Aedes aegypti* and *Aedes albopictus* (Diptera: Culicidae) larvae exposed to low temperatures in Taiwan. *J Med Entomol*. 2007;44(2):205–10.
26. Yang HM, Macoris ML, Galvani KC, Andrighetti MT, Wanderley DM. Assessing the effects of temperature on dengue transmission. *Epidemiol Infect*. 2009;4:1–9.
27. Yang HM, Macoris ML, Galvani KC, Andrighetti MT, Wanderley DM. Assessing the effects of temperature on the population of *Aedes aegypti*, the vector of dengue. *Epidemiol Infect*. 2009;4:1–15.
28. Canyon DV, Hii JL, Muller R. Adaptation of *Aedes aegypti* (Diptera: Culicidae) oviposition behavior in response to humidity and diet. *J Insect Physiol*. 1999;45(10):959–64.
29. Sota T, Mogi M. Interspecific variation in desiccation survival time of *Aedes* (stegomyia) mosquito eggs is correlated with habitat and egg size. *Oecologia*. 1992;90(3):353–8.
30. Lewis D. Observations on *Aedes aegypti* under controlled atmospheric conditions. *Bull Entomol Res*. 1933;24:363–72.
31. Garrett-Jones C. Prognosis for the interruption of malaria transmission through assessment of the mosquito’s vectoral capacity. *Nature*. 1964;204:1173–5.
32. Scott TW, Amerasinghe PH, Morrison AC, Lorenz LH, Clark GG, Strickman D, et al. Longitudinal studies of *Aedes aegypti* (Diptera: Culicidae) in Thailand and Puerto Rico: blood feeding frequency. *J Med Entomol*. 2000;37(1):89–101.

33. Costero A, Edman JD, Clark GG, Kittayapong P, Scott TW. Survival of starved *Aedes aegypti* (Diptera: Culicidae) in Puerto Rico and Thailand. *J Med Entomol.* 1999;36(3):272–6.
34. Focks DA, Haile DG, Daniels E, Mount GA. Dynamic life table model for *Aedes aegypti* (Diptera: Culicidae): simulation results and validation. *J Med Entomol.* 1993;30(6):1018–28.
35. Lumsden WH. Observations on the effect of microclimate on biting by *Aedes aegypti* (L.) (Dipt., Culicid.). *J Exp Biol.* 1947;24(3–4):361–73.
36. Watts DM, Burke DS, Harrison BA, Whitmire RE, Nisalak A. Effect of temperature on the vector efficiency of *Aedes aegypti* for dengue 2 virus. *Am J Trop Med Hyg.* 1987;36(1):143–52.
37. Sheppard PM, Macdonald WW, Tonn RJ, Grab B. The dynamics of an adult population of *Aedes aegypti* in relation to dengue haemorrhagic fever in Bangkok. *J Anim Ecol.* 1969;38:661–702.
38. Ooi EE, Goh KT, Gubler DJ. Dengue prevention and 35 years of vector control in Singapore. *Emerg Infect Dis.* 2006;12(6):887–93.
39. Scott TW, Morrison AC. *Aedes aegypti* density and the risk of dengue-virus transmission. In: Takken W, Scott TW, editors. Ecological aspects for application of genetically modified mosquitoes. Dordrecht: Kluwer Academic; 2003.
40. Focks DA, Brenner RJ, Hayes J, Daniels E. Transmission thresholds for dengue in terms of *Aedes aegypti* pupae per person with discussion of their utility in source reduction efforts. *Am J Trop Med Hyg.* 2000;62(1):11–8.
41. Focks DA, Haile DG, Daniels E, Mount GA. Dynamic life table model for *Aedes aegypti* (Diptera: Culicidae): analysis of the literature and model development. *J Med Entomol.* 1993;30(6):1003–17.
42. Southwood TR, Murdie G, Yasuno M, Tonn RJ, Reader PM. Studies on the life budget of *Aedes aegypti* in Wat Samphaya, Bangkok, Thailand. *Bull World Health Organ.* 1972;46(2):211–26.
43. Jury MR. Climate influence on dengue epidemics in Puerto Rico. *Int J Environ Health Res.* 2008;18(5):323–34.
44. Lenhart AE, Castillo CE, Oviedo M, Villegas E. Use of the pupal/demographic-survey technique to identify the epidemiologically important types of containers producing *Aedes aegypti* (L.) in a dengue-endemic area of Venezuela. *Ann Trop Med Parasitol.* 2006;100 Suppl 1:S53–9.
45. Romero-Vivas CM, Arango-Padilla P, Falconar AK. Pupal-productivity surveys to identify the key container habitats of *Aedes aegypti* (L.) in Barranquilla, the principal seaport of Colombia. *Ann Trop Med Parasitol.* 2006;100 Suppl 1:S87–95.
46. Dizon JJ. Philippine hemorrhagic fever—epidemiologic aspects. *J Philipp Med Assoc.* 1967;43(5):346–65.
47. Chareonviriyaphap T, Akratanakul P, Nettanomsak S, Huntamai S. Larval habitats and distribution patterns of *Aedes aegypti* (Linnaeus) and *Aedes albopictus* (Skuse), in Thailand. *Southeast Asian J Trop Med Public Health.* 2003;34(3):529–35.
48. Eamchan P, Nisalak A, Foy HM, Chareonsook OA. Epidemiology and control of dengue virus infections in Thai villages in 1987. *Am J Trop Med Hyg.* 1989;41(1):95–101.
49. Ghosh SN, Pavri KM, Singh KR, Sheikh BH, Dilma LV, Mahadev PV, et al. Investigations on the outbreak of dengue fever in Ajmer city, Rajasthan state in 1969 Part I. Epidemiological, clinical and virological study of the epidemic. *Indian J Med Res.* 1974;62(4):511–22.
50. Pontes RJ, Freeman J, Oliveira-Lima JW, Hodgson JC, Spielman A. Vector densities that potentiate dengue outbreaks in a Brazilian city. *Am J Trop Med Hyg.* 2000;62(3):378–83.
51. Brunkard JM, Cifuentes E, Rothenberg SJ. Assessing the roles of temperature, precipitation, and ENSO in dengue re-emergence on the Texas-Mexico border region. *Salud Publica Mex.* 2008;50(3):227–34.
52. Chowell G, Sanchez F. Climate-based descriptive models of dengue fever: the 2002 epidemic in Colima, Mexico. *J Environ Health.* 2006;68(10):40–4. 55.
53. Hurtado-Diaz M, Riojas-Rodriguez H, Rothenberg SJ, Gomez-Dantes H, Cifuentes E. Short communication: impact of climate variability on the incidence of dengue in Mexico. *Trop Med Int Health.* 2007;12(11):1327–37.

54. Moore CG, Cline BL, Ruiz-Tiben E, Lee D, Romney-Joseph H, Rivera-Correa E. *Aedes aegypti* in Puerto Rico: environmental determinants of larval abundance and relation to dengue virus transmission. *Am J Trop Med Hyg.* 1978;27(6):1225–31.
55. Keating J. An investigation into the cyclical incidence of dengue fever. *Soc Sci Med.* 2001;53(12):1587–97.
56. Arcari P, Tapper N, Pfueller S. Regional variability in relationships between climate and dengue/DHF in Indonesia. *Sing J Trop Geogr.* 2007;28(3):251–72.
57. National Research Council. *Under the weather: climate, ecosystems, and infectious disease.* Washington, D.C.: National Academy Press; 2001.
58. Johansson MA, Dominici F, Glass GE. Local and global effects of climate on dengue transmission in Puerto Rico. *PLoS Negl Trop Dis.* 2009;3(2):e382.
59. Thammapalo S, Chongsuwitwong V, McNeil D, Geater A. The climatic factors influencing the occurrence of dengue hemorrhagic fever in Thailand. *Southeast Asian J Trop Med Public Health.* 2005;36(1):191–6.
60. Muto R. Summary of dengue situation in WHO western pacific region. *Dengue Bull.* 1998;22:12–9.
61. Nagao Y, Thavara U, Chitnumsup P, Tawatsin A, Chansang C, Campbell-Lendrum D. Climatic and social risk factors for *Aedes* infestation in rural Thailand. *Trop Med Int Health.* 2003;8(7):650–9.
62. Cazelles B, Chavez M, McMichael AJ, Hales S. Nonstationary influence of El Niño on the synchronous dengue epidemics in Thailand. *PLoS Med.* 2005;2(4):e106.
63. Chantha N, Guyant P, Hoyer S. Control of DHF outbreak in Cambodia, 1998. *Dengue Bull.* 1998;22:69–74.
64. Bangs MJ, Larasati RP, Corwin AL, Wuryadi S. Climatic factors associated with epidemic dengue in Palembang, Indonesia: implications of short-term meteorological events on virus transmission. *Southeast Asian J Trop Med Public Health.* 2006;37(6):1103–16.
65. Corwin AL, Larasati RP, Bangs MJ, Wuryadi S, Arjoso S, Sukri N, et al. Epidemic dengue transmission in southern Sumatra, Indonesia. *Trans R Soc Trop Med Hyg.* 2001;95(3):257–65.
66. Gagnon AS, Bush ABG, Smoyer-Tomic KE. Dengue epidemics and the El Niño southern oscillation. *Clim Change.* 2001;19:35–43.
67. Poveda GJ, Graham NE, Epstein PR, Rojas W, Vélez DI, Quiñónez ML, Martnes P. Climate and ENSO variability associated to malaria and dengue fever in Columbia. 10th Symposium on global change studies, Boston: American Meteorological Society, Jan 10–15, 1999.
68. Amarakoon D, Chen A, Rawlins S, Chadee DD, Taylor M, Stennett R. Dengue epidemics in the Caribbean-temperature indices to gauge the potential for onset of dengue. *Mitig Adapt Strat Glob Change.* 2008;13(4):341–57.
69. Hales S, Weinstein P, Woodward A. Dengue fever epidemics in the south pacific: driven by El Niño southern oscillation? *Lancet.* 1996;348(9042):1664–5.
70. Hales S, Weinstein P, Souares Y, Woodward A. El Niño and the dynamics of vectorborne disease transmission. *Environ Health Perspect.* 1999;107(2):99–102.
71. Hales S, de Wet N, Mairionald J, Woodward A. Potential effect of population and climate changes on global distribution of dengue fever: an empirical model. *Lancet.* 2002;360(9336):830–4.
72. Jetten TH, Focks DA. Potential changes in the distribution of dengue transmission under climate warming. *Am J Trop Med Hyg.* 1997;57(3):285–97.
73. Patz JA, Martens WJ, Focks DA, Jetten TH. Dengue fever epidemic potential as projected by general circulation models of global climate change. *Environ Health Perspect.* 1998;106(3):147–53.
74. Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat. *The 2006 revisions and world urbanization prospects: The 2007 revision; 2007.*
75. Knudsen AB, Slooff R. Vector-borne disease problems in rapid urbanization: New approaches to vector control. *Bull World Health Organ.* 1992;70(1):1–6.
76. Gubler DJ. The changing epidemiology of yellow fever and dengue, 1900 to 2003: full circle? *Comp Immunol Microbiol Infect Dis.* 2004;27(5):319–30.

77. United Nations Human Settlements Program. The challenge of slums: global report on human settlements 2003. London: Earthscan; 2003.
78. World Health Organization, Regional Office for South-East Asia. Situation update of dengue in the SEA region, 2010; 2010.
79. Rogers DJ, Wilson AJ, Hay SI, Graham AJ. The global distribution of yellow fever and dengue. *Adv Parasitol.* 2006;62:181–220.
80. United Nations, Department of Economic and Social Affairs. World urbanization prospects, the 2011 revision. New York: UN DESA; 2011.
81. Cruz RV, Harasawa H, Lai M, Wu S. Asia. Cambridge: Cambridge University Press; 2007.
82. Confalonieri U, Menn B, Akhtar R, Ebi KL, Hauengue M, Kovats RS, et al. Impacts, adaptation and vulnerability. Cambridge: Cambridge University Press; 2007.
83. Beebe NW, Cooper RD, Mottram P, Sweeney AW. Australia's dengue risk driven by human adaptation to climate change. *PLoS Negl Trop Dis.* 2009;3(5):e429.
84. Christensen JH, Hewitson B, Busuioic A, Chen A, Goa X, Held I, et al. Regional climate projections. Cambridge: Cambridge University Press; 2007.
85. McMichael A, Woodruff R, Whetton P, Hennessy K, Nicholls N, Hales S, et al. Human health and climate change in Oceania: a risk assessment 2002. Canberra: Commonwealth of Australia; 2003.
86. Woofruff R, Hales S, Butler C, McMichael A. Climate change and health impacts in Australia: effects of dramatic CO₂ emission reductions. Canberra: Australian National University; 2005.
87. de Wet N, Ye W, Hales S, Warrick R, Woodward A, Weinstein P. Use of a computer model to identify potential hotspots for dengue fever in New Zealand. *N Z Med J.* 2001;114(1140):420–2.
88. Dengue in the Western Pacific Region [Internet]. http://www.wpro.who.int/health_topics/dengue/ 2009. Accessed 21 Aug 2009.
89. Calisher CH, Nuti M, Lazuick JS, Ferrari DM, Kappus KD. Dengue in the Seychelles. *Bull World Health Organ.* 1981;59(4):619–22.
90. Effler PV, Pang L, Kitsutani P, Vorndam V, Nakata M, Ayers T, et al. Dengue fever, Hawaii, 2001–2002. *Emerg Infect Dis.* 2005;11(5):742–9.
91. Mimura N, Burse L, McLean RF, Agard J, Briguglio L, Lefale P, et al. Small islands. Cambridge: Cambridge University Press; 2007.
92. Meehl GA, Stocker TF, Collins WD, Friedlingstein P, Gaye AT, Gregory JM, et al. Global climate projections. Cambridge: Cambridge University Press; 2007.
93. Amarsinghe A, Letson GW. Dengue in the middle east: a neglected, emerging disease of importance. *Trans R Soc Trop Med Hyg.* 2012;106(1):1–2.
94. Rathor HR. The role of vectors in emerging and re-emerging diseases in the Eastern Mediterranean region. *Dengue Bull.* 2000;24:103–9.
95. Suaya JA, Shepard DS, Beatty ME. Dengue: burden of disease and cost of illness. Geneva: WHO; 2006.
96. Dengue fever [Internet]. <http://www.cdc.gov/ncidod/dvbid/dengue/>. 2008. Accessed Aug 2009.
97. Amarasinghe A, Kuritsky J, Letson G, Margolis H. Dengue virus infection in Africa. *Emerg Infect Dis.* 2011;17(8):1349–54.
98. Population Reference Bureau. 2009 World population data sheet. 2009.
99. World Health Organization. World health statistics. 2009.
100. World Bank. Sub-Saharan Africa data profile. 2008.
101. Boko M, Niang I, Nyong A, Vogel C, Githeko A, Medany M, et al. Africa. In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE, editors. *Climate change 2007: impacts, adaptation and vulnerability. Contribution of working group II to the fourth assessment report of the intergovernmental panel on climate change.* Cambridge: Cambridge University Press; 2007. p. 433–67.
102. Reiter P. Climate change and mosquito-borne disease. *Environ Health Perspect.* 2001;109 Suppl 1:141–61.

103. Holstein M. Dynamics of *Aedes aegypti* distribution, density and seasonal prevalence in the Mediterranean area. *Bull World Health Organ.* 1967;36:541–3.
104. Caminade C, Medlock JM, Ducheyne E, McIntyre KM, Leach S, Baylis M, et al. Suitability of European climate for the Asia tiger mosquito *Aedes albopictus*: recent trends and future scenarios. *J R Soc Interface.* 2012;9(75):2708–17.
105. Senior K. Vector-borne diseases threaten Europe. *Lancet Infect Dis.* 2008;8(9):531–2.
106. Gardner L, Fajardo D, Waller S, Wang O, Sahotra S. A predictive spacial model to quantify the risk of air-travel-associated importation into the United States and Europe. *J Trop Med.* 2012;2012:103679.
107. Thomas ST, Fischer D, Fleischmann S, Bittner T, Beierkuhnlein C. Risk assessment of dengue virus amplification in Europe based on spatio-temporal high resolution climate change projections. *Erdkunde.* 2011;65(2):137–50.
108. Lambrechts L, Scott TW, Gubler DJ. Consequences of the expanding global distribution of *Aedes albopictus* for dengue virus transmission. *PLoS Negl Trop Dis.* 2010;4(5):e646.
109. San Martin JL, Braithwaite O, Zambrano B, Solorzano JO, Bouckenoghe A, Dayan GH, et al. The epidemiology of dengue in the Americas over the last three decades: a worrisome reality. *Am J Trop Med Hyg.* 2010;8(1):128–35.
110. Dengue regional information: Number of cases 2008–2010 [Internet]. http://new.paho.org/hq/index.php?option=com_content&task=view&id=264&Itemid=363. 2012. Accessed 20 May 2012.
111. Margin G, García CG, Choque DC, Giménez JC, Morenao AR, Nagy GJ, et al. *Latin America.* Cambridge: Cambridge University Press; 2007.
112. Ferranti DD, Perry GE, Ferreira FHG, Walton M, Coady D, Cunningham W, et al. *Inequality in Latin America and the Caribbean: breaking with history.* Washington, D.C.: The International Bank for Reconstruction and Development/The World Bank; 2004.
113. Ehrenkranz NJ, Ventura AK, Cuadrado RR, Pond WL, Porter JE. Pandemic dengue in Caribbean countries and the southern united states—past, present and potential problems. *N Engl J Med.* 1971;285(26):1460–9.
114. Ramos MM, Mohammed H, Zielinski-Gutierrez E, Hayden MH, Lopez JLR, Fournier M, et al. Epidemic dengue and dengue hemorrhagic fever at the Texas-Mexico border: results of a household-based seroepidemiologic survey, December 2005. *Am J Trop Med Hyg.* 2008;78(3):364–9.
115. Radke EG, Gregory CJ, Kintziger KW, Sauber-Schatz EK, Hunsperger EA, Gallgher GR, et al. Dengue outbreak in Key West, Florida, USA, 2009. *Emerg Infect Dis.* 2012;18(1):135–7.
116. CDC. Underdiagnosis of dengue—Laredo, Texas, 1999. *MMWR Morb Mortal Wkly Rep.* 2001;50(4):57–9.

Chapter 11

Impact of Climate Change on Vector-Borne Disease in the Amazon

William Pan, OraLee Branch, and Benjamin Zaitchik

Abstract Impending changes in climate regimes coupled with anthropogenic changes in land use and land cover change pose the most pressing challenges to human societies and natural ecosystems. Global climate change is predicted to disrupt seasonal periodicities and long-term trends in rainfall and temperature, altering natural climate cycles and variation. The impact of environmental change on disease transmission will determine who, when, and where human livelihoods flourish and fail. Vulnerable populations will be particularly affected—i.e., chronically disadvantaged populations who are typically poor, have limited economic opportunities and access to services, and few (if any) options to improve their quality of life. Immediate action is needed to better understand, adapt, and respond to disease burdens that will be affected by changing climate.

Keywords Climate change • Vector-borne disease in the Amazon and climate change • Climate change in the Amazon • Amazon climate change and vector-borne disease • Disease burden and climate change

Impending changes in climate regimes coupled with anthropogenic changes in land use and land cover change (LUCC) pose the most pressing challenges to human societies and natural ecosystems. Global climate change is predicted to disrupt

W. Pan, P.H., M.S., M.P.H.

Nicholas School of Environment and the Duke Global Health Institute, Duke University, Durham, NC, USA

O. Branch, Ph.D. (✉)

Department of Microbiology, NYU School of Medicine, 341 East 25th Street, OPH-610 10010, New York, NY, USA

e-mail: oralee.branch@nyumc.org

B. Zaitchik, Ph.D.

Department of Earth and Planetary Science, Johns Hopkins University, Baltimore, MD, USA

seasonal periodicities and long-term trends in rainfall and temperature, altering natural climate cycles and variation [1]. The impact of environmental change on disease transmission will determine who, when, and where human livelihoods flourish and fail. Vulnerable populations will be particularly affected—i.e., chronically disadvantaged populations who are typically poor, have limited economic opportunities and access to services, and few (if any) options to improve their quality of life. Immediate action is needed to better understand, adapt, and respond to disease burdens that will be affected by changing climate.

This chapter will discuss the impact of coupled climate–environment changes on disease transmission in the Amazon. The Amazon is the most bio-diverse region on the planet with over 50,000 plant, animal, fish, and reptile species and over one million insect species. It produces 20 % of the world’s freshwater discharge, and contains over 100 billion tons of carbon [2, 3]. Maintaining the integrity of this ecosystem has local, regional, and global implications. Given the multi-scale impacts of the environment and the rapid demographic and economic changes occurring in the Amazon, we focus on vector-borne and zoonotic diseases (VBZD). The goals of this chapter are to (1) describe the unique nature of climate change in the Amazonia context; (2) discuss climate and environment factors that influence the VBZD–climate relationship; (3) provide malaria in the Peruvian Amazon as a specific example of a climate-sensitive VBZD; and (4) provide recommendations for research and action to address complexities of climate impacts on VBZD.

Climate Change in the Amazon

The Amazon basin is characterized by a pronounced east to west humidity gradient, with relatively dry conditions, seasonal precipitation, and occasional water stress in the eastern portions of the basin and more consistent, frequently flooded conditions to the west [2, 4]. The basin also experiences significant climate variability at inter-annual and inter-decadal time scales. The El Niño Southern Oscillation (ENSO) has a profound impact on precipitation, particularly in the eastern and northern Amazon, with warm phase ENSO (El Niño) associated with hot temperatures, suppressed wet season precipitation, and reduced stream flow [5, 6]. ENSO cycles have been shown to explain seasonal malaria in several areas of the Amazon, but with variable predictive accuracy [7–11]. Climate teleconnections associated with Atlantic Ocean sea surface temperatures (SST) also have a significant influence on precipitation. Oscillations in the tropical Atlantic SST gradient influence dry season precipitation in the eastern and southern portions of the basin [12], while the North Atlantic Oscillation (NAO) has been implicated in recent drought events [13]. These remote drivers of variability are overlain by a 28-year precipitation cycle that is characteristic to the Amazon but has not been fully explained, and some of the largest flooding events have resulted from coincident timing of La Niña with the wet phase of the 28-year cycle [5, 6]. Against this background, anthropogenic climate change already appears to be affecting the Amazon. Temperatures rose at a rate of 0.25° per decade

between 1960 and 1998 [14], and projections from global climate models (GCMs) suggest that additional warming on the order of 2–5 °C is likely over the twenty-first century [1]. This already wide range is a basin average that includes a projection for greater warming in the Amazon interior during the dry season, and that could be amplified to a warming of up to 8 °C if significant biophysical feedbacks associated with forest dieback become active [15]. Projections for precipitation are even less certain. Observed precipitation trends in recent decades have been mixed, with evidence of a significant drying trend in the northern Amazon and a slight wetting trend in the southern Amazon [6], and with no evidence of a statistically significant trend in the eastern Amazon on the whole [14]. Nevertheless, there is reason to expect that a warming global climate, likely accompanied by continued deforestation within the basin, will alter precipitation patterns in coming decades. A number of GCMs, for example, suggest that El Niño events will become stronger and more frequent over the twenty-first century, which would be expected to effect a drying of the northern and eastern Amazon. In the less humid eastern portion of the basin such a reduction in precipitation could promote ecological change from forest to savannah-like conditions, which would reduce transpiration and enhance the drying trend [15, 16]. Deforestation could exacerbate these trends, as large scale removal of forest trees through burning and timber harvests reduces transpiration and can have a negative feedback on precipitation [17]. Somewhat paradoxically, while large scale deforestation is expected to lead to reduced precipitation and more frequent drought, the local effects of deforestation on the water cycle can lead to increases in flood intensity, as a reduction in transpiration leads to an increase in runoff [4]. The combined effects of climate change and deforestation, then, could well lead to long-term drying over the entire eastern Amazon but to more severe floods during high flow events.

On the ensemble average, the GCM simulations included in the fourth assessment of the Intergovernmental Panel on Climate Change (IPCC) project that dry seasons are likely to intensify across much of the basin and that water stress will, on average, increase in the eastern Amazon over the twenty-first century. This ensemble average must be interpreted with extreme caution, however, as individual ensemble members differ widely in the spatial and temporal character of projected precipitation change, and as all GCM simulations included in the ensemble are implemented at coarse spatial scale (typically at 1–5° resolution, or ~110–550 km) and with physics parameterizations that often neglect important biogeochemical feedbacks known to be important in the Amazon. As such the range of GCM projections for precipitation is best understood as an indicator of the potential sensitivity of Amazon precipitation to twenty-first century climate change.

Given these limitations, application of GCMs to predict specific impacts of climate on vector or animal seasonal and spatial distribution becomes problematic as the characteristics that define breeding, feeding, and living space are defined at smaller scales and with sensitivities that are often within the range of GCM uncertainty. For example, Ruiz et al. developed a system of coupled mathematical nonlinear models to help explain complexities between climate parameters and malaria transmission risk [10]. Their approach integrates human population with pre-imag

(larva/pupa) and imago (adult) stages of *Anopheles* that respond to temperature, water availability, relative humidity (RH), and climate anomalies (e.g., *El Niño* events). While informative, the model parameterizes climate over an area larger than 20,000 km², which seriously limits the inference one can make about how climate is impacting the distribution of vectors.

Coupled Environment–Climate Impacts on VBZD

Given the biodiversity of the Amazon, it is not surprising that a number of VBZD circulate in the region. While climate change will surely affect the incidence of several pathogens, there exist several enabling factors that couple with climate parameters to exacerbate the effects of climate on human disease [4, 18–20]. Among these, LUCC and flooding are among the most important. LUCC, particularly deforestation, is a widely studied topic that remains void of a synthesized theoretical framework due to the vast differences in causes that can occur across temporal and spatial scales. For example, Walsh et al. [21] demonstrated variations in the relationship between geophysical and socio-demographic characteristics with cultivated land in Thailand when predicted land cover varied from 30 to 1,050 m pixels in size [21]. Similarly, Verberg and Veldkamp demonstrated that low-resolution approaches (i.e., large geographic areas) are ideal for identifying “hot zones” of land cover change, but high-resolution approaches (small geographic areas) provide insight into evolving land patterns and ecological consequences [22]. This is particularly relevant for VBZD—not only do researchers have difficulty applying GCM parameters to vector-borne disease predictions, but many studies have used coarse land cover grids to characterize vector habitats and disease risk, such as the 0.5° latitude–longitude grid in [23]. As described by Messina and Pan [20], this is an important ontological difference that exists between epidemiology and land change science that impedes understanding of proximate drivers of disease risk. That is, epidemiology often posits hypotheses anchored in traditional health and place organizational strategies, which incorporate land–climate data as discrete realizations of a continuous surface product. In contrast, land science operates using a geography, space, and time paradigm that do not provide appropriate individual-level variables necessary for inputs to epidemiology models. This confusion leads to issues of ecological fallacy as environmental variables are modeled at the individual level.

Another challenge posed by land–climate coupling is the intimate relationship between LUCC and human population dynamics. Studies of LUCC highlight important economic, social, cultural, political, and demographic factors influencing land change processes [24–28]. With continued high fertility rates and in-migration, population growth throughout the Amazon will continue, ensuring that LUCC will occur as people look to improve their livelihoods [29–32]. Livelihood choices are particularly overlooked as a root cause for VBZD risk—malaria is a prime example: studies that have shown a clear relationship between deforestation, vector density, and

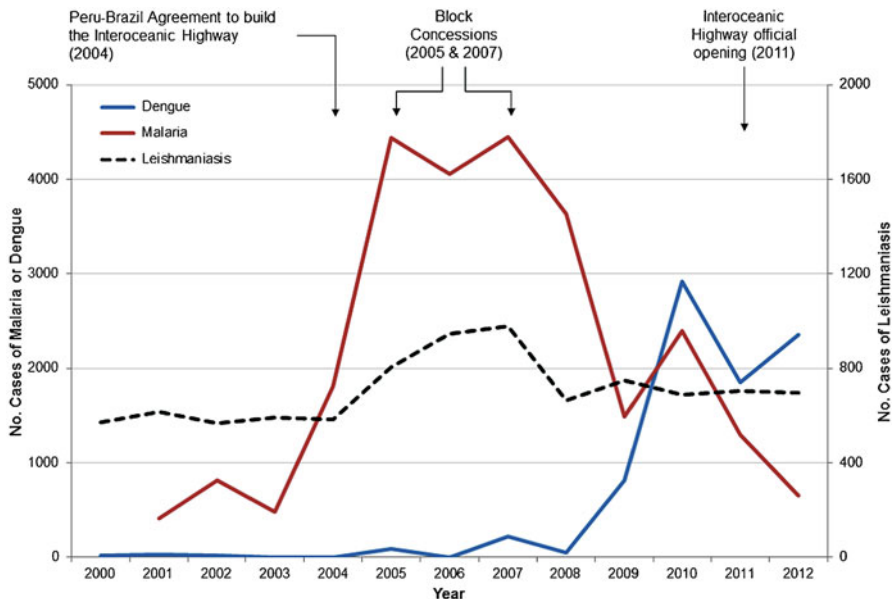


Fig. 11.1 Cases of malaria, dengue, and leishmaniasis in the region of Madre de Dios, Peru, between 2000 and 2012. Reported malaria cases are shown by the red line, dengue by blue line, and leishmaniasis by the dotted black line

malaria [33–35] are largely founded on the premise that ecological services are intimately tied to human dimensions of land cover change, as has been described by the Frontier Malaria Hypothesis [36, 37]. Castro et al. [37] clearly demonstrate the importance of Frontier Malaria as they related early features of colonization in Brazil with elevated malaria risk due to the establishment of new breeding sites for *Anopheles darlingi* and the introduction of a naïve human host. This is contrasted with recent research by Kosek and colleagues which demonstrated that epidemic malaria rates in northern Peru are associated with migration behaviors of families involved in occupational labor, primarily logging [38]. This human component of the land–climate relationship adds a layer of complexity that requires a comprehensive understanding of human livelihoods and vulnerability. Household livelihoods are tied to a number of factors that mediate household choices and, ultimately, drive malaria risk.

Perhaps the strongest mediating factor of VBZD in the Amazon is road construction and access. Roads alter the interface between humans and the environment by penetrating the forest like veins, pumping in migrants, occupational laborers, colonists, and altering species ecology and habitat. The multidimensional impact of roads is exemplified by the 2004 agreement between Brazil and Peru to construct the Interoceanic Highway connecting rural Amazonia farms in the western basin to Pacific and Atlantic Ocean ports. Construction began immediately with concession blocks awarded in 2005 and 2007. Figure 11.1 compares the timing of these large scale activities with malaria, dengue, and leishmaniasis disease rates reported by the Regional Ministry of Health of Madre de Dios (DIRESA-MDD) from 2000 to 2012.

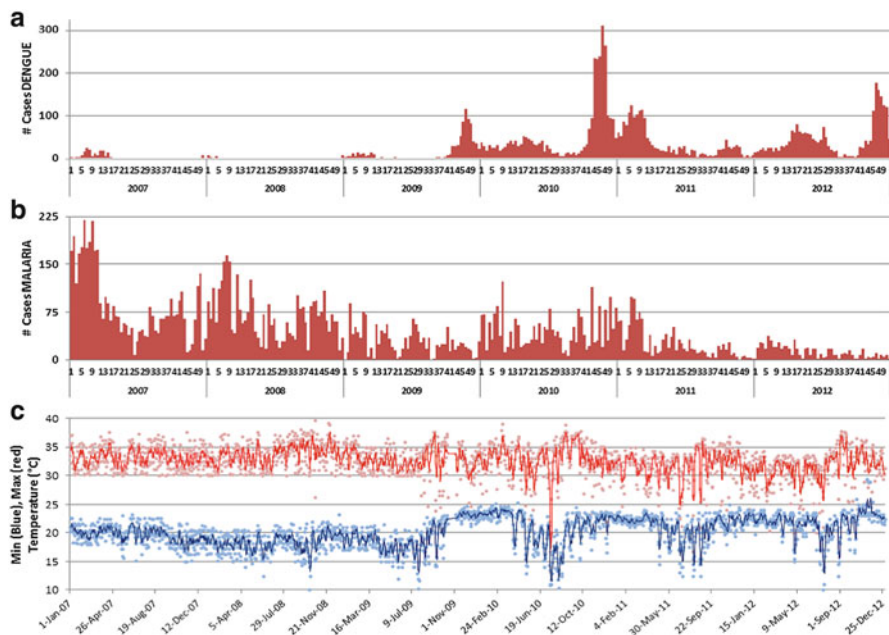


Fig. 11.2 Weekly surveillance case reports and daily minimum/maximum temperatures reported in Madre de Dios, Peru, between January 2007 and December 2012: (a) dengue cases; (b) malaria cases; and (c) temperature reported in Puerto Maldonado. Temperature reports were not available for the entire time series in Puerto Maldonado. For days with missing data, we used temperature reported from the Inapari weather station, which is approximately 180 km north

During construction, elevated rates of both malaria and leishmaniasis were experienced; in fact, between 2005 and 2008, reported cases were 470 % higher for malaria and 45 % higher for leishmaniasis compared to reported cases between 2000 and 2004. As road construction neared completion, reported cases of dengue skyrocketed, with cases primarily occurring in the city of Puerto Maldonado.

An important question is whether climate variability was related to these temporal patterns. Figure 11.2 compares the weekly surveillance reports for malaria and dengue from the DIRESA-MDD to daily minimum and maximum temperatures between 2007 and 2012. Dengue cases peaked in late 2009, 2010, and 2012, with a slight increase in late 2011 as well (i.e., November and December). Cases also appear to peak in early each year, roughly mid-January to March. This was true each year except 2012 when the peak appears to be shifted to April and May. Malaria trends are much more difficult to describe as they appear to have two distinct peaks in 2007 and 2008, but following widespread efforts to control malaria, the pattern begins to lack seasonality. Meteorological conditions appeared relatively stable between 2007 and mid-2009, but became much more erratic between 2010 and 2012 with less variance between minimum and maximum temperatures. The higher minimum temperature after 2010 may have contributed to the rise in dengue cases

seen regionally. There does not seem to be a strong signal between malaria and temperature during the high risk periods. However, finding an increase in malaria with overall VBZD is not expected at this gross-scale of analysis. Malaria infections in the Amazon are more long-lived in the human host than dengue (i.e., due to the nature of *Plasmodium vivax*), making it difficult to detect a strong signal between malaria and temperature at any given point in time. Also, this type of analysis lacks fine-scale resolution data on human mobility, urbanization, and land cover to fully evaluate the relationship. A specific demonstration of malaria associated with climate change will be discussed in the following section.

Epidemiology of Climate-Associated Vector-Borne Disease

Malaria is a prime example of a climate-sensitive VBZD disease. Malaria is endemic in over 100 countries with over 3.3 billion people at risk and, in 2010, caused an estimated 216 million episodes and 655,000 deaths [39]. It is transmitted by the female *Anopheles* mosquito and is the result of infection due to the presence of *Plasmodium* parasites (primarily, *P. vivax*, *P. falciparum*, *P. malariae*, and *P. ovale*) that cause fever, chills, fatigue, and headache, among other symptoms. In the Amazon, approximately 75 % of malaria is caused by *P. vivax*. Although less lethal when considering the acute infection, symptoms can be severe. There can be long-term pathology and *P. vivax* inflicts major impacts on human development ranging from impaired child growth and cognitive development, malnutrition, lower productivity in people of all ages, and disincentives for investment by industry and government [40–46]. As mentioned previously, malaria epidemiology in the Amazon can broadly be classified as either Frontier Malaria or occupational and migratory malaria. *Frontier Malaria* involves three stages of malaria risk in forest environments that follow stages of the frontier settlement process: [1] *Epidemic*, early years of agricultural colonization with high vector density, exposure, human population mobility, and weak institutional presence; [2] *Transition*, 3–10 years after settlement whereby land practices exhibit lower deforestation rates, population mobility slows, and residents begin to understand exposure risks; and [3] *Endemicity*, characterized by the integration of health services and infrastructure leading to improved socioeconomic status, such as urbanization, economic investments, and improvements in housing and income levels that result in less population mobility and environmental change [47, 48]. Occupational malaria refers to the idea that livelihood choices that involve human mobility for resource extraction, agriculture, or other activities far from one's home places that individual at greater risk for infection than if the individual stayed at home. Note that neither of these approaches directly integrate climate as a proximate determinant of infection.

Anopheles mosquitos are the vector capable of transmitting malaria. Only about 40 *Anopheles* species (of over 450) can transmit malaria to humans and only approximately 27 are effective transmitters [49, 50]. In the Amazon, the primary malaria vector is *A. darlingi* Root, 1926 [51–55]. *A. darlingi* is highly dependent on

water for its survival and breeding, is a typical riverine species that inhabits jungle and forest environments, and is mostly distributed in low altitude regions (<500 m above sea level) with high relative humidity [56, 57]. Vector competence is highly dependent on temperature and humidity, as *P. falciparum* and *P. vivax* are unable to develop at temperatures below 16 °C and 14.5 °C, respectively, but accelerate development when temperatures exceed 35 °C [50, 58–61]. High relative humidity (above 75 %) prolongs vector life and extends transmission (once infected), but below 35 % RH shortens their life span and prevents Plasmodium development. Vector density is directly proportional to the rate of malaria transmission, both from mosquitoes to humans and vice-versa [59]. Breeding site characteristics for *Anopheles* vary by species, but all depend on the presence of water for an average of 12–14 days to allow time to growth from egg to adult emergence.

Environmental determinants of larval and adult *Anopheles* habitat also vary by country [61]. For example, *A. darlingi* density has been reported to peak following maximum precipitation [62], the dry season [63], and wet–dry transition periods [34, 64]. Recently Barros et al. identified “microdams” (small obstructions to river flow such as tree trunks, branches, etc., that cause water to pool) to explain elevated adult and larval *A. darlingi* density during the dry season [65]. Ecologically altered landscapes (deforested, secondary forest, grass/cropland) [34, 53], forest fringes [66, 67], microclimate variation [62], as well as natural and artificial bodies of water (fish farms, rice fields, irrigation canals, etc.) [34] have all been identified as important breeding sites for anophelines, particularly *A. darlingi*.

Although climate change is not directly integrated into the human components of transmission, climate, coupled with land and flooding, is directly correlated with *Anopheles* species composition and abundance. Floods can significantly alter the epidemiology of disease transmission. Flood areas are predictable topographical features of the landscape where usual seasonal fluctuations are related to the usual patterns of infection. However, large floods, such as those that occurred throughout a large part of the western Amazon in 2012, dramatically increased the number of reported cases following several years of progress in reducing malaria burden. Between 2000 and 2006, reported cases of malaria in the region of Loreto averaged around 44,000 cases annually. During the ensuing years, reported cases dropped by about 10,000 each year and remained at 10,000–11,000 cases in 2010 and 2011. February and March of 2012 were among the wettest months in the Peruvian Amazon, resulting in one of the largest historical floods in Loreto. By the end of 2012, the number of reported cases of malaria had reached 25,000. Cases peaked between April and August, with an abnormally high second peak between October and November.

Climate and environment changes were associated with increases in malaria cases in the Peruvian Amazon region in three ways. First, elevated rainfall and high temperatures likely contributed to an expanded transmission season. With more water available, mosquito density could remain sufficiently high throughout the year, rather than dissipating between September and January. Second, flood waters altered the interface between people and *Anopheles* exposure. Families were displaced and moved into temporary housing, often without bednets or other

protective measures they may have against biting insects. Even animals, such as rodents, mice, and snakes, were similarly displaced from forested areas and moved into the populated communities. Third, public health efforts became focused on providing services to displaced families, and distracted from the prior surveillance interventions that gave some protection against VBZD transmission. All available resources were being used for dealing with injuries, snakebites, acute infections, and sanitation problems. This allowed malaria cases to go undetected and likely untreated, further enabling the increase of malaria transmission.

Malaria-Climate Change Case Study

During the 1990s, Loreto experienced an epidemic malaria outbreak that peaked at 121,268 cases in 1997 [68]. Cases were initially reported in the towns of Rumococha and Zungarococha in 1991, located about 10 and 20 km from the central city of Iquitos, respectively. Malaria rates subsequently transitioned to endemic levels ranging from 30,000 to 50,000 cases annually between 2000 and 2008, and then rapidly fell to approximately 10,000 cases annually in 2010 and 2011. The transition from epidemic malaria was aided by policies of the Peruvian Ministry of Health that focused intervention efforts on case detection, treatment, and bednet distribution. Specifically, the Ministry of Health focus is on febrile individuals because malaria infection is much more likely in individuals with fever than in individuals without fever. Individuals are instructed and reminded to go to their community health center if they have a fever, headache, or other symptoms that might indicate they have malaria. Individuals suspected to have malaria are diagnosed with a blood smear that is read by a trained microscopist. If the test is positive, treatment is prescribed *at no cost*, and treatment is specific to the malaria species found (i.e., *P. falciparum* or *P. vivax*). Malaria treatment drugs are not available in private pharmacies. In 2012, the number of malaria cases in Loreto unexpectedly rose to approximately 25,000 cases following massive flooding that significantly displaced a large proportion of the population.

A clinical study initiated in 2003 by Branch and colleagues in Zungarococha [69] is a microcosm of the underlying transition observed and new malaria epidemic. Zungarococha has an average population of around 2,200 persons across a 4 km² area that is organized into four villages. Beginning in 2004, the population was followed each full year until 2012. Although population size has been relatively stable, population turnover has been rapid. Seventy five percent of the 2,340 residents in 2012 who were older than 4 years old in 2007 participated in the 2007 (pop. 2,145) study. Most of the population works within the community in occupations related to local farming (agriculture and fish) and trading.

The Zungarococha study was designed as a year-round active case surveillance coupled with passive detection in the community health center [69]. Active case detection involved a minimum of six visits to each household during the (6-month) rainy season and testing a blood sample for malaria from all persons in the house

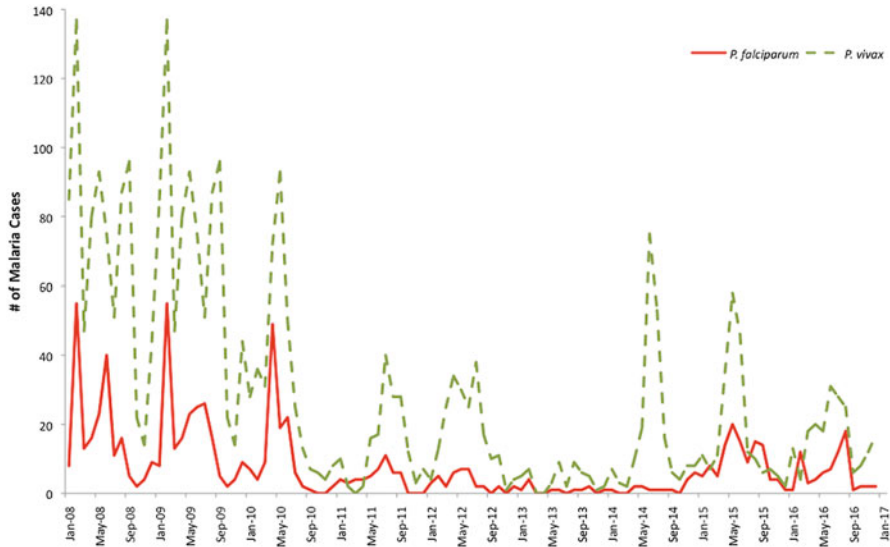


Fig. 11.3 Number of *P. falciparum* (solid red line) and *P. vivax* (hatched green line) in Zungarococha detected with active case surveillance between 2004 and 2012. Population size under surveillance varied from 2,145 to 2,340; however, the major flood event in April 2012 limited some surveillance and detection until June 2012

regardless of symptoms. Additionally, any person reporting to the community health center with fever or other malaria-like symptoms was tested for malaria parasites. More than 91 % of the community participated in the study each year (2004–2012).

Figure 11.3 shows the number of *P. falciparum* and *P. vivax* cases each month detected in Zungarococha. Strong seasonal patterns were observed between 2004 and 2006, followed by sharp declines in 2007 and 2008. These declines can be partly explained by increased treatment of symptomatic and asymptomatic cases identified during active case detection as well as some persons developing immunity [69–73]. Between 2009 and 2010, *P. falciparum* was nearly eradicated and *P. vivax* infections were at historically low levels in 2009.

In 2010 and 2011, *P. vivax* cases spiked during the normal transmission season (April–July), but by mid-2011 it was apparent that *P. falciparum* was also returning. Between 2011 and 2012, there was a dramatic increase in malaria infections, which resulted in the MOH declaring a new malaria epidemic in Loreto (Fig. 11.3). What happened during this interval? How was the climate changing? Were there coupled climate effects? While the study cannot answer all these questions, there are three distinct changes that occurred over the 8-year interval to begin understanding the underlying drivers. First, as mentioned previously, population turnover was rapid. Between 2004 and 2010 a large proportion of the population migrated out of Zungarococha, being replaced primarily by new births and unexposed new immigrants to the community. For example, after 2010, the study enrolled 124 new individuals who either immigrated or were born into the community. The number of

susceptible persons to malaria (either never been infected or infected more than 5 years prior) likely reached a maximum in 2009 and 2010 following several years of declining rates (Branch, unpublished).

Second, significant climate variation was experienced in the form of temperature change and water stress, resulting in some regions of the Amazon to have extensive droughts in 2005 and 2010 followed by a major flood in 2012. Notably, the 2005 drought at the time was considered one of the worst on record, but impacted primarily the southwest Amazon basin, leaving Loreto with relatively normal rainfall levels [74]. However, Loreto felt the full effect of the 2010 drought, which was much worse than 2005 and brought higher temperatures, fewer clouds, and less rainfall [75]. This was followed by one of the worst floods in the history of the Amazon that began in late 2011 with elevated precipitation. In Zungarococha, improved case detection and treatment, development of immunity, and extensive drought conditions likely translated into fewer infected persons and both fewer susceptible and infected *Anopheles*. This would reduce the force of infection over time. The higher temperatures over time might have caused a more rapid development of malaria in *Anopheles* mosquitoes. Because this is a riverine environment, there were at least some mosquito breeding sites available. As rivers began to swell in late 2011 and reached flood levels in 2012, *Anopheles* densities increased throughout the year, extending the malaria transmission season into usual non-malaria months of September, October, November, December, and January in Zungarococha, during which 24 and 33 cases of *P. falciparum* and *P. vivax* were found, respectively. Figure 11.4 shows the time series of *P. falciparum* cases and air temperature detected prior to, during, and after the drought in Zungarococha (2007–2013). As the figure shows, following the drought, there was high malaria incidence coupled with an expanded malaria transmission season.

This type of transmission season expansion can have long lasting consequences on VBZD epidemiology and evolution. Under normal seasonal conditions, there is a barrier to the spread and evolution of the malaria parasite by there being several months that are not hospitable to the malaria parasite and/or the mosquitoes; however, when transmission occurs continuously throughout the year, the malaria parasite can evolve to become more virulent [76]. Also, if high vector density persists throughout the year, this could facilitate reemergence of the disease as infected individuals reenter the area. The effect could make a balanced endemic transmission system change to one that either has higher endemic transmission or results in a new epidemic. In Zungarococha, the endemic transmission and near eradication that existed before 2011 became classified as epidemic in late 2011 due to the high number of *P. falciparum* cases in May 2011 (20 cases).

Third, the 2012 flood altered the structural response to malaria. In March 2012, the regional government of Loreto declared a state of emergency as several thousand people were displaced from their homes. At the regional level, the ability of the MOH to conduct passive detection of individuals with fever was significantly diminished. Local clinics and hospitals were distracted by injuries and illnesses other than malaria during these months (Sihuincha, unpublished). In Zungarococha, study protocols faltered as only 1,154 individuals were contacted and tested during

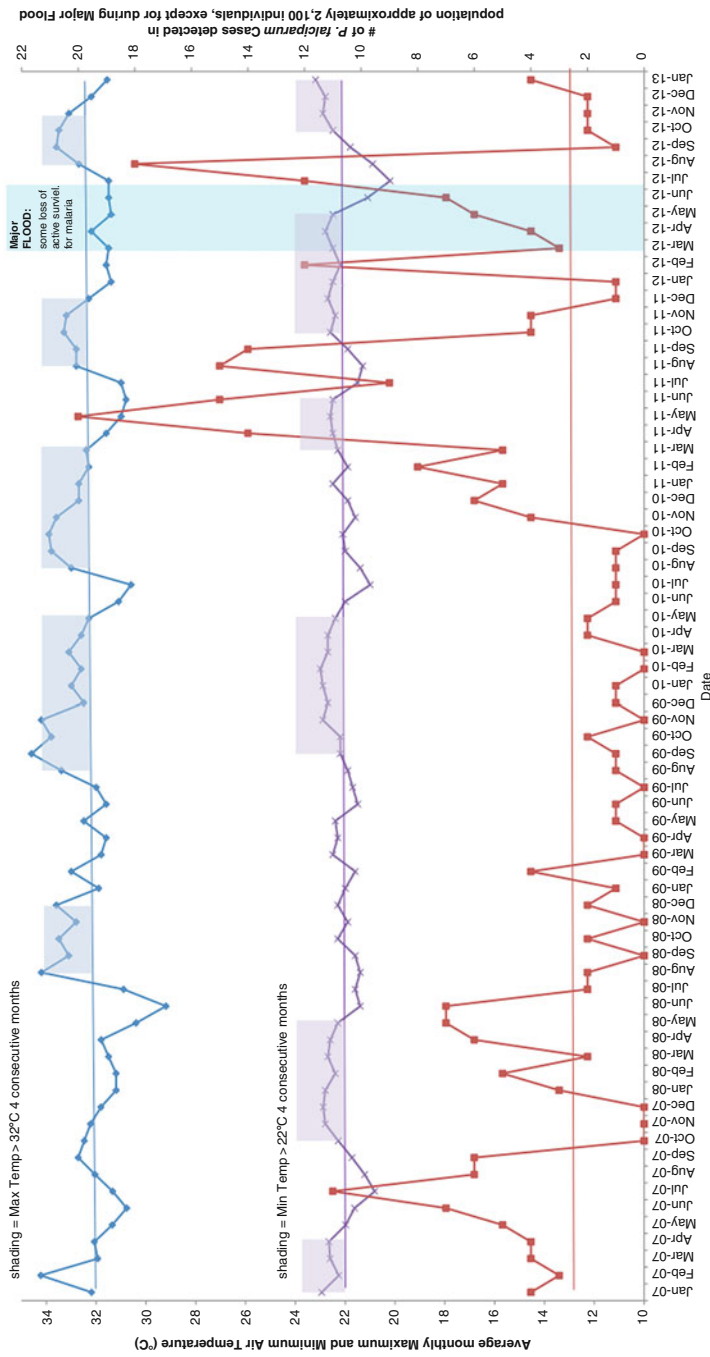


Fig. 11.4 Average monthly temperature maximum (*top blue line*) and minimum (*middle purple line*), and monthly *P. falciparum* malaria cases detected in Zungarococha. Temperature measures were collected by the National Weather Station located within the district of Zungarococha and provided by Dr. Moises Sihuincha and colleagues

this period, compared to an expected minimum of 1,800. Approximately 600 individuals were absent from their homes and could not be located; 532 individuals who returned to their homes by June 2012 were asked if they were diagnosed with malaria while they were away, but none reported a diagnosis or treatment. In prior years, the study found that approximately 30 % of the malaria infections detected were asymptomatic [69–73], all of which were treated with antimalaria drugs to help stop transmission. Even throughout active surveillance, the extent of the Iquitos flood likely resulted in some missed malaria detection due to displacement of individuals and distraction of health centers to focus on acute injuries, bites, and illnesses associated with the flood.

Here, we presented a specific case where malaria incidence responds positively to the climate–VBZD relationship through an expanded transmission season, flooding that contributed to *A. darlingi* breeding habitat and density, and disruption of active and passive case detection. Having an ongoing active case surveillance protocol in each year between 2004 and 2012 enabled us to observe this relationship. With more study, improved clarity of the associations between climate change and VBZD can be obtained. It is noteworthy that the climate–VBZD relationship does not have to increase disease incidence. For example, after the floods there was an increased detection of dengue virus infections in many communities, including Zungarococha. Dengue is spread by *Aedes aegypti*, which is considered hardier, more resistant to heavy rains, and more capable of breeding in small, artificial containers of water compared to anopheles [77]. Therefore, we might observe competition between vectors, resulting in differential VBZD risk. We are only at the beginning of understanding this complex climate–environment and disease relationship and ways they will impact the way we live, flourish, and fail.

Future Directions in Research

There have been several attempts to describe a generalized framework for understanding environmental relationships with disease transmission [18–20, 78]. Undoubtedly, studying VBZD is complex. Transmission results from interaction between humans, vectors, and pathogens that are mediated by environmental conditions operating at multiple geographic and temporal scales, which are likewise impacted by people [34, 49, 79–81]. Frontier Malaria is a clear example of how these interactions persist in the Amazon and how climate variation and change can significantly alter the natural course of disease transmission. Changes in climate not only alter the expected seasonal temperature and rainfall patterns, land–climate coupling means that if certain thresholds are breached, local and regional impacts in land surface characteristics and surface hydrology are also significantly affected. This was the case in Zungarococha, where declining malaria rates were impacted by major flooding, which caused a multitude of problems: extended vector breeding habitat over space and time, altered socio-demographic behavior of individuals, and

weakening of the health system to respond to malaria. Admittedly, positive human behavioral response can occur, such as increased awareness of the infection, prevention, or participation in intervention strategies; however, in resource-poor environments, the likelihood of these types of positive responses is, unfortunately, minimal.

One of the most challenging aspects of VBZD prevention and control is the interdisciplinary nature of transmission and causation. Collaborations between researchers in physical science, epidemiology, and social science to better understand disease dynamics have advanced considerably in recent years. These collaborations have been encouraged by interdisciplinary funding opportunities supported by NIH, NSF, NASA, and other funding agencies, and they have yielded significant improvements in integrated assessments of disease process, predictability, and prevention. For the most part, however, these collaborations have involved diverse experts bringing their traditional analytical tools and study designs to the problem of VBZD, with minimal feedback across disciplines that limit more effective integration of techniques. For example, the epidemiological triangle of disease causation (agent–host–environment) often characterizes disease risk as discrete events between agents and hosts. Environment, which is often a distant third wheel, is usually categorized as the place where agent–host interactions occur. This is where a large disconnect exists between epidemiology and land/climate scientists. In epidemiology, environment is a discrete space (e.g., community, political/administrative boundary) that is statistically modeled as a predictive variable of infection. However, land/climate scientists recognize that environmental characteristics are derived from modeled products (e.g., satellite imagery) and the inputs used in epidemiology are actually continuous in space and time. The severing of a continuous ecological biome to examine discrete events can result in ecological fallacies or at least spurious relationships between environment and disease.

In general, the application of satellite imagery to VBZD represents a core science of opportunity. While data from satellite sensors are of interest to VBZD risk monitoring and prediction, these sensors were almost never designed with any specific consideration for what measurement characteristics would be most useful for VBZD research or surveillance. Similarly, climate models are almost never optimized for VBZD applications in their resolution, periods of analysis, or even in the process simulations and model outputs. Of course, some limitations in these physical science techniques are difficult to overcome—high-resolution satellite-derived soil moisture measurements are extremely expensive and sometimes impossible to obtain, and climate models are computationally intensive and are plagued by possibly irreducible uncertainties for both seasonal prediction and future climate change projections. Recognizing this, epidemiologists might need to alter the study designs and/or surveillance networks to take full advantage of the model results and satellite observations that are available. This suggests that collaborations that currently occur primarily at the scale of small research teams need to be moved upstream into satellite mission design, climate model development, and planning for health monitoring systems, so that the interdisciplinary nature of VBZD problems is recognized in the design of the required research tools as well as in their application.

References

1. Christensen, J. H., B. Hewitson, et al. (2007). Regional Climate Projections. In: S. Solomon, D. Qin, M. Manning et al., editors. *Climate change 2007: The physical science basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*. Cambridge: Cambridge University Press.
2. Malhi Y, Wood D, et al. The regional variation of aboveground live biomass in old-growth Amazonian forests. *Glob Chang Biol*. 2006;12:1107–38.
3. Saatchi SS, Houghton RA, et al. Distribution of aboveground live biomass in the Amazon Basin. *Glob Chang Biol*. 2007;13:816–37.
4. Davidson EA, de Araujo AC, et al. The Amazon basin in transition. *Nature*. 2012;481(7381):321–8.
5. Coe MT, Costa MH, et al. Long-term simulations of discharge and floods in the Amazon Basin. *J Geophys Res*. 2002;107(D20):1–17.
6. Marengo JA. Interdecadal variability and trends of rainfall across the Amazon basin. *Theor Appl Climatol*. 2004;78(1–3):79–96.
7. Bouma MJ, Dye C. Cycles of malaria associated with El Nino in Venezuela. *JAMA*. 1997;278(21):1772–4.
8. Bouma MJ, Poveda G, et al. Predicting high-risk years for malaria in Colombia using parameters of El Nino Southern Oscillation. *Trop Med Int Health*. 1997;2(12):1122–7.
9. Gagnon AS, Smoyer-Tomic KE, et al. The El Nino southern oscillation and malaria epidemics in South America. *Int J Biometeorol*. 2002;46(2):81–9.
10. Ruiz D, Poveda G, et al. Modelling entomological-climatic interactions of Plasmodium falciparum malaria transmission in two Colombian endemic-regions: contributions to a National Malaria Early Warning System. *Malar J*. 2006;5:66.
11. Hanf M, Adenis A, et al. The role of El Nino Southern Oscillation (ENSO) on variations of monthly Plasmodium falciparum malaria cases at the Cayenne General Hospital, 1996–2009, French Guiana. *Malar J*. 2011;10:100.
12. Li W, Fu R, et al. Rainfall and its seasonality over the Amazon in the 21st century as assessed by the coupled models for the IPCC AR4. *J Geophys Res*. 2006;111(D2):2156–202.
13. Marengo JA, Nobre CA, et al. Hydroclimate and ecological behaviour of the drought of Amazonia in 2005. *Philos T Roy Soc B*. 2008;363:1773–8.
14. Malhi Y, Wright J. Spatial patterns and recent trends in the climate of tropical rainforest regions. *Philos T Roy Soc B*. 2004;359(1443):311–29.
15. Betts RA, Cox PM, et al. The role of ecosystem-atmosphere interactions in simulated Amazonian precipitation decrease and forest dieback under global climate warming. *Theor Appl Climatol*. 2004;78:157–75.
16. Salazar LF, Nobre CA, et al. Climate change consequences on the biome distribution in tropical South America. *Geophys Res Lett*. 2007;34(9), L09708.
17. Coe MT, Costa MA, et al. The influence of historical and potential future deforestation on the stream flow of the Amazon river: land surface processes and atmospheric feedbacks. *J Hydrol*. 2009;369:165–74.
18. Patz JA, Daszak P, et al. Unhealthy landscapes: policy recommendations on land use change and infectious disease emergence. *Environ Health Perspect*. 2004;112(10):1092–8.
19. Patz JA, Campbell-Lendrum D, et al. Impact of regional climate change on human health. *Nature*. 2005;438(7066):310–7.
20. Messina J, Pan WK. Different ontologies: land change science and health research. *Curr Opin Environ Sust*. 2013;5:1–7.
21. Walsh S, Evans TP, et al. Scale dependent relationships between population and environment in Northeast Thailand. *Photogramm Eng Remote Sensing*. 1999;65(1):97–105.
22. Verberg PH, Veldkamp A. Projecting land use transitions at forest fringes in the Philippines at two spatial scales. *Landsc Ecol*. 2004;19:77–98.

23. Ermert V, Fink AH, et al. The impact of regional climate change on malaria risk due to greenhouse forcing and land-use changes in tropical Africa. *Environ Health Perspect*. 2012;120(1):77–84.
24. Contreras-Hermosillo A. The underlying causes of forest decline. CIFOR Occasional Paper 30. Bogor Barat: Center for International Forestry Research; 2000.
25. Geist HJ, Lambin EF. What drives tropical deforestation? A meta-analysis of proximate and underlying causes of deforestation based on sub-national case study evidence. Louvain-la-Neuve: LUC International Project Office; 2001. p. 116.
26. Pan WK, Walsh SJ, et al. Farm-level models of spatial patterns of land use and land cover dynamics in the Ecuadorian Amazon. *Agric Ecosyst Environ*. 2004;101:117–34.
27. Asner GP, Knapp DE, et al. Selective logging in the Brazilian Amazon. *Science*. 2005;310(5747):480–2.
28. Swenson JJ, Carter CE, et al. Gold mining in the Peruvian Amazon: global prices, deforestation, and mercury imports. *PLoS One*. 2011;6(4):e18875.
29. Carr DL, Pan WK, et al. Declining fertility on the frontier: The Ecuadorian Amazon. *Popul Environ*. 2006;28:17–39.
30. de Sherbinin A, Vanwey LK, et al. Rural household demographics, livelihoods, and the environment. *Glob Environ Chang*. 2008;18:38–53.
31. Ishida K, Stupp P, et al. Stalled decline in fertility in Ecuador. *Int Perspect Sex Reprod Health*. 2009;35(4):203–6.
32. Potter JE, Schmertmann CP, et al. Mapping the timing, pace, and scale of the fertility transition in Brazil. *Popul Dev Rev*. 2010;36(2):283–307.
33. Yasuoka J, Levins R. Impact of deforestation and agricultural development on Anopheline ecology and malaria epidemiology. *Am J Trop Med Hyg*. 2007;76(3):450–60.
34. Vittor AY, Pan WK, et al. Linking deforestation to malaria in the Amazon: Characterization of the breeding habitat of the principal malaria vector, *Anopheles darlingi*. *Am J Trop Med Hyg*. 2009;81(1):5–12.
35. Olson SH, Gangnon R, et al. Deforestation and malaria in Mancio Lima County, Brazil. *Emerg Infect Dis*. 2010;16(7):1108–15.
36. Sawyer D. Economic and social consequences of malaria in new colonization projects in Brazil. *Soc Sci Med*. 1993;37(9):1131–6.
37. de Castro MC, Monte-Mor RL, et al. Malaria risk on the Amazon frontier. *Proc Natl Acad Sci USA*. 2006;103(7):2452–7.
38. Parker B, Olortegui MP, et al. Hyperendemic malaria transmission in areas of occupation-related travel in the Peruvian Amazon. *Malar J*. 2013;12:178.
39. World Health Organization. World malaria report: 2011. Geneva: WHO, Global Malaria Programme; 2011. p. 248.
40. Mendis K, Sina BJ, et al. The neglected burden of *P. vivax* malaria. *Am J Trop Med Hyg*. 2001;164:97–106.
41. Breman JG, Alilio MS, et al. Conquering the intolerable burden of malaria: what's new, what's needed: a summary. *Am J Trop Med Hyg*. 2004;71(2 Suppl):1–15.
42. Duarte EC, Gyorkos TW, et al. Epidemiology of malaria in a hypoendemic Brazilian Amazon migrant population: a cohort study. *Am J Trop Med Hyg*. 2004;70(3):229–37.
43. Vitor-Silva S, Reyes-Lecca RC, et al. Malaria is associated with poor school performance in an endemic area of the Brazilian Amazon. *Malar J*. 2009;8:230.
44. Fernando SD, Rodrigo C, et al. The “hidden” burden of malaria: cognitive impairment following infection. *Malar J*. 2010;9:366.
45. Lee G, Yori P, et al. Comparative effects of vivax malaria, fever and diarrhoea on child growth. *Int J Epidemiol*. 2012;41(2):531–9.
46. Pan, W. K. (unpublished). Population, Health and Environment Dynamics in the Peruvian Amazon (ongoing research). <https://globalhealth.duke.edu/projects/population-environment-dynamics-influencing-malaria-risk-peruvian-amazon>.

47. Sawyer DO, Sawyer DR. Malaria on the Amazon frontier: economic and social aspects of transmission and control. In: Chen LL, Kleinman A, Ware NC, editors. *Advancing health in developing countries*. Belo Horizonte, Brazil: Centro de Desenvolvimento e Planejamento Regional (CEDEPLAR); 1987. p. 116.
48. Sawyer DR, Sawyer DO. The malaria transition and the role of social science research. In: Chen LL, Kleinman A, Ware NC, editors. *Advancing health in developing countries*. New York: Auburn House; 1992. p. 105–27.
49. Service MW. Mosquitoes (Culicidae). In: Lane RP, Crosskey RW, editors. *Medical insects and arachnids*. London: Chapman & Hall; 1993. p. 120–240.
50. Wernsdorfer WH. Global challenges of changing epidemiological patterns of malaria. *Acta Trop*. 2012;121(3):158–65.
51. de Arruda M, Carvalho MB, et al. Potential vectors of malaria and their different susceptibility to *Plasmodium falciparum* and *Plasmodium vivax* in northern Brazil identified by immunoassay. *Am J Trop Med Hyg*. 1986;35(5):873–81.
52. Deane LM. Malaria vectors in Brazil. *Mem Inst Oswaldo Cruz*. 1986;81(Suppl II):5–14.
53. Vittor AY, Gilman R, et al. The effect of deforestation on the human biting rate of *Anopheles darlingi*, the primary vector of falciparum malaria in the Peruvian Amazon. *Am J Trop Med Hyg*. 2006;74(1):3–11.
54. Sinka ME, Rubio-Palis Y, et al. The dominant *Anopheles* vectors of human malaria in the Americas: occurrence data, distribution maps and bionomic precis. *Parasit Vectors*. 2010;3:72.
55. Hiwat H, Bretas G. Ecology of *Anopheles darlingi* Root with respect to vector importance: a review. *Parasit Vectors*. 2011;4:177.
56. Rozendaal J. Observations on the distribution of anophelines in Suriname with particular reference to the malaria vector *Anopheles darlingi*. *Mem Inst Oswaldo Cruz*. 1990;85(2):221–34.
57. Rubio-Palis Y, Zimmerman RH. Ecoregional classification of malaria vectors in the neotropics. *J Med Entomol*. 1997;34(5):499–510.
58. Wernsdorfer WH. *The importance of malaria in the world*. New York: Academic; 1980.
59. Gage KL, Burkot TR, et al. Climate and vectorborne diseases. *Am J Prev Med*. 2008;35(5):436–50.
60. Gething PW, Van Boeckel TP, et al. Modelling the global constraints of temperature on transmission of *Plasmodium falciparum* and *P. vivax*. *Parasit Vectors*. 2011;4:92.
61. Montoya-Lerma J, Solarte YA, et al. Malaria vector species in Colombia: a review. *Mem Inst Oswaldo Cruz*. 2011;106 Suppl 1:223–38.
62. Magris M, Rubio-Palis Y, et al. Vector bionomics and malaria transmission in the Upper Orinoco River, Southern Venezuela. *Mem Inst Oswaldo Cruz*. 2007;102(3):303–11.
63. Camargo LM, Dal Colletto GM, et al. Hypoendemic malaria in Rondonia (Brazil, western Amazon region): seasonal variation and risk groups in an urban locality. *Am J Trop Med Hyg*. 1996;55(1):32–8.
64. Galardo AK, Zimmerman RH, et al. Seasonal abundance of anopheline mosquitoes and their association with rainfall and malaria along the Matapi River, Amapa, [corrected] Brazil. *Med Vet Entomol*. 2009;23(4):335–49.
65. Barros FS, Arruda ME, et al. Spatial clustering and longitudinal variation of *Anopheles darlingi* (Diptera: Culicidae) larvae in a river of the Amazon: the importance of the forest fringe and of obstructions to flow in frontier malaria. *Bull Entomol Res*. 2011;101(6):643–58.
66. Hutchings RS, Sallum MA, et al. Mosquito (Diptera: Culicidae) diversity of a forest-fragment mosaic in the Amazon rain forest. *J Med Entomol*. 2011;48(2):173–87.
67. Manguin S, Roberts D, et al. Characterization of *Anopheles darlingi* (Diptera: Culicidae) larval habitats in Belize, Central America. *J Med Entomol*. 1996;33(2):205–11.
68. Aramburu J, Asayag CR, et al. Malaria reemergence in the Peruvian Amazon region. *Emerg Infect Dis*. 1999;5(2):209–15.
69. Branch O, Casapia WM, et al. Clustered local transmission and asymptomatic *Plasmodium falciparum* and *Plasmodium vivax* malaria infections in a recently emerged, hypoendemic Peruvian Amazon community. *Malar J*. 2005;4:27–33.

70. Torres KJ, Clark EH, et al. Antibody response dynamics to the *Plasmodium falciparum* conserved vaccine candidate antigen, merozoite surface protein-1 C-terminal 19kD (MSP1-19kD), in Peruvians exposed to hypoendemic malaria transmission. *Malar J.* 2008;7:173.
71. Branch OH, Sutton PL, et al. *Plasmodium falciparum* genetic diversity maintained and amplified over 5 years of a low transmission endemic in the Peruvian Amazon. *Mol Biol Evol.* 2011;28(7):1973–86.
72. Sutton PL, Torres LP, et al. Sexual recombination is a signature of a persisting malaria epidemic in Peru. *Malar J.* 2011;10:329.
73. Clark EH, Silva CJ, et al. *Plasmodium falciparum* malaria in the Peruvian Amazon, a region of low transmission, is associated with immunologic memory. *Infect Immun.* 2012;80(4):1583–92.
74. Saleska SR, Didan K, et al. Amazon forests green-up during 2005 drought. *Science.* 2007;318(5850):612.
75. Lewis SL, Brando PM, et al. The 2010 Amazon drought. *Science.* 2011;331(6017):554.
76. Mackinnon MJ, Read AF. Virulence in malaria: an evolutionary viewpoint. *Philos Trans R Soc Lond B Biol Sci.* 2004;359(1446):965–86.
77. Wong J, Morrison AC, et al. Linking oviposition site choice to offspring fitness in *Aedes aegypti*: consequences for targeted larval control of dengue vectors. *PLoS Negl Trop Dis.* 2012;6(5):e1632.
78. Lambin EF, Tran A, et al. Pathogenic landscapes: Interactions between land, people, disease vectors, and their animal hosts. *Int J Health Geogr.* 2010;9:54.
79. van Lieshout M, Kovats RS, et al. Climate change and malaria: analysis of the SRES climate and socio-economic scenarios. *Global Environ Change.* 2004;14(1):87–99.
80. da Silva-Nunes M, Codeco CT, et al. Malaria on the Amazonian frontier: transmission dynamics, risk factors, spatial distribution, and prospects for control. *Am J Trop Med Hyg.* 2008;79(4):624–35.
81. da Silva-Nunes M, Moreno M, et al. Amazonian malaria: asymptomatic human reservoirs, diagnostic challenges, environmentally driven changes in mosquito vector populations, and the mandate for sustainable control strategies. *Acta Trop.* 2012;121(3):281–91.

Chapter 12

Climate Variability and Change: Food, Water, and Societal Impacts

Jonathan Patz

Abstract Climatologists now state with a high degree of certainty that global climate change is real, is advancing more rapidly than expected, and is caused by human activities, especially through fossil fuel combustion and deforestation. Environmental public health researchers, in assessing future projections for Earth's climate, have concluded that, on balance, adverse health outcomes will predominate under these changed conditions. The number of pathways through which climate change can affect the health of populations makes this environmental hazard one of the most perilous and intricate challenges that we face in this century. By contrast, the potential health co-benefits from departing from our current fossil fuel-based economy may offer some of the most beneficial health opportunities in over a century.

Keywords Climate change • Societal impact of climate change • Climate change and food and water • Sea level rise in climate change • Land use effects on health • Human health and climate change

Long-term climate change can be observed as a signal against a background of natural climate variability. Since instrument records are available only for the recent past (a period of less than 150 years), previous climates must be deduced from paleoclimatic records such as tree rings, pollen series, faunal, and floral abundances in deep-sea cores, isotope analyses of coral and ice cores, and diaries and other documentary evidence. Surface temperatures in the mid to late twentieth century appear to have been higher than they were during any similar period in the last 600 years in most regions, and in at least some regions, warmer than in any other century for several thousand years [1].

J. Patz, M.D., M.P.H. (✉)
Global Health Institute, University of Wisconsin in Madison, Madison, WI, USA
e-mail: patz@wisc.edu

Temperature changes are accelerating rapidly. During the century 1906–2005, global average temperature warmed by 0.74 °C. According to the United Nations Intergovernmental Panel on Climate Change (IPCC), this trend is accelerating, and by 2,100 average global temperatures are projected to increase between 1.8 and 4.0 °C. The rate of change in climate is faster now than in any period in the last thousand years.

Climate Change, Sea Level Rise, and Extremes in Climate Variability

Changing temperatures are only part of the story. Hot temperatures evaporate soil moisture more quickly, thereby leading to severe droughts, while warmer air can hold more moisture and result in heavy precipitation events; such “hydrologic extremes” (floods and droughts) accompany warming temperatures within future climate change scenarios; both extremes are a concern to global public health.

The insurance and reinsurance industry is also worried about climate change. In 2011, the United States experienced 14 weather-related disasters exceeding \$1 billion each in damage costs, a new record. Weather disasters since 1996 have been nearly twice as numerous and costly compared to the period from 1980 to 1995 [2]. Of course, this growth is in part a result of development in vulnerable (especially coastal) areas, but the trend in weather extremes is part of the story.

The extent of Arctic sea ice has declined by 7.4 % per decade and snow cover and glaciers have diminished in both hemispheres. In 2012, Arctic sea ice fell below four million square kilometers, having melted to its lowest amount in the satellite record (Fig. 12.1) [3]. Terrestrial glaciers and Antarctic ice sheets are also melting, releasing vast amounts of water into the oceans, raising sea levels and potentially altering the flow of ocean currents. Sea levels have risen on average approximately 2 mm per year since 1961. According to the IPCC, in 90 years sea levels will rise between 18 and 59 cm.

Sea Surface Temperatures and Hurricanes

Sea surface temperatures have steadily increased over the last century, and more sharply over the last 35 years. The highest average sea surface temperatures were recorded from 1995 to 2004 [4]. Warmer ocean surface temperatures affect wind velocities in hurricanes. Hurricanes form only in regions where sea surface temperatures are above 26 °C [5, 6]. Since the 1950s, overall hurricane activity in the North Atlantic has doubled and the Caribbean has experienced a fivefold increase [7]. Hurricane intensity may also be associated with warmer temperatures [8, 9]. As Hurricane Katrina demonstrated in 2005, such events have enormous significance for public health.

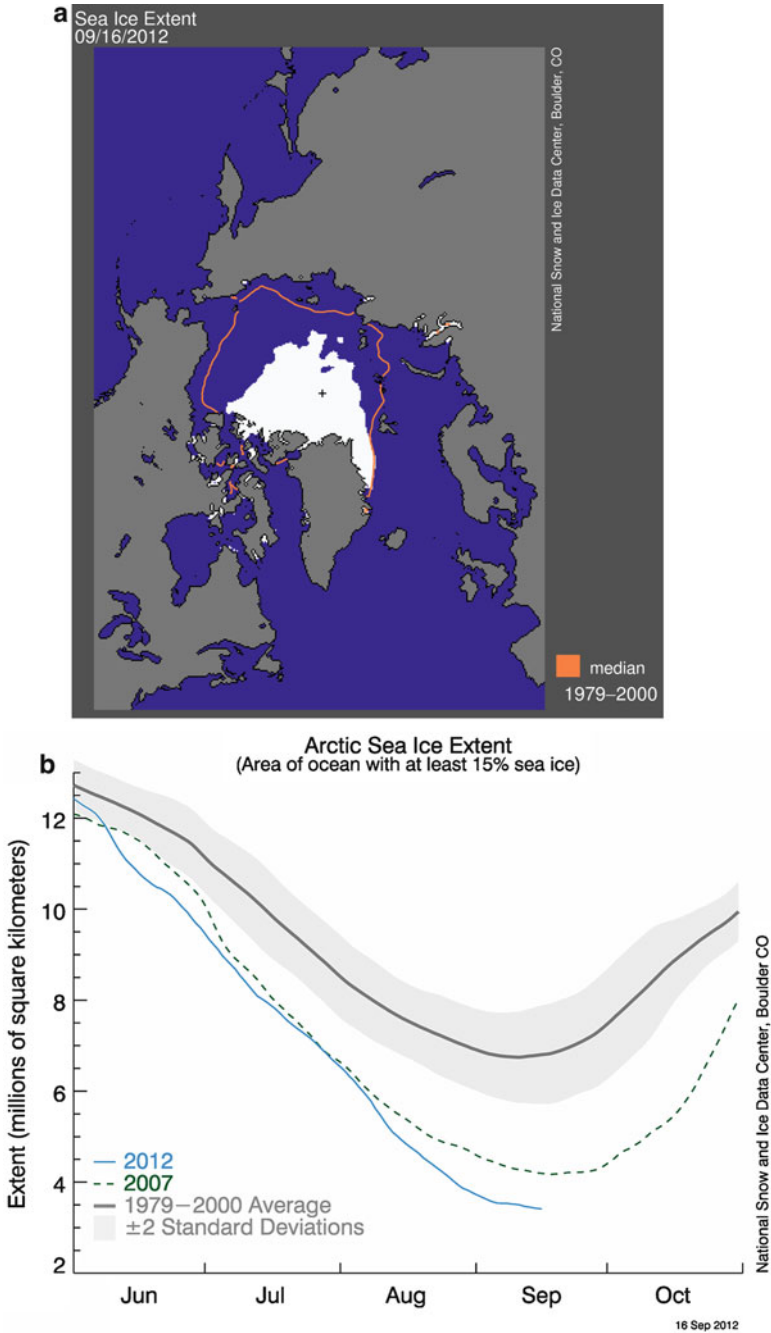


Fig. 12.1 Sea ice record melting. (a) Extent of sea ice extent on 9/16/12 compared with median ice extent for 1979–2000 (orange line). *Source:* National Snow and Ice Data Center, Boulder, CO. (b) Arctic sea ice extent during years 2007 and 2012, compared with average ice extent from 1979 to 2000 (*source:* National Snow and Ice Data Center, Boulder, CO.)

Vulnerable Geographic Regions

Certain regions and populations are more vulnerable to the health impacts of climate change [10]: areas bordering regions with a high endemicity of climate-sensitive diseases such as malaria; areas with an observed association between disease epidemics and weather extremes as with El Niño-linked epidemics; areas at risk from the combined impacts of climate relevant to health, such as stress on food and water supplies or risk of coastal flooding; and areas at risk from concurrent environmental or socioeconomic stresses, for example, local stresses resulting from land-use practices or an impoverished or undeveloped health infrastructure, with little capacity to adapt.

Vulnerability can also vary by neighborhood. For example, Uejio et al. [11] found that the number of heat distress calls in Phoenix, Arizona, was higher where city blocks had more impervious surfaces (indicating asphalt or concrete roads and buildings), and this was the primary cause of localized “urban heat island” intensification of temperatures.

Increases in floods and droughts, decreased food security, and biodiversity loss are special concerns for parts of Africa, Latin America, and Asia. Coastal and delta regions are at special risk even without climate change. These include coastal China, Bangladesh, Egypt, and especially densely populated, low-lying, small island states such as coral reef atolls throughout Polynesia. Arid regions such as Eastern Africa and Central Asia that already suffer from drought are likewise at increased risk. These risks are elevated even more as global climate warms [12].

Throughout this book, we will document many direct and indirect implications for human health due to climate change. This chapter focuses on threats to nutrition and safe water, risks from weather extremes and sea-level rise, and water- and food-borne infectious diseases. We then address public health responses to climate change and potential health “co-benefits” of greenhouse gas mitigation. Finally, the ethical dimensions of climate change and health are discussed.

Food Productivity and Malnutrition

It is no surprise that projected increases in frequency and intensity of climate extremes will have a major impact on crop and livestock production, as well as on the viability of fisheries [13–15]. Of course, the net effect on food production will vary from place to place. Changes will depend on several factors; their agents include temperature, precipitation, CO₂ levels (relating to the fertilization effect, for example), extreme climate variability, and sea-level rise. But indirect effects of climate-induced changes in soil quality, incidence of plant diseases, and increased weed and insect populations could have just as large an effect on world food supplies. Higher heat and humidity will also increase food spoilage (discussed below). The last 2 decades have seen continuing deterioration of food production in Africa,

caused in part by persistent drought. For some foods, nutritional quality (e.g., their protein content) will diminish as climate changes. Finally, the extent to which adaptive responses are available to farmers must be taken into account.

Food Production and Drought

Malnutrition remains one of the world's largest challenges to health. Eight hundred million people are currently undernourished [16]. Developing countries struggle with large and expanding populations and are particularly vulnerable to threats to food production. Projections forecast that drought-affected areas will increase, thereby exacerbating threats to agriculture, water supplies, energy production, and human health [17]. One-third of the world's population currently live in water-stressed countries and that number is predicted to increase to five billion people by 2025.

In Central Asia and Southern Africa, stream flows are expected to fall, and this may affect the food supply. Mountain snow pack, glaciers, and small ice caps play a crucial role in freshwater availability at regional sites. Large losses from glaciers and reductions in snow cover over recent decades are likely to accelerate throughout the twenty-first century. This will reduce water availability and hydropower potential and will change the seasonality of flows in regions supplied by melt water from major mountain ranges (e.g., the Hindu-Kush, the Himalayas, the Andes), where more than one-sixth of the world population resides [18]. Diarrhea and such diseases as scabies, conjunctivitis, and trachoma are associated with poor hygiene and can result from a breakdown in sanitation when water resources become depleted [19].

Despite significant agricultural technological advances, including irrigation, food production strongly depends on weather conditions. Most cultivars are growing close to their thermal optimum. Data from 23 global climate models show a high probability that the *average* growing season temperatures by the end of the century will exceed the *hottest* temperatures on record from 1900 to 2006 (Fig. 12.2) [20]. Lower yields are expected to occur at low latitudes due to heat stress, and crops will be subject to damage from flooding, erosion, and wildfires. The potential for global food production is projected to increase with increases in local average temperature over a range of 1–3 °C, but above this it is likely to decline [17]. Effects on global agricultural productivity will vary regionally; reductions will be especially acute in sub-Saharan Africa and South Asia [21].

According to one study [22], by the 2050s climate change would increase the risk of hunger from 34 % currently to a level of 64–72 %, unadjusted for potential adaptive interventions. Battisi and Naylor [20] found that reductions in regional productivity could destabilize food security to the extent that the number of people at risk for malnutrition could double by mid-century [20]. A recent study took the next step by estimating the human toll of such changes in worldwide malnutrition: Lloyd et al. [23] estimate that by 2030 climate change would lead to over 1.2 million malnutrition-related deaths.

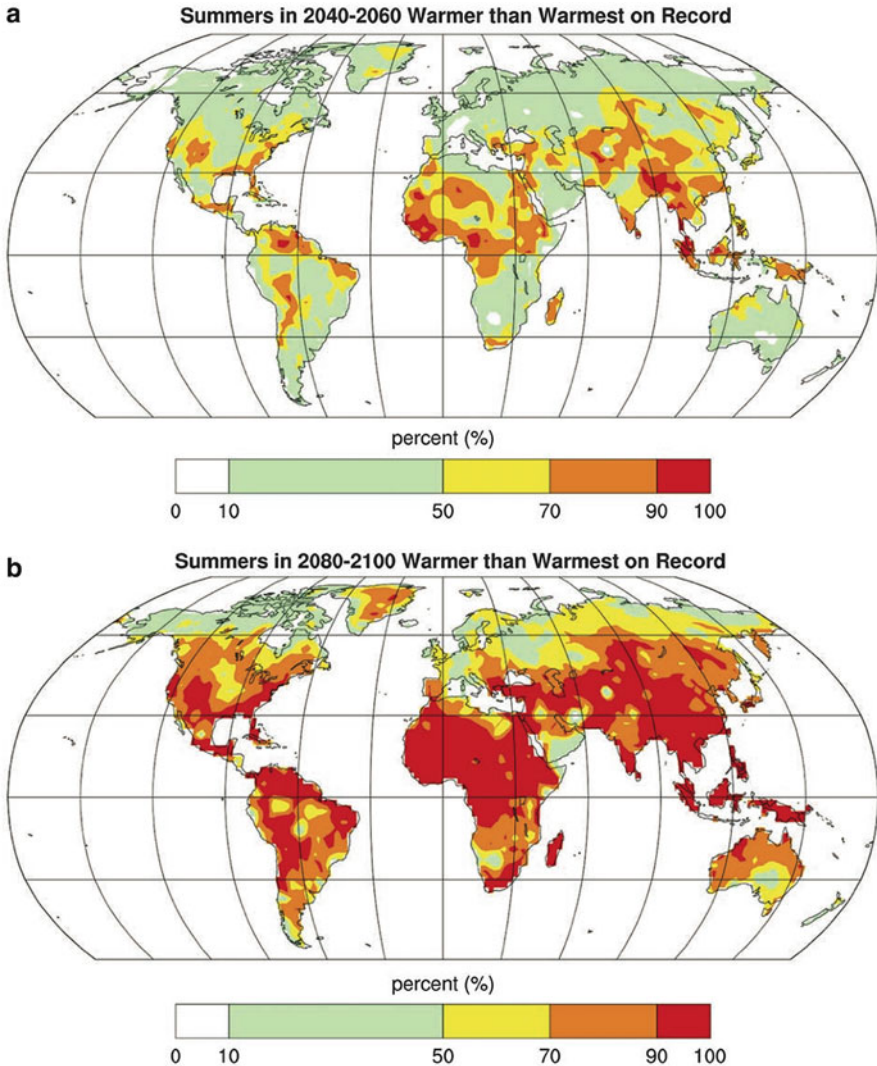


Fig. 12.2 Likelihood (in percent) that future summer average temperatures will exceed the highest summer temperature observed on record (a) for 2050 and (b) for 2090. For example, for places shown in red there is greater than a 90 % chance that the summer-averaged temperature will exceed the highest temperature on record (1900–2006). Reprinted with permission from Science. *Source:* Battisti, D.S. and Naylor, 2009

Crop yields are not the only concern. Nutritional value must be considered as well under a future climate regime. Some crops incorporate less nitrogen when CO₂ levels are elevated, resulting in lower protein content. Studies of barley, wheat, rice, potatoes, and soybeans show this reduced protein when crops are grown under

high-CO₂ conditions. The magnitude of the effect varies with soil conditions, air quality, and other factors [24]. For populations that depend on these crops for their protein, the high CO₂ effect could further threaten their nutritional status.

Of course, effects of climate change on malnutrition must be viewed in a broader context that takes into account other trends, such as the greater portion of crops now used either to feed livestock or supply feedstock for biofuels. In addition, when climate change affects the prevalence of bacterial or parasitic infectious diseases—where nutrient absorption is limited—this indirect pathway will affect nutritional benefits [25].

Fisheries

The most abundant greenhouse gas, CO₂, when absorbed by the ocean, leads to acidification. Over the past 250 years, the uptake of anthropogenic carbon has reduced ocean pH by 0.1 units, a trend that is continuing. IPCC scenarios predict a drop in global surface ocean pH of between 0.14 and 0.35 units over the twenty-first century. While the effects of ocean acidification are not fully understood, this process may threaten marine shell-forming organisms (e.g., corals) and their dependent species [18]. Of course, climate change may also threaten fish populations through other mechanisms. The recent slowing of the North Atlantic Gulf Stream, for instance, may lower the abundance of plankton that support many fish larvae [26]. Declining larval populations will threaten recovery of overexploited fish species.

Sea surface temperature change is the dominant driving force that shifts the geographical distribution of marine species. Warmer waters are oxygen-poor; when coupled with CO₂-induced ocean acidification, they pose substantial risks to marine ecosystems [27].

Coastal and island populations that rely on fish as their main source of protein could be threatened if global fisheries are further stressed. Worldwide, fish represent 16 % of the animal protein consumed by people, with a higher proportion in some regions, for example, 26 % in Asia. Climate change, together with such other pressures as overfishing, may have a serious impact on this source of nutrition.

Extreme Weather Events and Health

Natural Disasters

Droughts, floods, and violent storms have claimed millions of lives during the past 2 decades, threatened many more millions of people and caused billions of dollars in property damage. On average, such disasters have killed 123,000 people worldwide each year between 1972 and 1996 [28]. Africa suffers the highest rate of deaths related to disasters, although 80 % of the people affected by natural disasters are in

Asia. For every person killed in a natural disaster, 1,000 people are estimated to be affected, either physically, mentally, or through loss of property or livelihood [29].

Mental health problems such as post-traumatic stress disorder (PTSD) can be pervasive after a disaster. Their persistence depends on how unexpected the event was, the intensity of the experience, the degree of personal and community disruption, and long-term exposure to the visual signs of the catastrophe [30]. PTSD symptoms have been found as high as 75 % in refugee children and adolescents [31].

In poor countries, disasters can trigger large-scale dislocation of populations, often to jurisdictions ill prepared to receive them. Malnutrition and communicable diseases are prevalent in refugee populations. Displaced groups are also subjected to violence, sexual abuse, and mental illness. Generally, crude mortality rates in displaced populations may reach as high as 30 times the baseline with substantial mortality occurring in children under 5 [32]. Even in the United States, system failures were evident in the aftermath of hurricanes Katrina and Rita. Over 2,000 Americans were killed during that hurricane season, more than double the average number lost to hurricanes in the United States [33]. The survivors of Katrina suffered twice the rate of mental illness after the disaster when compared to a similar New Orleans population prior to that hurricane [34].

Floods

The heaviest 1 % of rain events falling in the United States increased by 20 % in the past century, while total precipitation increased by 7 %. During the same period, there was a 50 % increase in the frequency of days with precipitation over 4 inches in the upper Midwest [35]. Other regions, notably the South, have also seen strong increases in heavy downpours, with most of these coming in the warm season and almost all of the increase coming in the last few decades.

Populations are more vulnerable in floodplains and coastal zones. Degradation of the local environment can also contribute significantly to risk. Hurricane Mitch serves as one example; it was the most deadly hurricane to strike the Western Hemisphere in the last two centuries; the hurricane caused 11,000 deaths in Central America, with thousands of other people still recorded as missing. Many fatalities occurred from mudslides in deforested areas [36].

Wildfires

Hot temperatures combined with drought induce wildfires that threaten health both directly and through reduced air quality. Fire smoke carries a large amount of fine particulate matter that exacerbates cardiac and respiratory problems, such as asthma and chronic obstructive pulmonary disease (COPD). Drought-induced fires in Florida in 1998 were associated with increased hospital emergency room visits for

asthma, bronchitis, and chest pain [37]. The incidence of extensive wildfires in the Western United States (counting those over 400 ha) increased fourfold from the period 1970–1986 to 1987–2003 [37]. Higher springtime temperatures (0.87 °C warmer) that hasten spring snowmelt and result in a drop in soil moisture are considered driving factors that explain this increase in fires [38, 39]. Fire and climate change modeling for California has shown that the most severe effects of global climate change would occur in the Sierra Foothills, where potentially catastrophic fires could increase by 143 % in grassland and 121 % in chaparral [40]. The same study showed that greater burn intensity would result from a predicted change in fuel moisture and wind speeds.

Sea Level Rise and Health

Thermal expansion of salt water alone (without adding glacial melt water) causes sea level rise. One anticipated effect is an increase in flooding and coastal erosion in low-lying coastal areas. This will endanger large numbers of people; at present, 13 of the world's 20 megacities are situated at sea level. Midrange estimates project a 40 cm sea level rise by the 2080s. Under this scenario, coastal regions at risk from storm surges will expand and the population at risk will increase from the current 75 million to 200 million [12]. Greater sea level rise would mean even more devastation. Nicholls and Leatherman showed that the extreme case of a 1-m rise in sea level could inundate numerous low-lying areas, and impact 18.6 million people in China, 13 million in Bangladesh, 3.5 million in Egypt, and 3.3 million in Indonesia [41]. Countries similar to Egypt, Vietnam, and Bangladesh, as well as small island nations, are especially vulnerable, for several reasons. Coastal Egypt is already subsiding due to extensive groundwater withdrawal, and Vietnam and Bangladesh have heavily populated low-lying deltas along their coasts. In the United States, an estimated 20 million people will be affected by sea level rise by the year 2030, either directly or indirectly by migration networks linking inland and coastal areas and their populations [42]. In addition, rising sea levels heighten storm surges and cause salination of coastal freshwater aquifers, and they disrupt stormwater drainage and sewage disposal. Armed conflict may be among the worst results emerging from forced population migrations [43].

Water- and Food-Borne Diseases

Water-borne diseases, from both freshwater and coastal marine waters, are likely to become a greater problem through climate change-related weather extremes. In freshwater systems, both water quantity and quality can be affected. In marine waters, changes in temperature and salinity will affect coastal ecosystems in ways that may increase the risk of certain diseases, and as cholera and food poisoning from toxic algal blooms.

Freshwater

Water quantity and quality play a large role in water-borne diseases, which are therefore particularly sensitive to changes in the hydrologic cycle.

The impact of climate change on water *quantity* is relatively straightforward. In some regions precipitation is expected to increase, whereas in others it is predicted to decline, even to the point of ongoing drought. Water shortages contribute to poor hygiene, and that in turn contributes to diarrheal disease, especially in poor countries. At the other extreme, flooding can contaminate drinking water across watersheds with runoff from sewage lines, containment lagoons (such as those used in livestock feeding operations), or nonpoint source pollution (such as agricultural fields).

Extreme weather events can affect water *quality* in more complex ways. Many community water systems are already overwhelmed by extreme rainfall events. Runoff can exceed the capacity of the sewer system or treatment plants, and these systems are designed to discharge the excess wastewater directly into surface water bodies [44, 45]. Urban watersheds receive more than 60 % of their annual contaminant loads during storm events [46]. Turbidity also increases during storms, and studies have linked turbidity with illness in many communities [47, 48].

Disease outbreaks from most water-borne pathogens are distinctly seasonal, clustered in key watersheds, and associated with heavy precipitation [49]. In Walkerton, Ontario, in May 2000, heavy precipitation combined with failing infrastructure contaminated drinking water with *E. coli* 0157:H7 and *Campylobacter jejuni* resulting in seven deaths and an estimated 2,300 illnesses [50].

Intense rainfall can also contaminate recreational waters and increase the risk of human illness through higher bacterial counts [51]. This association is strongest at the beaches closest to rivers [52]. Enteric viruses are found at higher levels in both surface and ground water following heavy rainfall [53].

Cryptosporidiosis, one of the most prevalent diarrheal diseases in the world, is illustrative. Associated with domestic livestock, *cryptosporidium* is a protozoan that can contaminate drinking water during periods of heavy precipitation. The oocyst is resistant to chlorine treatment. The 1993 cryptosporidiosis outbreak in Milwaukee, during which an estimated 403,000 people were exposed to contaminated water, followed unusually heavy spring rains and runoff from melting snow [54]. Similarly, studies of the Delaware River have shown that *Giardia* and *Cryptosporidium* oocyst counts correlate with the amount of rainfall [55]. In Walkerton, Ontario, in May 2000, heavy precipitation combined with failing infrastructure contaminated drinking water with *E. coli* 0157:H7 and *Campylobacter jejuni* resulting in seven deaths and an estimated 2,300 illnesses [50].

A nationwide analysis of water-borne disease outbreaks in the United States from 1948 to 1994 demonstrated a distinct seasonality, a spatial clustering in key watersheds and an association with heavy precipitation; 67 % of reported outbreaks were preceded by unusually rainy months (defined as rainfall in the upper 80th percentile based on a 50-year local baseline) [49]. A recent study from a pediatric hospital in Milwaukee found that admissions for acute gastrointestinal illness

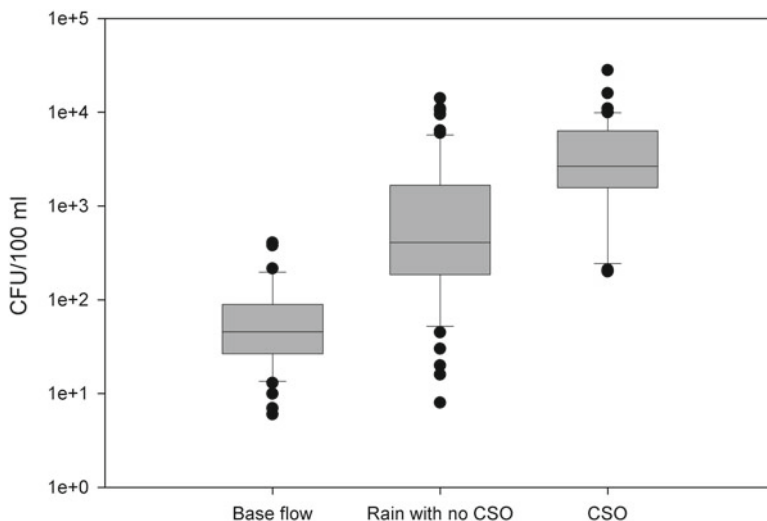


Fig. 12.3 Relationship between precipitation and *E. coli* counts. From 2001 to 2007 levels of *E. coli* in the Milwaukee estuary, which discharges to Lake Michigan, during base flow ($n=46$), following rain events with no CSO ($n=70$), and following CSO events ($n=54$). There were significant differences in *E. coli* levels following rainfall and CSOs compared to base flow ($p \leq 0.05$) (data from the Great Lakes Water Institute in Patz, JA, Vavrus S, Uejio C, McClellan S. Climate Change and Waterborne Disease Risk in the Great Lakes Region of the US. *Am J Preventive Medicine* 2008;35(5):451–458, with permission.)

increased following rain [56]. Certain watersheds, by virtue of associated land-use patterns and the presence of human and animal feces, are at high risk of surface water contamination after heavy rains, and this seriously threatens the purity of drinking water.

Recreational waters are also contaminated by heavy rainfall. For example, extensive runoff leads to higher bacterial counts in rivers and at beaches in coastal areas and is strongest at the beaches closest to rivers [52]. This suggests that the public health risk of swimming at beaches increases with heavy rainfall, a predicted consequence of climate change.

Heavy rains can lead to flooding, which can also raise the risk of water-borne diseases such as *Cryptosporidium* and *Giardia*. In many communities where sewage and stormwater runoff are handled in a combined system, when heavy rainfall overwhelms storm drainage infrastructures a combined sewer overflow (or CSO) event ensues. The highest levels of *E. coli* bacteria occur in surface waters in such cases (Fig. 12.3). Using 2.5 in. (6.4 cm) of daily precipitation as the threshold for initiating a CSO event, the frequency of these occurrences in Chicago is expected to rise by 50–120 % by the end of this century [57]. This will pose increased risk to drinking and recreational water quality. The worldwide average for diarrheal diseases in the future is projected to rise 20 % for the period 2040–2069 and 29 % for 2070–2099 [58].

Marine Environments

The impact of climate change on the extent of sea ice melt is well documented [17]. Data since 1978 show average annual Arctic sea ice area has declined by 2.7 % per decade (2.1–3.3), with decreases of 7.4 % in summer (5.0–9.8) [17]. Atkinson et al. combined net sampling data on Antarctic krill from 1926 to 2003 to demonstrate the effect of the scope of sea ice on krill populations [59]. After controlling for populations of top-down predators and bottom-up resources, they found temporal links between summer krill density and the extent of winter sea ice the preceding year, perhaps related to larval overwintering. Krill have enormous effects on the entire Arctic ecosystem; they are one of the primary food sources for penguins, albatrosses, seals, and whales [59]. Subsequently, the humans who rely on these species for their food and livelihood are affected by krill prevalence.

Blooms of marine algae that can release toxins into the marine environment, including two groups, dinoflagellates and diatoms, are enhanced by warm water and elevated nitrogen levels. These harmful algal blooms—sometimes referred to as red tides—can cause acute paralytic, diarrhetic, and amnesic poisoning in humans, as well as extensive die-offs of fish, shellfish, and marine mammals and birds that depend on the marine food web. Over the past 3 decades the frequency and global distribution of harmful algal blooms appears to have increased, and more human intoxication from algal sources has occurred [60]. For example, during the 1987 El Niño, a bloom of *Gymnodinium breve*, previously confined to the Gulf of Mexico, extended northward after warm Gulf Stream water flowed far up the eastern United States coast. This resulted in human neurological poisonings from shellfish and in substantial fish kills [61]. Similarly that year, an outbreak of amnesic shellfish poisoning occurred on Prince Edward Island when warm eddies of the Gulf Stream neared the shore and heavy rains increased nutrient-rich runoff [62].

By the year 2100, a 4 °C increase in summer temperatures in combination with water column stratification would double growth rates of several species of harmful algal blooms in the North Sea [63]. Biotoxins associated with warmer waters also include ciguatera (fish poisoning), which could extend its range to higher latitudes. An association has been found between ciguatera and sea surface temperature in some Pacific Islands [64].

Vibrio species are especially prolific in warm marine waters. Copepods (zooplankton), which feed on algae, can serve as reservoirs for *Vibrio cholerae* and other enteric pathogens. For example, in Bangladesh cholera follows seasonal warming of sea surface temperatures, which can enhance plankton blooms [65], and cholera cases fluctuate with temperature in coastal Africa as well [66, 67].

Vibrio species have expanded in northern Atlantic waters in association with warm water [68]. For example, in 2004 an outbreak of *V. parahaemolyticus* shellfish poisoning was reported from Prince William Sound in Alaska [69]. This pathogenic species of *Vibrio* had not previously been isolated from Alaskan shellfish due to the frigidity of Alaskan waters [69]. Water temperatures during in the 2004 shellfish harvest remained above 15 °C and mean water temperatures were significantly higher than the previous

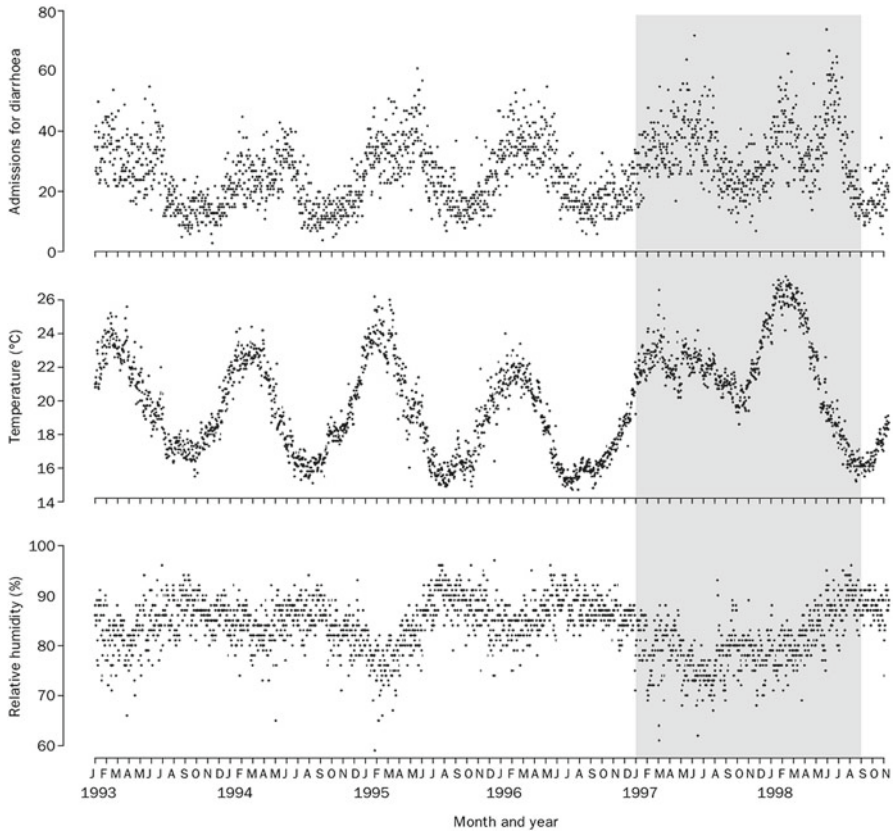


Fig. 12.4 Temperature and cholera relationship, Peru. Daily time-series between 1993 and 1998 of hospital admissions for pediatric diarrhoea, mean ambient temperature, and relative humidity in Lima, Peru (from Checkley, W., and others. Effects of El Niño and Ambient Temperature on Hospital Admissions for Diarrhoeal Diseases in Peruvian Children. *Lancet*, 2000, 355, 442–450, with permission from *The Lancet*.)

6 years [69]. Northern expansion of *V. parahaemolyticus* has been documented in Europe [70] and a rising trend of *Vibrio* bacteria and shifts in plankton abundance have paralleled warming trends in the North Sea since 1987 [71]. Such evidence suggests the potential for warming sea surface temperatures to increase the geographic range of shellfish poisoning and *Vibrio* infections into temperate and even Arctic zones.

The 1997 and 1998 El Niño event provided a natural experiment to examine temperature effects on diarrheal diseases, when winter temperatures in Lima, Peru, increased more than 5 °C above normal, and the daily hospital admission rates for diarrhoea more than tripled the rates over the prior 5 years [72] (Fig. 12.4). Long-term studies of the El Niño Southern Oscillation or ENSO have confirmed this pattern. ENSO refers to natural year-to-year variations in sea surface temperatures, surface air pressure, rainfall, and atmospheric circulation across the equatorial Pacific Ocean. This cycle provides a model for observing climate-related changes in

many ecosystems. ENSO has had an increasing role in explaining cholera outbreaks in recent years, perhaps because of concurrent climate change [73]. Overall there is growing evidence that climate change contributes to the risk of water-borne diseases in both marine and freshwater ecosystems.

Food-Borne Diseases

Changes in temperature and/or humidity can alter the incidence of food-borne infectious diseases. In the UK, researchers have found a strong correlation between the incidence of food-borne disease and temperatures in the month preceding the illness [74], suggesting food poisoning or spoilage. Reported cases of food poisoning across Australia, Western and Central Europe, and Canada follow a near linear relationship to each degree of increase in weekly temperature [17]. Temperature contributed to an estimated 30 % of cases of salmonellosis in much of continental Europe, especially when they exceeded a threshold of 6 °C above average [75]. Monthly incidence of food poisoning in Britain is most strongly associated with temperatures during the previous 2–5 weeks [74]. Other food-borne agents, such as *campylobacter*, are also seasonal but not as strongly linked to temperature fluctuations. Food spoilage is temperature-dependent since pest species, especially flies, rodents, and cockroaches, increase their contact with food as temperatures rise [76].

Land-Use Effects on Weather and Health

The center or core of most large cities is often much hotter than surrounding areas. The term “urban heat island” defines parts of a city that generate and/or retain heat as a result of roads, buildings, and industrial activities. Black asphalt and other dark surfaces on roads, parking lots, and roofs have a low albedo (reflectivity); they absorb and retain heat, reradiating it at night when the area would otherwise cool down. In addition, trees are relatively sparse in urban areas, so they provide less of the cooling effect associated with evapotranspiration. Global warming is expected to increase both heat and humidity, which will aggravate the effect of heat islands and increase heat stress on urban populations [77]. One study estimates the mean surface warming due to urban sprawl and land-use change to be 0.27 °C (0.49 °F) for the continental United States [78]. Urban areas may therefore face a compounded problem as they experience both global warming and localized warming from the heat island effect (Fig. 12.5). Urban residents in developing countries may be especially vulnerable to morbidity and mortality during heat waves.

By the end of the twenty-first century, the number of heat wave days could double in Los Angeles [79] and quadruple in Chicago [80], if emissions are not reduced. A recent analysis of 21 United States cities found that the average number of deaths due to heat waves would more than double by 2050, even after controlling for acclimatization [81].

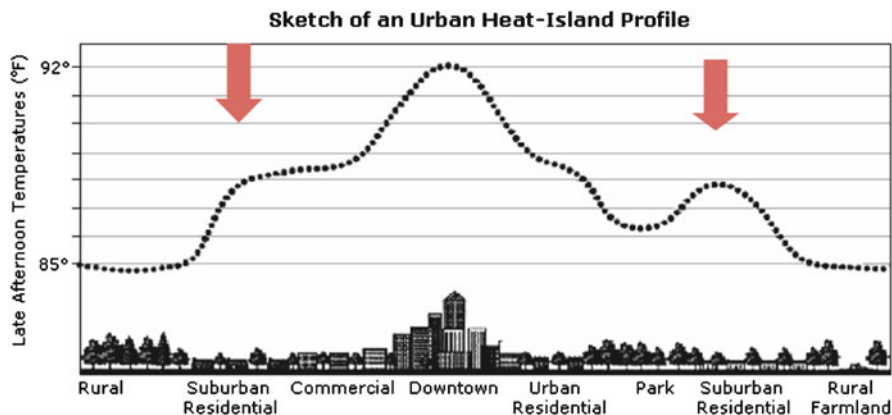


Fig. 12.5 Urban Heat Island Effect. Dark surfaces such as asphalt roads or rooftops can reach temperatures 30–40 °C higher than surrounding air (*source*: US Environmental Protection Agency, Washington, DC.)

To understand the relationship between vector-borne disease and climate, local landscapes need to be included in the analyses. For example, in the Amazon Basin, malaria incidence fluctuates with rainfall levels. Yet regional differences in the range of wetlands and surface water modify the effect of rainfall so much that in upland locations with sparse wetlands, malaria increases with rainfall, whereas in areas with abundant wetlands, it decreases [82]. In essence, climate effects must take into account local land cover data.

Ecosystems that preserve landscape integrity and biodiversity form the basis of many essential environmental services. However, global vegetation cover is changing far more rapidly than climate. Land cover is disrupted by such forces as deforestation, urban sprawl, industrial development, road construction, large water control projects—dams, canals, irrigation systems, and reservoirs—and climate change. Natural landscapes are being damaged or destroyed everywhere on a very large scale. A global pattern of landscape fragmentation has emerged.

The Public Health Response

Disentangling relationships between human health and climate change remains complex. The relationship is not always discernible, especially over short time spans. To understand and address such links requires systems thinking and consideration of many factors that range beyond health to such sectors as energy, transportation, agriculture, and development policy. Interdisciplinary collaboration is critical. A wide range of tools is needed, including innovative public health surveillance methods, geographically based data systems, classical and scenario-based risk assessment, and integrated modeling.

Mitigation and Adaptation

Two strategic approaches may be considered to address climate change. The first, mitigation, corresponds to primary prevention, and the second, adaptation, corresponds to secondary prevention.

Mitigation involves efforts to stabilize or reduce the production of greenhouse gases (and perhaps to sequester those gases that are produced). This goal can be achieved through policies and technologies that result in more efficient energy production and reduced energy demand. For example, sustainable energy sources such as wind and solar energy do not contribute to greenhouse gas emissions. Similarly, transportation policies that rely on walking, bicycling, mass transit, and fuel-efficient automobiles result in fewer greenhouse gas emissions than are produced by the current United States reliance on large automobiles with high fuel consumption for most transportation. Much energy use occurs in buildings, and green construction that emphasizes energy efficiency, together with electrical appliances that conserve energy, also plays a role in reducing greenhouse gas emissions (see Chap. 19). A final aspect of mitigation does not aim to reduce the production of greenhouse gases, but rather to accelerate their removal. Carbon dioxide sinks such as forests are effective in this regard, so land-use policies that preserve and expand forests are an important doctrine to mitigate global climate change.

Adaptation (or preparedness) refers to efforts to reduce the public health impact of climate change. For example, if we anticipate severe weather events such as hurricanes, then preparations by emergency management authorities and medical facilities can minimize morbidity and mortality. Similarly, public health surveillance systems can detect outbreaks of infectious diseases in vulnerable areas, a prerequisite to early control. Many of today's current challenges, such as deaths from heat waves, floods, and air pollution, will be exacerbated by climate change. Much preparedness can therefore be constructed from analyses of the strengths and weaknesses of current prevention efforts, and a rethinking of potential thresholds that may change in the future. Examples are expected changes in the volume of storm-water runoff and the frequency of heat waves.

“Co-benefits” from Mitigating Climate Change

While the steps needed to address the evolution of climate may appear formidable, some of them—reducing greenhouse gas emissions, developing and deploying sustainable energy technologies, and/or adapting to climate change—yield multiple benefits [83]. This can make them especially attractive, cost-effective, and politically feasible. For example, urban tree planting helps reduce CO₂ levels, while at the same time it reduces the heat island effect and local energy demand, improves air quality, dampens noise levels, and provides an attractive venue for physical activities and social interaction [84]. Another example is the reduction of fossil fuel use

in power plants. This is a principal strategy to reduce greenhouse gas emissions, and also a strategy to reduce air pollution [85]. A third example is sustainable community design [86]. Box 1 (below) shows the substantial health benefits gained by facilitating active transport (walking and bicycling) in and around urban settings.

In planning solutions such as sustainable communities, it is essential that the communities themselves be involved. Poor communities and communities of color bear a disproportionate vulnerability to many environmental health threats. These groups must be included when solutions are planned in order to preclude the possibility that the already large gap in access to healthy and desirable neighborhoods be widened. New areas must also be designed with cultural sensitivity and diversity in mind so that all people can be afforded a realistic opportunity to enjoy healthy new neighborhoods where environmental justice issues are considered at every level.

Health Co-benefits of Greenhouse gas Mitigation

Energy

A recent study by Shindell et al. addressed tropospheric ozone and black carbon (BC) contribution to both degraded air quality and global warming [87]. The authors identified 14 best interventions targeting methane and BC emissions that reduce projected global mean warming ~ 0.5 °C by 2050. The resulting “co-benefit” was the avoidance of 0.7–4.7 million annual premature deaths from outdoor air pollution and increases annual crop yields by 30–135 million metric tons due to ozone reductions in 2030 and beyond. The valuation was dominated by health effects from reduced BC in the air. While this study was global in nature, the findings apply to any location with coal-fired power plants, the most substantial contributor to black carbon particulates.

Transportation

Midwest Region Case study: Co-Benefits of Alternative Transportation Futures from improving air quality and physical fitness. The transportation sector produces one-third of the US greenhouse gas emissions. Automobile exhaust contributes not only to GHGs but also contains precursors to fine particulate matter (PM_{2.5}) and ozone (O₃), posing public health risks. Adopting a low carbon transportation system with fewer automobiles, therefore, could have immediate health “co-benefits” via improved air quality [88]. Modeled census tract-level mobile emissions for two comparative scenarios: current baseline versus a low carbon scenario where automobile trips shorter than five miles round-trip would be removed for the 11 largest metropolitan areas in the Midwestern United States. These relatively short car trips comprised approximately 20 % of vehicle miles traveled for the region.

Across the upper Midwest study region of approximately 31.3 million people and 37,000 total square miles, mortality would decline by nearly 575 deaths per

year from the benefit of improved air quality. Health benefits would also accrue in rural settings as well, with 25 % air quality-related health benefits to populations outside metropolitan areas.

An active transport scenario was then added, with the assumption that 50 % of the short trips (<5 miles) could be achieved by bicycle during the 4 months of most favorable weather conditions in the region. This theoretical maximum level of biking was selected because some locations in Europe have achieved this amount of bicycle commuting, and there already exists an observed trend of increasing bicycle share across all of the 11 Midwestern metropolitan areas [89]. This active transport scenario alone yielded savings of another 700 lives/year and approximately \$3.8 billion/year from avoided mortality costs (95 % CI: \$2.7, \$5.0 billion).

In summary, the estimated benefits of improved air quality and physical fitness from a green transportation scenario would 1,295 (95 % CI: 912, 1,636) lives saved and \$8 billion in avoided mortality and health care costs per year for the upper Midwest region alone. Nationally, there is already evidence that the US cities with enhanced levels of active transport experience large health benefits; one study found that cities with the highest rates of commuting by bike or on foot have obesity and diabetes rates 20 and 23 % lower, respectively, than cities with the lowest rates of active commuting [90].

Side Effects and Unintended Consequences

When solutions are attempted through interventions that are too narrowly focused or lack involvement by the local community, steps taken to address climate change can have unintended consequences. A cautionary example is biofuel production, a rapidly growing industry driven by economic incentives and public policies. Worldwide biofuel production may quadruple within the next 15–20 years [91–93].

However, critics claim that large-scale production of biofuels diverts crops from use as food, thus creating scarcity and driving food prices higher [94, 95]. The extent of humanitarian food aid from the United States that is available for extremely impoverished countries is inversely correlated with commodity prices [96]. Demand for biofuels may also accelerate the conversion of forests to cropland. Paradoxically, this could increase carbon dioxide levels [97–100] and threaten biodiversity in sensitive areas [101]. It is quite surprising to learn that a full life cycle analysis for biofuels showed slightly higher particulate matter levels for corn-based ethanol compared to gasoline and cellulosic ethanol; growing corn for ethanol involves intense use of fertilizers and farm machinery, and may simply shift air pollution from urban locations toward rural areas [102]. Critics further claim that biofuel production is economically inefficient, and relies heavily on subsidies [103]. Each of these claims is controversial; for example, some argue that food scarcity results from inadequate or inefficient distribution rather than from scarcity per se. In sum, the biofuel debate illustrates the potential to bring about unintended consequences that are especially harmful for vulnerable populations, and the need for careful analysis of each strategy proposed to address climate change [104].

Ethical Considerations

Climate change poses monumental ethical concerns in several ways. First, on a global scale, the nations that are responsible for most carbon emissions to date represent a small proportion of the world's population; they are relatively resilient to the effects of climate change. By contrast, the large population of the southern hemisphere—the poor countries—account for a relatively small cumulative share of carbon emissions, and present a very low per capita emission rate (although total emissions from developing nations are growing rapidly, and China surpassed the United States in 2006). The United States, with 5 % of the global population, produces 25 % of total greenhouse gas emissions. This discrepancy exemplifies the ethical implications posed by climate change on a global scale [105]. Poor populations in the developing world have little by way of industry, transportation, or intensive agriculture. They contribute only a fraction of the per capita greenhouse gases that the developed countries produce, and their capacity to protect themselves against the adverse consequences of emissions caused mostly by others is quite limited. Of course, if developing nations do not choose pathways that use more efficient energy technology, global climate change trends will intensify even as equity between rich and poor nations improves [43].

Within the United States, and within many other nations, a similar disparity exists. Poor and disadvantaged people will in many cases bear the brunt of climate change impacts, including those on health. This was graphically demonstrated in the aftermath of Hurricane Katrina, a disaster typical of those expected to increase with climate change. The poor populations of New Orleans and the nearby Gulf region were disproportionately likely to fail to evacuate, to suffer catastrophic disruption following the storm, and to be unable to recover [106–108].

Finally, an ethical issue arises with respect to intergenerational justice. Climate change holds the potential for enormous impacts on the health and well-being of future generations. Ethical and religious thinkers have pointed this out, and have argued that our generation owes a moral obligation to those who will follow to restore a sustainable climate.

Financial Considerations

Every weather-related disaster has large economic costs as well as health cost. For example, estimates for Russia's 2010 heat wave are 55,000 deaths, 25 % of annual crop failure, more than one million hectares of land destroyed by fire, and economic losses at about \$15 billion—or 1 % gross domestic product (GDP) [109]. The most comprehensive economic analysis to emerge on climate change probably comes from the UK's Stern report.

According to the Stern Report [110] 5–6 °C warming would result in an average 5–10 % loss in global GDP, with poor countries experiencing in excess of 10 % loss

of GDP. The report further describes analyses that include the full range of both impacts and possible outcomes, and under a Business as Usual scenario climate change would pose economic risks of between 5 and 20 % per capita.

Summary

Climatologists now state with a high degree of certainty that global climate change is real, is advancing more rapidly than expected, and is caused by human activities, especially through fossil fuel combustion and deforestation. Environmental public health researchers, in assessing future projections for Earth's climate, have concluded that, on balance, adverse health outcomes will predominate under these changed conditions. The number of pathways through which climate change can affect the health of populations makes this environmental hazard one of the most perilous and intricate challenges that we face in this century. By contrast, the potential health co-benefits from departing from our current fossil fuel-based economy may offer some of the most beneficial health opportunities in over a century.

References

1. Nicholls N, et al. Observed climate variability and change. In: Houghton J, et al. (editors), *Climate change 1995: the science of climate change. Contribution of working group I to the second assessment report of the intergovernmental panel on climate change*. New York: Cambridge University Press; 1996.
2. Tomanio J. Billion dollar weather. *National Geographic*. Sept 2012; issue: 53–4.
3. NSIDC. http://nsidc.org/news/press/20120827_2012extentbreaks2007record.html. 2012.
4. Trenberth K. Uncertainty in hurricanes and global warming. *Science*. 2005;308:1753–4.
5. Gray W. Hurricanes: their formation, structure and likely role in the tropical circulation. In: Shaw DB, editor. *Meteorology over the tropical oceans*. London: Royal Meteorology Society; 1979.
6. Miller STK, Keim BD, Talbot RW, Mao H. Sea breeze: Structure, forecasting, and impacts. *Review of Geophysics*. 2003;41(3):1011.
7. Goldenberg SB, Landsea CW, Mestas-Nuñez AM, Gray WM. The recent increase in Atlantic hurricane activity: causes and implications. *Science*. 2001;293:474–9.
8. Webster PJ, Holland GJ, Curry JA, Chang H-R. Changes in tropical cyclone number, duration, and intensity in a warming environment. *Science*. 2005;309:1844–6.
9. Emanuel K, Sundararajan R, Williams J. Hurricanes and global warming: results from down-scaling IPCC AR4 simulations. *Bull Am Meteorol Soc*. 2008;89:347–67.
10. Hess JJ, Malilay JN, Parkinson AJ. Climate change: the importance of place. *Am J Prev Med*. 2008;35:468–78.
11. Uejio CK, Wilhelmi OV, Golden JS, Mills DM, Gulino SP, Samenow JP. Intra-urban societal vulnerability to extreme heat: the role of heat exposure and the built environment, socioeconomics, and neighborhood stability. *Health Place*. 2011;17(2):498–507.
12. McCarthy J et al. (editor) *Climate change 2001: impacts, adaptation, and vulnerability. Contribution of working group II to the third assessment report of the intergovernmental panel on climate change*. New York: Cambridge University Press; 2001.

13. Schimhuber J, Tubiello FN. Global food security under climate change. *Proc Natl Acad Sci.* 2007;104:19703–8.
14. Tubiello FN, Soussana J-F, Howden SM, Easterling W. Crop and pasture response to climate change. *Proc Natl Acad Sci.* 2007;104:19686–90.
15. Brown ME, Funk CC. Food security under climate change. *Science.* 2008;319:580–1.
16. World Health Organization. *World Health Report 2002: reducing risks, promoting healthy life.* Geneva: World Health Organization; 2002.
17. IPCC. *Climate change 2007.* In: Core Writing Team, Pachauri RK, Reisinger A, editors. *Contribution of working groups I, II and III to the fourth assessment report of the intergovernmental panel on climate change.* Geneva: IPCC; 2007.
18. IPCC. *Summary for policymakers.* In: *Climate Change 2007: impacts, adaptation and vulnerability. Contribution of working group II to the fourth assessment report of the intergovernmental panel on climate change.* Cambridge, UK: IPCC; 2007.
19. Patz JA. Public health risk assessment linked to climatic and ecological change. *Hum Ecol Risk Assess.* 2001;7(5):1317–27.
20. Battisti DS, Naylor RL. Historical warnings of future food insecurity with unprecedented seasonal heat. *Science.* 2009;323:240–4.
21. Cline WR. *Global warming and agriculture impact estimates by country.* Washington, DC: Center for Global Development and Peterson Institute for International Economics; 2007. p. 186.
22. Butt TA, McCarl BA, Angerer J, Dyke PT, Stuth JW. The economic and food security implications of climate change in Mali. *Clim Chang.* 2005;68(3):355–78.
23. Lloyd SJ, Kovats RS, Chalabi Z. Climate change, crop yields, and malnutrition: development of a model to quantify the impact of climate scenarios on child malnutrition. *Environ Health Perspect.* 2011;119(12):1817–23.
24. Taub D, Miller B. Effects of elevated CO₂ on the protein concentration of food crops: a meta-analysis. *Glob Chang Biol.* 2008;14:565–75.
25. Humphrey JH. Child undernutrition, tropical enteropathy, toilets, and handwashing. *Lancet.* 2009;374:1032–5.
26. Pauly D, Alder J. Marine fisheries systems. In: Scholes R, Hassan R, Ash N, editors. *Ecosystems and human well-being: current state and trends, vol. 1.* Washington, DC: Island Press; 2005.
27. Portner HO, Peck MA. Climate change effects on fishes and fisheries: towards a cause-and-effect understanding. *J Fish Biology.* 2010;77(8):1745–79.
28. Loretto A, Tegegn Y. Disasters in Africa: old and new hazards and growing vulnerability. *World Health Stat Q.* 1996;49:179–84.
29. International Federation of Red Cross and Red Crescent Societies. *World disaster report 1997.* New York: Oxford University Press; 1998.
30. Green BL. Assessing levels of psychological impairment following disaster: consideration of actual and methodological dimensions. *J Nerv Ment Dis.* 1982;170:544–8.
31. McCloskey LA, Southwick K. Psychosocial problems in refugee children exposed to war. *Pediatrics.* 1996;97:394.
32. Toole M, Waldman R. The public health aspects of complex emergencies and refugee situations. *Annu Rev Publ Health.* 1997;18:283–312.
33. NOAA, Unified Synthesis Product. *Global climate change impacts in the United States; 2009.*
34. Kessler RC. Mental illness and suicidality after Hurricane Katrina. *Bull World Health Organ.* 2006;84:930–93.
35. Kunkel KE, Easterling DR, Redmond K, Hubbard K. 2003: temporal variations of extreme precipitation events in the United States: 1895–2000. *Geophys Res Lett.* 2003;30:1900. doi: [10.1029/2003GL018052](https://doi.org/10.1029/2003GL018052).
36. National Climatic Data Center. *Mitch: the deadliest Atlantic Hurricane Since 1780.* 1999. <http://www.ncdc.noaa.gov/ol/reports/mitch/mitch.html>.

37. Centers for Disease Control and Prevention. Surveillance of morbidity during wildfires: Central Florida, 1998. *Morbidity and mortality weekly report*, 1999;48(4):78–9.
38. Westerling AL, Hidalgo HG, Cayan DR, Swetnam TW. Warming and earlier spring increase western U.S. forest wildfire activity. *Science*. 2006;313:940–3.
39. Running SW. Is global warming causing more, larger wildfires? *Science*. 2006;313:927–8.
40. Torn MS, Mills E, Fried J. Will climate change spark more wildfire damage? LBNL Report no. 42592. Lawrence, CA: Lawrence Berkeley National Laboratory; 1998.
41. Nicholls R, Leatherman S. Global sea-level rise. In: Strzepek K, Smith J, editors. *As climate changes: international impacts and implications*. New York: Cambridge University Press; 1995. p. 92–123.
42. Curtis KJ, Schneider A. Understanding the demographic implication of climate change: estimates of localized population predictions under future scenarios of sea-level rise. *Popul Environ*. 2011. doi:[10.1007/s11111-011-0136-2](https://doi.org/10.1007/s11111-011-0136-2).
43. Patz JA, Kovats RS. Hotspots in climate change and human health. *Br J Med*. 2002;325:1094–8.
44. Perciasepe R. Combined sewer overflows: where are we four years after adoption of the CSO control policy? Report of the Environmental Protection Agency; 1998.
45. Rose JB, Simonds J. Kings County water quality assessment: assessment of public health impacts associated with pathogens and combined sewer overflows. Olympia: Washington State Department of Natural Resources; 1998.
46. Fisher GT, Katz BG. Urban stormwater runoff: selected background information and techniques for problem assessment with a Baltimore, Maryland, case study. 1988. U.S. Geological Survey Water-Supply Paper 2347, p. 30.
47. Morris RD, Naumova EN, Levin R, et al. Temporal variation in drinking water turbidity and diagnosed gastroenteritis in Milwaukee. *Am J Public Health*. 1996;86(2):237–9.
48. Schwartz J, Levin R, Hodge K. Drinking water turbidity and pediatric hospital use for gastrointestinal illness in Philadelphia. *Epidemiology*. 1997;8(6):615–20.
49. Curriero F, Patz JA, Rose J, Lele S. The association between extreme precipitation and waterborne disease outbreaks in the United States, 1948–1994. *Am J Public Health*. 2001;91:1194–9.
50. Hrudevy SE, Payment P, Huck PM, et al. A fatal waterborne disease epidemic in Walkerton, Ontario: comparison with other waterborne outbreaks in the developed world. *Water Sci Technol*. 2003;47(3):7–14.
51. Schuster CJ, Ellis AG, Robertson WJ, et al. Infectious disease outbreaks related to drinking water in Canada, 1974–2001. *Can J Public Health*. 2005;96(4):254–8.
52. Dwight RH, Semenza JC, Baker DB, et al. Association of urban runoff with coastal water quality in Orange County, California. *Water Environ Res*. 2002;74(1):82–90.
53. Borchardt MA, Spencer SK, Kieke BA, Lambertini E, Loge FJ. Viruses in nondisinfected drinking water from municipal wells and community incidence of acute gastrointestinal illness. *Environ Health Perspect*. 2012;120:1272–9.
54. MacKenzie WR, et al. A massive outbreak in Milwaukee of cryptosporidium infection transmitted through the public water supply. *New Engl J Med*. 1994;331(3):161–7.
55. Atherholt TB, LeChevallier MW, Norton WD, Rosen JS. Effect of rainfall on Giardia and Crypto. *J Am Water Works Assoc*. 1998;90(9):66–80.
56. Drayna P, McLellan SL, Simpson P, Li SH, Gorelick MH. Association between rainfall and pediatric emergency department visits for acute gastrointestinal illness. *Environ Health Perspect*. 2010;118:1439–43.
57. Patz J, Campbell-Lendrum D, Gibbs HK, Woodruff R. Health impact assessment of global climate change: expanding upon comparative risk assessment approaches for policy making. *Annu Rev Publ Health*. 2008;29:27–39.
58. Kolstad EW, Johansson KA. Uncertainties associated with quantifying climate change impacts on human health: a case study for diarrhea. *Environ Health Perspect*. 2011;119:299–305.

59. Atkinson A, Siegel V, Pakhomov E, Rothery P. Long-term decline in krill stock and increase in salps within the Southern Ocean. *Nature*. 2004;432:100–3.
60. Van Dolah FM. Marine algal toxins: origins, health effects, and their increased occurrence. *Environ Heal Perspect*. 2000;108 suppl 1:133–41.
61. Tester PA, et al. An expatriate red tide bloom, transport, distribution, and persistence. *Limnol Oceanogr*. 1991;36:1053–61.
62. Hallegraeff GM. A review of harmful algal blooms and their apparent global increase. *Phycologia*. 1993;32(2):79–99.
63. Peperzak L. Future increase in harmful algal blooms in the North Sea due to climate change. *Water Sci Technol*. 2005;51(5):31–6.
64. Hales S, Weinstein P, Woodward A. Ciguatera fish poisoning, El Niño and Pacific sea surface temperatures. *Ecosyst Heal*. 1999;5:20–5.
65. Colwell RR. Global climate and infectious disease: the cholera paradigm. *Science*. 1996;274:2025–31.
66. Myers SS, Patz JA. Emerging threats to human health from global environmental change. *Annu Rev Environ Resour*. 2009;34:223–52.
67. IPCC Assessment Report 5, 2011. http://www.ipcc.ch/publications_and_data/publications_and_data_reports.shtml
68. Thompson JR, Randa MA, Marcelino LA, Tomita-Mitchell A, Lim E, Polz MF. Diversity and dynamics of a North Atlantic coastal *Vibrio* community. *Appl Environ Microbiol*. 2004;70(7):4103–10.
69. McLaughlin JB, DePaola A, Bopp CA, Martinek KA, Napolilli NP, Allison CG, Murray SL, Thompson EC, Bird MM, Middaugh JP. Outbreaks of *Vibrio parahaemolyticus* gastroenteritis associated with Alaskan oysters. *N Engl J Med*. 2005;353(14):1463–70.
70. Martinez-Urtaza J, Simental L, Velasco D, et al. Pandemic *Vibrio parahaemolyticus* 03:K6, Europe. *Emerg Infect Dis*. 2005;11:1319–20.
71. Vezzulli L, Brettar I, Pezzati E, et al. Long-term effects of ocean warming on the prokaryotic community: evidence from the vibrios. *ISME J*. 2012;6:21–30.
72. Checkley W, et al. Effects of El Nino and ambient temperature on hospital admissions for diarrhoeal diseases in Peruvian children. *Lancet*. 2000;355:442–50.
73. Rodo X, Pascual M, Fuchs G, Faruque AS. ENSO and cholera: a nonstationary link related to climate change? *Proc Natl Acad Sci USA*. 2002;99(20):12901–6.
74. Bentham G, Langford IH. Climate change and the incidence of food poisoning in England and Wales. *Int J Biometeorol*. 1995;39:81–6.
75. Kovats RS, Edwards SJ, Hajat S, et al. The effect of temperature on food poisoning: a time-series analysis of salmonellosis in ten European countries. *Epidemiol Infect*. 2004;132(3):443–53.
76. Goulson D, Derwent LC, Hanley ME, Dunn DW, Abolins SR. Predicting calyprate fly populations from the weather, and probable consequences of climate change. *J Appl Ecol*. 2005;42:795–804.
77. Kattenberg A., et al. (1996) Climate models: projections of future climate. In: Houghton J et al. (editor) *Climate change 1995: the science of climate change. Contribution of working group I to the second assessment report of the intergovernmental panel on climate change*. New York: Cambridge University Press.
78. Kalnay E, Cai M. Impact of urbanization and land-use change on Climate. *Nature*. 2003;423:528–31.
79. Hayhoe K, et al. Emissions pathways, climate change, and impacts on California. *Proc Natl Acad Sci USA*. 2004;101(34):12422–7.
80. Vavrus S, Van Dorn J. Projected future temperature and precipitation extremes in Chicago. *J Great Lakes Res*. 2009. http://ccr.aos.wisc.edu/resources/publications/pdfs/CCR_986.pdf
81. Kalkstein LS, Greene JS, Mills DM, Perrin AD, Samenow JP, Cohen JC. Analog European heat waves for U.S. cities to analyze impacts on heat-related mortality. *Bull Am Meteorol Soc*. 2008;89(1):75–85.

82. Olson SH, Gangnon R, Eiguero E, Durieux L, Guegan JF, Foley JA, Patz JA. Surprises in the climate-malaria link in the Amazon. *J Emerg Infect Dis.* 2009;15(4):659–62.
83. Corfee-Morlot J, Agrawala S. The benefits of climate change policies: analytical and framework issues. Paris: Organization for Economic Co-operation and Development; 2004.
84. Frumkin H, McMichael AJ. Climate change and public health: thinking, communicating, acting. *Am J Prev Med.* 2008;35:403–10.
85. Cifuentes L, Borja-Aburto VH, Gouveia N, Thurston G, Davis DL. Assessing the health benefits of urban air pollution reductions associated with climate change mitigation (2000–2020): Santiago, São Paulo, México City, and New York City. *Environ Health Perspect.* 2001;109(3S):419–25.
86. Wilkinson P, Smith KR, Beevers S, Tonne C, Oreszczyn T. Energy, energy efficiency, and the built environment. *Lancet.* 2007;370:1175–87.
87. Shindell D, Johan CI, Kuylenstierna JCI, Vignati E, et al. Simultaneously mitigating near-term climate change and improving human health and food security. *Science.* 2012;335:183–9.
88. Grabow ML, Spak SN, Holloway TA, Stone B, Mednick AC, Patz JA. Air quality and exercise-related health benefits from reduced car travel in the Midwestern United States. *Environ Heal Perspect.* 2012;120:68–76.
89. U.S. Census Bureau. American Community Survey. American Factfinder. 2008. <http://factfinder.census.gov>. Accessed 1 Oct 2009.
90. Pucher J, Buehler R, Bassett DR, Dannenberg AL. Walking and cycling to health: a comparative analysis of city, state, and international data. *Am J Public Health.* 2010;100(10):1986–92.
91. International Energy Agency. Biofuels for transport 2004. http://www.iea.org/publications/freepublications/publication/biofuels_roadmap.pdf
92. Himmel ME, Ding SY, Johnson DK, et al. Biomass recalcitrance: engineering plants and enzymes for biofuels production. *Science.* 2007;315(5813):804–7.
93. Fairless D. Biofuel: the little shrub that could—maybe. *Nature.* 2007;449(7163):652–5.
94. Food and Agriculture Organization. Biofuels: prospects, risks and opportunities. The state of food and agriculture 2008. Rome: FAO; 2008.
95. United Nations-Energy. 2007. http://forestpolicy.typepad.com/ecoecon/2007/05/unenergy_on_bio.html
96. Naylor RL, Liska AJ, Burke MB, et al. The ripple effect: biofuels, food security, and the environment. *Environment.* 2007;49(9):30–43.
97. Fearnside PM. Soybean cultivation as a threat to the environment in Brazil. *Environ Conserv.* 2001;28(1):23–38.
98. Morton DC, DeFries RS, Shimabukuro YE, et al. Cropland expansion changes deforestation dynamics in the southern Brazilian Amazon. *Proc Natl Acad Sci USA.* 2006;103(39):14637–41.
99. Koh LP, Wilcove DS. Is oil palm agriculture really destroying tropical biodiversity? *Conserv Letts.* 2008;2(1):1–5.
100. Gibbs HK, Johnston M, Foley JA, et al. Carbon payback times for crop-based biofuel expansion in the tropics: the effects of changing yield and technology. *Environ Res Lett.* 2008;3:34.
101. Keeney D, Nanninga C. Biofuel and global biodiversity. Minneapolis: Institute for Agriculture and Trade Policy; 2008. http://www.iatp.org/files/258_2_102584_0.pdf.
102. Hill J, et al. Climate change and health costs of air emissions from biofuels and gasoline. *Proc Natl Acad Sci.* 2009;106(6):2077–82.
103. Organization for Economic Cooperation and Development. Biofuel support policies: an economic assessment. Paris: OECD. 2008. www.oecd.org/tad/bioenergy.
104. Patz JA, Vavrus S, Uejio C, McClellan S. Climate change and waterborne disease risk in the great lakes region of the US. *Am J Prev Med.* 2008;35(5):451–8.
105. Patz JA, Gibbs HK, Foley JA, Rogers JV, Smith KR. Climate change and global health: quantifying a growing ethical crisis. *EcoHealth.* 2007;4(4):397–405.

106. Dyson ME. *Come hell or high water: Hurricane Katrina and the color of disaster*. New York: Basic Books; 2007.
107. Elliott JR, Pais J. Race, class, and Hurricane Katrina: social differences in human responses to disaster. *Soc Sci Res*. 2006;35:295–321.
108. Pastor M, Bullard RD, Boyce JK, Fothergill A, Morello-Frosch R, Wright B. *In the wake of the storm: environment, disaster and race after Katrina*. New York: Russell Sage Foundation; 2006.
109. World Bank. *Turn down the heat: why a 4°C warmer world must be avoided*. Washington, DC: The World Bank; 2012.
110. Stern N. *The economics of climate change: the stern review*. Cambridge: Cambridge University Press; 2007.

Chapter 13

Household Air Pollution from Cookstoves: Impacts on Health and Climate

William J. Martin II, John W. Hollingsworth,
and Veerabhadran Ramanathan

Abstract Household air pollution (HAP) is an exposure of poverty. The success in having a sustainable reduction in HAP requires an understanding of the traditions and culture of the family as well as the causes of poverty that place the family at the bottom of the energy ladder. An integrated approach to reducing HAP with efforts also aimed at correcting other poverty-related issues is challenging but offers the hope for addressing root causes of poverty in a community setting that provides a more comprehensive and sustainable approach to improving health, the environment, and, ultimately, the global climate. From one perspective, research that provides detailed exposure-responses to HAP may seem superfluous to the obvious need for poor families to breathe cleaner air at home. One can argue that we already have decades of information on the health risks from outdoor air pollution or the products of incomplete combustion from tobacco smoke and so further research is not needed. However, there is a compelling need to know how clean a stove or fuel must be to significantly reduce health risks, so that with proper use, major implementation of such new technology may reasonably provide the intended benefits for improved health, the regional environment, and the global climate. The alternative of providing electrification or use of clean fuels such as LPG may not be realistic for the

W.J. Martin II, M.D. (✉)

Disease Prevention and Health Promotion, Eunice Kennedy Shriver National Institute of Child Health and Human Development, National Institutes of Health, 31 Center Drive, Building 31, Room 2A32, MSC 2425, Bethesda, MD 20892-2425, USA
e-mail: wjmartin@mail.nih.gov

J.W. Hollingsworth, M.D.

Department of Medicine and Immunology, Duke University Medical Center, Durham, NC 27710, USA

V. Ramanathan, M.S., Ph.D.

Center for Atmospheric Sciences, Scripps Institution of Oceanography, 8622 Kennel Way, La Jolla, CA 92037, USA

University of California at San Diego, La Jolla, CA, USA

world's poor for decades to come, if ever. Addressing the key scientific gaps related to HAP and its reduction will provide critical new information that can inform large scale implementation programs to provide sufficiently clean household air for families living in poverty, such that diseases are prevented, a healthier lifestyle is promoted, and a reduction in global warming trends buys more time for a planet in peril from climate change.

Keywords Biomass • Household air pollution • Climate change • Poverty and climate change

Household air pollution (HAP) from cooking fires in mostly low and middle income countries contributes to major health and environmental risks [1–3]. HAP is a result of incomplete combustion of solid fuels such as biomass and coal that is typically used for cooking, heating, and lighting in homes of those living at the bottom of the energy ladder. Biomass fuels consist of wood, crop residues, charcoal, or dung. Almost three billion people on the planet rely on use of solid fuels with the exposure to HAP contributing to almost four million deaths annually [3]. In addition, the consumption of these solid fuels causes regional environmental degradation through deforestation and the household emissions at scale represent a sizeable fraction of the outdoor air pollution in villages and cities [4]. Furthermore, some of these emissions such as black carbon are short-lived climate forcers that can contribute to global warming [5]. HAP is both a major health risk for the poorest people on the planet and a major risk for global climate change; thus, its remedies which are possible today offer the unique opportunity to improve the health and quality of life of the world's poor and, at the same time, provide hope that the global warming trends can be mitigated by reducing the impact of the short-lived climate forcers.

Cooking Fires and the Role of Women

Use of cooking fires goes back to the origins of our species and likely contributes to our evolutionary success as an intelligent species through improved nutrition [6]. Many of us harbor pleasant memories of camping fires and perceive cooking even in primitive sites as a warm and nurturing experience. Cooking fires in poor households reflect generations and centuries of traditions and cultural practices that reinforce patterns of behavior that often contribute to defining the role of women in a social and familial context. Cooking is not only a duty that falls almost exclusively to women, they are also responsible for the fuel gathering, a form of drudgery that occupies significant time in their daily routine and places the women and accompanying children at considerable personal risk, if they must walk miles from their villages to gather fuel [7]. Thus, because of the role of women in cooking and fuel gathering, women are at the center of this environmental issue. Therefore, women are additionally the key to the success of the proposed solutions to address HAP.

Proposed interventions to reduce HAP require the successful adoption and use of new stove or fuel technology, which can only be achieved through support from women. The ability of women to have a voice in the family decisions and to adopt the behavior changes necessary for reducing HAP requires fundamental changes in social and cultural practices. We emphasize this message early in this chapter lest the new and increasingly affordable technologies to reduce HAP suggest that the health and environmental risks are easily managed and implemented; they are not. Failure is always more likely than success. This is well demonstrated in a recent Ted Lecture by David Damberger, a member of engineers without borders, who articulates the need in any development enterprise to carefully evaluate the long-term outcome of any intervention, as most will fail [8]. Current efforts of large scale implementation of improved cookstove technology targeting the world's poor will require involvement of women in all levels of participation to achieve success. Implementation programs require constant evaluation and research to be certain that expected health, environmental, and climate benefits are in fact realized.

Cooking Using Solid Fuels and Possible Cooking Solutions

The three billion people who use solid fuels for cooking or heating typically use a variation of a three-stone fire with fuel being pushed into the fire gradually from the sides or, if affordable, use a primitive stove that provides the basic needs of cooking [9]. If a stove exists, it is often without a chimney or flue as they typically require detailed construction and maintenance to function properly. Over time, efforts for adequate removal of cookstove emissions are often not sustainable and emissions are simply released into the household [10]. This is how almost half the planet lives.

For decades, nongovernmental organizations (NGOs), local and multinational manufacturers, development agencies, host country governments, and foundations have struggled with improving the quality of cookstoves in lower and middle income countries, which is where the majority of the world's poor live. Some of these efforts have had substantial success such as in China [11] or more limited success as in the case of India [12]. In the majority of implementation studies around the world, there has been little study of the impact of "improved cookstove" programs on health or environment. Most implementations are often conducted at such a small scale and in such different cultural settings, that benefits are assumed and comparisons across programs are difficult.

In the past several years, there are increasing efforts to develop better coordination of the efforts of implementation and to develop a common knowledge base about the principles of stove efficiencies, affordability, and successful implementation. The best example of this effort is the Partnership for Clean Indoor Air (PCIA) led by the US Environmental Protection Agency (EPA) that has more than 500 members including NGOs, manufacturers, governments, academic institutions, and others (<http://www.pciaonline.org>). Since there are many "improved" cookstoves on the world market, the PCIA has focused much its attention on improving the understanding of what is an efficient and clean burning stove.

There are two types of cookstove efficiencies that impact health and the environment: (1) fuel efficiency and (2) combustion efficiency [9, 13]. Fuel efficiency reflects the amount of fuel required to achieve a specific task, such as a controlled water boiling test [9, 13, 14]. Fuel efficiency is critically important to households as stoves with improved fuel efficiency save the family fuel costs and time lost in fuel gathering. Reductions in time required to gather fuel are important for both women and children because saved time could be redirected to enhance educational and economic growth. Improved fuel efficiency will reduce the quantity of solid fuel burned and, thus, the quantity of CO₂ released from cooking fires. The second type of efficiency relates to the efficiency of combustion itself, and is necessary for reducing particulate matter (PM) that impairs health. PM_{2.5} is that fraction of aerosol particles that is smaller than 2.5 μm and poses special risks to human subjects due to its access to the lower respiratory tract and alveolar structures of the lung, where gas exchange occurs [15, 16]. Many outdoor air quality standards rely on PM_{2.5} and PM_{10.0} to reflect the risk of these air pollutant fractions to human health. Carbon monoxide (CO) is also a very dangerous pollutant, especially with the use of charcoal as a fuel [17]. Improving the combustion efficiency of a stove is key to reducing harmful emissions such as PM_{2.5} and CO. Black carbon is part of the PM_{2.5} fraction and is reflected as “soot” to the observer. Successful reduction of these pollutants will reduce human exposures and improve human health. There can be considerable differences in combustion and fuel efficiencies between stove testing sites and the household setting related to many factors including choice of fuel, ventilation, location of stoves, and human behavior. Therefore, demonstration of cookstove fuel and combustion efficiencies requires validation in the households of low to middle income countries to achieve desired benefits with implementation.

Another critical component to reducing exposures to household members is to understand how human behavior or cultural traditions may impact level of exposures. For example, the solid fuels collected (or purchased) for the stove must be sufficiently dry and combustible to perform the cooking task to achieve the reduced levels of emissions. Often, people will collect anything that burns easily such as leaves or crop residues which contain excessive moisture, and, when burned in even the most advanced stoves, will result in a very smoky indoor environment. In addition, there are special challenges during the transition from a traditional fire to use of an improved stove. Many families continue to use both types of stoves at the same time. In this common scenario, there may be some minor reduction in emissions with new cookstove technology. However, the reduction in indoor ambient pollution may be far less than that required to significantly improve human health. Often, the new improved stove or fuel is not properly designed to meet the complex cooking and cultural needs of the household including the absence of a traditional smoky flavor which makes it less desirable. Improving the efficiency of stoves or fuels offers the potential for multiple benefits to both households and the environment. However, implementation at large scale requires thoughtful interaction and participation with families and communities with a sensitivity to cultural traditions to ensure adoption of new technologies and realization of the co-benefits.

In an effort to bring together the diverse interests that surround HAP and its multiple adverse impacts, the United Nations Foundation launched the Global Alliance for Clean Cookstoves in September 2010 (<http://cleancookstoves.org>). The [Global Alliance for Clean Cookstoves](http://cleancookstoves.org) is a public–private partnership with a mission “to save lives, empower women, improve livelihoods, and combat climate change by creating a thriving global market for clean and efficient household cooking solutions.” The Global Alliance has a stated goal to have 100 million homes adopt clean and efficient stoves and fuels by 2020. The US government is a key partner with a commitment of more than \$50 million with almost half representing research and training efforts by the National Institutes of Health [1]. The Alliance has already developed hundreds of partners to help meet its mission and goals including other governments around the world, multinational companies, foundations, and NGOs. If successful, the Global Alliance will provide a forum for major implementation of new technology to reduce HAP and its health and atmospheric impacts that will use ongoing research and evaluation to validate whether such impacts occur at the scale expected. This ambitious effort is potentially a “game-changer” in bringing recognition and resources to address this global threat to human health and the environment.

Stove Testing

There are multiple sites today where stoves can be tested for fuel and combustion efficiencies. The US EPA offers rigorous stove testing at its facility in Research Triangle Park, North Carolina, USA, to determine emission patterns under controlled conditions [18]. In addition, Aprovecho Research Center in Cottage Grove, Oregon, offers similar testing but also offers a portable stove testing lab that can be used anywhere in the world [14]. Similarly, Berkeley Air in Berkeley, California, offers state of the art testing of stoves that complement a number of technologies related to HAP and stove use including exposure monitoring devices [19]. The PCIA web site <http://www.pciaonline.org> keeps up to date information on available stove testing facilities around the world as this technology moves into the host countries where stove testing is so critical to assess the potential benefits of an “improved stove.” Today, the standard practice is to test stoves both under laboratory conditions and in the field, where the testing more closely replicates family use and exposures.

Health Impacts of Household Air Pollution

HAP is the number one environmental cause of death in the world. These deaths are primarily from respiratory conditions including acute lower respiratory tract infection (ALRI) in children under age 5, chronic obstructive pulmonary disease (COPD), and lung cancer as reported in 2009 for the year 2004 [2] as well as from inclusion of cardiovascular diseases as reported in the recent update of the Global Burden of

Disease (GBD) 2010 [3]. The lung cancer risks are almost exclusively related to coal use for cooking and heating in China [20], although the GBD 2010 report now includes lung cancer from biomass HAP exposure [3].

Outdoor and HAP share many of the same products of incomplete combustion, although typically the household levels of these pollutants are of much higher concentration [21, 22]. Also, the same is true of emissions from burning of tobacco, the other “biomass.” Studies of health risks from HAP may well be informed from these related exposures, especially if exposure-response data are comparable across the different exposures.

A trans-US Government workshop held in May of 2011 addressed the state of the science of health impacts from HAP and offered a number of recommendations for future research related to health risks [23]. These findings relate to additional health risks from a small number of studies of HAP that may require replication but also include human health risks related to what we know from outdoor air pollution and tobacco smoke. Some of these putative risks will require further study in populations living with HAP, but the underlying rationale for these studies based on similar exposures is strong.

Examples of probable health risks attributable to HAP include cardiovascular disease, other respiratory diseases such as asthma or interstitial lung diseases, pregnancy outcomes such as birth weight, prematurity, or perinatal complications such as sepsis, infectious diseases such as acute pneumonia in older children or adults or tuberculosis, cancers related to HAP from non-coal sources such as biomass, and ocular disorders such as cataracts or trachoma. Of course, some health risks from indoor fires are unrelated to HAP. Burns and scalding are often under-reported and yet represent a life-changing risk for women and children that can include death [24]. Thus, stoves must not only be more efficient to promote health but also be tested for safety to reduce risk of burns.

Potential Host Risk Factors That Predict Adverse Health Effects Associated with Household Air Pollution

There are numerous studies supporting adverse health effects of chronic exposure to HAP related to use of cookstoves and exposure to incomplete combustion of solid fuels. The average of particulate exposure with use of indoor cookstoves is in the range of milligrams per cubic meter and peak levels can reach 10–30 mg/m³ [25]. This level is orders of magnitude higher than current EPA regulatory standards for outdoor air pollution, which is currently a 24-h average of 35 µg/m³ which is solely based on considerations for adverse health outcomes above this regulatory standard. There are limited studies of susceptible or vulnerable populations that are specifically associated with HAP related to solid fuel use. However, based on our fundamental understanding of the biological response to outdoor PM exposure and the extremely high levels of exposure encountered in homes with indoor fires, it is not unreasonable to consider that similar risk factors *may* contribute to adverse health outcomes associated with HAP. However, vulnerable and susceptible populations in

the LMIC associated with this common environmental exposure have not yet been identified through rigorous scientific investigations.

There are specific groups that may be at increased risk of adverse health outcomes based on our current understanding of the biological response to particulate matter. Previous work with outdoor particulate matter exposure identifies that both life-stage (children and older adults) and low socio-economic status represent characteristics associated with increased health risk. This is highly relevant as individuals exposed to HAP include children of low socio-economic status. Studies consistently demonstrate an exposure–response relationship (duration of daily exposure and number of years of exposure with health risk). Additionally, because of the cultural role of women in many regions of the world, both women and children experience a highest level of daily exposure. Therefore, both women and children are at the highest risk of health-related complications from HAP. For example, strong associations between biomass exposure and COPD have been demonstrated in never-smoking women, but not in men [26]. These findings do not necessarily demonstrate sex-dependent differences in *intrinsic susceptibility*, but rather are more likely related to cumulative duration of exposure to HAP. Together these observations support that both women and children are likely at the highest risk of adverse health effects related to level of exposure to HAP.

The role of host genetics in the response to HAP has received little attention. However, we recognize that exposure to PM is associated with specific host genetics. Previous studies of PM have identified a potential role for genes associated with regulation of oxidative stress (GSTM1, GSTP1, GSTT1, HMOX1, CAT, MNSOD), detoxifying enzymes (NQO1, EPHX1), and inflammation (TNF, TGF) [27]. It remains unclear whether the response to HAP is dependent on similar host genetic factors as the specific components of HAP are different than those associated with outdoor PM. However, a major component of HAP is black carbon and recent evidence supports that exposure to black carbon is associated with adverse effects on blood pressure [28]. In addition to the role of host genetics in response to PM, it is now clear that exposure to PM can modify host DNA. Exposure to PM can result in both DNA damage [29, 30] and shortening of telomere length [31]. Future studies will be invaluable to better understand the role of host genetics in response to HAP and the potential impact of this exposure on damaging host genes.

Our current appreciation of the effect of ambient environment on disease susceptibility extends beyond classic genetics. We now appreciate that common environmental exposures can modify epigenetic marks that include DNA methylation, histone modification, chromatin structure, and short regulatory RNA. These nongenetic (*non-code*) heritable changes can impact genetic expression and can have a profound lasting impact on human health. For example, exposure to traffic-related PM can result in rapid changes in DNA methylation [32] and exposure to ambient PM can result in modified site-specific DNA methylation [33]. Exposure to black carbon is associated with specific changes in micro-RNAs (regulatory short fragments of RNA) [34] and DNA methylation [32, 35]. The implications of these observations are that exposure to air pollution could have immediate impact on disease risk in a manner independent of changes in genetic code.

Based on the current understanding of epigenetics, pregnancy likely represents a unique window of susceptibility in programming epigenetic marks [36]. We *speculate* that early life (in utero and childhood) may represent a vulnerable population to the effects of HAP. During this period of development, HAP exposures may have a lasting effect on health through modification of epigenetic marks. One example of this long-term impact of early life exposures is that HAP may represent a major contributor to risk for noncommunicable diseases later in life, even if subsequent years of childhood and adulthood are lived in an environment free of this hazard. The core principle of developmental origins of health and disease as initially proposed by David Barker is that these windows of susceptibility in early life impact lifelong risk of disease [37]. Future work should focus on the impact of HAP on modification of the epigenome and its role in long-term risk for health and disease.

Currently there are limited available studies that identify host risk factors for adverse health effects associated with exposure to HAP. There is strong evidence supporting an exposure–risk association with HAP, which identifies both women and children at the highest risk of adverse health consequences. We speculate that similar to outdoor particulate matter exposure, undefined host genetics likely contribute to the biological response to cookstove emissions. Ambient exposures can modify host epigenetic marks that could alter disease risk and should be considered in future studies of biomass exposure. Identification of both *susceptible* and *vulnerable* populations for the health effects of indoor biomass exposure will require multidisciplinary studies integrating quantification of environmental exposures, genetic/epigenetic marks, and social context.

Regional Environmental Degradation

Fuel gathering is necessary for most of the world's poor to maintain a supply of fuel for cooking, heating, and lighting within their homes. It may reflect a range from walking long distances to collect wood in areas that are deforested, to picking up burnable debris along the roadside to pilfering discarded chunks of coal, where available. As noted previously, fuel gathering long distances from the safety of the village places women and their accompanying children at risk from gender-based violence, as well as injuries from heavy lifting, animal attacks, and insect bites [7]. Progressive deforestation due to uncontrolled consumption of wood for fuel has enormous social, environmental, and climate consequences as the loss of trees directly impacts biodiversity with loss of habitats for animals as well as loss of plant life required for a balanced ecosystem [4, 7, 38–40]. This, in turn, begins a cascade which can impair effective water management that can result in pooling of water that exacerbates the environmental degradation as well as puts human subjects at risk for illness including infectious diarrhea and vector-borne disease such as malaria. As a “picture is worth a thousand words,” there are several aerial photographs of national boundaries around the world that reflect differing environmental policies between countries that exist in nearly identical geographic circumstances.



Fig. 13.1 Island of Hispaniola demonstrating impact of deforestation in Haiti compared with Dominican Republic (DR). Haiti is the poorest country in the Western Hemisphere and shares the Island of Hispaniola with its neighbor, the DR. The population of Haiti relies on household fuel principally in the form of charcoal. There has been virtually no formal governmental policy in Haiti to protect its forests as fuel needs have increased over the past decades. The resulting deforestation results in a marked visual difference apparent in this NASA satellite photograph of the island with Haiti appearing largely barren and the DR that has federal policies regarding forest management, demonstrating a significant retention of its forests and biodiversity. <http://earthobservatory.nasa.gov/IOTD/view.php?id=5352>

One such example is the island of Hispaniola in the Caribbean, that is home to both the Dominican Republic and Haiti (Fig. 13.1). Haiti relies almost entirely on charcoal as its primary energy source for residential use of solid fuels and the environmental consequences are self-evident, placing the country at major risk for repeated flooding and with a loss of its once rich biodiversity.

Contribution of Household Air Pollution to Outdoor Air Pollution

The contribution of HAP on the level and composition of outdoor air pollution remains poorly characterized. However, given the global prevalence of households that use solid fuels as the primary source of household energy needs and the

extremely high level of HAP, it is highly probable that HAP significantly contributes to outdoor air pollution. For example, one remarkable historical event is the London smog of 1952 that resulted in 12,000 excess deaths and was attributed, in part, to HAP from the myriad homes that relied on residential burning of coal [41]. It is recognized that black carbon is an important component of HAP. The relative contribution of HAP as source of black carbon in outdoor air pollution, when compared to industrial emissions, remains unknown. Future studies should focus on the contribution of HAP on ambient outdoor air pollution. Current global efforts to replace traditional cookstoves provide an opportunity to better understand the contribution of household incomplete fuel combustion on external environment. The Surya project described later in this chapter offers the first such opportunity to address this issue. Interventions on household stoves on a large scale could have the potential co-benefits of improved indoor environment and reduce emissions that may impact outdoor air pollution.

Role of Black Carbon and Other Short-Lived Climate Forcers

Rapid and meaningful progress on slowing global warming is achievable if we recognize that global warming is caused by two different types of pollutants. The first is the long-lived carbon-dioxide released by fossil fuel combustion, which stays in the atmosphere for a century to thousand years. Most climate policies have focused on CO₂, but it will take decades and trillions of dollars to reduce emissions significantly. The world cannot afford to lose such decades. The planet has already warmed by more than 0.8 °C and the resulting symptoms are being perceived in rising sea levels, melting mountain glaciers including in the Himalayas and the Alps, large scale retreat of the Arctic sea ice and warming of the ocean waters penetrating to a depth of 1,000 m or more, and such extreme weather as droughts, floods, and heat waves. Worse, humans have already dumped enough greenhouse gases in the atmosphere to warm the planet by more than 2 °C [42]. Even if we were to replace half of all fossil fuel use with renewables, the warming will continue to increase for decades, because roughly half of the CO₂ molecules live for a century or more once released.

Fortunately, the world can get out of this seemingly hopeless predicament by broadening its focus to the *second* type of pollutants. Roughly half of total global warming is due to the release of four of these: dark soot particles called black carbon; and the gases methane, lower atmospheric ozone, and the halocarbons (CFCs, HCFCs, and HFCs). These pollutants (except CFCs, which are already banned and few other halocarbons) stay in the atmosphere for only weeks to a few decades and hence are referred to as short-lived climate forcers. Cutting these short-lived climate warming pollutant levels in half, which is feasible with current technologies—as UNEP’s Report on black carbon and ozone has recently demonstrated [43]—would quickly reduce the warming trend by 50 % [44] and give the world 2–4 decades for the effects of CO₂ reductions to take hold. In addition such measures can save 0.7–4.7 million lives annually and protect more than 100 million tons of crops from air

pollution-related damages [45]. The effects will also be quickly realized. For example, if we were to eliminate black carbon emissions by diesel vehicles today, their warming effect would disappear within weeks to a month. The cost of such reductions would not cripple economies; for example, between 1989 and 2007, California reduced its black carbon emissions by as much as 50 %.

Black carbon and ozone in the atmosphere have major regional climate effects, including melting the Himalayan glaciers and decreasing the monsoon rainfall over S. Asia [43, 46, 47]. In addition, both these climate warming agents lead to melting of Arctic sea ice [43]. China and India have a common interest in cutting the black carbon and ozone that is melting their shared glaciers, killing millions and destroying millions of tons of crops. The United States and Europe share common interest in the Arctic where black carbon and other short-lived pollutants are responsible for almost half of the melting ice. Modest steps that attack these short-lived climate forcers, with fast and measurable responses, are the best way to jump-start the stalled climate mitigation actions.

Improved Cookstoves or Fuels as Interventions to Reduce Health Impacts

As the majority of HAP is from cooking fires, it is reasonable to pursue interventions with more efficient stoves and fuels that will result in dramatic reductions in emissions and in exposures to family members. The challenge to date has been that although many “improved stoves” have demonstrated improved fuel efficiency with expected savings in fuels from 30 to 50 %, exposure reductions have been more modest. The recently published RESPIRE study from Guatemala suggests that exposures may need to be reduced by 50 %, and perhaps as much as 90 %, to reduce the risk of pneumonia in young children [48]. These findings were the result of a controlled trial with improved built-in stoves with added chimneys that physically replaced the traditional stoves, thereby removing the risk that the families might continue to use the traditional stoves as well. Participants in the study were trained in the proper use and maintenance of the stoves and chimneys and community workers and investigators were available to monitor the intervention as well as the exposure assessments. Thus, multiple factors reinforced the correct use of the intervention to achieve the results of dramatic exposure reduction.

It is challenging to consider how to achieve similar results from implementation of cookstoves that are sold in local markets but will not have the support systems in place similar to that of a controlled trial that reinforces proper stove adoption and use. NGOs or government programs working closely with communities can develop village-level training and educational programs to provide many of the same support systems, if well planned and implemented. There are many, perhaps thousands, of cookstove types available at local markets in lower and middle income countries. Examples of many of these stoves have been tested for various performance measures including fuel and combustion efficiencies (Fig. 13.2) [9, 13, 14]. Typical



Fig. 13.2 Display of multiple cookstove types used around the world. This photograph shows the wide variety of cookstoves using solid fuels in LMIC including: Open “3-stone” fire, wood fuel, Berkeley Darfur, wood fuel, Envirofit G-3300, wood fuel, Onil, wood fuel, Philips HD4008, wood fuel, Philips HD4012, wood fuel, Sampada, wood fuel, StoveTec GreenFire, wood fuel, Upesi Portable, wood fuel, GERES, charcoal fuel, Gyapa, charcoal fuel, Jiko, ceramic, charcoal fuel, Jiko, metal, charcoal fuel, KCJ Standard, charcoal fuel, Kenya Uhai, charcoal fuel, StoveTec prototype, charcoal fuel, Belonio Rice Husk Gasifier, rice hull fuel, Mayon Turbo, rice hull fuel, Oorja, biomass pellet fuel, StoveTec TLUD prototype, wood pellet fuel, Jinqilin CKQ-80I, corn cob fuel, and Protos, plant oil fuel (Courtesy of James Jetter, U.S. EPA, National Risk Management Research Laboratory Air Pollution Prevention and Control Division, Stove Testing Center, Research Triangle Park, North Carolina, USA)

“rocket stoves” achieve reasonable fuel efficiency with reductions in fuel use of about 30 %, but the exposure reductions will be less than the 50–90 % noted in the RESPIRE trial to achieve risk reduction for acute pneumonia. Additions of fans to the rocket stoves, the so-called fan stoves, offer greater efficiencies for both fuel use and emissions [13, 14]. The Philips stove was one of the first examples of a successful commercially available fan stove produced at scale. And many of the liquid fuel-based stoves, such as LPG, propane, biogas, or alcohol, offer the opportunity for being ultraclean with exposure reductions greater than 90 % [13, 14, 49, 50]. There are also natural draft, “top loading updraft” (TLUD), and other gassifier stoves, all of which offer opportunity for marked reduction in emissions [13, 14]. And finally, solar-based stoves offer the advantage of zero emissions and no fuel costs [13]. However, there can be issues with solar stoves such as the timing of cooking (early morning and evening) when sunlight is not available, or during rainy seasons when alternatives are needed or, finally, the adoption of solar cooking from traditional

cooking methods may be too great a change for some families. Nonetheless, solar cookers are a viable alternative as the primary means of cooking or as a supplement to an “improved” solid fuel stove. An additional strategy that can extend the cooking cycle without additional energy input is heat-retention cooking [9]. This method uses devices such as a “haybox” that is insulated and houses a cooking pot recently removed from a cookstove that limits loss of heat and permits the food to continue to cook. Such an integrated approach to cooking makes sense from both an energy usage perspective and a health and climate perspective. The diversity of cookstoves on the market in the absence of widespread testing creates confusion to consumers, NGOs, and governments that wish to address this problem. In 2012, there was an international working agreement for setting standards for cookstove performance from a meeting of stakeholders hosted by the International Organization for Standardization (ISO) in The Hague [13]. This is a major advance as both companies manufacturing stoves and consumers buying stoves can be guided by internationally accepted standards to ensure a “clean cookstove” is what it says it is.

The challenge facing investigators and implementers (mostly NGOs, manufacturers, and governments) is to select cookstoves that are affordable and acceptable to households; and, yet, are sufficiently clean as to achieve dramatic reductions in both emissions and exposures. Exposure reduction of 50–90 % is critical to reducing health risks [48] and emission reduction is necessary for mitigating climate risks. Currently, the commercially available stoves most likely to provide both reduced emissions and exposures from use of solid fuels include fan stoves that use a predictable and reliable fuel source such as pellets or properly dried wood. As noted previously, there is a rapidly emerging class of stoves that are gassifiers, natural draft, or other stove types that are also available but not necessarily world-wide as yet. Commercially available charcoal stoves typically have lower PM emissions than rocket or traditional stoves but can create dangerous levels of CO as families are less aware of the dangers absent the higher PM emissions. As noted previously, liquid fuels such as LPG, propane, biogas, and alcohol offer very low emissions but ongoing cost of fuels can represent an unrealistic financial burden to a family in poverty. The key to any of these strategies is to develop a monitoring and evaluation system that documents stove use and, where possible, exposure levels in and around the household. The stove unit monitoring system (SUMS) developed by Berkeley Air offers one approach to quantitatively assess stove use for both improved and traditional stoves [51]. Personal and area exposure monitoring on a selected basis are also essential to determining whether improved stoves or fuels are delivering the impacts expected.

Surya Project as Model of Potential Interventions to Reduce Climate Impacts

Black carbon and ozone, two potent short-lived climate forcers, are also great targets for developing nations because they have other known consequences apart from their health effects. They contribute to global warming (about 25–50 % of the CO₂ warming

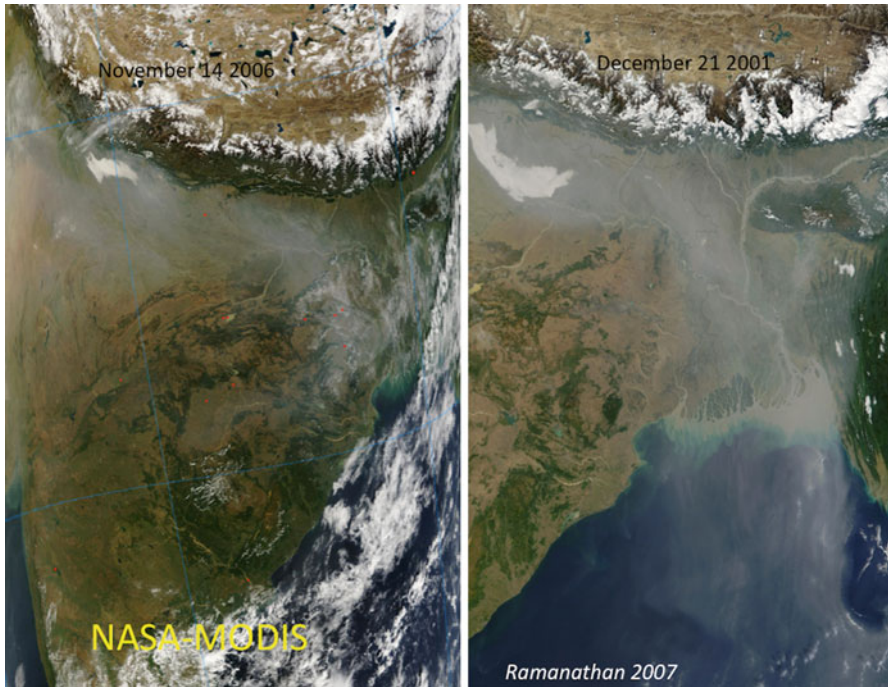


Fig. 13.3 Evidence of warming over the elevated Himalayan-Tibetan region comparing aerial photographs of 2001 with 2006. The interception of sunlight by black carbon leads to about 30–50 % of the warming effect of this region with evidence for deglaciation

as of 2005). In addition, they perturb regional climate in major ways. Interception of sunlight by black carbon leads to about 30–50 % of the warming over the elevated Himalayan-Tibetan region (Fig. 13.3) [43, 52, 53]. Black carbon interception of sunlight also weakens the monsoon circulation and reduces monsoon rainfall [43, 54, 55]. In addition both these pollutants lead to widespread destruction of crops, both directly [43] and indirectly through their effects on monsoon precipitation [56].

The world has an unprecedented opportunity to mitigate some of the disastrous effects of black carbon and ozone on climate, agriculture, water, and health with a simple act: replacing traditional cookstoves with energy-efficient and pollution-free cooking technologies. This work has already begun with international initiatives like the Global Alliance for Cookstoves, but challenges remain. The numerous cookstove initiatives that have taken place all over the world have demonstrated time and again that catalyzing widespread adoption of such clean cooking technologies will require innovative and affordable solutions.

This is where Project Surya, an internationally recognized cookstove project sponsored by the United Nations Environment Programme, comes in [52, 57]. Its goal is to demonstrate scientifically the environmental and health benefits of introducing clean cooking technologies and, ultimately, provide a rigorous evidence base for

large scale action. It aims to deploy improved cooking technologies in a contiguous region with a population of approximately 50,000, thus creating a “black carbon hole” in the otherwise omnipresent pollution cloud which will be measured across space and time to quantify the multi-sector impacts of better cooking technologies. Project Surya will use cell phones, instrument towers, and satellites, and will empower village youth to work with world-class experts in documenting the impacts.

A pilot phase was successfully completed in 2010 in a village in one of the poorest and most polluted regions in the Indo-Gangetic plains. It has already achieved some ambitious and measurable outcomes including documenting the connection between indoor air pollution from cooking and ambient outdoor pollution levels [58]; identifying improved cooking technologies that reduce pollution significantly [59]; deploying improved cookstoves in all the 500 or so households in the pilot village; and verifying that we will be able to measure the impacts of a larger-scale intervention using cell phones [60]. Another, parallel pilot test has been started in Nairobi, Kenya.

Our recent data has also shown that the measured black carbon concentrations are three to five times higher than the concentrations simulated by climate models, making it all the more urgent to take action now to target it and other short-lived climate forcers [61]. Fortunately, there is a great success story to draw upon. The enormous greenhouse effect of CFC-11 and CFC-12 was discovered only in 1975 [62]. CFCs were regulated by the 1987 Montreal Protocol, because of their negative effects on stratospheric ozone, but if this had not happened they would have added enough heat energy to warm the planet by about 1 °C or more.

Value of Co-benefits for Human Health and Climate

Improved and more efficient stoves or fuels can significantly reduce stove emissions that reduce HAP but also reduce outdoor air pollution that contributes to atmospheric changes that influence the climate. Simply displacing stove emissions through a chimney or flue without improving stove or fuel efficiencies not only continues to place a family or village at risk for HAP as the pollution reenters the home from the outside, its contribution to atmospheric change remains unabated. There are additional strategies needed to augment household exposure reduction. Obviously, the technology used to reduce HAP in any intervention being studied is critical to the impact on health and climate outcomes. However, the new technology must be acceptable to the user as significant reductions in HAP require exclusive use of the new stoves or fuels by the user, as opposed to shared use with the traditional means of cooking that can generate emissions that overwhelm the benefits of a new stove or fuel. There has been too little focus on the importance that human behavior and cultural traditions play in household approaches to energy use. When large scale implementation programs with improved stoves or fuels are being conducted, there is a need to measure the impact on household and outdoor exposures either directly or indirectly that reflect the impact of the improved stove or fuel. In the absence of such measurements, the impact on human health, environment, and climate remains unknown and speculative. It is the

responsibility of investigators, implementers, communities, and governments to work together to validate that major implementation programs with improved cooking solutions have the intended effects, and, if not, make the necessary changes in the implementation to ensure that the health of human subjects in poverty and the health of the planet are finally realized as true co-benefits.

Summary

HAP is an exposure of poverty. The success in having a sustainable reduction in HAP requires an understanding of the traditions and culture of the family as well as the causes of poverty that place the family at the bottom of the energy ladder. An integrated approach to reducing HAP with efforts also aimed at correcting other poverty-related issues is challenging but offers the hope for addressing root causes of poverty in a community setting that provides a more comprehensive and sustainable approach to improving health, the environment, and, ultimately, the global climate [63]. From one perspective, research that provides detailed exposure-responses to HAP may seem superfluous to the obvious need for poor families to breathe cleaner air at home. One can argue that we already have decades of information on the health risks from outdoor air pollution [64] or the products of incomplete combustion from tobacco smoke [65] and so further research is not needed. However, there is a compelling need to know how clean a stove or fuel must be to significantly reduce health risks, so that with proper use, major implementation of such new technology may reasonably provide the intended benefits for improved health, the regional environment, and the global climate. The alternative of providing electrification or use of clean fuels such as LPG may not be realistic for the world's poor for decades to come, if ever. Addressing the key scientific gaps related to HAP and its reduction will provide critical new information that can inform large scale implementation programs to provide sufficiently clean household air for families living in poverty, such that diseases are prevented, a healthier lifestyle is promoted, and a reduction in global warming trends buys more time for a planet in peril from climate change.

Acknowledgments This research was supported [in part] by the Intramural Research Program of the NIH, Eunice Kennedy Shriver National Institute of Child Health and Human Development (W.J.M.). Support is provided by NIH grants ES016126, ES020426, AI081672 and the Duke Provost's Fund, which support the Duke Cookstove Initiative (to J.W.H.).

References

1. Martin II WJ, Glass RI, Balbus JM, Collins FS. A major environmental cause of death. *Science*. 2011;334(6053):180.
2. Quantifying environmental health impacts: global estimates of burden of disease caused by environmental risks. Geneva: WHO. 2009. www.who.int/quantifying_ehimpacts/global/globalair2004/en/index.html

3. Lim SS, Vos T, Flaxman AD, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden Of Disease Study 2010. *Lancet*. 2012;380:2224–60.
4. Household cookstoves, environment, health and climate change: a new look at an old problem (63217, Washington, DC: World Bank). 2011. <http://climatechange.worldbank.org/climatechange/content/cookstoves-report>
5. US Climate Change Science Program. Climate projections based on emissions scenarios for long-lived and short-lived radiatively active gases and aerosols. 2008. <http://www.climate-science.gov/Library/sap/sap3-2/final-report/#finalreport>
6. Calvin WH. *The ascent of mind: ice age climates and the evolution of intelligence*. New York: Bantam; 1990.
7. Patrick E. Sexual violence and firewood collection in Darfur. *Forced Migr Rev*. 2007;27:40–1.
8. Damberger D. Ted lecture: what happens when an NGO admits failure. 2011. http://www.ted.com/talks/david_damberger_what_happens_when_an_ngo_admits_failure.html
9. Winiarski L. Design principles for wood burning cook stoves. Aprovecho Research Center, Partnership for Clean Indoor Air (PCIA), Shell Foundation. 2005.
10. Ramakrishna J, Durgaprasad MB, Smith KR. Cooking in India: the impact of improved stoves on indoor air quality. *Environ Int*. 1989;15(1–6):341–52.
11. Sinton JE, Smith KR, Peabody JW, et al. An assessment of programs to promote improved household stoves in China. *Energy Sustain Dev*. 2004;8(3):33–52.
12. Venkataraman C, Sagar AD, Habib G, Lam N, Smith KR. The Indian national initiative for advanced biomass cookstoves: the benefits of clean combustion. *Energy Sustain Dev*. 2010;14(2):63–72.
13. Partnership for clean indoor air. Test results of cook stove performance. 2010. <http://www.pciaonline.org/files/Test-Results-Cookstove-Performance.pdf>. Accessed 27 Jan 2012.
14. MacCarty N, Still D, Ogle D. Fuel use and emissions performance of fifty cooking stoves in the laboratory and related benchmarks of performance. *Energy Sustain Dev*. 2010;14(3):161–71.
15. Agency; USEP. PM_{2.5} NAAQS implementation. 2008. http://www.epa.gov/ttnnaqs/pm/pm25_index.html. Accessed 27 Jan 2012.
16. Ostro B, Lipsett M, Reynolds P, et al. Long-term exposure to constituents of fine particulate air pollution and mortality: results from the California Teachers Study. *Environ Health Perspect*. 2010;118(3):363–9.
17. Kituyi E, Marufu L, Wandiga SO, Jumba IO, Andreae MO, Helas G. Carbon monoxide and nitric oxide from biofuel fires in Kenya. *Energy Convers Manag*. 2001;42(13):1517–42.
18. Jetter JJ, Kariher P. Solid-fuel household cook stoves: characterization of performance and emissions. *Biomass Bioenergy*. 2009;33(2):294–305.
19. Berkeley Air Monitoring Group. Protecting health and climate. 2011. <http://berkeleyair.com/>. Accessed 27 Jan 2012.
20. Hosgood 3rd HD, Boffetta P, Greenland S, et al. In-home coal and wood use and lung cancer risk: a pooled analysis of the International Lung Cancer Consortium. *Environ Health Perspect*. 2010;118(12):1743–7.
21. Balakrishnan K, Sambandam S, Ramaswamy P, Mehta S, Smith KR. Exposure assessment for respirable particulates associated with household fuel use in rural districts of Andhra Pradesh, India. *J Expo Anal Environ Epidemiol*. 2004;14 Suppl 1:S14–25.
22. The Health Effects Institute. Outdoor air pollution and health in the developing countries of Asia: a comprehensive review. Special report 18. 2010. <http://pubs.healtheffects.org/view.php?id=349>. Accessed 27 Jan 2012.
23. Martin WJ, Glass RI, Araj H, et al. Household air pollution in low- and middle-income countries: health risks and research priorities. *PLoS Med*. 2013;10:e1001455.
24. Peck MD, Kruger GE, van der Merwe AE, Godakumbura W, Ahuja RB. Burns and fires from non-electric domestic appliances in low and middle income countries. Part I. The scope of the problem. *Burns*. 2008;34(3):303–11.
25. Smith KR, Samet JM, Romieu I, Bruce N. Indoor air pollution in developing countries and acute lower respiratory infections in children. *Thorax*. 2000;55(6):518–32.

26. Eisner MD, Anthonisen N, Coultas D, et al. An official American Thoracic Society public policy statement: novel risk factors and the global burden of chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 2010;182(5):693–718.
27. Romieu I, Moreno-Macias H, London SJ. Gene by environment interaction and ambient air pollution. *Proc Am Thorac Soc*. 2010;7(2):116–22.
28. Mordukhovich I, Wilker E, Suh H, et al. Black carbon exposure, oxidative stress genes, and blood pressure in a repeated-measures study. *Environ Health Perspect*. 2009;117(11):1767–72.
29. Risom L, Moller P, Loft S. Oxidative stress-induced DNA damage by particulate air pollution. *Mutat Res*. 2005;592(1–2):119–37.
30. Rubes J, Rybar R, Prinosilova P, et al. Genetic polymorphisms influence the susceptibility of men to sperm DNA damage associated with exposure to air pollution. *Mutat Res*. 2010;683(1–2):9–15.
31. Hoxha M, Dioni L, Bonzini M, et al. Association between leukocyte telomere shortening and exposure to traffic pollution: a cross-sectional study on traffic officers and indoor office workers. *Environ Health*. 2009;8:41.
32. Baccarelli A, Wright RO, Bollati V, et al. Rapid DNA methylation changes after exposure to traffic particles. *Am J Respir Crit Care Med*. 2009;179(7):572–8.
33. Tarantini L, Bonzini M, Apostoli P, et al. Effects of particulate matter on genomic DNA methylation content and iNOS promoter methylation. *Environ Health Perspect*. 2009;117(2):217–22.
34. Wilker EH, Baccarelli A, Suh H, Vokonas P, Wright RO, Schwartz J. Black carbon exposures, blood pressure, and interactions with single nucleotide polymorphisms in MicroRNA processing genes. *Environ Health Perspect*. 2010;118(7):943–8.
35. Madrigano J, Baccarelli A, Mittleman MA, et al. Prolonged exposure to particulate pollution, genes associated with glutathione pathways, and DNA methylation in a cohort of older men. *Environ Health Perspect*. 2011;119(7):977–82.
36. Breton CV, Byun HM, Wenten M, Pan F, Yang A, Gilliland FD. Prenatal tobacco smoke exposure affects global and gene-specific DNA methylation. *Am J Respir Crit Care Med*. 2009;180(5):462–7.
37. Hanson M, Godfrey KM, Lillycrop KA, Burdge GC, Gluckman PD. Developmental plasticity and developmental origins of non-communicable disease: theoretical considerations and epigenetic mechanisms. *Prog Biophys Mol Biol*. 2011;106(1):272–80.
38. World Health Organization. WHO air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Global update 2005. Summary of risk assessment. Geneva: WHO; 2005. http://whqlibdoc.who.int/hq/2006/WHO_SDE_PHE_OEH_06.02_eng.pdf
39. World Health Organization. Fuel for life: household energy and health. Geneva: WHO; 2006.
40. World Health Organization. Indoor air pollution: national burden of disease estimates. Geneva: WHO; 2007. http://www.who.int/indoorairepublications/indoor_air_national_burden_estimate_revised.pdf. Accessed 3 Feb 2012.
41. Bell ML, Davis DL. Reassessment of the lethal London fog of 1952: novel indicators of acute and chronic consequences of acute exposure to air pollution. *Environ Health Perspect*. 2001;109 Suppl 3:389–94.
42. Ramanathan V, Feng Y. On avoiding dangerous anthropogenic interference with the climate system: formidable challenges ahead. *Proc Natl Acad Sci USA*. 2008;105(38):14245–50.
43. United Nations Environment Programme. Integrated assessment of black carbon and tropospheric ozone: summary for decision makers. 2011. http://www.unep.org/dewa/Portals/67/pdf/Black_Carbon.pdf. Accessed 30 Jan 2012.
44. Ramanathan V, Xu Y. The Copenhagen accord for limiting global warming: criteria, constraints, and available avenues. *Proc Natl Acad Sci USA*. 2010;107(18):8055–62.
45. Shindell D, et al. Simultaneously mitigating near-term climate change and improving human health and food security. *Science*. 2012;335:183–9.
46. Ramanathan V, Carmichael G. Global and regional climate changes due to black carbon. *Nat Geosci*. 2008;1(4):221–7.

47. Pontifical Academy of Sciences, Ajai LB, Breashears D, Crutzen PJ, Fuzzi S, Haeberli W, Immerzeel WW, Kaser G, Kennel C, Kulkarni A, Pachauri R, Painter TH, Rabassa J, Ramanathan V, Robock A, Rubbia C, Russell L, Sánchez Sorondo M, Schellnhuber HJ, Sorooshian S, Stocker TF, Thompson LG, Toon OB, Zaelke D. Fate of mountain glaciers in the Anthropocene. Vatican City: Pontifical Academy of Sciences; 2011.
48. Smith KR, McCracken JP, Weber MW, et al. Effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE): a randomised controlled trial. *Lancet*. 2011;378(9804):1717–26.
49. Smith KR, Rogers J, Cowlin SC. Household fuels and ill-health in developing countries: what improvements can be brought by LP gas? Paris: World LP Gas Association and Intermediate Technology Development Group; 2005.
50. Po JY, FitzGerald JM, Carlsten C. Respiratory disease associated with solid biomass fuel exposure in rural women and children: systematic review and meta-analysis. *Thorax*. 2011;66(3):232–9.
51. Ruiz-Mercado I, Lam N, Canuz E, Davila G, Smith KR. Low-cost temperature data loggers as Stove Use Monitors (SUMs). *Boiling Point*. 2008;55:16–8.
52. Ramanathan V, Balakrishnan K. Project Surya: Reduction of air pollution and global warming by cooking with renewable sources—a controlled and practical experiment in rural India: a white paper. <http://ramanathan.ucsd.edu/files/SuryaWhitePaper.pdf> (2007). Accessed 30 Jan 2012.
53. Flanner MG, Zender CS, Hess PG, et al. Springtime warming and reduced snow cover from carbonaceous particles. *Atmos Chem Phys Discuss*. 2008;8(6):19819–59.
54. Ramanathan V, Chung C, Kim D, et al. Atmospheric brown clouds: impacts on South Asian climate and hydrological cycle. *Proc Natl Acad Sci USA*. 2005;102(15):5326–33.
55. Meehl GA, Arblaster JM, Collins WD. Effects of black carbon aerosols on the Indian monsoon. *J Climate*. 2008;21(12):2869–82.
56. Auffhammer M, Ramanathan V, Vincent JR. Integrated model shows that atmospheric brown clouds and greenhouse gases have reduced rice harvests in India. *Proc Natl Acad Sci USA*. 2006;103(52):19668–72.
57. Ramanathan V, Ramanathan N. An unprecedented opportunity. *Our Planet*. 2011:28–29. http://www.unep.org/pdf/op_dec_2011/EN/OP-2011-12-EN-ARTICLE8.pdf. Accessed 1 Feb 2012.
58. Rehman IH, Ahmed T, Praveen PS, Kar A, Ramanathan V. Black carbon emissions from biomass and fossil fuels in rural India. *Atmos Chem Phys*. 2011;11(14):7289–99.
59. Kar A, Siva P, Suresh R, Rehman IH, Singh L, Singh VK, Ahmed T, Burney J, Ramanathan N, Ramanathan V. Real-time assessment of black carbon pollution in Indian households due to traditional and improved biomass cook stoves. *Environ Sci Technol*. 2012;46:2993–3000.
60. Ramanathan N, Lukac M, Ahmed T, et al. A cellphone based system for large-scale monitoring of black carbon. *Atmos Environ*. 2011;45(26):4481–7. *Atmos Environ*. 2011;45(39):7536.
61. Praveen PS, Ahmed T, Kar A, Rehman IH, Ramanathan V. Link between local scale BC emissions and large scale atmospheric solar absorption. *Atmos Chem Phys Discuss*. 2011;11(7):21319–61.
62. Ramanathan V. Greenhouse effect due to chlorofluorocarbons: climatic implications. *Science*. 1975;190(4209):50–2.
63. Bodereau PN. Peruvian highlands, fume-free. *Science*. 2011;334(6053):157.
64. Pope 3rd CA, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA*. 2002;287(9):1132–41.
65. U.S. Department of Health and Human Services. How tobacco smoke causes disease. In: U.S. Department of Health and Human Services CfDCAp, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, editors. *The biology and behavioral basis for smoking-attributable disease: a report of the surgeon general*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2010.

Chapter 14

Biomass Fuel and Lung Diseases: An Indian Perspective

Rajendra Prasad and Rajiv Garg

Abstract One half of the world's population relies on biomass fuel as the primary source of domestic energy. Biomass fuel exposure inflicts a high degree of morbidity and mortality upon the human race. This is especially true in the context of developing countries especially for India where biomass fuel is main source of the domestic energy. It is estimated that three-quarters of Indian households use biomass fuel as the primary means for domestic cooking. According to WHO estimate more than half of Indian population depends on solid fuel for domestic purpose. There are wide variations between the rural and urban households regarding the specific kind of biomass fuel used. Biomass fuel attributes for 5–6 % of the national burden of disease. Many respiratory diseases have been found to be associated with the exposure of biomass fuels such as acute lower respiratory infections, chronic obstructive pulmonary disease, lung cancer, pulmonary tuberculosis, and asthma. Published evidences from India suggest association of the respiratory diseases particularly tuberculosis, COPD, and lung cancer with biomass fuel exposure and also it contribute heavily to the burden of diseases. It is hoped that in future, studies on biomass exposure-associated morbidities and its prevention would claim priority. Concerted efforts in improving stove design and transition to high efficiency with low emission fuels may reduce respiratory disease associated with biomass fuel exposure.

Keywords Biomass fuels, indoor air pollution, lung diseases, solid fuel use in cooking • India experience with biomass fuel use • Lung disease and biomass fuel use

R. Prasad, M.D., FAMS, FCCP(USA), FNCCP (✉)
Department of Pulmonary Medicine, Vallabhbhai Patel Chest Institute, University of Delhi,
Delhi 110007, India
e-mail: rprasadkmc@gmail.com

R. Garg
Department of Pulmonary Medicine, K.G's Medical University UP, Lucknow,
UP 226003, India

Biomass fuels are primary source of domestic energy for about half of the world's population [1]. Biomass fuel consists of firewood, dung cakes, agricultural crop residues (straw, grass, shrubs etc.), coal fuels, and kerosene. Together they supply 75 % of the domestic energy in India. The rest of the country relies on cleaner fuels namely liquefied petroleum gas (LPG) and natural gas [2]. The biomass fuels and coal are sources of high level indoor air pollution as these are used for cooking and heating on traditional stoves or open fires which results in incomplete combustion and heavy smoke production.

Biomass Fuel Use in India

It is estimated that three-quarters of Indian households use biomass fuel as the primary means for domestic cooking. Ninety percent of the rural households and 32 % of the urban households cook their meals on a biomass stove. Only 25 % of the cooking is done with the cleaner gases. Ninety percent households using biomass fuels cook on an open fire (Figs. 14.1 and 14.2). There are wide variations between the rural and urban households regarding the specific kind of biomass fuel used. In rural India, 62 % households use firewood, 14 % cook with dung cakes while 13 % use straw, shrubs, grass, and agricultural crop residues to fire their stoves. In urban India, 22 % use firewood, 8 % use kerosene, and the rest uses cleaner fuels like LPG or natural gas [2]. According to World Health Organization estimate in 2010, more than half of Indian population (58 %) depends on solid fuels for domestic purpose [3]. It can also be stated that 75 % of rural households reported firewood as their primary cooking fuel as compared to only 22 % of urban households. It is apparent that factors such as affordability, awareness, ease of availability, cooking space constraints, social customs, and demographics (for example, working women) play a significant role in the choice of fuel in urban locality [4].



Fig. 14.1 This photo shows the fire wood used as a cooking medium in open space



Fig. 14.2 Crop residue used as cooking medium indoors

Morbidity and Mortality

Globally, almost two million deaths per year are attributable to solid fuel use with more than 99 % of these occurring in developing countries [1]. The number of disability adjusted life years (DALYs) attributable to indoor air pollution from solid fuel use for all causes account to 40 million. India's figures are very alarming. Within yearly death toll of 662,000 attributed to biomass fuel exposure, India tops the list of the South Asian region [2]. Biomass fuel attributes for 5–6 % of the national burden of disease [5]. It has been estimated that indoor air pollution from solid fuel use in all developing countries accounted for about 1.6 million deaths annually in 2004 and about 500,000 in India in 2010 suggesting serious impact on health [6, 7].

The Emissions from a Biomass Stove and Exposure Determining Factors

Biomass fuel combustion results in production of numerous physical and chemical products which affect the health of the lung. When firewood is burnt, the combustion efficiency is far less than 100 % [8]. The biomass cook stoves wastes 74 % of the carbon as dissipated heat and only 18 % is used for real cooking [9]. Burning biomass

fuels emits toxic fumes into the air which is mix of small solid particles, Carbon Monoxide, Poly organic and poly aromatic hydrocarbons (PAH), Formaldehyde.

The small solid particles are the particulate matter having size less than 10 μm (PM10) and particles having size less than 2.5 μm are more hazardous for health as they can penetrate the lung [10]. The concentration of indoor particles less than 10 μm (PM10) measured over 24 h in Indian solid-fuel-using households is over 2,000 $\mu\text{g}/\text{m}^3$ compared to 30 $\mu\text{g}/\text{m}^3$ in USA [10].

The carbon monoxide produced during burning of various biomass fuels produces various short-term health effects like dizziness, headache, nausea, feeling of weakness, etc. and the long-term exposure can be likened to carbon monoxide from cigarette smoke which can lead to heart disease and fetal development anomaly [11, 12].

The PAH include a large class of compounds released during the incomplete combustion of organic matter [13]. Benzopyrene is one of the most important carcinogens of this group. The PAHs are fluorine, pyrene, chrysene, benzoanthracene, benzofluoranthene, benzopyrene, dibenzanthracene, benzoperylene, and indenopyrene. All these PAHs except the first three have been classified as possible carcinogens [14]. Formaldehyde is well recognized to be an acute irritant and long-term exposure can cause a reduction in vital capacity and chronic bronchitis. In an epidemiological study in UK, significantly excess mortality from lung cancer was observed in workers exposed to high levels of formaldehyde [15].

The overall pathological effect of biomass smoke can be taken as mutagen, immune system suppressant, severe irritant, blood poison, inflammatory agent, CNS depressant, cilia toxin, endocrine disruptures, and neurotoxin. They have also been firmly established as human carcinogens. Several toxic inorganic chemicals are known to cause asphyxiation, stillbirth, infant death, heart disease, and severe acute and chronic lung disease. Many mechanisms of cell injury are still unexplained.

Architecture of the House and Biomass Smoke Exposure

The level of exposure to these toxic fumes from a biomass stove varies widely with the house architecture and household composition. Quantitative exposure assessments in various households have been conducted in different parts of India for development of exposure–response relationships. The climatic and cultural variations between the northern and southern Indian regions have influenced outcome significantly. Cooking areas in many Indian households tend to be poorly ventilated, and about one half of all households do not have separate kitchen (Figs. 14.3 and 14.4). Most of the households lack a chimney or any other ventilatory measures. One study conducted in Porur, Chennai reported that 36 % households used biomass fuels for cooking in indoor kitchens without partitions, 30 % in separate kitchens inside the house, 19 % in separate kitchens outside the house, and 16 % in outdoor kitchens [16]. The personal exposure of cooks to the respirable particles in biomass smoke was not significantly different between indoor kitchens with or without



Fig. 14.3 Agricultural crop residues are used as a cooking medium indoors



Fig. 14.4 Another example of firewood used as a cooking medium indoors

partitions and separate kitchens outside the house but was significantly different from exposures of cooks using open outdoor kitchens as dispersion of emissions is greater outdoors as compared to indoors and therefore cooks cooking in open outdoors experience lower exposures compared to those in enclosed kitchens (Figs. 14.5 and 14.6). Households with kitchens without partitions experienced the highest



Fig. 14.5 Cooking is done in open space on coal



Fig. 14.6 Agricultural crop residues are used as a cooking medium outdoors

levels of living area concentrations as compared to other types. It was also observed that young children and the elderly who mostly occupy the living room are exposed to higher levels of smoke in unpartitioned indoor kitchens. Among non-cooks in households using solid fuels, women not involved in cooking and men with outdoor jobs have the lowest exposures, while women involved in assisting the cook and

men staying home have the highest exposure. There seems to be no significance for the cooking duration, the number of meals cooked, outdoor area measurements or the presence or absence of chimneys [9, 16].

Respiratory Health Effects of Biomass Fuels

Many respiratory diseases have been found to be associated with the exposure of Biomass fuels. The strength of association varies for such diseases like acute lower respiratory tract infections (ALRI), chronic obstructive pulmonary disease (COPD), lung cancer, pulmonary tuberculosis, asthma, and interstitial lung diseases. The evidence relating to their strength of association for ALRI in children <5 years is strong (relative risk 2.3, C.I. 1.9–2.7), for COPD in women more than 30 years age is strong (relative risk 3.2, C.I. 2.3–4.8), for lung cancer with coal smoke exposure the strength of association is also strong in women ≥ 30 years (relative risk 1.9, C.I. 1.1–3.5). For tuberculosis and asthma the strength of association is moderate, the relative risk being 1.5 (C.I. 1.0–2.4) and 1.2 (C.I. 1.0–1.5), respectively [17]. There are also studies including meta-analyses depicting association between solid fuel and risk of common respiratory disease from India as summarized in Table 14.1.

Acute Lower Respiratory Infection in Children Under 5 Years of Age

Acute lower respiratory infection (ALRI) contributes to 13 % of deaths and 11 % of the national burden of diseases [5]. This is one of the major diseases associated with the indoor air quality. There are many studies to date that show various respiratory symptoms (coughing, wheezing etc) to be associated with solid fuel smoke exposures. However none of them provide sufficient evidence to calculate odds ratio. A host of odd's ratios ranging from 1.9 to 2.7 have been worked out [17]. These ratios pertain to children with ALRI younger than 5 years only. Other factors might strongly influence ALRI incidence like housing type, location of cooking, and other cultural practices [18]. Some of the studies carried out in India have reported no association between use of biomass fuels and ALRI in children. In a case–control study in children under 5 years of age in southern Kerala, India where children with severe pneumonia (ascertained by WHO criteria) were compared with those having non-severe ALRI attending out patient department, cooking fuel was not a severe risk factor for severe ALRI [19]. Also Sharma et al. in a cross-sectional study involving 642 infants dwelling in urban slums of Delhi and using wood and kerosene, respectively, did not find a significant difference in the prevalence of ALRI infections and the fuel types [18].

Table 14.1 Major Indian studies depicting association between current solid fuel use relative to cleaner burning fuel or electricity and risk of common respiratory diseases

Respiratory disease	Authors	Study type	Outcome	Odd's ratio/ incidence risk ratio (95 % CI)	References	
Tuberculosis	Gupta et al. (1997)	Case-control India	Clinical pulmonary	2.54 (1.07-6.04)	[21]	
	Mishra et al. (1999)	Cross-sectional: India (National Family Health Survey)	Self-reported	2.58 (1.98-3.37)	[28]	
ALRI	Shetty et al. (2006)	Case-control India	Clinical pulmonary	3.26 (1.25-8.46)	[33]	
	Magheswari et al. (2008)	Case-control India	Clinical pulmonary	0.22 (0.12-0.41)	[34]	
	Kolappan et al. (2009)	Case-control India	Clinical pulmonary	2.9 (1.8-4.7)	[27]	
	Behera et al. (2010)	Case-control India	Clinical pulmonary	0.60 (0.22-1.63)	[35]	
	Lakshmi et al. (2012)	Case-control India	Clinical pulmonary	2.33 (1.18-4.59)	[29]	
	Mishra et al. (1999)	Cross-sectional survey	Self-reported symptoms	1.58 (1.28-1.95)	[28]	
	Dherani et al. (2008)	Meta-analysis	24 studies for calculation of OR	1.78 (1.45-2.18)	[36]	
	Ramaswamy et al. (2011)	Longitudinal cohort	Clinical symptoms and estimation of the incidence risk ratio among children from households using biomass fuels relative to cleaner fuels	1.33 (1.02-1.73)	[37]	
	Lung cancer	Gupta et al. (2001)	Case-control India	Clinical, radiological and histopathological assessment	1.52 (0.33-6.98)	[38]
		Behera et al. (2005)	Case-control India	Clinical, radiological and histopathological assessment	3.59 (1.07-11.97)	[24]
Sapkota et al. (2008)		Case-control India	Clinical, radiological and histopathological assessment	3.76 (1.64-8.63)	[25]	
COPD	Hosgood et al. (2011)	Meta-analysis	25 studies for estimation of OR	2.15 (1.61-2.89)	[39]	
	Behera et al. (1991)	Descriptive study	Clinical assessment	3.04 (2.15-4.31)	[40]	
	Qureshi et al. (1994)	Case-control India	Clinical assessment	2.10 (1.50-2.94)	[41]	
	Kurmi et al. (2010)	Meta-analysis	12 studies for estimation of OR	2.80 (1.85-4.0)	[42]	

Chronic Obstructive Pulmonary Disease

COPD accounts for 1.5 % of deaths and forms 0.9 % of national burden of diseases in India [5]. The incidence of chronic cor pulmonale is similar in both men and women. This is despite the fact that only 10 % women are smokers compared to 75 % men. Another point to note is that chronic cor pulmonale occurs 10–15 years earlier in women compared to men [20]. A relative risk of 2–4 has been arrived at for biomass fuel exposure in various Indian studies [5]. Despite the progress made in highlighting the association between biomass fuel exposure and *COPD*, many shortcomings still exist. Smoking is an important confounding variable for *COPD* and particularly so when men are included in the analyses. Another major confounding factor is age. The risk for *COPD* increases with age and many age-matched studies have provided insufficient quantitative evidence to develop an odds ratio (OR). The overall risk of *COPD* in women exposed to biomass fuel has been estimated as 3.2 (95 %CI 2.3–4.8) [17]. There is much less evidence available about the impact on men, but the risk seems to be lower with OR of 1.8 (95 %CI 1.0–3.2). This may be attributed to the lower exposure to biomass fumes in men [21].

Lung Cancer

Lung cancer in women is a well-demonstrated outcome of cooking with open coal stoves in China [22]. Indian women generally have low lung cancer rates [23]. This may be in a way attributed to the minimal use of coal for cooking in Indian households. Nevertheless a few studies from India have suggested an association with lung cancer even after adjusting for active and passive smoking. An odds ratio of 3.59 (95 %CI 1.07–11.97) has been worked out [24]. In conclusion, it may be inferred that there is a general lack of epidemiological evidence relating lung cancer with biomass fuel exposure. The limited cases reported have been linked with exposure to coal fires [25].

Tuberculosis

Tuberculosis (TB) is a major public health problem in India. Out of the 9.4 million new cases recorded globally 1.98 million are reported from India [26]. It is estimated that 276,000 deaths occur annually due to TB in India. There is a strong association between the use of biomass fuel and pulmonary TB. A high risk of pulmonary TB exists in those using wood and cow dung cake as cooking fuel (Figs. 14.7, 14.8, and 14.9) [27]. It is suggested that lowered immunodefense mechanisms of the lung may be the reason for disease presentation. Biomass fuel poses a higher risk (969/100,000) of TB compared to cleaner fuels (378/100,000). It is



Fig. 14.7 The preparation of cow dung cakes is shown



Fig. 14.8 A woman and child using agricultural crop residues as a cooking medium indoor

believed that 51 % of active TB in age group more than 20 years is attributable to cooking smoke from biomass fuels [28].

A recent study done from northern part of India among adult women having sputum positive pulmonary tuberculosis as cases and age-residence area matched controls revealed OR for biomass fuel compared with LPG was 2.33 (C.I. 1.18–4.59).



Fig. 14.9 A man using fire wood as a cooking medium indoors

Adjustment for confounding factors (education, type of kitchen, smoking tobacco, and TB in family member) and interaction between cooking fuel and smoker in family revealed an OR of 3.14 (C.I. 1.15–8.56) [29]. Given the importance of TB in India, because it is both prevalent and likely to increase with HIV epidemic, these findings need to be followed up with more detailed studies.

Pneumoconiosis and Interstitial Lung Diseases

Pneumoconiosis has been reported from Ladakh, a hilly terrain in northern most part of India [30]. This place is completely devoid of industries or mines. Yet cases have been reported of diseases resembling miner's pneumoconiosis. Another factor considered responsible for the development of this respiratory morbidity is the exposure to dust from dust storms. In spring, dust storms blanket the villages in fine dust. The practice of not allowing the wood to burn quickly and smouldering for longer duration to conserve fuel adds to the high level of respirable particles indoors. Low oxygen levels or some other factor associated with high altitude may be an important contributory factor in causation of pneumoconiosis because it has been reported that the miners working at high altitude are more prone to develop pneumoconiosis than their counterparts exposed to the same levels of dust and working in the mines at normal altitude [31]. The causal role of biomass fuel exposure is however not established [30]. Similarly, a few case reports linking ILD and biomass fuel exposure have been documented. But here too, the veracity of the association is still debatable [32].

Biomass Fuel Exposure and Lung Functions

There are only three studies throwing light on the effect on lung functions from use of domestic cooking fuels including biomass fuel from India. Out of this one study included children from north India which revealed forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV1) lowest in boys, whose households used biomass fuel ($p < 0.05$) and peak expiratory flow rate (PEFR) and forced expiratory flow (FEF) 25 and 50 % also lowest in boys with their homes using kerosene as fuels. All these were the best for LPG fuel [33–43]. The other two studies carried out from northern part of India revealed different outcomes; the first study carried out in 3,318 rural nonsmoking women using different domestic cooking fuel like biomass fuel, LPG, kerosene, and mixed revealed biomass fuel users had FVC values less than 75 % predicted whereas in other groups it was more than 75 % of predicted, though less than 80 % of the predicted values. The absolute values of all the three parameters (FVC, FEV1, and PEFR) of lung functions were the lowest in the biomass and mixed fuel users [44]. The second study was comparative study to see the lung functions of healthy nonsmoking women who used either biomass or liquified petroleum gas (LPG) as their sole cooking fuel. The effects of passive smoking, ventilation, overcrowding and cooking index were also taken into account. The results of this study revealed no statistically significant differences in lung functions in the two groups except for the PEFR, which was significantly lower ($P < 0.01$) in women using biomass. No correlation was observed between different variables and pulmonary functions. The stepwise multivariate linear regression analysis showed no correlation between cooking fuel and the pulmonary functions. Authors concluded that the absence of the expected adverse effects of biomass on pulmonary functions was possibly due to better ventilation in the kitchens of subjects in the biomass group compared to previous studies [45]. These studies indicate that the lung functions are adversely affected by the use of biomass as domestic cooking fuel in comparison to cleaner fuels but have the linear relationship with the duration of cooking, overcrowding, poor ventilation and has negative correlation with better ventilated kitchens.

Conclusion

In conclusion, biomass fuel exposures contribute heavily to the burden of diseases in India. Despite heterogeneity of the published literature, available evidences suggest significant associations with diseases like ALRI and COPD. Other diseases, where solid fuel smoke is thought to play a role, needs more evidence to establish the association and there are evidences that the lung functions are adversely affected with the use of biomass fuel as domestic cooking fuel as compared to the cleaner fuel and there is negative correlation with better ventilated kitchens. Therefore, exposure reduction strategy should be adopted. It is also believed that selection of

the strategies to withdraw or reduce the exposure is very challenging and probable need of the hour for country like ours. Exposure reduction strategy will require consideration of not only the personal exposure but also cultural and economic aspects, both at individual as well as local levels including the level of development, resources, technical capacity, the domestic needs of energy, the sustainability of the considered sources of energy, and the protection of the environment. Substantial improvement can be desired by health education and cultural modification, modification of stove design and switching over to cleaner fuels or other high efficiency with low emission fuels for cooking. There should be increased awareness regarding the health effects of solid fuel smoke inhalation among physicians and health administrators, which may improve not only research but also preventive actions as well as diagnosis and treatment of affected patients in future. It is hoped that in future studies on biomass exposure-associated morbidities and its prevention would claim priority. This is highly desired considering the mammoth risk the solid fuels pose in rural India.

References

1. World Health Organization. The world health report: 2004—changing history. <http://www.who.int/whr/2004/en/> Accessed 12 Feb 2012.
2. International Institute of Population Sciences (IIPS). National Family Health Survey (MCH and family planning): India 2005–06. Bombay: International Institute of Population Sciences; 2007.
3. World Health Organization. The world health statistics: 2012. <http://www.who.int/whosis/who-stat/2012/en/> Accessed 24 Sept 2012.
4. GoI. Service sector implementation, NSSO enterprises (extending trade) and consumer expenditure. National Sample Survey report, 63rd round 2006–2007. Government of India, Ministry of Statistics and Program, New Delhi, India
5. Smith KR. National burden of disease in India from indoor air pollution. *Proc Natl Acad Sci USA*. 2000;97:13286–93.
6. Smith KR, Mehta S, Maeusezahl-Feuz M. Indoor smoke from household solid fuels. In: Ezzati M, Rodgers AD, Lopez AD, et al., editors. Comparative quantification of health risks: global and regional burden of disease due to selected major risk factors, vol. 2. Geneva: World Health Organization; 2004. p. 1435–93.
7. Wilkinson P, Smith KR, Davies M, et al. Household energy, series on the impact on public health of strategies to reduce greenhouse gases. *Lancet*. 2009;374:1917–29.
8. Smith KR. Biomass, air pollution, and health; a global review. New York: Plenum; 1987.
9. Balakrishnan K, Sankar S, Padmavathi R, Smith KR. Respirable particulate levels in rural households of Andhra Pradesh, India: daily concentrations and exposures. *J Environ Stud Policy*. 2002;5:87–97.
10. Smith KR. Indoor air pollution in developing countries: growing evidence of its role in the global disease burden. In: K. Ikeda T. Iwata, editors. *Indoor Air '96: Proceedings of 7th International Conference on indoor air and climate*. Tokyo: Institute of Public Health; 1996. p. 33.
11. World Health Organization, International Programme on Chemical Safety. Environmental health criteria 202: selected non-heterocyclic, polycyclic aromatic hydrocarbons, vol. 3. Geneva: WHO; 1998. p. 63.
12. USDHHS. The health consequences of smoking—cardiovascular diseases. A report of the Surgeon General. Rockville, MD: US Department of Health and Human Services; 1983.

13. Perera FP, Hemmlinki K, Young TL, Brenner D, Kelly G, Santella RM. Detection of poly-aromatic hydrocarbon- DNA adducts in white blood cells of foundry workers. *Cancer Res.* 1988;48:2288–91.
14. Schwarz-Miller J, Rom WN, Brandt-Rauf PW. Polycyclic aromatic hydrocarbons. In: Rom RN, editor. *Environmental and occupational health*, vol. 2. London: Little Brown; 1991. p. 873.
15. Acheson ED, Barnes HR, Gardner MJ, Osmond C, Pannet B, Taylor CP. Formaldehyde process, workers and lung cancer. *Lancet.* 1984;1:1066–7.
16. Balakrishnan K, Parikh J, Sankar S, Padmavathi R, Srividya K, Venugopal V, et al. Daily average exposures to respirable particulate matter from combustion of biomass fuels in rural households of southern India. *Environ Health Perspect.* 2002;110:1069–75.
17. Desai MA, Mehta S, Smith KR. Indoor smoke from solid fuels: assessing the environmental burden of disease at national and local levels, *Environmental burden of disease series no. 4.* Geneva: WHO protection of the human environment; 2004.
18. Sharma S, Sethi GR, Rohtagi A, Chaudhary A, Shankar R, Bapna JS, Joshi V, Sapir DG. Indoor air quality and acute lower respiratory infection in Indian urban slums. *Environ Health Perspect.* 1998;106(5):291–7.
19. Shah N, Ramankutty V, Pramila PG, Sathy N. Risk factors for severe pneumonia in children in south Kerala: a hospital based case–control study. *J Trop Paediatr.* 1994;40:201–6.
20. Padmavati S, Pathak SN. Chronic cor pulmonale in Delhi. *Circulation.* 1959;20:343–52.
21. Gupta B, Mathur N, Mahendra P, Srivastava A, Swaroop V, Agnihotri M. A study of the household environmental risk factors pertaining to respiratory disease. *Energy Environ Rev.* 1997;13:61–7.
22. Smith KR, Liu Y. Indoor air pollution in developing countries. In: Samet J, editor. *The epidemiology of lung cancer*, vol. 3. New York: Dekker; 1994. p. 151–84.
23. Alberg AJ, Samet JM. Epidemiology of lung cancer. *Chest.* 2003;123:21S.
24. Behera D, Balamugesh T. Indoor air pollution as a risk factor for lung cancer in women. *J Assoc Physicians India.* 2005;3:190–2.
25. Sapkota A, Gajalakshmi V, Jetli DH, Chowdhary SR, Dikshit RP, Brennan P, Hashib M, Boffetta P. Indoor air pollution from solid fuels and risk of hypopharyngeal/laryngeal and lung cancers: a multicentric case–control study from India. *Int J Epidemiol.* 2008;2:1–8.
26. GoI. RNTCP TB status report 2010. Government of India, Central TB Division, Ministry of Health and Family Welfare, New Delhi, India.
27. Kolappan C, Subramani R. Association between biomass fuel and pulmonary tuberculosis: a nested case–control study. *Thorax.* 2009;64:705–8.
28. Mishra VK, Ratherford RD, Smith KR. Biomass cooking fuels and prevalence of tuberculosis in India. *Int J Infect Dis.* 1999;3:119–29.
29. Lakshmi PV, Virdi NK, Thakur JS, Smith KR, Bates MN, Kumar R. Biomass fuel and risk of tuberculosis: a case–control study from Northern India. *J Epidemiol Community Health.* 2012;66(5):457–61. doi:10.1136/jech.2010.115840.
30. Saiyed HN, Sharma YK, Sadhu HG, Norboo T, Patel PD, Patel TS, Venkaiah K, Kashyap SK. Non-occupational pneumoconiosis at high altitude villages in central Ladakh. *Br J Ind Med.* 1991;48:825–9.
31. Odinaev FI. The characteristics of development and course of pneumoconiosis under the conditions of a mountain climate. *Gig Tr Prof Zabol.* 1992;7:13–4 (Russian).
32. Dhar SN, Pathania AGS. Bronchitis due to biomass fuel burning: ‘Gujjar lung’ an extreme effect. *Semin Respir Med.* 1991;12:69–74.
33. Shetty N, Shemko M, Vaz M, D’Souza G. An epidemiological evaluation of risk factors for tuberculosis in South India: a matched case control study. *Int J Tuberc Lung Dis.* 2006;10:80–6.
34. Magheswari U, Johnson P, Ramaswamy P, Balakrishnan K, Jenny A, Bates M, et al. Exposure to biomass fuel smoke and tuberculosis—a case–control study in India. *Epidemiology.* 2007;18:s122.

35. Behera D, Aggarwal G. Domestic cooking fuel exposure and tuberculosis in Indian women. *Indian J Chest Dis Allied Sci.* 2010;2:139–43.
36. Dherani M, Pope D, Mascarenhas M, Smith KR, Weber M, Bruce N. Indoor air pollution from unprocessed solid fuel use and pneumonia risk in children aged under five years: a systematic review and meta-analysis. *Bull World Health Organ.* 2008;86:390–8.
37. Ramaswamy P, Balakrishnan K, Ghosh S, et al. Indoor air pollution due to biomass fuel combustion and acute respiratory infection in children under 5 in Trichy district of rural Tamilnadu, India. *Epidemiology.* 2011;22:s104.
38. Gupta D, Boffetta P, Gaborieau V, Jindal SK. Risk factors of lung cancer in Chandigarh, India. *Indian J Med Res.* 2001;113:142–50.
39. Hosgood III HD, Wei H, Sapkota A, Choudhury I, Bruce N, Smith KR, et al. Household coal use and lung cancer: systematic review and meta-analysis of case–control studies, with an emphasis on geographic variation. *Int J Epidemiol.* 2011;40:1–10.
40. Behera D, Jindal SK. Respiratory symptoms in Indian women using domestic cooking fuels. *Chest.* 1991;100:385–8.
41. Qureshi KA. Domestic smoke pollution and prevalence of chronic bronchitis/asthma in a rural area of Kashmir. *Indian J Chest Dis Allied Sci.* 1994;36:61–72.
42. Kurmi OP, Semple S, Simkhada P, Smith WCS, Ayres JG. COPD and chronic bronchitis risk of indoor air pollution from solid fuel: a systematic review and meta-analysis. *Thorax.* 2010;65:221–8.
43. Behera D, Sood P, Singh S. Passive smoking, domestic fuels and lung functions in north Indian children. *Indian J Chest Dis Allied Sci.* 1998;40(2):89–98.
44. Behera D, Jindal SK, Malhotra HS. Ventilatory function in nonsmoking rural Indian women using different cooking fuels. *Respiration.* 1994;61(2):89–92.
45. Reddy TS, Guleria R, Sinha S, Sharma SK, Pande JN. Domestic cooking fuel and lung functions in healthy non-smoking women. *Indian J Chest Dis Allied Sci.* 2004;46(2):85–90.

Chapter 15

The Effects of Climate Change and Air Pollution on Children and Mothers' Health

Roya Kelishadi and Parinaz Poursafa

Abstract Air pollution and climate change have numerous health hazards for pregnant mothers and children. Therefore, environmental protection activities should be considered a health priority. The importance of environmental factors on maternal and child health care should be considered one of the main public health priorities for primordial/primary prevention of chronic diseases.

Keywords Children's health and climate change • Climate change and effects on mothers and children • Air pollution and children's health • Air pollution effects on mothers' and children's health • Health effects of ecological changes

According to the World Health Organization (WHO), the burden of climate-related disease and air pollution is greater for children than adults [1]. This issue is of special importance for low- and middle-income countries [2] as climate change alters air pollutant concentrations and transport [3, 4]. Groups especially vulnerable to these changes include pregnant mothers, neonates, infants, and children.

The health impacts of climate change have different underlying mechanisms. These impacts may be direct via cold or heat stress or indirect through influences on natural systems. Climate and weather extremes may cause floods, famine, food insecurity, social disturbance, and population displacement. Consequently, these environmental factors are predisposing factors for both communicable and non-communicable diseases [3].

R. Kelishadi, M.D. (✉)

Department of Pediatrics, Child Growth and Development Research Center, Isfahan University of Medical Sciences, Hezar Jerib Avenue, Isfahan, Iran

P. Poursafa, Ph.D.

Environment Research Center, Isfahan University of Medical Sciences, Isfahan, Iran

Based on climate models, it is proposed that increased greenhouse gas emissions lead to higher mean temperatures, which in turn promote stronger storms and droughts, with serious health implications [5].

Health Effects of Ecological Changes

Climatic and ecological changes present several health threats that can be classified as primary, secondary, and tertiary. The primary effects of climate change include increased heat waves and wildfires. Secondary effects are indirect, such as altered distribution of arthropod vectors and intermediate hosts and pathogens along with consequent change in the epidemiology of many infectious diseases. Health impacts related to future health consequences, such as famine and substantial population shift, are tertiary effects. Air pollution can be used to provide an example of all three classifications. Consider that high levels of air pollution cause the development of acute respiratory and irritant symptoms (primary effect). As a result, hospitalization and mortality rates increase (secondary effect). Long-term consequences of the exposure include low birth weight, endothelial dysfunction and genetic disorders (tertiary effects) [6]. Key adverse effects of climate change and air pollution on maternal and child health are summarized below:

Climate Change

Effects of Climate Change on Maternal Health

Climate change puts maternal health at risk, with the major health effects of climate change and warming increasing pregnancy complications and renal disorders [7].

- *Association of climate change with toxemia of pregnancy:* Some environmental factors, such as increased humidity, may increase the risk of preeclampsia and eclampsia [8].
- *Association of climate change with premature labor:* Exposure of pregnant mothers to extreme heat, mainly in the second and third trimesters, increases the risk of premature birth and or low birth weight [9].
- *Climate change and renal effects in mothers:* Temperature influences body fluid and urine volume. An association between increasing temperature in summer time and hospitalization rates for renal disorders, such as renal stones, is well documented. The climate-related burden of renal diseases is expected to grow globally [10–13]. Pregnant and lactating women, who require extra hydration, are especially vulnerable to renal disorders related to climate change.

Effects of Climate Change on Child Health

It is estimated that more than 88 % of the burden of disease related to climate change occurs in children less than 5 years of age [14]. Greenhouse gas emissions promote water cycle intensification [15], which is especially important for children who are most prone to water-borne diseases, injury, food insufficiencies [14], exposure to toxic compounds [15], emotional disturbance, and renal [16] and mental disorders [17].

Climate Change and Food Safety for Mothers and Children

One of the most important effects of climate change on maternal and child health is decreased food quantity and quality [18]. Agricultural production must double by 2,050 to supply worldwide demand [19]. In turn, a doubling of food production puts a strain on water supplies; increases fertilizer application and its impact on groundwater; and drives air pollution as well as acidification of soils and freshwater [20]. Climate change may also compromise the nutritional quality of certain foods because grains grown at elevated concentrations of carbon dioxide have the potential for decreased content of protein and nutrients, such as iron and zinc [21, 22].

Air Pollution

Effects of Air Pollutants on Maternal Health

There is a growing body of evidence that air pollutants are associated with low birth weight and prematurity. Individuals who experienced intrauterine growth retardation, low birth weight [23], and/or prematurity [24] are at an increased risk for non-communicable diseases, such as obesity, hypertension and cardiovascular disease, later in life.

Effects of Air Pollutants on Child Health

Short-Term Effects of Air Pollutants on Child Health

Respiratory and allergic symptoms are the most common short-term effects of exposure to air pollution. Infants and children are most likely the most vulnerable age group to air pollutants because they have greater respiratory rates than adults and, therefore, greater exposure to air pollutants. The mouth breathing of infants

and children may bypass the filtering effect of the nose; consequently, children would inhale higher levels of pollutants than adults. Furthermore, children generally spend more time outdoors than adults, and their immune system and organs are immature [25].

Long-Term Effects of Air Pollutants on Child Health

Exposure to air pollution during early life is associated with several harmful health effects, such as perinatal disorders, infant mortality, malignancies, cardiovascular disorders, oxidative stress, endothelial dysfunction, mental disorders and vitamin D deficiency [26]. The long-term effects of air pollutants and their impact on chronic disease may be mediated by systemic inflammation [27, 28]. An association between air pollution and consequent inflammatory and oxidative stress can be found in early life [29, 30].

Conclusion

Air pollution and climate change have numerous health hazards for pregnant mothers and children. Therefore, environmental protection activities should be considered a health priority. The importance of environmental factors on maternal and child health care should be considered one of the main public health priorities for primordial/primary prevention of chronic diseases.

References

1. WHO (World Health Organization) Global burden of disease: 2004 Update. 2008. Available:http://www.who.int/healthinfo/global_burden_disease/GBD_report_2004update_full.pdf. Accessed 20 June 2012.
2. Haines A, Kovats RS, Campbell-Lendrum D, Corvalan C. Climate change and human health: impacts, vulnerability, and mitigation. *Lancet*. 2006;367:2101–9.
3. Confalonieri U, Menne B, Akhtar R, Ebi KL, Hauengue M, Kovats RS, Revich B, Woodward A. Human health. In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE, editors. Contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press; 2007. p. 391–431.
4. Greenough G, McGeehin M, Bernard SM, Trtanj J, Riad J, Engelberg D. The potential impacts of climate variability and change on health impacts of extreme weather events in the United States. *Environ Health Perspect*. 2001;109 Suppl 2:191–8.
5. CCSP (2008). *Analyses of the effects of global change on human health and welfare and human systems*. A Report by the U.S. Climate Change Science Program and the Subcommittee on Global Change Research. Gamble JL, editors, KL Ebi, FG Sussman, TJ Wilbanks (Authors). U.S. Environmental Protection Agency, Washington, DC.
6. Poursafa P, Kelishadi R. What health professionals should know about the health effects of air pollution and climate change on children and pregnant mothers. *Iran J Nurs Midwifery Res*. 2011;16:257–64.

7. Zhang Y, Bi P, Hiller JE. Climate change and disability-adjusted life years. *J Environ Health*. 2007;70:32–6.
8. Subramaniam V. Seasonal variation in the incidence of preeclampsia and eclampsia in tropical climatic conditions. *BMC Womens Health*. 2007;7:18.
9. Deschênes O, Greenstone M, Guryan J. Climate change and birth weight. *Am Econ Rev*. 2009;99:211–7.
10. Chen Y-Y, Roseman JM, DeVivo MJ, Huang C-T. Geographic variation and environmental risk factors for the incidence of initial kidney stones in patients with spinal cord injury. *J Urol*. 2000;164:21–6.
11. Cramer JS, Forrest K. Renal lithiasis: addressing the risks of austere desert deployments. *Aviat Space Environ Med*. 2006;77:649–53.
12. Kovats RS, Hajat S, Wilkinson P. Contrasting patterns of mortality and hospital admissions during hot weather and heat waves in Greater London, UK. *Occup Environ Med*. 2004;61(11):893–8.
13. Brikowski TH, Lotan Y, Pearle MS. Climate-related increase in the prevalence of urolithiasis in the United States. *Proc Natl Acad Sci USA*. 2008;105(28):9841–6.
14. Sheffield PE, Landrigan PJ. Global climate change and children's health: threats and strategies for prevention. *Environ Health Perspect*. 2011;119:291–8.
15. Ohl CA, Tapsell S. Flooding and human health. *BMJ*. 2000;321:1167–8.
16. Mandeville JA, Nelson CP. Pediatric urolithiasis. *Curr Opin Urol*. 2009;19(4):419–23.
17. McLaughlin K, Fairbank J, Gruber M, et al. Serious emotional disturbance among youths exposed to hurricane Katrina 2 years postdisaster. *J Am Acad Child Adolesc Psychiatry*. 2009;48:1069–78.
18. Dean J, Stain H. Mental health impact for adolescents living with prolonged drought. *Aust J Rural Health*. 2010;18:32–7.
19. Alexandratos N. World food and agriculture: outlook for the medium and longer term. *Proc Natl Acad Sci USA*. 1999;96:5908–14.
20. Tilman D. Forecasting agriculturally driven global environmental change. *Science*. 2001;292:281–4.
21. Idso SB, Idso KE. Effects of atmospheric CO₂ enrichment on plant constituents related to animal and human health. *Environ Exp Bot*. 2001;45:179–99.
22. Taub DR, Miller B, Allen H. Effects of elevated CO₂ on the protein concentration of food crops: a meta-analysis. *Global Chang Biol*. 2008;14:565–75.
23. Sinclair KD, Lea RG, Rees WD, Young LE. The developmental origins of health and disease: current theories and epigenetic mechanisms. *Soc Reprod Fertil Suppl*. 2007;64:425–43.
24. Evensen KA, Steinshamn S, Tjonna AE, et al. Effects of preterm birth and fetal growth retardation on cardiovascular risk factors in young adulthood. *Early Hum Dev*. 2008;85:239–45.
25. Kim JJ, American Academy of Pediatrics Committee on Environmental Health. Ambient air pollution: health hazards to children. *Pediatrics*. 2004;114:1699–707.
26. Kelishadi R, Poursafa P. Air pollution and non-respiratory health hazards for children. *Arch Med Sci*. 2010;6:483–95.
27. Brook RD, Franklin B, Cascio W, Expert Panel on Population and Prevention Science of the American Heart Association. Air pollution and cardiovascular disease: a statement for health-care professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. *Circulation*. 2004;109:2655–71.
28. Huang SL, Hsu MK, Chan CC. Effects of submicrometer particle compositions on cytokine production and lipid peroxidation of human bronchial epithelial cells. *Environ Health Perspect*. 2003;111:478–82.
29. Chuang KJ, Chan CC, Su TC. The effect of urban air pollution on inflammation, oxidative stress, coagulation, and autonomic dysfunction in young adults. *Am J Respir Crit Care Med*. 2007;176:370–6.
30. Poursafa P, Kelishadi R, Lahijanzadeh A, Modaresi M, Javanmard SH, Assari R, Amin MM, Moattar F, Amini A, Sadeghian B. The relationship of air pollution and surrogate markers of endothelial dysfunction in a population-based sample of children. *BMC Public Health*. 2011;11:115.

Chapter 16

Climate Change and Public Health in Small Island States and Caribbean Countries

Muge Akpinar-Elci and Hugh Sealy

Abstract According to popular perspective the small island states in the Caribbean, Indian Ocean, and Pacific regions are regarded as tropical paradises, far from the rest of the world and with pristine environments unharmed from climate change. Unfortunately this assumption is not true. Small island communities are the most vulnerable populations susceptible to the effects of climate change including warmer temperatures, rising sea levels, rainfall changes, and more severe extreme weather events such as hurricanes and tropical storms. Climate change impacts coral reefs, fisheries, and other marine-based resources and leads to the replacement of some local species, which negatively affects tourism, the most important source of income. Many small islands are located in tropical zones, where the climate is prone to increased risk from heat stress, asthma, and vector-, food-, and waterborne diseases. Changing climate conditions can worsen the impacts of these diseases. Thus, the effects of climate change create significant risks to public health, food security, natural resources, and the general economies of small island communities. This chapter highlights the impacts of climate change among small island communities and discusses their vulnerabilities.

Keywords Small island states and climate change • Global warming • Public health in Caribbean • Disease exacerbation in small island states • Sea level rise and small island states • Caribbean and global warming

The effects of climate change (CC) on human health are a growing global issue. Small island states are more susceptible to CC related with public health, food security, natural resources, and fragile economies of the developing world. In the small

M. Akpinar-Elci, M.D., M.P.H (✉) • H. Sealy, M.Sc., Ph.D.
Department of Public Health and Preventative Medicine, St. George's University Medical School, St. George, Grenada
e-mail: makpinarelei@gmail.com; hsealy@sgu.edu

island states, CC exacerbates the impacts of many diseases such as heat stress, asthma, and vector-, food-, and waterborne diseases. The small island states from the Caribbean to the Pacific believe that if the world does not act quickly enough to identify and implement solutions for mitigation or adaptation for CC, then the small islands will suffer more in the near future. In this chapter, we will summarize current knowledge on the physical, socioeconomic, and health effects of CC from the small island states' perspective. We also share our experience as small islanders. The final section outlines the chapter's conclusions.

The Physical Context: Current and Expected Physical Effects of Climate Change on Small Island States

Tropical or subtropical small islands exhibit variable climates but can be generally characterized by distinct wet and dry seasons (precipitation) and small seasonal variations in temperature. They are also characteristically threatened by periodic extreme weather events such as cyclones. In the following section of this chapter, the current and predicted (by 2100) physical manifestations of CC on small islands, with emphasis on those in the Caribbean Sea, are discussed.

Atmospheric Temperatures

Until the next, much anticipated, definitive report is issued by the Intergovernmental Panel on Climate Change (IPCC); sometime in late 2014, the 2007 4th Assessment Report (AR4) remains the primary source of scientific prediction of the physical manifestations of CC [1]. However, it is noted that many scientists now believe that CC is occurring at a faster rate and with greater intensity than was predicted by the models used in the AR4. The models used in the AR4 provide a range of predicted increases (1.1–6.4 °C) for average atmospheric temperatures near the Earth's surface (over land and over water) until the end of the twenty-first century.

The predicted average global atmospheric temperature increase resulting from a doubling of CO₂ concentrations is between 2 and 4.5 °C. The latest data indicate that the world has already seen a 0.8 °C increase in average atmospheric temperatures, and the Special Report on Emissions Scenarios (SRES) models in the IPCC AR4 predict further warming at a rate of ~0.2 °C per decade. There is little evidence of changes in diurnal temperature ranges with both day and night temperatures increasing at the same rate. The latest update to the "Bridging the Emissions Gap" report published by the United Nations Environment Programme (UNEP [2]) predicts that the world is on an emission pathway that will take temperature increases past 3 °C [2]. The International Energy Agency has indicated that more CO₂ was released from the combustion of fossil fuels globally in 2010 than in any other year in the history of mankind [3].

The temperature increases are not and will not be the same around the globe. More warming will occur at the poles than at the equator. More warming of the atmosphere is occurring over land than over water, although greater than 80 % of the increased heat is being absorbed by the oceans. This implies that for tropical small island states, the increase in atmospheric temperatures may be different to the global average [4].

Data analysis by Trenberth et al. indicates consistent but nonlinear warming trends in all small island regions (Caribbean, Mediterranean, Indian Ocean, and Pacific) during the period 1901–2004 [5]. Ocean surface and island air temperatures have increased by between 0.6 and 1.0 °C with decadal increases between 0.3 and 0.5 °C in the Pacific as compared to 0–0.5 °C increases per decade in the Caribbean, Indian Ocean, and Mediterranean islands between 1971 and 2004.

The seven Atmospheric Ocean General Circulation Models used in the IPCC AR4 all predict increases in surface air temperatures for all of the island regions. Surface air temperatures are predicted to be at least 2.5 °C higher than 1990 levels by 2100 in the South Pacific [6].

Sea Level Rise and Ocean Acidification

Perhaps the greatest existential threat posed by CC to small islands and low-lying coastal states is that of Sea Level Rise (SLR) and the acidification of the oceans. Currently, the majority of the SLR is predicted to be from thermal expansion, with temperature increases already being evident at depths greater than 3,000 m [1].

The amount of SLR that islands will experience by 2100 will be dependent upon the degree of further warming that will occur, with nonlinear responses and tipping points potentially occurring. For example, loss of the Greenland and West Antarctica ice sheets would significantly increase the magnitude and public health threat of SLR to small islands. If temperature increases are between 3 and 5 °C by the end of the century, as was the case during the last interglacial period about 125,000 years ago, an associated SLR of 4–6 m could be expected.

Observed SLR has varied across regions and even across individual islands (e.g., 1 mm/year in the north as compared to 0.4 mm/year in southern Trinidad) as a result of tectonic shifts [7]. Pulwarty et al. claim that sea levels rose in the Caribbean an average of 10 cm during the twentieth century [8]. However, it is noted that islands like Barbados are also experiencing tectonic uplift, which would ameliorate the effect of SLR.

According to the fourth Assessment Report, sea levels have been rising at ~1.8 mm/year between 1961 and 2003 and at ~3.1 mm/year between 1993 and 2003, primarily due to thermal expansion with some contribution from glacier and ice cap melt [1]. It is unclear whether the increased rate of SLR from 1993 is due to decadal variability or represents a trend. Six SRES predict a range of SLR between 0.18 and 0.59 m as a global average for the last decade in the twenty-first century. These models do not include a significant increase in ice melt from Greenland or Antarctica. There have been estimates that global mean sea levels may rise in excess

of 5 m above 1990 levels if there is significant loss of the Greenland and West Antarctica ice sheets. It is noted that current emissions (early 2012) have us on target for a temperature increase of between 3 and 4 °C with the concomitant increased likelihood of considerable ice melt.

The latest data from the Arctic Monitoring and Assessment Programme predicts that sea levels will rise on average 0.9–1.6 m by 2100 (as compared to the 0.18–0.59 m in the AR4) and provided evidence that the melting of glaciers, sea ice, the Greenland ice sheet, and ice caps had further increased in the last decade [9].

The least reported but perhaps the most insidious physical impact of CC and with potential far-reaching economic impacts on Small Island Developing States (SIDS) is the acidification of the world's oceans. The lowering of the pH is directly related to the concentration of carbon dioxide dissolved in the oceans. The concentration of carbon dioxide in the oceans is directly related to the concentration of carbon dioxide in the atmosphere. The acidity of the oceans restricts the storage of carbon by coral species and affects all calciferous marine species.

It has been predicted that if the carbon dioxide levels stabilize in the atmosphere at 450 ppm (which is the CO₂ target associated with the <2 °C temperature target being negotiated under the United Nations Framework Convention on Climate Change- UNFCCC), coral reefs will cease to grow. Atmospheric stabilization at 550 ppm or greater (which is possible under current global emission pathways) would result in dissolution of all existing corals [10]. The loss of the coral reefs would result in a loss of fisheries, irreversible damage to a very diverse ecosystem, loss of mechanical protection from waves, and loss of replenishment sand for beaches. Ultimately, most if not all sand beaches would be lost with the resulting socioeconomic impacts, which are discussed later in this chapter.

Hydrology and Storm Events

According to Pulwarty et al. if countries were ranked for the number of disaster events per unit area, small islands would occupy 19 out of the top 20 natural disaster prone regions in the world [8]. The following quote is from the Human Development Report 2011: “Of the 10 countries suffering the greatest number of natural disasters per capita from 1970 to 2010, 6 were SIDS” [11].

Countries like Barbados have argued that despite their very high Human Development Index, their vulnerability to disasters should always be taken into account when assessing the economic status of small islands and determining eligibility for concessionary developmental financing. SIDS have consistently argued that special loss and insurance mechanisms for SIDS need to be constructed specifically to address this vulnerability.

Small islands have little resilience to major disasters. Hurricane Ivan, in 2004, damaged 90 % of Grenada's built environment, destroyed 95 % of its nutmeg production, and set back that industry by at least a decade. Haiti, perhaps the most unfortunate of all small islands when it comes to disasters, will not recover for perhaps decades from the earthquake in January 2010, which killed over 300,000 people.

The AR4 could not determine a trend in the number of cyclones but did determine that the intensity of tropical cyclones had increased in the North Atlantic since 1970 [1]. Pulwarty et al. were unable to predict with any high degree of certainty how precipitation patterns will vary in the Caribbean as a result of CC [8]. There is some indication that the southern Caribbean and Central America region is drying, with predicted 20 % less precipitation by 2100 [8]. The general indications for Caribbean islands are for shorter wet seasons, with more intense rainfall events, longer dry seasons, and increased periods of drought.

It is likely that freshwater resources management will become a constraint to further economic development in some of the more water-stressed islands (e.g., Barbados, Antigua, and Barbuda). The use of desalination, wastewater reclamation, and rainwater harvesting is likely to become more prominent, with associated public health implications, which are discussed later in this chapter.

There are few studies on the potential impacts of the changes in hydrological patterns on agriculture and food security in SIDS. However, it is noted that the primary crops in the Caribbean—sugar, bananas, and cocoa—are not particularly drought tolerant.

Ecosystem Structural Changes

As discussed earlier, as a result of CC, there will be changes to precipitation patterns, and SLR will result in changes to coastal morphology and bathymetry. Both inland and coastal ecosystems are likely to be impacted structurally. It now appears inevitable, no matter what global mitigation pathways are chosen in the future, that most small islands will face at least 0.5–1.0 m SLR before the end of the twenty-first century.

Entire coastlines will become submerged. Mangroves, sea grass beds, and coral reefs will be affected. Built infrastructure will be destroyed (airports, seaports, oil terminals, roads, hotels, schools, electricity generating plants, sewerage systems) and will have to be relocated inland with the possible concomitant deterioration of green-field sites inland. Pressures on land use, including previously protected habitats, will become even more intense. It is noted that terrestrial watersheds in Caribbean countries have already lost, on average, 90 % of their primary vegetative cover [8].

The Socioeconomic Situation: Potential Socioeconomic Effects of Climate Change on Caribbean Small Island Developing States

CC is an existential issue for many low-lying SIDS and atolls (e.g., Kiribati, Tuvalu, the Maldives). However, most SIDS have already begun to suffer from the socioeconomic impacts of CC as a result of extreme weather effects and loss of agricultural yields due to prolonged droughts or frequent flooding. Some of the socioeconomic impacts may be indirectly caused by the responses of others to CC. For example, the

introduction of a “carbon tax” on airline travel into and out of Europe is likely to affect tourism revenues in the Caribbean. In the following section, the potential impacts of CC on three sectors of an economy (energy, water, and waste) are discussed.

Energy, Water, and Waste Management

Energy, water, and waste management/sanitation can be considered as three key crosscutting issues to be considered when any country is trying to achieve sustainable development. Energy is perhaps the most crucial of all three, as access to abundant, reliable, and affordable sources of energy solves the water management problem if it allows for desalination and pumping of the desalinated water to the areas of demand.

Global efforts to mitigate CC, coupled with an increase in the cost of fossil fuels (oil reached US\$148/barrel in June 2008), have led to considerable interest in the deployment of renewable energy and energy efficiency technologies worldwide. In 2011, even with the world still recovering from the economic recession, according to Bloomberg Energy Finance, there was a record high investment of US\$260 billion in renewable energy [12].

However, to a large extent the investment in renewable energy has not yet taken place in most small islands, even though these islands possess abundant sources of renewable energy (e.g., wind, solar, oceans). Perhaps two of the reasons for the lack of market penetration and continued reliance on imported fossil fuels are (1) the lack of interest of the private sector, including the carbon markets, due to the smallness of the individual islands and (2) the lack of public capital to invest in renewable energy technologies.

Island states have prioritized adaptation rather than mitigation, perhaps conscious that island states can make very little contribution numerically to the global CC mitigation effort. However, the sheer economics of sustained high fossil fuel prices and the potential for revenue streams from carbon markets such as the Clean Development Mechanism (CDM), even in a post Kyoto Protocol world, will likely drive small islands towards low carbon developmental pathways.

There are potential public health impacts, both adverse and beneficial, of renewable energy and energy efficiency interventions that may be implemented as a result of CC. Examples include, inter alia, disposal of the mercury contained with compact fluorescent light bulbs or disposal of batteries from electric vehicles, potential contamination of groundwater from geothermal operations, improvement in indoor air quality by switching to modern efficient cooking stoves, and of course mitigation of CC, perhaps the greatest public health threat faced by mankind. Public health departments and those agencies responsible for environmental health management and monitoring will need to be cognizant of these potential impacts.

Water resources management is likely to become more problematic for many SIDS. Without adequate storage capacity due to their small landmass, SIDS are inherently vulnerable to changes in precipitation patterns and to saltwater intrusion into

freshwater aquifers as a result of SLR. If, as it is predicted for the Caribbean, the rainy season will be shorter but individual rainfall events more intense and sea levels will rise by up to 1 m, considerable strain will be placed on the water utility companies to maintain adequate supplies of fresh clean water to households and businesses.

It is likely that the costs of water supply and distribution will significantly increase as utility companies and individual businesses turn to desalination, rainwater harvesting, and wastewater reclamation. The increased costs of water will impact upon agriculture and other sectors of the economy. Governments in severely water-stressed islands will have to prioritize the provision of minimum quantities of fresh clean water to their citizens to maintain public health standards. Significant investment will be required to augment above groundwater storage capacity and to reduce leakage in aging existing water distribution infrastructure. CC will place additional technical and financial strain on water utilities that are already challenged by freshwater resource constraints, particularly in Caribbean SIDS. The provision of adequate amounts of clean, potable water for cooking and sanitation is a prerequisite for good public health.

Waste management at both the household and country level in small islands will also be impacted by CC. Valuable land space will become even more precious. Landfilling as the primary or only means of waste disposal will not be sustainable. Coastal landfills (e.g., Perseverance in Grenada) will have to be sealed and relocated inland. The interrelationship between waste management and disaster management will need to be appreciated by public health planners. For example, hurricane and storm events create significant quantities of inorganic and organic waste that can swiftly become public health and environmental hazards post event if not adequately managed. Improper disposal of waste in watercourses and drains can greatly exacerbate the effects of flood events.

It is recommended that the public health implications associated with CC are identified and addressed in the integrated water resource management plans, the integrated solid waste management plans, and the disaster management plans of small island states.

Agriculture and Fisheries: Food Security

According to the Director of the Caribbean Council, the Caribbean food import bill in 2009 may have been as high as US\$5 billion [13]. Even without CC the Caribbean SIDS have faced food security issues. The Caribbean's agricultural model is geared towards export crops—sugar cane, bananas, cocoa, nutmeg, and spices. These crops are grown and exported to earn vital foreign exchange to then allow for the import of, among other things, the foodstuffs to feed resident populations and visiting tourists.

Barbados, one of the most developed and prosperous SIDS, with a very high Human Development Index, has already transitioned from an agrarian- to a service-based economy [11]. However, Barbados is highly dependent upon food imports and is vulnerable to world food prices and transport costs.

The yields from the major export crops of the region, sugar and bananas, are highly vulnerable to the changes in precipitation patterns that are a result of CC. One of the greatest economic impacts of Hurricane Ivan on Grenada in 2004 was the destruction of the nutmeg trees on the plantations and small farms. Recovery has taken years (a nutmeg tree matures in ~5 years), with nutmeg exports in 2011 (~350 tons) being still just a fraction (<15 %) of production pre-Ivan (2,500 tons/year) [14, 15].

There have been few studies on the potential impacts of CC on fisheries in the Caribbean region. A recent article by Nurse of the University of the West Indies concludes that the impacts of CC on Caribbean fisheries are likely to be generally negative [16]. Nurse cites increasing sea surface temperatures and persistent warm El Niño phases of the El Niño Southern Oscillation (ENSO) as having implications for increased coral bleaching in Caribbean waters. Loss of this habitat would affect all reef fisheries.

Ocean acidification will impact coral reefs and all calciferous marine species, including the “conch,” which has been part of the traditional diet of the Caribbean.

Several studies have been conducted on the impact of CC on coastal upwelling of zooplankton [17]. There is some evidence that the migration patterns of pelagic species may vary according to the CC-induced changes in the productivity of zooplankton [18]. There is evidence of fish moving closer to the poles as the oceans warm [19]. However, there is little specific data for the Caribbean. Nurse notes that the fisheries sector employs ~200,000 people, is responsible for 10 % of the protein intake and generates over US\$5 billion in annual revenues for the countries within the Caribbean Community (CARICOM) [16].

Tourism

Tourism is a major industry contributor to the national economy in many small islands. According to World Travel and Tourism Council data, the direct contribution of tourism to Gross domestic product (GDP) is USD 15.8 billion, and tourism generates around 700,000 jobs in the Caribbean region [20]. However, CC has a direct and significant impact on tourism.

Sea level rise, beach erosion, bleaching of coral reefs, change in rainfall, lost natural resources and biodiversity, and severe hurricanes decrease the touristic attractiveness of small islands. For example, tourism contributed 33 % to the Maldives' GDP. However SLRs will affect Maldives dramatically, and they will lose their land and tourism revenue [21]. In 2004, the Indian Ocean tsunami reduced tourist visits to southern Thailand, especially the provinces of Phuket, and showed how island communities are vulnerable to natural disasters [22].

According to the UNFCCC Report, “In Barbados, 70 % of the hotels are located within 250 m of the high water mark. This suggests that many hotels are almost exclusively within the 1 in 500 and 1 in 100 inundation zones, placing them at risk of major structural damage” [23]. CC affects the water resources, thus shortage of water or the emergence of vector-borne diseases may also cause a negative impact

on tourism in small islands. Related with CC warmer weather in the north might also decrease the number of tourists in the tropical regions. Uyarra et al. studied effects of CC on tourism in small islands, and 654 tourists from Bonaire and Barbados participated their study. Their results concluded “CC might have a significant impact on Caribbean tourism economy through alteration of environmental features important to destination selection” [24].

The negative impact of CC on the tourism industry may cause unemployment, financial crises, rising external debt, and rising incidence of poverty and political instability in small island states.

Infrastructure and Population Displacement

In the Pacific and Caribbean islands, large populations and infrastructures are located in coastal areas that are more vulnerable for CCs. Severe hurricanes easily destroy buildings, damage infrastructure, disrupt public services, and cause billions of dollars in damage. For instance, Puerto Rico, the Virgin Islands, Turks and Caicos, and Barbados are susceptible to floods often resulting from severe storms because of their unique topography [25].

Hurricane Ivan landed in Grenada in 2004 and is a perfect example of small island vulnerability [26]. Grenada’s socioeconomic infrastructure such as housing (90 % of damaged), utilities, touristic facilities (90 % of damaged), and agricultural production (90 % of nutmeg trees—main agricultural product) were destroyed in less than 8 h during this category four hurricane. According to the IPCC report, “Prior to Hurricane Ivan, Grenada was on course to experience an economic growth rate of approximately 5.7 % per annum but negative growth of around -1.4 % per annum is now forecast” [1]. In the future, CC may create more intense and frequent hurricanes; therefore island communities will have less time to recover.

This vulnerability and economic devastation from extreme events cause extensive migration from the small island states to metropolitan countries. According to Docquier and Marfouk, the Caribbean and Pacific regions are the most affected regions from skilled migration [27]. Currently, the Caribbean region has the highest emigration rates in the world; around 12 % of the labor force has migrated to other countries [28]. In 2002, there were approximately 750,000 refugees from the Americas and the Caribbean and 900,000 refugees from East Asia and the Pacific [29]. Effects of large migrations might lead to many serious health problems in the communities. Unfortunately the quality of life of immigrants does not also improve when they arrive in their new country, and they will be most vulnerable to health difficulties in a new place [29]. According to McMichael’s review, “displacements can cause varied health risks: undernutrition, exposures to infectious diseases, conflict situations, mental health problems, and altered health-related behaviors such as alcohol consumption, tobacco smoking, and transactional sex” [30]. At the same time, population displacement, especially the loss of skilled workers, impacts economic growth and social stability of the small island states negatively [31].

Potential Public Health Consequences Related with Climate Change in the Caribbean

The health impacts of CC are complex and comprehensive; the real health burden is rarely recognized. According to the estimation of the World Health Organization (WHO), 200,000 deaths happen each year in the world's low-income countries from a climate-related health problem such as crop failure and malnutrition, diarrheal disease, malaria, and flooding [30]. Many small islands are located in tropical zones, which have climates already suitable for heat stress; asthma; vector-borne, food-borne, and waterborne diseases; and morbidity/mortality from extreme weather events. Incidence and prevalence of chronic diseases are increasing in the Caribbean region with unclear reasons. As stated by the United States Agency for International Development (USAID) 2009 report: "The burden of disease associated with non-communicable chronic diseases (NCDs) is greater than the burden of disease associated with communicable diseases or injuries in Latin America and the Caribbean (LAC); however, much less attention has been given to NCDs. In LAC, approximately 50 % of all years of life lost are related to NCDs" [32].

Because of poor public health practices and inadequate infrastructure, these problems are already escalating in small island states [33]. Changing climate conditions will increase these health-related problems and burdens [34]. Before talking about an effective adaptation action, we need to understand the consequences of CC on health in the small island states.

As we discussed in the first part of this chapter, average annual temperatures in the Pacific Islands have increased by about 0.25 °C and in the Caribbean have increased by more than 0.5 °C approaching 1 °C over the last 100 years [35]. Continued temperature rise will be a risk to human societies and cause heat-related health problems among the small island communities. Mortality, morbidity, and hospital admissions show that death rates increase during extreme heat [30]. Patients with cardiopulmonary problems, outside workers, elderly, and the very young can be especially vulnerable to extreme heat. Remember that depending on culture and infrastructure (housing), some communities are more vulnerable than others [36]. Exposure to extreme heat can result in heat stroke, sunburn, heat exhaustion, heat cramps, heat rashes, and dehydration [37].

Rising temperatures, changing rainfall patterns and precipitation increase the rate of vector-borne and waterborne diseases. Costello et al. stated in their paper "Schistosomiasis, fascioliasis, alveolar echinococcosis, leishmaniasis, Lyme borreliosis, tick-borne encephalitis, and hantavirus infections are all projected to increase as a result of global CC" [38]. Malaria, dengue fever, filariasis, and schistosomiasis already exist in tropical small island states; however, they are increasing because of changing climate conditions in addition to poor public health practices, inadequate infrastructure, and poor waste management practices [34]. Vector reproduction, parasite maturation, and bite frequency mostly rise with temperature; as a result, malaria, tick-borne encephalitis, and dengue fever will become prevalent. Dengue fever is especially sensitive to climate conditions. Rawlins et al. reported that the incidence

of dengue fever rises during the warm years of the ENSO in the Caribbean [39]. An outbreak of dengue fever in Fiji simultaneously occurred with increased temperatures during the El Niño, and the cost of the outbreak was US\$3–6 million [34].

Vector-borne diseases may not be the only infectious disease caused by CC impact. CC and warm weather causes increases in pathogen microorganism development and survival rates, disease transmission, and host susceptibility. For example, when ocean temperatures rise, cholera risk might be increased because of higher plankton activity (algal blooms) that supplies nutrients for *Vibrio cholerae* [40]. Increased rainfalls and flooding may cause leptospirosis or cryptosporidiosis outbreaks [38]. In the WHO Synthesis Workshop on Climate Variability, CC and Health in Small-Island States report, it was stated that “the Epidemiology Centre and the Water and Sewage Authority of Trinidad and Tobago found that 18.6 % of samples of potable water taken after heavy rainfall events were positive for *Cryptosporidium*” [33].

In the small island states, freshwater resources are predicted to reduce in relation to increased demand, decreased rainfall, and saltwater invasion due to hurricanes and SLR [4]. Singh et al. also showed that “the incidence of diarrhoeal diseases is associated with annual average temperature and negatively associated with water availability in the Pacific” [35]. Thus, rising temperatures and decreasing water resources related with CC may increase outbreak of diarrheal and other infectious diseases and negatively impact quality of life and the economy of small island states. However, rising temperatures, decreased water resources, and hurricanes also cause a loss of agricultural productivity and seriously affect food security among island communities. SLR, rising temperatures, and acidification of the oceans will lead to a loss of mangroves and coral reefs, and reduced fish stocks and warm ocean temperatures cause fish populations to move to higher latitudes which will also affect food security of the islands [26]. This food insecurity will affect livelihoods in coastal populations and result in malnutrition. For example, during extreme drought, micronutrient deficiencies were found in pregnant women in Fiji. Therefore CC can exacerbate undernutrition and starvation.

CC also contributes to air quality problems; higher temperatures and/or humidity impacts the frequency of smog events, seasonality of pollens, spores, and formation of various air pollutants [30]. Sunlight and high temperatures combine with nitrogen oxides and volatile organic compounds to increase ground-level ozone, which can damage respiratory systems. This effect may cause an increase in respiratory disorders, especially asthma and other chronic lung diseases [1]. CC may affect the concentration of particulate matter (PM) pollution in the air by affecting natural or “biogenic” sources of PM such as wildfires and dust from dry soils [41]. Forest fires in Indonesia occur annually and increase significantly related with a strong El Niño. The Indonesian island of Sumatra faced massive forest fires caused by El Niño-driven droughts and caused an increase in respiratory illnesses and allergy symptoms among islanders [34].

Published data from the Caribbean region stated that chronic respiratory diseases are a significant public health problem in the Caribbean; [42–44] however, causality is largely unknown. One of the studies showed that climatic variables are associated with seasonal acute asthma admissions in emergency rooms in Trinidad [45]. Monteil et al. also reported Sahara dust as a risk factor for asthma in the Caribbean

[46, 47]. In our recent study, we also found hospital visits due to asthma attack were correlated with Sahara dust exposure and the monthly mean rainfall level ($p < 0.05$) [48]. Considering that the region is prone to tropical rain, high humidity, hurricanes, and flooding, mold should be considered as an important respiratory risk factor in the Caribbean. In one of our other community-based studies, we found that the flooding caused asthma-like symptoms among the occupants of water damp buildings in Guyana after a 2008 flood [49]. This study found objective evidence of dampness and mold in 32.8 % of the households.

Extreme events including hurricane, storm, flooding, and drought have also short-term effects on human health such as drowning, injuries, and mental disorders [1]. In 2001–2002 there were more than 50 deaths related to storms and hurricanes in the Caribbean [33].

Conclusion and Recommendations

CC is already physically affecting small island states in the Caribbean and worldwide. Due to the longevity of carbon in the atmosphere, further physical effects are inevitable such as significant SLR no matter what global mitigation efforts are taken in the near future. Changes in rainfall patterns would also appear to be inevitable. A coordinated adaptive response will be required involving, inter alia, economists, engineers, physical planners, and public health professionals.

To manage the health effects of CC, we need to understand the consequences of CC on health and the solution for adaptation. Therefore building awareness and expanding knowledge through regional-based research will be an important step for developing adaptation and prevention strategies. Establishing effective monitoring, early warning, and data management systems is critical for management of the health effects of CC.

References

1. Anon. IPCC 2007. Climate change 2007: the physical science basis. Summary for policy makers. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change (AR4). 2007.
2. UNEP. Bridging the emissions gap. United Nations Environment Programme (UNEP). 2011. www.unep.org/pdf/UNEP_bridging_gap.pdf
3. Anon. International Energy Agency. Prospect of limiting the global increase in temperature to 2°C is getting bleaker. International Energy Agency. 2011. http://www.iea.org/index_info.asp?id=1959. Accessed 27 Jan 2012.
4. Mimura N, Nurse L, McLean R, et al. Small Islands. In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE, editors. Climate change 2007: impacts, adaptation and vulnerability. Contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press; 2007. p. 687–716.
5. Trenberth K, Jones P, Ambenje P, et al. Observations: surface and atmospheric climate change. In: Solomon S, Qin D, Manning M, Chen Z, Marquis M, Averyt KB, Tignor M, Miller HL,

- editors. *Climate change 2007: the physical science basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*. Cambridge: Cambridge University Press; 2007. p. 235–336.
6. Lal M. Climate change and small Island developing countries of the South Pacific. *Fijian Studies*. 2004;2:15–31.
 7. Miller KM. Variations in sea level on the West Trinidad coast. *Mar Geod*. 2005;28(3):219–29.
 8. Pulwarty RS, Nurse LA, Trotz UO. Caribbean Islands in a changing climate. *Environment*. 2010;52(6):16–27.
 9. Anon. Snow, Water, Ice and Permafrost in the Arctic (SWIPA) 2011—executive summary. 2011. <http://amap.no/swipa/>. Accessed 27 Jan 2012.
 10. AOSIS Technical Negotiating Team. AOSIS frequently asked questions—technical document 12; 2009.
 11. UNDP. Human development report, 2011. Sustainability and equity: a better future for all. 2011:36. <http://hdr.undp.org/en/reports/global/hdr2011/download/>
 12. Anon. Renewable energy investments worldwide set record high in 2011. Power engineering. 2012. <http://www.power-eng.com/articles/2012/01/renewable-energy-investments-worldwide-set-record-high-in-2011.html>. Accessed 27 Jan 2012.
 13. Anon. Agriculture and food security. BBC Caribbean.com. 2009. http://www.bbc.co.uk/caribbean/news/story/2009/05/090505_jessop_caribbean_agriculture.shtml. Accessed 27 Jan 2012.
 14. Anon. Grenada nutmeg farmers receive Christmas bonus—Caribbean360. <http://www.caribbean360.com/index.php/business/526067.html#axzz1kZpRZFLL>. Accessed 27 Jan 2012.
 15. Anon. Grenada celebrates rare, massive nutmeg production—Yahoo! 2011. <http://sg.news.yahoo.com/grenada-celebrates-rare-massive-nutmeg-production-201916386.html>. Accessed 27 Jan 2012.
 16. Nurse LA. The implications of global climate change for fisheries management in the Caribbean. *Climate and Development*. 2011;3(3):228–41.
 17. Wiafe G, Yaqub HB, Mensah MA, Frid CLJ. Impact of climate change on long-term zooplankton biomass in the upwelling region of the Gulf of Guinea. *ICES J Mar Sci*. 2008;65(3):318–24.
 18. Rijnsdorp AD, Peck MA, Engelhard GH, Möllmann C, Pinnegar JK. Resolving the effect of climate change on fish populations. *ICES J Mar Sci*. 2009;66(7):1570–83.
 19. Rose G. On distributional responses of North Atlantic fish to climate change. *ICES J Mar Sci*. 2005;62(7):1360–74.
 20. World Travel&Tourism Council. Caribbean-key facts at a glance. World Travel&Tourism Council. 2011. <http://www.wttc.org/research/economic-impact-research/regional-reports/caribbean/>. Accessed 27 Jan 2012.
 21. Bigano A, Hamilton JM, Tol RSJ. The impact of climate change on domestic and international tourism: a simulation study. Research unit sustainability and global change working paper FNU-58. Hamburg: Hamburg University and Centre for Marine and Atmospheric Science; 2005.
 22. Athukorala P, Resosudarmo BP. The Indian Ocean tsunami: economic impact, disaster management, and lessons. *Asian Economic Papers*. 2005;4(1):1–39.
 23. UNEP (United Nation Environment Programme). *Climate change in the Caribbean and the challenge of adaptation*. Panama City: UNEP Regional Office for Latin America and the Caribbean; 2008.
 24. Uyarra MC, Cote IM, Gill JA, et al. Island-specific preferences of tourists for environmental features: implications of climate change for tourism-dependent states. *Environ Conserv*. 2005;32(1):11–9.
 25. Anon. Pan American Health Organization—area on emergency preparedness and disaster relief. <http://www.amro.who.int/english/dd/ped/PED-about.htm>. Accessed 27 Jan 2012.
 26. Nurse L, Moore R. Adaptation to global climate change: an urgent requirement for small Island developing states. *Review of European Community & International Environmental Law*. 2005;14(2):100–7.
 27. Docquier F, Marfouk A. International migration by educational attainment (1990–2000)—Release 1.1. March; 2005.

28. Mishra P. Emigration and brain drain: Evidence from the Caribbean. Western Hemisphere Department. International Monetary Fund. 2006;1–37. <http://www.imf.org/external/pubs/ft/wp/2006/wp0625.pdf>
29. Thomas SL, Thomas SDM. Displacement and health. *Br Med Bull.* 2004;69(1):115.
30. McMichael AJ, Haines A, Slooff R, Kovats S. Climate change and human health. Geneva: WHO; 1996. p. 51–2.
31. Thomas-Hope E. Skilled labour migration from developing countries: study on the Caribbean region. Geneva: International Migration Programme, International Labour Office; 2002.
32. Anderson GF, Waters H, Pittman P, Herbert R. Non-communicable diseases in Latin America and the Caribbean. In: Latin America and the Caribbean. Bloomberg School of Public Health, Johns Hopkins University, Baltimore, MD. 2009. <http://www.healthycaribbean.org/publications/documents/NCD-in-LAC-USAID.pdf>
33. WHO. Synthesis workshop on climate variability, climate change and health in small-island states. Geneva: World Health Organization; 2004.
34. Ebi KL, Lewis ND, Corvalan C. Climate variability and change and their potential health effects in small island states: information for adaptation planning in the health sector. *Environ Health Perspect.* 2006;114(12):1957.
35. Singh B. Climate changes in the greater and southern Caribbean. *Int J Climatol.* 1997;17(10):1093–114.
36. Kovats RS, Hajat S. Heat stress and public health: a critical review. *Annu Rev Public Health.* 2008;29:41–55.
37. Anon. CDC—heat stress—NIOSH workplace safety and health topic. <http://www.cdc.gov/niosh/topics/heatstress/>. Accessed 19 Jan 2012.
38. Costello A, Abbas M, Allen A, et al. Managing the health effects of climate change. *Lancet.* 2009;373(9676):1693–733.
39. Rawlins S, Chen A, Ivey M, Amarkoon D, Polson K. The impact of climate change/variability events on the occurrence of dengue fever in parts of the Caribbean: a retrospective study for the period 1980–2002. *West Indian Med J.* 2004;53:54.
40. Patz JA, Campbell-Lendrum D, Holloway T, Foley JA. Impact of regional climate change on human health. *Nature.* 2005;438(7066):310–7.
41. EPA. Climate change—health and environmental effects. U.S. EPA. <http://epa.gov/climate-change/effects/health.html>. Accessed 27 Jan 2012.
42. Bayona M, Montealegre F, Gomes de Andrade VL, Treviño F. Prognostic factors of severe asthma in Puerto Rico. *PR Health Sci J.* 2002;21(3):213–9.
43. Gyan K, Henry W, Lacaillie S, et al. African dust clouds are associated with increased paediatric asthma accident and emergency admissions on the Caribbean Island of Trinidad. *Int J Biometeorol.* 2005;49(6):371–6.
44. Homa DM, Mannino DM, Lara M. Asthma mortality in U.S. Hispanics of Mexican, Puerto Rican, and Cuban heritage 1990–1995. *Am J Respir Crit Care Med.* 2000;161(2 Pt 1):504–9.
45. Ivey MA, Simeon DT, Monteil MA. Climatic variables are associated with seasonal acute asthma admissions to accident and emergency room facilities in Trinidad, West Indies. *Clin Exp Allergy.* 2003;33(11):1526–30.
46. Monteil MA. Saharan dust clouds and human health in the English-speaking Caribbean: what we know and don't know. *Environ Geochem Health.* 2008;30(4):339–43.
47. Monteil MA, Antoine R. African dust and asthma in the Caribbean: medical and statistical perspectives. *Int J Biometeorol.* 2009;53(5):379–81; author reply 383–5.
48. Martin F, Elci OC, Hage R, Akpinar-Elci M. Climate change, Sahara Dust and the emergency room visits due to asthma in Grenada, the Caribbean. *Am J Respir Crit Care Med.* 2011;183(1 Meeting Abstracts):A3752.
49. Rose S, Elci OC, Hegamin-Younger C, Akpinar-Elci M. Evaluation of respiratory and general symptoms among occupants of households affected by flooding: the case of Cove & John, Guyana; 2010.

Chapter 17

Global Climate Change, Desertification, and Its Consequences in Turkey and the Middle East

Hasan Bayram and Ayşe Bilge Öztürk

Abstract Climate change and desertification is a global problem, and Turkey and the Middle East region are among the mostly affected areas of the world. By the end of this century, Turkey and the Middle East region are expected to have an increased mean temperature about 3–5 °C and a 20–40 % decline in precipitation. The Intergovernmental Panel on Climate Change (IPCC) warns that desertification is likely to become irreversible, if the environment becomes drier and the soil becomes further degraded through erosion and compaction. According to United Nations Environment Program (UNEP), most of areas in Turkey are under desertification and/or high potential for desertification and only small parts of the areas in Turkey are non-risky places. Climate models predict a hotter, drier and less predictable climate for the Middle East region, and degradation and desertification are expected to accelerate due to global warming. Climate change and desertification is acting as a risk for water loss, decline in agriculture, and loss of biodiversity. Climate change has a negative impact on human health by indirect effects including air, water, and food supplies and by direct effects on especially elderly, children, and chronically ill population. This chapter examines the potential impacts of climate change and desertification on the environmental parameters and human health in Turkey and the Middle East.

Keywords Climate change in the desert • Desertification • Sandstorms • Water • Precipitation • Biodiversity • Human health • Turkey • Middle East

H. Bayram, M.D., Ph.D. (✉)

Department of Chest Diseases, School of Medicine, University of Gaziantep,
Gaziantep 27310, Turkey
e-mail: bayram@gantep.edu.tr

A.B. Öztürk, M.D.

Adult Allergy Unit, Göztepe Education and Research Hospital, Medeniyet University
Goztepe Training and Research, Kadıköy, Istanbul, Turkey

Climate has been changed due to increases in the average global surface temperature of the earth from preindustrial period to present times. All areas of the world are expected to be affected by consequences of climate change, however; the Middle East countries including Turkey seem to feel these effects more severe because of the long hot seasons they live and their limited natural reserves of water. Turkey is located in the Mediterranean macroclimatic zone that lies between the temperate and the subtropical zones at western parts, allowing the country to have widely diverse regional and/or seasonal variations ranging from extremely cold winters to very hot dry summers. Due to climate change impacts, widespread increases in summer temperatures are expected to be recorded in the future. Summer temperatures have been increasing mostly in the western and southwestern parts of Turkey. Also, winter precipitation in the western parts of the Turkey has been decreased significantly in the last 5 decades [1]. According to United Nations Environment Program (UNEP), most of areas in Turkey are under desertification and/or high potential for desertification, and only small parts of the areas in Turkey are non-risky places [2].

Climate models are also predicting a hotter, drier and less predictable climate for Middle East region. The region is expected to get hotter across all seasons; models predict an increase of 2.5–3.7 °C in summer and 2.0–3.1 °C in winter [3]. By the end of this century, this region is expected to have an increased mean temperature about 3–5 °C and a 20 % decline in precipitation. Most of the region is expected to remain as very hot deserts under climate change scenarios. According to United Nations Development Program (UNDP) Human Development Report 2007/2008, the Middle East is considered as one of the most water-stressed regions of the world [4]. The Middle East countries including Iraq, Iran, Israel, Jordan, Lebanon, Syria, and Saudi Arabia are also under the threat of desertification [5]. Increased temperature is expected to cause greater seasonal variability, more severe weather events, and significant sea level rises. Furthermore, Mediterranean region is expected to shift 300–500 km northward if a 1.50 °C warming will occur, which would mean that Mediterranean ecosystem would become desert [6].

In this chapter, we review the published papers and the governmental and non-governmental reports on global climate changes including changes in temperature, green house gas emissions, desertification and their consequences on sandstorms, water use, and loss of biodiversity in the Middle East countries including Turkey. The impact of such changes on human health will also be reviewed in the view of limited number of published studies and reports referring this region.

Greenhouse Gas Emissions

Turkey's energy need and demand are increasing over the years. The country's demand for general energy and electricity has increased by an annual rate of 3.7 % and 7.2 % for the period of 1990–2004, respectively [1]. In 2004, the ratios for coal, biomass, oil and natural gas, hydro-geothermal and wind electricity, and other

renewable sources in the total energy production were as 43 %, 23 %, 12 %, 17 %, and 5 %, respectively [1].

Turkey lies in a sunny belt between 36 and 42 N latitudes. The yearly average solar radiation is 3.6 kWh/m²/day and average sunshine duration is 2,640 h, corresponding to 30 % of the year. Although the use of solar technologies is limited, solar water heaters are commonly used. Turkey is one of the leading countries in the world with a total installed capacity of 8.2 million m² collector area as of 2001. In Turkey, the solar energy has a technical potential of 8.8 million tons of oil equivalent (Mtoe) electricity generation and 26.4 Mtoe heating capacity [7]. However, 66 % of Turkey's energy consumption is based on fossil fuels [1]. Turkey's carbon dioxide (CO₂) emission has increased by 98 % between 1998 and 2009. Although the country's CO₂ emission was 20.59 million tons in the year 1990, it reached to 30.90 million tons in 2004. According to the estimates in 2000, 34 % of CO₂ emission was produced by electricity generation, 32 % by industry, 17 % by transportation, and 16 % by other sectors. However, by the year 2020, it is estimated that 41 % of CO₂ emission will be produced by generation of electricity, 33 % by industry, 13 % by transportation, and 13 % by other sectors [7]. When Turkey is compared with other countries with respect to basic CO₂ indicators, Turkey is ranked 23rd in total CO₂ emissions, 75th in CO₂ emissions per capita, 60th in the ratio of CO₂ emissions to the gross domestic product (GDP), and 55th in the ratio of CO₂ emissions to the GDP, measured on the basis of purchasing power parity [5].

It is thought that solar energy has the potential to equip the Middle East with centuries of sustainable, clean electricity [8]. It has been reported that the Middle East receives 3,000–3,500 h of sunshine per year, with more than 5.0 kW/m² of solar energy per day, and that average solar radiation is about 19.23 M joules per square meter in Iran. In Israel, over 700,000 households are reported to have solar water heaters [8].

As a region, the Middle East produces a tiny fraction of global emissions (less than 1 % of the world total), but on per capita basis, Israel's emissions (11.8 metric tons per capita) exceed the European average (10.05 tons) [3]. The amounts of CO₂ emissions of Jordan, Syria, and Iraq are 4.9, 3.3, and 4.1 metric tons per capita, respectively [3]. However, the 88 % growth of CO₂ emissions in the Middle East was the third largest in the world in 1990–2004 and more than 3 times faster than the world average; most of that growth came from fuel combustion [6].

Climate Change

There have been widespread increases in summer temperatures in Turkey [1] (Fig. 17.1). These increases are mostly recorded in the western and southwestern parts of Turkey [1]. A recent study using the regional climate model, Providing Regional Climates for Impacts Studies (PRECIS), suggests that the average temperature in 2071–2100 will be 4–5 °C higher for coastal regions and 5–6 °C higher for inland Turkey comparing to the average for 1961–1990, respectively [9].

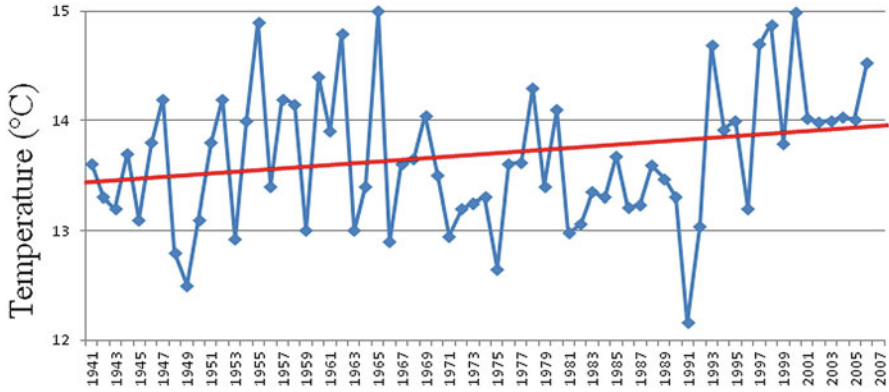


Fig. 17.1 Mean annual temperature trend in Turkey (°C) (1941–2007). Trend $Y=0.0064x+13.474$; $R^2=0.0422$ (from Demir İ, Kılıç G, Coşkun M. PRECIS bölgesel İklim Modeli ile Türkiye için İklim Öngörülürleri: HadAMP3 SRES senaryosu, IV. Atmosfer Bilimleri Sempozyumu, 2008, Bildiriler Kitabı, 365–373 (in Turkish). Available at: <http://www.mgm.gov.tr/FILES/iklim/iklimongoruleri.pdf>, with permission)

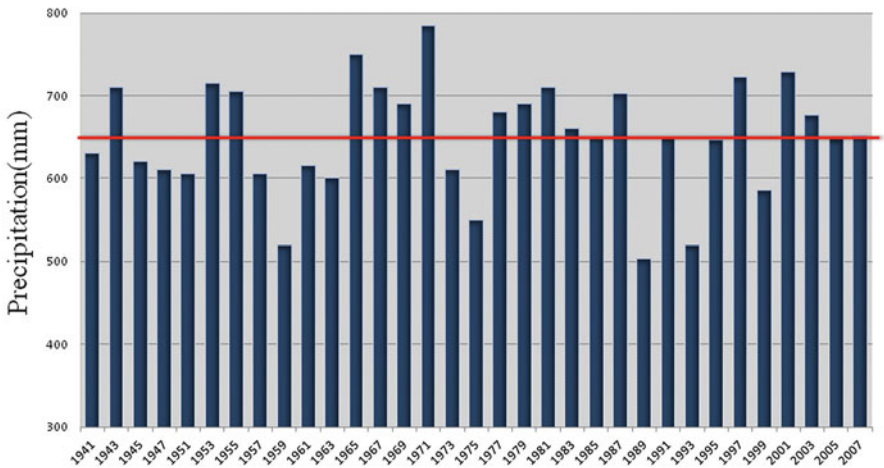


Fig. 17.2 Annual mean precipitation and its trend in Turkey (1941–2007). Trend $Y=-0.2917x+656.92$; $R^2=0.0079$ (from Demir İ, Kılıç G, Coşkun M. PRECIS bölgesel İklim Modeli ile Türkiye için İklim Öngörülürleri: HadAMP3 SRES senaryosu, IV. Atmosfer Bilimleri Sempozyumu, 2008, Bildiriler Kitabı, 365–373 (in Turkish). Available at: <http://www.mgm.gov.tr/FILES/iklim/iklimongoruleri.pdf>)

Furthermore, winter precipitation in the western provinces of Turkey has decreased significantly in the last 5 decades [1]. Although precipitation has decreased along the Aegean and Mediterranean coasts, it has increased along the Black Sea coast of Turkey (Fig. 17.2). The rainfall is also expected to be 40 % less in the West and 5 % less in the East and the eastern Black Sea Regions, respectively [9]. On the other

hand, high mountains in Turkey started to lose their glaciers, large lakes have become smaller, and shallow lakes have vanished [10]. According to projections, nearly 20 % of the surface water will be lost by the year 2030. By the year 2050 and 2100, the percentage of water loss is expected to increase up to 35 % and more than 50 %, respectively [11].

The Intergovernmental Panel on Climate Change (IPPC) estimates an increase in temperature in the Middle East up to 2 °C in the next 15–20 years and over 4 °C for the end of the century [6]. For example, the main climate change scenarios projected for Israel by the year 2100 include a mean temperature increase of 1.6–1.8 °C, a reduction in precipitation by –8 to –4 %, an increase in evapotranspiration by 10 %, and a sea level rise of 12–88 cm [12]. According to reports from Iran, temperature has risen between 2.5 and 5 °C on average with the increase in minimum temperature being more widespread [13]. It has been reported that southwestern parts of the Caspian Sea, northwest and west of Iran, have experienced the highest rate of reduction in the amount of their annual precipitation [13]. On the basis of climate change scenarios from Saudi Arabia, the average warming in the country for the year 2041 will be higher than the global average, and the highest warming (2.2–2.7 °C) is expected to occur during summer in the northwestern regions. The precipitation is also expected to decrease in the entire Kingdom from December to June [14]. According to similar climate change scenarios, the average warming in Syria for the year 2041 will be higher than the global average. The greatest increase (2.0–2.1 °C) will be expected to occur in the northwest and the southeast region of the country [15]. The IPCC projections indicate that the anticipated increase in surface temperature and reduction in rainfall will result extreme desiccation in Middle East region [6]. It is also expected that these changes will result in a global increase in sea levels, which are expected to rise between 0.1–0.3 m by 2050 [3].

In the Middle East, total available water resources are 262.9 billion cubic meter (Bcm) [6]. The water deficit is likely to increase from 28.3 Bcm in the year 2000 to 75.4 Bcm in 2030. According to projections, a temperature increase of 5 °C will reduce the snow cover from 170,000 to 33,000 km² in the upland section of Euphrates and Tigris watersheds. This is expected to reduce the discharge of the Euphrates and Tigris rivers. An increase in temperature of Jordan by 2–4 °C is expected to reduce the flow of Azraq River by 12–40 % [6].

Desertification

Climatic factors that may lead to desertification in Turkey were investigated by analysis of the spatial and temporal variations of the precipitation and aridity index series, for the period of 1930–1993. Severe and widespread dry conditions have occurred, particularly in 1973, 1977, 1984, 1989, and 1990. Southeastern Anatolia and the continental interiors of Turkey have been affected by desertification processes as a result of deterioration in the climatic factors. Significant trends from normal to drier conditions in annual precipitation and winter precipitation and

towards dry subhumid or semiarid climatic conditions have been climatic factors that lead to desertification in the Mediterranean and Aegean regions of Turkey [16]. Climatic changes impacts were also investigated in the Büyük Menderes and Gediz River basins, and rivers' runoff trend was analyzed between the year 1960 and 2000. It was found that the water potency of these rivers was decreased dramatically [17]. Moreover, the salt reserve and water in Salt Lake has decreased between 1987 and 2005 as a result of a 1 °C increase in temperature between 1993 and 2005 as compared with 1970–1992 [10]. However, The Mesopotamia Basin in Turkey is expected to suffer more drastically from desertification, since this area receives only 150–300 mm of rainfall annually but experiences 1,500–2,500 mm of evaporation per year [18].

In addition to changes in climate, the factors that lead to loss of land (i.e., erosion), deforestation, and soil pollution contribute to desertification in Turkey. It is estimated that 54 % of the forest land and a 59 % of prime agricultural land are thought to be prone to erosion [19]. The total forest area in Turkey is about 21.2 million ha (27.2 % of total land); however, 49 % of this is estimated to be degraded and unproductive [20]. On the other hand, Turkey is losing 11,500 ha of her forests every year with an average of 1,900 fires annually [21].

Desertification is an important threat for the whole Middle East region [5, 6, 12–14]. In Iraq, areas subject to desertification are estimated to exceed to 92 % of the total surface area. Since 1981, the percentage has increased, and this was partly due to military operations, which had detrimental effects on the environment including plants and the soil [5]. Syria has 25.79 km³ renewable freshwater potential per year, and the available freshwater amount per capita is estimated to decrease from an amount of 2.089 m³ in 1990 to 546 m³ in 2050. In 1955, freshwater availability as per cubic meter/inhabitant in Lebanon and Syria was 3.084 and 6,501 m³ per capita, respectively. These values were decreased to 1949 and 2.089 m³ in 1990 for Lebanon and Syria, respectively. The estimated values for years 2025 and 2050 for Syria are thought to be 1,126 and 960 m³, whereas the corresponding figures for Lebanon are expected to be 770 and 546 m³ for years 2025 and 2050, respectively [5]. The percentage of desertified land ranges from 10 % in Syria to nearly 100 % in the United Arab Emirates. It is estimated that the cost of soil degradation in Syria is equivalent to about 12 % of the value of the country's agricultural output. In Lebanon degradation is reported to be serious on steppe mountainous land [5].

In Iran, the level of annual precipitation has decreased in the southwestern parts of the Caspian Sea, northwest and west of the country. The amount of degradation was reported to be 1.5 million ha in the country. If the rate of desertification continues in the present trend, the amount of affected land for the year 2050 is expected to be 75 million ha in Iran [13]. Desertification is also expected to be exacerbated by climate change in Israel, particularly in the Judean Desert highlands and the northern Negev [12]. Saudi Arabia is particularly vulnerable to desertification, as about 76 % of the country's territory is nonarable lands, of which 38 % is made up by deserts. The yearly temperature increase is expected to be 0.8–6.0 °C in the year 2100, and as a result the rate of desertification is expected to rise in this country [14].

Consequences of Climate Change and Desertification

Sandstorms and Dust Storms

Arid lands are considered as the significant contributors of dust. The phenomenon of sand dunes is thought to be one of the most dangerous consequences of desertification, due to its negative impact on every vital aspect of life. Sand dunes lead to increased sandstorms and dust storms, increased soil salinity and water logging, and widespread rangeland degradation [5]. Sandstorms and dust storms pollute the environment and agricultural production by disrupting the physiological functions of plants, especially during pollination and inflorescence. Sandstorms blow from the dune fields in central and southern areas of the Middle East region. It has been reported that their incidence has increased during recent years, and although dust storms are reported to be most common in the central plain region in Iraq and Syria [5], they have started to affect all Middle East countries. Studies suggest that Middle East countries such as Iraq face a severe desertification problem that jeopardizes their food security through the effects of soil salinity, water logging, loss of vegetative cover, shifting sand dunes, and severe sandstorms/dust storms [5].

It has been suggested that the introduction and expansion of rain-fed agriculture in the Syrian steppe led to environmental consequences including formation of dust, dust storms, sand accumulation on roads and railroads, and formation of sand sheets, sand hummocks, and sand dunes [5]. Furthermore, dust frequency and intensity are reported to have remarkably increased during the last few years in the eastern part of the country. The frequency and amount of sandstorms and dust storms in Turkey and Lebanon are reported to be less than in Iraq and Syria [5]. However, in recent years, Turkey, in particular the southeast parts of the country, has faced to more sandstorms coming from over Syrian and Saharan deserts.

Water Use

According to estimations of population growth rate of Turkey, per capita available, water was 250 L/day in the year 2000. With the assumption that Turkey will continue to grow and develop, this amount is expected to increase to 500 L/day in 2030 [22]. The total water requirement for domestic and industrial consumption is predicted to be 25.3 and 13.2 billion m³, respectively. Per capita of potential water resources was estimated as 3,070 m³/year in 1990, however; according to climate change scenarios, the per capita of water potential will be decreased to 700–1,910 m³/year in 2050. Gross irrigatable area in Turkey is 8.5 million ha, and the whole of this area will be irrigated by the year 2030. Water requirement for this area is estimated to be 71.5 billion m³; however, in total consumption, the percentage of irrigation is expected to drop from 75 to 65 % due to the water shortage [22] (Table 17.1).

Table 17.1 Gross total amount and consumable water in Turkey

Surface water	Rainfall (mm)	Water amount (billion m ³ /year)	Gross water potential (billion m ³ /year)	Exploitable (billion m ³ /year)
Turkey	643	501	186	95
From bordering countries			7	3
Groundwater			41	12
Total			234	110

From Sekercioglu CH, Anderson S, Akçay E, et al. Turkey's globally important biodiversity in crisis. *Biol. Conserv.* 2011; 144:2752–2769, with permission

In global-scale assessments, basins are defined as being water-stressed if they have either per capita water availability below 1,000 m³ per year. Middle East is one of the regions where water-stressed basins are located. The Arab region receives an estimated 2282 billion m³ of rainwater each year compared to estimated 205 billion m³/year of surface water and 35 billion m³/year of groundwater [6]. Lebanon, Syria, and southern Sudan receive as much as 1,500 mm of rainfall. Reduced stream flow and groundwater recharge are expected to decrease water supply 10 % by 2050 [6]. Recent estimates of water resources in Middle East region indicate that total available natural water resources are 262.8 Bcm, of this; 226.5 Bcm is made up by surface water and 36.3 Bcm by groundwater including 11.874 Bcm of nonrenewable groundwater. Per capita renewable water resources in the region have decreased from 4,000 m³ per year (year 1950) to 1,100 m³ per year in recent years. The water deficit is expected to increase from about 28.3 Bcm for the year 2000 to 75.4 Bcm in the year 2030 due to climatic and non-climatic factors [6]. Lebanon is one of the richest countries with water in the Middle East region. The total amount of available water is 3.992 million cubic meters in Lebanon. According to studies conducted by the Food and Agriculture Organization of the Nations and by the UNDP, the irrigated area of Lebanon is expected to rise to 170.000 ha by 2015 [5]. Syria has 25.79 km³ renewable freshwater potential per year, and the available freshwater amount per capita is predicted to decrease from 2.089 m³ (in the year 1990) to 546 m³ in the year 2050 [5]. According to the UNDP Human Development Report 2007/2008, the Middle East is among the most water-stressed regions of the world [15].

Loss of Biodiversity

The Earth is made up of an ecosystem and ecological features, which are supported by biodiversity. Higher temperatures may result in a reduction in soil fertility due to higher rates of decomposition and losses of organic matter and may adversely affect nutrient cycling. As a result, climate change is expected to cause the loss of biodiversity and undermine ecological system. Turkey is considered as one of the richest countries of Europe and the Middle East with respect to biodiversity. The country contains 5 % of the plant species found in the continent of Europe. Studies have reported that there are 163 plant families covering 1,225 types, which in turn cover

about 9,000 species [18]. Turkey is also reported to be rich as biodiversity with 120 mammals, 400 fishes, 469 bird species, and 130 reptiles. Turkey has 33 % of endemic species of totally 9,000 plant species. By factors result from climate changes, of 3,504 endemic plants in Turkey, 12 are reported to be extinct, and 3,492 are considered to be under threat [23].

Iranian habitat supports 8,200 plant species, of which 2,500 are endemic, over 500 species of birds, 160 species of mammals, and 164 species of reptiles [13]. Although no systematic review has been conducted to show linkage between climate change and biodiversity in Iran, national documents in biodiversity have addressed that climate change has a negative impact on biodiversity [13]. The National Syria Strategy for Biodiversity indicates that the country has more than 3,000 animal species and 3,077 species of flowering plants. Syria is considered as a poor country with respect to its forests, which cover only 3 % of the total land area. There has also been a decrease in the wooded areas of Jebel Abdel Aziz, Abou Rajmein, and Balaas mountains, which were in the past ecosystem rich in ecological biodiversity [15]. It has also been suggested that desertification, further exacerbated by climate change, will widen the desert barrier to be crossed by the birds and will make Israel less hospitable for migration of the migrants. Many Red Sea species have colonized the Mediterranean Sea following migration through the Suez Canal. With increased warming, more Red Sea immigrants are expected to colonize, reproduce, and persist in the eastern Mediterranean [12]. In conclusion, the biodiversity is expected to further deteriorate due to climate change in the Middle East region [3].

Human Health

Human health is adversely influenced by the direct and indirect effects of climate change, and preliminary research has shown climate change has potentially direct and indirect adverse impacts [24, 25]. Changes in pollen releases impact asthma and allergic rhinitis; heat waves may cause critical care-related diseases; climate-driven air pollution increases may lead to exacerbations of asthma and chronic obstructive pulmonary disease; desertification increases particulate matter (PM) exposures; and climate-related changes in food and water security impact infectious disease through malnutrition [24, 25]. Although all countries will be affected by climate change, low-resource countries including some of the Middle East countries are expected to be more effected by climate due to low-resource countries often lacking economic resources, having a close dependence on natural systems for basic food and water provision, and suffering from inadequate housing, energy, and waste management [25].

Quantifying the full impact of climate change on health is extremely difficult. This is partly because many modeling techniques are still in their infancy, but partly because impacts will depend on numerous interacting factors including other environmental trends, social resources, and preexisting health status. In the twenty-first century, the Mediterranean area is expected to be one of the most prominent and

vulnerable climate change regions that will experience a large number of extremely hot temperature events, an increase of summer heat wave frequency and duration, and increasing summer temperature variability [26]. An increase in the frequency and severity of heat waves is expected to enhance both illness and death rates. Using models that estimate climate change for the years 2020 and 2050, it is predicted that summer mortality will increase dramatically; the winter mortality will decrease slightly, even if people acclimatize to the increased warmth [27].

However, there are only a limited number of studies investigating effects of climate change on human health [28–33]. During the 2006 California Heat Wave, emergency visits for heat-related diseases and hospitalization were reported to have increased statewide. Children (0–4 years of age) and elderly (≥ 65 years of age) were found to be at the greatest risk. Emergency visits also showed significant increases for acute renal failure, cardiovascular disease, diabetes, electrolyte imbalance, and nephritis [28]. Al Eskan disease, reported in Military Medicine in 1992, is a novel and previously unreported condition triggered by the exceptionally fine sand dust of the central and eastern Saudi Arabian peninsula [29, 30]. It has been suggested that the mixture of the fine Saudi sand dust and pigeon droppings triggered a hyper-allergic lung condition [29]. It was concluded that sand particles less than $1\ \mu\text{m}$ ($0.1\text{--}0.25\ \mu\text{m}$) in diameter were present in substantial quantities in the Saudi sand and that these were the cause of the disease. Following the Gulf War in 1990, a similar clinicopathological entity was defined as “Persian Gulf syndrome” [30]. A wide range of acute and chronic symptoms have included fatigue, musculoskeletal pain, cognitive problems, respiratory symptoms, skin rashes, and diarrhea [31]. It was concluded that exposure to sand particles less than $1\ \mu\text{m}$ also contributed to pathogenesis of the syndrome, which was associated with the Gulf War factors [30]. Moreover, recent studies have reported that sandstorms increase hospitalization of children for asthma exacerbation [32].

It has been suggested that climate change may also lead to increased levels of air pollutants such as ozone. For example, according to projections made by the North American Regional Climate Change Assessment Program, an increase of 0.43 ppb in average ozone concentration is expected for the year 2040 comparing to the year 2000, and this was estimated to correspond to a 0.01 % increase in mortality rate and 45.2 premature deaths in the study communities attributable to the increase in future ozone levels [33].

Warmer conditions may lead to increases in the incidence and extent of infectious diseases such as malaria, dengue fever, schistosomiasis, and yellow fever. In Istanbul, Turkey, leptospirosis cases increased at the warmer periods of April–May–June, as compared to the cooler period of January–February–March in years 2004–2006 [17]. Within the last 3 decades, the number of malaria cases was increased in the two periods of 1977–1987 and 1993–1998 in Turkey, and this was in parallel with increased temperature [1]. In Iran, leishmaniasis diseases showed an outbreak during the period of 1995–2005 [13]. Furthermore, leishmaniasis is an endemic disease in all regions of Syria since nineteenth century, and the World Health Organization (WHO) classified border areas of the country with Iraq and Turkey as malarial high-risk areas [15].

Other consequences of climate change are expected to be the decreases in food production and increases in the cost that could lead to the risk of widespread malnutrition and hunger in the Middle East countries. A rise in sea levels and sea temperatures could also decrease the seafood stocks. Water shortages together with the higher temperatures may increase the risk of infectious diseases such as cholera, salmonella, and dysentery [25]. According to climate model scenarios, Iran will experience a maximum of 1.4 °C increase in temperature during the years 2010–2039, which is expected to increase the number of hospitalizations for diarrhea and cholera [13]. The loss of biodiversity and temperature changes may possess a risk for allergic airway diseases. Hence, a recent study in Turkey evaluated the effects of geo-climatic factors on the prevalence of allergic disease in a general adult population, and it has been demonstrated that high temperatures are associated with higher levels of allergens, higher asthma prevalence, longer pollen seasons, and diversity in pollens [34].

Conclusion

Global climate change is a serious problem and has adverse impacts on the environment and human health. However, some parts of the world such as the Middle East region suffer more from the detrimental effects of climate change. The region faces heat waves, water shortage, desertification, dust storms, loss of biodiversity, and their health consequences at a much severe scale. The resident countries, in addition to their contribution to the global combat against factors leading to climate changes, need to take local and regional adaptation and mitigation measures. Furthermore, more research is needed to understand the scale of the problem and its impacts on human health.

References

1. First National Communication of Turkey on Climate Change. In: Apak G, Ubay B, editors. Turkish Ministry of Environment and Forestry; 2007:1–263. http://www.dsi.gov.tr/docs/iklim-degisikligi/iklim_degisikligi_%C4%B1_ulusal_bildirim_eng.pdf?sfvrsn=2 (English)
2. United Nations Environment Programme (UNEP). World atlas of desertification. London: Edward Arnold; 1992.
3. International Institute for Sustainable Development (IISD). Rising temperatures, rising tensions. In: Brown O, Crawford A, editors. Climate change and the risk of violent conflict in Middle East; 2009:1–41. <http://www.iisd.org/publications/pub.aspx?pno=1130>
4. United Nations Development Programme (UNDP). Human development report 2007/2008. hdr.undp.org/en/media/HDR_20072008_EN_Complete.pdf
5. Haktanır K, Karaca A, Omar SM. The prospects of the impact of desertification on Turkey, Lebanon, Syria and Iraq. In: Marquina A, editor. Environmental challenges in the Mediterranean 2000–2050. Netherlands: Kluwer Academic; 2004. p. 139–54.
6. Elasha BO. Arab human development report. Mapping of climate change threats and human development impacts in the Arab region. 2010. <http://www.arab-hdr.org/publications/other/ahdrps/paper02-en.pdf>

7. Budak DB. Analysis of renewable energy and its impact on rural development of Turkey. 2009. <http://euroqualityfiles.net/AgriPolicy/Report%202.2/AgriPolicy%20WP2D2%20Turkey%20Final.pdf>
8. Meisen P, Hunter L. Global energy network institute. Renewable energy potential of the Middle East, North Africa vs The Nuclear Development Option. 2007. <http://www.geni.org/globalenergy/research/middle-east-energy-alternatives/MENA-renewable-vs-nuclear.pdf>
9. Demir İ, Kılıç G, Coşkun M. PRECIS bölgesel İklim Modeli ile Türkiye için İklim Öngörülürü: HadAMP3 SRES senaryosu, IV. Atmosfer Bilimleri Sempozyumu, 2008, Bildiriler Kitabı, 365–3 (in Turkish). <http://www.mgm.gov.tr/FILES/iklim/iklimongoruleri.pdf>
10. Ekerin S, Örmeci C. Evaluating climate change effects on water and salt resources in Salt Lake, Turkey using multitemporal SPOT imagery. *Environ Monit Assess.* 2010;163:361–8.
11. The Ministry of Environment and Forest, Republic of Turkey. Devlet Su İşleri Genel Müdürlüğü. İklim Değişikliği ve Yapılan Çalışmalar. 2008. http://www.dsi.gov.tr/docs/iklim-degisikligi/iklim_degisikligi_ve_yap%C4%B1lan_calismalar_ekim_2008.pdf?sfvrsn=2 (in Turkish).
12. Climate Change Israel National Report under the United Nations Framework Convention on Climate Change Impact, Vulnerability, Adaptation. 2000. <http://www.bgu.ac.il/BIDR/rio/Global91-editedfinal.html>
13. United Nations Development Programme. Iran Second National Communication to United Nations Framework Convention on Climate Change (UNFCCC). 2010. unfccc.int/resource/docs/natc/snc_iran.pdf
14. The United Nations Framework Convention on Climate Change (UNFCCC). Second National Communication Kingdom of Saudi Arabia. 2011. http://unfccc.int/resource/docs/natc/snc_report_2011_kingdom_of_saudi_arabia.pdf
15. Meslmani Y. United Nations Framework Convention on Climate Change (UNFCCC) Climate Change Initial National Communication of Syrian Arab Republic. 2010. http://unfccc.int/files/national_reports/non-annex_i_natcom/submitted_natcom/application/pdf/syria_initial_national_communication_03feb2011.pdf
16. Türkeş M. Vulnerability of Turkey to desertification with respect to precipitation and aridity conditions. *Tr J Eng Environ Sci.* 1999;23:363–80.
17. Birleşmiş Milletler Kalkınma Programı İklim Değişikliği ve Türkiye. Etkiler, Sektörel Analizler, Sosyoekonomik Boyutlar. 2007. <http://www.tobb.org.tr/Documents/yayinlar/iklim-degisikligiveturkiye.pdf> (in Turkish).
18. Sekercioglu CH, Anderson S, Akçay E, et al. Turkey's globally important biodiversity in crisis. *Biol Conserv.* 2011;144:2752–69.
19. TC Orman ve Su İşleri Bakanlığı Çölleşme ve Erozyonla Mücadele Genel Müdürlüğü. Türkiye'deki orman durumu ve erozyon. <http://www.cem.gov.tr/erozyon/AnaSayfa/faliyetler/erozyon/genelbilgiler.aspx?sflang=tr> (in Turkish).
20. TC Orman ve Su İşleri Bakanlığı Çölleşme ve Erozyonla Mücadele Genel Müdürlüğü. United Nations Convention Combat Desertification (UNCCD). Turkish National Action Programme on Combating Desertification. 2006. http://www.cem.gov.tr/erozyon/Files/faaliyetler/collesme/Belgeler/Turkey_s_National_Action_Program_on_Combating_Desertification.pdf (in Turkish and English).
21. General Directorate of Forestry. 2009 Forestry statistics. 2009. <http://web.ogm.gov.tr/Dkmanlar/istatistikler/OrmancilikIst2009.rar> (in Turkish).
22. Tahmiscioglu MS, Karaca Ö, Özdemir AD, Özgüler H. Possible effect of the global climate change on water resources and floods in Turkey. International conference on climate change and the middle east past, present and future, 2006, Istanbul, Turkey. http://www.dsi.gov.tr/docs/iklim-degisikligi/possible_effect_of_the_global_climate_change_on_water_resources_and_floods_in_turkey.pdf?sfvrsn=2
23. Kahraman A, Önder M, Ceyhan E. The importance of bioconservation and biodiversity in Turkey. *IJBBS.* 2012;2:95–9.

24. IPCC Fourth Assessment Report: climate change 2007. http://www.ipcc.ch/publications_and_data/publications_ipcc_fourth_assessment_report_wg3_report_mitigation_of_climate_change.htm
25. Pinkerton KE, Rom WN, Akpınar-Elci M, et al. An official American Thoracic Society workshop report: climate change and human health. *Proc Am Thorac Soc.* 2012;9:3–8.
26. Kuglitsch FG, Toreti A, Xoplaki E, et al. Heat wave changes in the eastern Mediterranean since 1960. *Geophys Res Lett.* 2010;37:1–5.
27. 29-Kalkstein LS, Greene SC. An evaluation of climate/mortality relationships in large US cities and possible impacts of climate change. *Environ Health Perspect.* 1997;105(1):84–93.
28. Knowlton K, Rotkin-Ellman M, King G, et al. The 2006 California heat wave: impacts on hospitalizations and emergency department visits. *Environ Health Perspect.* 2009;117(1):61–7.
29. Korényi-Both AL, Korényi-Both AL, Molnár AC, Fidelus-Gort R. Al Eskan disease: desert storm pneumonitis. *Mil Med.* 1992;157:452–62.
30. Korényi-Both AL, Korényi-Both AL, Juncer DJ. Al Eskan disease: Persian Gulf syndrome. *Mil Med.* 1997;162:1–13.
31. Coker WJ, Bhatt BM, Blatchley NF, Graham JT. Clinical findings for the first 1000 Gulf war veterans in the Ministry of Defence's medical assessment programme. *BMJ.* 1999;318:290–4.
32. Kantani TK, Ito I, Al-Delaimy WK, et al. Desert dust exposure is associated with increased risk of asthma hospitalization in children. *Am J Respir Crit Care Med.* 2010;182:1475–81.
33. Chang HH, Zhou J, Fuentes M. Impact of climate change on ambient ozone level and mortality in southeastern United States. *Int J Environ Res Public Health.* 2010;7:2866–80.
34. Metintaş M, Kurt E, PAFRAIT study group. Geo-climate effects on asthma and allergic diseases in Turkey: results of PAFRAIT study. *Int J Environ Health Res.* 2010;20:189–99.

Chapter 18

Assessing the Health Risks of Climate Change

Kristie Ebi

Abstract Climate change challenges the assumptions underlying traditional risk assessment approaches for estimating the health risks of climate change, starting with the most basic assumption that a defined exposure to a particular agent causes a specific adverse health outcome. These and other challenges are discussed, followed by a description of the process used by the Intergovernmental Panel on Climate Change to conduct assessments based on expert judgment evaluations of the literature combined with the collective experience and judgment of the authors.

Keywords Climate change and health • Challenges of traditional risk assessment • Determinants of health risk • IPCC assessment • Complexity of health risk evaluation • Climate-sensitive health outcomes

Climate change is altering everyday weather patterns, including changing averages and extremes of temperature and precipitation; the frequency, intensity, duration, and spatial extent of some extreme weather and climate events; and sea level [1, 2]. Health impacts can arise from:

- Climate change-related alterations in weather patterns that affect natural and physical systems that, in turn, affect the number of people at risk of malnutrition; the geographic range and incidence of vectorborne, zoonotic, and food and waterborne diseases; and the prevalence of diseases associated with air pollutants and aeroallergens. Additional climate change in coming decades is projected to significantly increase the number of people at risk of these major causes of ill health [3].

K. Ebi, Ph.D., M.P.H. (✉)
Department of Medicine, Stanford University, 424 Tyndall Street, Los Altos,
CA 94022, USA
e-mail: krisebi@stanford.edu

- Climate change-related alterations in the frequency, intensity, spatial extent, and duration of extreme weather events (e.g., heat waves, floods, droughts, and windstorms). Each year, these events affect millions of people, damage critical public health infrastructure, and cause billions of dollars worth of economic losses [2]. The frequency and intensity of some types of extreme weather events are expected to continue to increase over coming decades as a consequence of climate change, suggesting that the associated health impacts could increase without additional prevention actions.
- Climate change can affect population health through climate-related migration, as well as through damage to critical public health infrastructure and livelihoods by extreme weather events that affect development pathways [4].

Policy and decision-makers, public health and health-care agencies and institutions, and the general public want to understand to what extent these changes could affect their health and that of their families. Providing the answer is more complex than for more traditional health risks. This chapter discusses some challenges with using traditional risk assessment approaches to estimating the health risks of climate change. It then presents a conceptual approach for thinking about assessing health risks, followed by a discussion of the process used by the Intergovernmental Panel on Climate Change (IPCC) to conduct their assessments.

Challenges with Using Traditional Risk Assessment Approaches to Estimate the Health Risks of Climate Change

Public health has a long history of determining whether an agent presents a risk to health, where risk is defined as probability times consequence. Methods and tools to assess whether an agent could harm human health range from the Bradford-Hill criteria [5] to the International Agency for Research on Cancer [6] to toxicological risk assessments for environmental stressors [7]. At their simplest, these approaches ask whether this is sufficient information to determine if an agent is a hazard to health and, if so, to determine exposure-response relationships and to characterize the extent of human exposures. These are combined, often including safety factors designed to protect those most vulnerable to adverse impacts, to produce a quantitative or qualitative statement about the probability and degree of harm to the exposed populations [8].

Climate change challenges the assumptions underlying these traditional approaches to assessing risk [9]. The challenges start with the basic assumption—that a defined exposure to a specific agent causes an adverse health outcome to identifiable exposed populations, with specific groups at particular risk. This implicitly assumes the health outcome from an exposure is distinctive, and the association between immediate cause (e.g., cigarette smoking) and health impact (e.g., lung cancer) can be determined fairly clearly. Further, studies of the associations are based on comparing an unexposed, control group to a group with, preferably, quantified levels of exposure, in order to develop quantitative exposure-response

relationships. Both the assumptions and the types of studies conducted to support traditional risk assessments do not easily apply to the health risks of climate change.

Any health outcome sensitive to weather and climate could be affected by climate change, including morbidity and mortality associated with temperature extremes, other weather- and climate-related extreme events, ground-level ozone, infectious diseases, malnutrition, and migration [4]. The range of outcomes is increasing with more research focusing on the health risks of climate change. For example, ambient temperature may influence survival during gestation, affecting the sex ratio at birth and male longevity in some regions [10]. In addition, a changing climate can result in key weather variables crossing thresholds that result in large changes in the geographic range or incidence of a health outcome. One example is the 2004 outbreak of *Vibrio parahaemolyticus* in Alaska, the leading cause of seafood-associated gastroenteritis in the USA; outbreaks are typically associated with the consumption of raw oysters gathered from warm-water estuaries [11]. The consumption of raw oysters was the only significant predictor of illness. The attack rate among people who consumed oysters was 29 %. All oysters associated with the outbreak were harvested when mean daily water temperatures exceeded 15.0 °C (the theorized threshold for the risk of *V. parahaemolyticus* illness from the consumption of raw oysters). Between 1997 and 2004, mean water temperatures in July and August at the implicated oyster farm increased 0.21 °C per year. 2004 was the first year during which mean daily temperatures did not drop below 15.0 °C. The outbreak extended by 1,000 km the northernmost documented source of oysters that caused illness due to *V. parahaemolyticus*. Anticipating these types of events requires a better understanding of how weather could affect the incidence and geographic range of health outcomes, including thresholds.

A further complexity is that multiple weather variables may be associated with a single health outcome, with the variables varying geographically. The geographic distributions and seasonal variations of many infectious diseases indicate the potential importance of weather and seasonal to interannual climate variability in disease patterns [12]. Temperature, precipitation, and humidity can affect vector survival, reproduction, development, and biting rates, as well as pathogen reproduction and development, thus affecting the timing and intensity of outbreaks. Further, the weather variables of importance can vary geographically. For example, Thomson [13] found a geographically complex association between malaria incidence and the timing of the onset and retreat of seasonal rains in Nigeria, with rainfall onset related to the El Niño Southern Oscillation and the Northern Annular Mode, and retreat related to the North Atlantic Oscillation and East Pacific or West Pacific circulation index.

Climate-sensitive health outcomes often have many, interrelated causes, of which weather is only one factor; feedback mechanisms also may be important. The causal chain between exposure to a pathogen and disease is complex; exposure is necessary but not sufficient to cause disease. Therefore, weather and climate are often not the primary drivers of a health outcome, which is not to discount their importance, but to acknowledge the importance of studying health outcomes using systems-based approaches that include the social, economic, and political factors influencing disease risk.

Although impact assessments generally state their goal is to evaluate the magnitude and extent of the health risks of climate change, nearly all actually assess the possible impacts of weather and seasonal to interannual variability on health outcomes today and in the future. Climate change takes place over decades or longer, affecting local weather patterns differentially across temporal and spatial scales [14]. For example, as global mean surface temperatures have increased, warming most parts of the world, some areas cooled [2]. Further, the extent of temperature increase varied across regions, generally with higher latitudes warming more.

Analyzing relationships between climate change and health outcomes requires decades long data sets; such data sets are available for meteorological data but are rare in the health sector. Analyzing these data sets requires selecting a baseline for comparison because there is no natural baseline in a changing climate. Analyses in the IPCC fourth Assessment Reports often used 1961–1990 as the baseline, while acknowledging the climate during that period differed from the climate a century earlier when greenhouse gas emissions rapidly accelerated with the industrial revolution [15]. A standard approach is to analyze whether there was a statistically significant trend in climate and in the outcome of interest from the baseline and then to determine whether some of the observed changes can be attributed to climate change [16]. Such detection and attribution studies are rare in the health sector [17].

Determinants of Health Risk of Climate Change

Approaches to assess the possible impacts of climate change typically focus on identifying communities and places vulnerable to observed or projected changes in weather patterns and a range of possible adaptation and mitigation options to reduce risks and increase resilience. One challenge has been that vulnerability is conceptualized differently across and even within sectors [18], with some sectors, such as the health sector, viewing vulnerability as the initial state before considering changing exposure patterns. Other sectors, such as natural hazards, tend to view vulnerability as a description related to the residual impacts after considering changes in exposure and implementation of response options. At their most basic, the various definitions consider vulnerability to be the propensity or predisposition to be adversely affected [2]. A large number of factors determine vulnerability, including poverty (although all poor people are not equally at risk), demographics (although not all population groups are equally vulnerable to each outcome), wealth and income distribution, status of the public health infrastructure, access to medical care, behavioral factors, individual physiological factors, and a wide range of social and cultural factors. A key message from the IPCC *Special Report on Managing the Risks of Extreme Events and Disasters to Advance Climate Change Adaptation* is that vulnerability can be much more important than climate change in determining impacts [2]. Policies to address these vulnerabilities may have commonalities across regions and sectors but need to be tailored to specific circumstances. Further, policies need to balance competing demands, such as water needs across agriculture, other economic sectors and tourism, urban areas, recreational use, health concerns, and others.

Therefore, a framework for assessing the health risks of climate change is that the magnitude and extent of impacts of climate change on the incidence and geographic range of climate-sensitive health outcomes is a function of the interactions between exposure(s) to climate change-related alterations in weather patterns (and the implications of the associated changes, such as changing crop yields) and the vulnerabilities of the exposed human and natural systems (e.g., changing crop yields can have differential consequences depending on the availability of other food sources). Exposures are changes in mean and variability of temperature, precipitation, and other weather variables associated with climate-sensitive health outcomes.

This framing highlights another major challenge to applying traditional risk assessment approaches to assessing the health risks of climate: the magnitude and extent of risks depend on the vulnerability of a community or place. Crucially, this means one exposure-response relationship may not be applicable across all temporal and spatial scales. A very few examples illustrate the challenge. Heat waves kill unnecessarily; the extent to which a heat wave is a risk depends on the population acclimatization to hot weather, the number of previous heat waves that season, the proportion of the population with increased sensitivity (e.g., older adults, the prevalence of diabetes, the proportion using certain drugs), mortality rates the previous winter, the effectiveness of the local early warning system, etc. [19, 20]. In some regions of Africa, malaria follows the rains, in others it follows drought [21]. The same magnitude typhoon hitting Japan will have very different consequences from one hitting the Philippines [22]. An evaluation of the flood risk in Sri Lanka depends on the question being asked; storm surges affect coastal regions fairly infrequently, with large consequences when they do [22]. Inland areas have more frequent and less intense flooding events that affect more communities and their livelihoods. Presumably, increased investment in risk reduction activities, from strengthening housing to moving buildings at particular risk to early warning systems, could reduce vulnerability over time, so the consequences of a heavy precipitation event would change over time. National level estimates of vulnerability will average over very different sets of circumstances that affect local to regional vulnerability.

Finally, climate change may be increasing the probability of not just individual but also of joint extreme events [2]. The combination of a long-standing drought, heat wave, and wildfire in Melbourne, Australia had devastating consequences.

Understanding differences in vulnerability is necessary for estimating the possible health risks of climate change, from which effective and efficient adaptation options can be designed and implemented [23].

Assessments by the Intergovernmental Panel on Climate Change

The IPCC conducts periodic assessments of the scientific, technical, and socioeconomic information relevant for understanding anthropogenic climate change, its potential impacts, and options for mitigation and adaptation. These assessments are

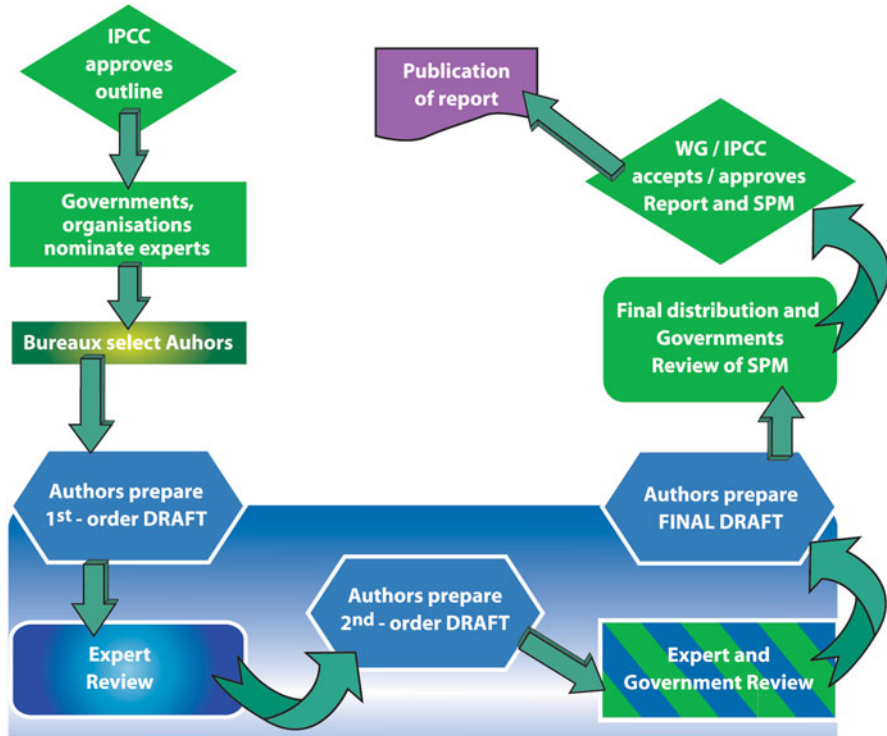


Fig. 18.1 Peer-reviewed and internationally available scientific technical and socioeconomic literature, manuscripts made available for IPCC review, and selected non-peer-reviewed literature produced by other relevant institutions including industry

based on expert judgment evaluation of the literature (peer- and non-peer-reviewed) combined with the collective experience and judgment of a group of individuals chosen because of their diverse and relevant expertise (Fig. 18.1) [24]. The chapters produced are thoroughly reviewed by the worldwide scientific community to ensure the assessments reflect the literature base.

The United Nations Environment Programme and the World Meteorological Organization established the IPCC as a unique collaboration between the scientific community and policymakers, with governments (through their Focal Points) providing guidance and input at several stages during the process to the scientists conducting an assessment. IPCC reports are mandated to be comprehensive, objective, and balanced [25]. Additional requirements are to describe different scientific, technical, and socioeconomic views on a subject and for an assessment to be policy relevant and policy neutral. The assessments aim to inform national governments about the most up-to-date scientific thinking and to highlight possible policy options to address current and projected risks, without promoting one set of options over another. The members of the IPCC are the world governments.

When the governments decide to initiate an assessment, the first step is for governments to select individuals to lead the three Working Groups (WGs) in the IPCC: WGI assesses the science of climate change; WGII assesses impacts, adaptation, and vulnerability; and WGIII assesses mitigation options. It is the Panel that decides whether to prepare a report, including its scope, outline, and work plan, in consultation with the respective WG. Policymakers and other users of IPCC Reports may be consulted to identify key policy-relevant issues. For example, the outlines for the Working Groups contributions to the AR5 were developed during a scoping meeting in Venice, Italy in July 2009 (http://ipcc.ch/meeting_documentation/workshops-experts-meetings-ar5-scoping.shtml). Participants in the meeting included leading scientists and government representatives who considered advances in scientific knowledge since the literature cutoff date for the fourth Assessment Report (early 2006) as well as emerging issues and perspectives. Once an outline is agreed, Governments and IPCC Observer Organizations are requested to nominate experts to be coordinating lead authors (CLAs), lead authors (LAs), and review editors (REs). Author teams were constructed with attention to scientific qualifications, the needed range of institutional and disciplinary perspective, and adequate regional and gender balance, while also involving the next generation of climate scientists.

As required by the IPCC Principles and Procedures, there are two reviews of a report, the Expert Review (First Order Draft) and the Government and Expert Review (Second Order Draft). These reviews involve hundreds of reviewers who submit thousands of comments. There were more than 18,000 review comments in the two review periods for the nine-chapter IPCC Special Report on *Managing the Risks of Extreme Events and Disasters to Advance Climate Change Adaptation*, with a range of several hundred to more than a thousand per chapter per review period. For the WGII contribution to the AR4, more than 40,000 review comments were submitted. A requirement of the IPCC process is that authors must provide written responses to all comments submitted during these review periods; a considerable task. Review Editors, a unique feature of IPCC reports, are involved in the process starting with the First Order Draft review, representing the reviewers and ensuring that each comment is considered and appropriately addressed.

The last step in the process for a WG contribution to an assessment cycle is for the Summary for Policymakers (SPM) to be approved line by line in a Working Group session. Every sentence in a SPM is discussed and agreed (by consensus) between the authors who drafted the SPM and governments. Authors and governments want to ensure the SPM is not only an accurate assessment of the state of knowledge but also that it communicates key findings clearly in understandable language to policymakers. When a SPM is approved, the governments then accept the underlying report [25]. This close and ongoing dialogue at the science-policy interface ensures an assessment achieves its mandate and requirements and is a unique feature of IPCC reports. This process was intensively reviewed and endorsed with some modifications by the InterAcademy Council [26].

Science is only one input into decision-making [27]; policymakers also take into consideration social and cultural values and perspectives, practical issues (from technological to political), and other factors when developing and implementing a

policy. Policies need to be specific to a national (or subnational) context, including level of development, current and projected vulnerabilities, current and projected climate variability and change, and many other factors. For example, policies to enhance food security in a changing climate will depend on a wide range of issues, such as causes of food insecurity in the region of interest, crops grown, water availability, transport, trade policies, and others.

IPCC CLAs and LAs are not only experts in their field; they also willingly donate considerable time and intellect to an IPCC assessment. The letters of invitation to participate in the WGII contribution to the AR5 estimated that CLAs could expect to commit approximately 6 months of full-time activity between appointment and the WGII approval session in 2014; LAs were told to expect approximately 4 months of full-time activity. The time committed is voluntary; the IPCC does not support the time scientists spend working on an assessment. Steve Schneider frequently referred to the IPCC as his pro bono job. A WG Technical Support Unit provides support for limited aspects of report development, but not for reviewing literature and writing text. The IPCC has a Trust Fund that covers travel and per diem to lead author meetings for authors and review editors from developing countries and countries with economies in transition. Developed country governments are expected to cover travel and per diem for their authors and review editors.

WGII author teams generally have two CLAs and six LAs to deliver comprehensive assessments on a broad range of topics. No chapter team has experts for every issue that will be covered. Many chapters will cite roughly 1,000 references. For example, the human health chapter will include topics such as the current burden of climate-sensitive health outcomes; vulnerability of children and older adults; projected changes in malnutrition, infectious diseases, emerging zoonotic diseases, and morbidity and mortality due to extreme weather events; experience with adaptation; costs of action and of inaction on climate change; and co-benefits of mitigation policies. Therefore, expertise is drawn from authors on other chapters and from the wider scientific community through selection of Contributing Authors (CAs). CAs are selected as needed to write about a specific topic or contribute a case study to illustrate a particular point.

In reporting the key conclusions from their chapter, authors describe the certainty in those findings using calibrated uncertainty language [28]. This language aims to facilitate clear communication of the degree of certainty in assessment findings, including findings that span a range of possible outcomes. It also aims to avoid descriptions of uncertainties using casual terms that may imply different meanings to different disciplines and/or in different languages.

The process used in the fifth Assessment Report provides authors from all WGs with a common approach for considering key findings in the assessment process, for supporting key findings with traceable accounts in the chapters, and for characterizing the degree of certainty in key findings using two metrics [28]:

- Confidence in the validity of a finding, based on the type, amount, quality, and consistency of evidence (e.g., mechanistic understanding, theory, data, models, expert judgment) and the degree of agreement. Confidence is expressed qualitatively.

- Evidence and agreement are each categorized on a three-point scale (for nine combinations). The author team’s evaluation of evidence and agreement provides the basis for any key finding it develops and also the foundation for determining the author team’s degree of certainty in those findings. The description of the author team’s evaluation of evidence and agreement is called a traceable account. Each key finding presented in a chapter’s Executive Summary will include reference to the chapter section containing the traceable account for the finding.
- When there is sufficient evidence and agreement, they can be synthesized into one metric to describe a qualitative level of confidence, where level of confidence is designated as very low, low, medium, high, and very high.
- Quantified measures of uncertainty in a finding, such as a probabilistic estimate of a specific occurrence or range of outcomes. Probabilistic information may originate from statistical or modeling analyses, expert elicitation of views, or other quantitative information.

Discussion

Climate change presents a wide range of risks to human health that vary spatially and temporally. Traditional risk assessment approaches are ill suited to understanding the complex interactions leading to adverse health impacts when exposure is one of many factors affecting the health burden. At this time, expert judgment processes, such as that used by the IPCC, can provide more nuanced understanding of risks and how they could change over time with changes in climate, development, and other factors. As the literature base on the health impacts of climate change expands, meta-analytic and other techniques may be possible that would provide more robust key findings.

Whatever the approach used to assess the health risks of climate change, the goal should be to provide information relevant for developing strategies, policies, and measures to protect the most vulnerable, today and in the future.

References

1. IPCC Climate Change. Summary for policy makers. Cambridge: WMO/UNEP; 2007.
2. IPCC. Summary for policymakers. In: Field CB, Barros V, Stocker TF, Qin D, Dokken DJ, Ebi KL, Mastrandrea MD, Mach KJ, Plattner GK, Allen SK, Tignor M, Midgley PM, editors. Managing the risks of extreme events and disasters to advance climate change adaptation. A special report of Working Groups I and II of the Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press; 2012. p. 1–19.
3. Confalonieri U, Menne B, Akhtar R, Ebi KL, Hauengue M, Kovats RS, Revich B, Woodward A. Human health. In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hansson CE, editors. Climate change 2007: impacts, adaptation and vulnerability. Contribution of Working

- Group II to the fourth assessment report of the Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press; 2007.
4. McMichael AJ, Lindgren E. Climate change: present and future risks to health, and necessary responses. *J Int Med.* 2011;270:401–13.
 5. Bradford-Hill A. The environment and disease: association or causation? *Proc R Soc Med.* 1965;58:295–300.
 6. International Agency for Research on Cancer (IARC). Preamble. IARC monographs on the evaluation of carcinogenic risks to humans. Lyon, France: World Health Organization, IARC. <http://monographs.iarc.fr/ENG/Preamble/index.php> (2006). Accessed 22 Apr 2012.
 7. National Research Council. Risk assessment in the Federal Government: managing the process. Washington, DC: National Academy Press; 1983.
 8. National Research Council Committee on Risk Assessment of Hazardous Air Pollutants. Science and judgment in risk assessment. Washington, DC: National Academy Press; 1994.
 9. Bernard SM, Ebi KL. Comments on the process and product of the health impacts assessment component of the United States national assessment of the potential consequences of climate variability and change. *Environ Health Perspect.* 2001;109 Suppl 2:177–84.
 10. Catalano R, Bruckner T, Smith KR. Ambient temperature predicts sex ratios and male longevity. *Proc Natl Acad Sci USA.* 2008;105:2244–7.
 11. McLaughlin JB, DePaola A, Bopp CA, Martinek KA, Napolilli NP, Allison CG, Murray SL, Thompson EC, Bird MM, Middaugh JP. Outbreak of *Vibrio parahaemolyticus* gastroenteritis associated with Alaskan oysters. *N Engl J Med.* 2005;353:1463–70.
 12. National Research Council. Under the weather: climate, ecosystems, and infectious diseases. Washington, DC: National Academy Press; 2001.
 13. Thomson AJ. Climate indices, rainfall onset and retreat, and malaria in Nigeria. *J Vector Borne Dis.* 2010;47:193–203.
 14. IPCC. Climate change 2007: the physical science basis. In: Solomon S, Qin D, Manning M, Chen Z, Marquis MC, Averyt KB, Tignor M, Miller HL, editors. Contribution of Working Group I to the fourth assessment report of the Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press; 2007.
 15. Stott PA, Gillett NP, Hegerl GC, Karoly DJ, Stone DA, Zhang X, Zwiers F. Detection and attribution of climate change: a regional perspective. *Wiley Interdiscip Rev Clim Change.* 2010;1:192–211.
 16. Hegerl GC, Hoegh-Guldberg O, Casassa G, Hoerling MP, Kovats RS, Parmesan C, Pierce DW, Stott PA. Good practice guidance paper on detection and attribution related to anthropogenic climate change. In: Stocker TF, Field CB, Qin D, Barros V, Plattner G-K, Tignor M, Midgley PM, Ebi KL, editors. Meeting report of the Intergovernmental Panel on Climate Change Expert Meeting on detection and attribution related to anthropogenic climate change. Bern, Switzerland: IPCC Working Group I Technical Support Unit, University of Bern; 2010. p. 1–8.
 17. Ebi KL, Kovats RL. Detection and attribution of health effects to global climate change. *Environ Health Perspect.* In: Abstracts of the 23rd annual conference of the international society of environmental epidemiology (ISEE), Barcelona, Spain, 13–16 Sep 2011. 2011. <http://dx.doi.org/10.1289/ehp.isee2011>. Accessed 25 Apr 2012.
 18. Cardona O-D, van Aalst MK, Birkmann J, Fordham M, McGregor G, Perez R, Pulwarty RS, Schipper ELF, Sinh BT. Determinants of risk: exposure and vulnerability. In: Field CB, Barros V, Stocker TF, Qin D, Dokken DJ, Ebi KL, Mastrandrea MD, Mach KJ, Plattner G-K, Allen SK, Tignor M, Midgley PM, editors. Managing the risks of extreme events and disasters to advance climate change adaptation. A special report of Working Groups I and II of the Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press; 2012. p. 65–108.
 19. Kovats RS, Hajat S. Heat stress and public health: a critical review. *Annu Rev Public Health.* 2008;29:41–55.
 20. Rocklöv J, Forsberg B, Meister K. Winter mortality modifies the heat-mortality association the following summer. *Eur Respir J.* 2009;33:245–51.

21. Parham PE, Michael E. Modeling climate change and malaria transmission. *Adv Exp Med Biol.* 2010;673:184–99.
22. United Nations International Strategy on Disaster Reduction Secretariat. Global assessment report on disaster risk reduction. <http://www.preventionweb.net/english/hyogo/gar/report/index.php?id=9413> (2009). Accessed 25 Apr 2012.
23. Ebi KL. Public health responses to the risks of climate variability and change in the United States. *J Occup Environ Med.* 2009;51:4–12.
24. Parry M, Carter T. Climate impact and adaptation assessment: a guide to the IPCC approach. London: Earthscan Publications; 1998.
25. IPCC. Decisions taken with respect to the review of IPCC processes and procedures: procedures. IPCC 34th Session, 18–19 Nov 2011, Kampala, Uganda. http://ipcc.ch/pdf/ipcc-principles/appendix_a_decision.pdf (2011). Accessed 25 Apr 2012.
26. InterAcademy Council. Climate change assessments, review of the processes and procedures of the IPCC. Amsterdam, The Netherlands: InterAcademy Council. <http://reviewipcc.interacademycouncil.net/> (2010). Accessed 20 Apr 2012.
27. Scheraga J, Ebi K, Moreno AR, Furlow J. From science to policy: developing responses to climate change. In: McMichael AJ, Campbell-Lendrum D, Corvalan CF, Ebi KL, Githeko A, Scheraga JD, Woodward A, editors. *Climate change and human health: risks and responses.* Geneva, Switzerland: WHO/WMO/UNEP; 2003.
28. Mastrandrea MD, Field CB, Stocker TF, Edenhofer O, Ebi KL, Frame DJ, Held H, Kriegler E, Mach KJ, Matschoss PR, Plattner G-K, Yohe GW, Zwiers FW. Guidance note for lead authors of the IPCC fifth assessment report on consistent treatment of uncertainties. Intergovernmental Panel on Climate Change (IPCC). <http://www.ipcc-wg2.gov/meetings/CGCs/index.html#UR> (2010). Accessed 25 Apr 2012.

Chapter 19

Federal Programs in Climate Change and Health Research

Maya Levine and John Balbus

Abstract The federal government plays an integral role in supporting climate change science and health research in the USA. Federally funded climate change research initially focused on science to understand climate and earth systems change during the 1970s and 1980s. Today, federally supported climate change research involves numerous agencies pursuing a wide range of climate change science topics and applications, including research exploring the connections between human health and climate change. Because each federal agency has a different mandate and range of scientific expertise, the focus and goals of various agencies' climate change and human health research vary. For example, the National Oceanic and Atmospheric Administration (NOAA) emphasizes the use of weather and climate forecasts and oceanographic data for public health applications, while the National Aeronautics and Space Administration (NASA) emphasizes health applications of remotely sensed data from its satellites. This chapter provides a brief history of federally funded climate research and includes a survey of the relevant agencies, programs, tools, and datasets to illustrate the diversity of health and climate change research supported by the federal government.

Keywords Federal programs in climate change and health research • Health research and climate change • Climate change and federal programs • Research in climate change • Global Change Research Program

M. Levine, M.A. • J. Balbus, M.D., M.P.H. (✉)
National Institute of Environmental Health Sciences, 31 Center Drive, Room B1C02,
Bethesda, MD 20892-2256, USA
e-mail: balbusjm@niehs.nih.gov

Background

The federal government plays an integral role in supporting climate change science and health research in the USA. Federally funded climate change research initially focused on science to understand climate and earth systems change during the 1970s and 1980s. Today, federally supported climate change research involves numerous agencies pursuing a wide range of climate change science topics and applications, including research exploring the connections between human health and climate change. Because each federal agency has a different mandate and range of scientific expertise, the focus and goals of various agencies' climate change and human health research vary. For example, the National Oceanic and Atmospheric Administration (NOAA) emphasizes the use of weather and climate forecasts and oceanographic data for public health applications, while the National Aeronautics and Space Administration (NASA) emphasizes health applications of remotely sensed data from its satellites. This chapter provides a brief history of federally funded climate research and includes a survey of the relevant agencies, programs, tools, and datasets to illustrate the diversity of health and climate change research supported by the federal government.

Federal Climate Change Research Prior to 1990

In 1978, Congress established the Federal Interagency Climate Program through the National Climate Act to “assist in the understanding and response to natural and human-induced climate processes and their implications” [1]. The National Climate Act required the program to conduct studies to understand the impacts of human activities on climate, to promote scientific understanding of climate change, to improve forecasts and data collection of climate processes, and to encourage international cooperation in climate research [2]. The National Oceanic and Atmospheric Administration (NOAA) of the U.S. Department of Commerce (DOC) coordinated the Interagency Climate Program. As a result of the Climate Program's emphasis and design, NOAA, NASA, and National Science Foundation (NSF) became leaders of climate change science at the federal level with each agency developing its own climate change-related programs during the 1980s. While the Climate Program received positive evaluations from the NSF, many agreed that the program's limited scope and emphasis on atmospheric and climate-related science disciplines was failing to produce the range of understanding required to inform the growing demand for policy responses and “global change”-related research [3].

Prompted by increasing concerns about climate change from the international and domestic scientific communities, Congress held a series of hearings, beginning in late 1985, further increasing public and legislative interest in climate change. In 1987, Congress passed The Global Climate Protection Act (P.L. 100–204), which designated the Environmental Protection Agency (EPA) and U.S. Department of

State as leads for climate change policy development. Even with the passing of the Climate Protection Act of 1987, the Bush Administration had not articulated a national strategy or set of goals related to global climate change [4]. As scientific interest and public concern about climate change continued to grow, some members of Congress became frustrated with the U.S. government's inability to coordinate research efforts to inform climate policy decisions [5].

The Global Change Research Act of 1990 and the Global Change Research Program

The U.S. Global Change Research Program (GCRP) has coordinated federal research and observation on global environmental change and societal impacts since its inception. The Global Change Research Act of 1990 (GCRA), signed by President George H. W. Bush, in November 1990, established the GCRP with the explicit aim “to provide for development and coordination of a comprehensive and integrated United States research program, which will assist the Nation and the world to understand, assess, predict, and respond to human-induced and natural processes of global change” (P.L. 10–606). The GCRP's initial research agenda focused on developing a predictive understanding of the earth's climate [6].

The National Research Council (NRC) reviews the GCRP to provide a politically neutral assessment of the program's accomplishments and activities. A 1999 NRC report to the GCRP found that understanding the economic and social impacts of “the human consequences of global environmental change on key life-support systems, such as water, health, energy natural ecosystems, and agriculture” need to be prioritized within the GCRP research agenda [7]. In 2001, the NRC report on the GCRP noted, “in order to address the consequences of climate change and better serve the Nation's decision makers, the research enterprise dealing with environmental change and environment-society interactions must be enhanced” [8]. With growing concerns about the impacts of climate change and the need for more information to inform policy response strategies, the GCRP began to integrate environment-society interactions into its research priorities more aggressively over the past decade.

The most recent NRC assessment in 2009 found that the GCRP's reinvigorated mission of 2003 was particularly relevant to the societal needs of 2010 and beyond. At the same time, the NRC recommended that the program once again broaden its scope to better meet the needs of decision makers and stakeholders by including additional research focusing on the human dimensions of climate change [9]. Human dimensions of climate change include research about potential social and economic impacts, adaptation and mitigation strategies, and vulnerabilities of particular subpopulations [10]. This information is critical for accurate climate change economic assessments and timely policy responses. In response to this guidance from the NRC, the GCRP has broadened its focus from science to understand earth's systems and their functions to the development of information for comparative

analysis of adaptation and mitigation strategies, capacity building, and the implication of climate change for multiple society-environment interactions, including human health [11]. Under the societal needs sections of the GCRP, research priorities shifted to include the following five elements: urban systems, energy systems, land use change, water resources, and human health.

In the 2012 Strategic Plan, the GCRP mission is “to build a knowledge base that informs human responses to climate and global change through coordinated and integrated federal programs of research, education, communication, and decision support” [12]. The 2012 Strategic Plan outlines four strategic goals for GCRP coordination of federal climate change research [13]:

- Goal 1. Advance Science: advance scientific knowledge of the integrated natural and human components of the Earth system.
- Goal 2. Inform Decisions: provide the scientific basis to inform and enable timely decisions on adaptation and mitigation.
- Goal 3. Conduct Sustained Assessments: build sustained assessment capacity that improves the Nation’s ability to understand, anticipate, and respond to global change impacts and vulnerabilities.
- Goal 4. Communicate and Educate: advance communications and education to broaden public understanding of global change and develop the scientific workforce of the future.

Thirteen federal agencies and departments participate in the USGCRP. Program activities are coordinated through interagency working groups organized around cross-disciplinary climate and global change themes [14]. These groups focus on the following program elements: Integrated Observation, Integrated Modeling, Multidisciplinary Research on the Human and Natural Components of the Earth System, Conduct Sustained Assessments, Informing Decisions, International Cooperation, Communication and Education, and Climate Change and Human Health [15]. In order to foster better integration among the sciences (biological, social, behavioral, and economic), participation in the working groups extends beyond the 13 agencies formally represented in the GCRP. Through these efforts and collaborations with other national and international research programs, the GCRP has made substantial advances in these critical areas:

- Observing and understanding short- and long-term changes in climate, the ozone layer, and land cover
- Identifying the impacts of these changes on ecosystems and society
- Estimating future changes in the physical environment and vulnerabilities and risks associated with those changes
- Providing scientific information to enable effective decision making to address the threats and opportunities posed by climate and global change [12].

GCRP-supported research contributes to influential international reports such as the Intergovernmental Panel on Climate Change’s (IPCC) climate change assessment as well as various national climate change assessments greatly enhancing our

understanding of climate and global change science [16]. GCRP documents, program results, and plans are compiled in an annual report entitled “Our Changing Planet” [17].

The Interagency Climate Change and Human Health Group

As part of the effort to reorient the GCRP to better meet societal needs, and specifically to provide greater focus on understanding the human health implications of climate change, the GCRP chartered a new interagency working group, the Climate Change and Human Health Group (CCHHG) in December 2009. The CCHHG is intended to pilot the “end-to-end” approach to science described in the NRC report “Restructuring Federal Climate Research to Meet Societal Needs.” To that end, the composition of the CCHHG includes agencies that have not traditionally participated in climate research but are translators and users of the scientific information on climate and health produced within the GCRP, like the Centers for Disease Control and Prevention (CDC) and the Department of Homeland Security (DHS). The breadth of the CCHHG’s charge is reflected by the agencies serving as cochairs of the workgroup: NIEHS representing a research focus, CDC representing public health programmatic work, and NOAA representing atmospheric and oceanic research as well as coastal and oceanic programmatic work.

The roles of the CCHHG as described in the group’s charter include:

- Coordinating federal research efforts on climate change and human health and ensuring research agendas are informed by end users of the information developed
- Serving as a conduit of information between the GCRP and stakeholders on climate change and health issues
- Providing expertise to reports and assessments
- Representing GCRP on health issues to international organizations
- Reporting back to the GCRP on the effectiveness of the CCHHG’s structure and approaches to meeting societal needs for climate change and human health information

The charter also specifies that the CCHHG will apply a “one health” concept in its work, integrating science on the health of domestic and wild animals and ecosystems with the health of humans [18].

In addition to coordinating and communicating the work of its constituent agency members, as described below, the CCHHG has served the GCRP and its charter through a number of activities and accomplishments, including:

- Serving as a workgroup for the President’s Climate Adaptation Task Force and producing recommendations for health adaptation
- Supporting the National Climate Assessment (NCA) by conducting workshops, reviewing the scientific literature, and providing leadership and input to the health sector and other author teams
- Developing a metadata portal to improve access to climate and health datasets

- Conducting town hall meetings and other events at conferences to promote two-way communication with critical stakeholder groups
- Conducting a self-evaluation and reporting the results back to the GCRP leadership

The CCHHG continues to prioritize the new GCRP goals by creating workstreams that support the GCRP efforts to develop scientific information on climate and health to meet user needs [19]. The 2012 Strategic Plan states that the GCRP will address the challenges of climate change and human health by “building the integrated knowledge base needed to understand, predict, respond, and adapt.” Particular areas of interest include [20]:

- Develop models and tools to assess the environmental, social, human health, and economic outcomes of alternative adaptation and mitigation options.
- Enhance the integration of new socioeconomic, health, and ecological observations with integrated observations of the climate system to address the vulnerability of ecosystems and human systems to global change and inform national adaptation and mitigation efforts.
- Through the NCA, analyze the effects of global change on the natural environment, agriculture, energy production and use, land and water resources, transportation, human health and welfare, human social systems, and biological diversity.

Climate Change and Human Health Group Priority Workstreams

The CCHHG is presently organized around priority workstreams to carry out activities for the USGCRP and the broader climate change and human health community. Identified priority workstreams include:

- Adaptation—including ongoing support for the Interagency Climate Change Adaptation Task Force
- Assessment—including technical input and stakeholder engagement support for the development of the NCA report
- Communication, education, and engagement—including coordination with broader USGCRP communication, education, and engagement (CEE) activities
- Data integration—including development of an interactive Internet-based metadata access tool, early warning systems, and monitoring tools related to the health impacts of global climate change
- Joint research and funding planning—including development of a human health and climate change research framework, gap analysis, prioritization of research needs, and coordination of joint funding opportunities
- International—including review of international health adaptation plans and assessments to capture lessons learned and engagement with the global health community on climate change and health

Climate Change and Human Health Group Workstream Activity Spotlight: Regional Climate Change and Health Assessments

The USGCRP's Climate Change and Human Health Working Group (CCHHG) convened two regional climate change and human health workshops in February 2012 as part of the NCA process. The workshops, held in the Southeast and Northwest regions, explored regional climate change impacts on health and aimed to foster collaboration and dialogue. Over 50 regional climate change experts, public health experts, and other relevant stakeholders attended the meetings. The workshops were supported by the NOAA Oceans and Human Health Initiative (OHHI), the CDC National Center for Environmental Health, the National Institutes of Health (NIH), and National Institute for Environmental Health Sciences (NIEHS). Workshop goals included:

- Inform the 2013 U.S. NCA report.
- Increase the level of understanding of climate and health science in the region.
- Raise awareness of ongoing climate and health activities in the region.
- Improve tools for public health decision making by providing a forum for scientists and decision makers to share information and develop new or improve existing partnerships.
- Serve as a pilot for how to sustain an ongoing assessment process for understanding, predicting, and adapting to the human health impacts of climate change across time scales.

The synthesis reports and regional project inventories for each were submitted as technical input to the NCA 2013 Report [21].

Tool: Metadata Access Tool for Climate and Health Geportal

Metadata Access Tool for Climate and Health (MATCH) is an interactive, searchable, web-based clearinghouse of publicly available federal metadata including monitoring and surveillance datasets, early warning systems, and tools for characterizing the health impacts of global climate change. MATCH serves as a gateway to information that can be used to identify opportunities in climate change and health research, enable scientific collaboration using a "One Health" approach, promote data sharing, and encourage good data stewardship to enhance the quality and application of climate and health research. match.globalchange.gov is currently being beta-tested by USG partners, and a public launch is planned for FY2013.

Climate Change Adaptation Task Force

In 2009, the Obama Administration convened the Interagency Climate Change Adaptation Task Force, cochaired by the Council on Environmental Quality (CEQ),

the Office of Science and Technology Policy (OSTP), and the National Oceanic and Atmospheric Administration (NOAA), and including representatives from more than 20 federal agencies to help the federal government strengthen policies and programs to better prepare the nation to adapt to the current and future impacts of climate change. The Climate Change Adaptation Task Force develops both the international and domestic dimensions of a federal climate change adaptation strategy which is then used to inform and align agency initiatives and activities. The Task Force integrates information on federal adaptation activities and supports the development of tools for decision makers and recommendations for future actions [22]. The 2010 Progress Report highlighted the need for crosscutting, collaborative approaches to climate change adaptation at the national level. The 2011 report provides an update on actions in key areas of federal adaptation, including building resilience in local communities, safeguarding critical natural resources such as freshwater, and providing accessible climate information and tools to help decision makers manage climate risks. Key strategies to date include:

- A *National Action Plan* for managing freshwater resources in a changing climate to assure adequate water supplies and protect water quality, human health, property, and aquatic ecosystems.
- A draft *National Ocean Policy Implementation Plan*, released in January of 2012 by the National Ocean Council, which includes a series of actions to address the Resiliency and Adaptation to Climate Change and Ocean Acidification priority objective.
- A *National Fish, Wildlife, and Plants Climate Adaptation Strategy* for safeguarding our Nation's species and natural resources. The draft Strategy was released in January of 2012 [22].

Federal Agency Adaptation Plans

On October 5, 2009, President Obama signed Executive Order 13514—*Federal Leadership in Environmental, Energy, and Economic Performance*—establishing sustainability goals for federal agency operations and directing agencies to improve their environmental, energy, and economic performance and to create agency-specific climate change adaptation plans. Section 8 (i) of the order directs federal agencies to “evaluate agency climate-change risks and vulnerabilities to manage the effects of climate change on the agency’s operations and mission in both the short and long term” [23]. The 2010 Climate Change Adaptation Task Force report provided recommendations for how federal agencies can better prepare for the impacts of climate change. Individual agencies are in the process of creating implementation plans based on these recommendations and Executive Order 13514. Agencies are required to submit their adaptation plans to the Office of Management and Budget in 2012 as a portion of their Strategic Sustainability Performance Plans. Once approved, these plans will be publically available [22, 24].

Current Federal Agency Activities in Climate Change and Health

Environmental Protection Agency

The Environmental Protection Agency's (EPA) GCRP aims to develop scientific information to help stakeholders, policy makers, and communities respond to climate change and associated impacts on human health, ecosystems, and socioeconomic systems in the USA. EPA's research focus is informed by the Agency's mission and statutory requirements and includes (1) improving the scientific understanding of global change effects on air quality, water quality, ecosystems, and human health in the context of other stressors; (2) assessing and developing adaptation options to effectively respond to global change risks, increase resilience of human and natural systems, and promote their sustainability; and (3) developing an understanding of the potential environmental impacts and benefits of greenhouse gas emission reduction strategies to support sustainable mitigation solutions. EPA's program emphasizes the integration of knowledge across the physical, chemical, biological, and social sciences into decision support frameworks that recognize the complex interactions between human and natural systems at national, regional, and local scales. Research activities include efforts to connect continental-scale temperature and precipitation changes to regional and local air quality and hydrology models to better understand the impacts of climate change on air quality and water quality and to examine how watersheds will respond to large-scale climate and other global changes to inform decisions about management of aquatic ecosystems and expand understanding of the impacts of global change. This information is leveraged by EPA Program Offices and Regions in support of mitigation and adaptation analyses, decisions, and efforts and to promote communication with external stakeholders and the public.

The EPA's National Center for Environmental Assessment's Global Change Impacts and Adaptation program assesses the options for adaptation and vulnerability of EPA's federal, regional, and tribal efforts to protect air, land, water, and human health to climate change. This information is provided to help decision makers make climate-informed policy choices and management decisions. The NCEA works with policy and decision makers to design context-appropriate scientific questions to create relevant information. The program relies on a traditional risk assessment model to understand long-term climate change.

Tool: BenMAP

EPA's BenMAP is a Geographic Information System (GIS)-based computer tool to estimate the health and economic impacts of air quality change [44]. The system runs health impact functions, which incorporate information about ambient air pollution levels, health effects estimates, baseline incidence rates of particular health

endpoints, and the exposed population. Individual users can input potential changes in air quality resulting from policy measures or climate change impacts on air pollution concentrations and calculate the changes in health impacts that would result. BenMAP has also been modified in a pilot study to estimate impacts of temperature changes on warm season heat-related mortality [45]. This provides an example of using geospatially organized data to estimate climate and weather-related health impacts.

National Aeronautics and Space Administration

The 2010 National Space Policy stated that the National Aeronautics and Space Administration (NASA) plays a crucial role in global change research and sustained monitoring capabilities and advances scientific knowledge of the global, integrated Earth system through satellite observations and development of new Earth observing satellites. These systems provide information related to climate change including solar activity, sea level rise, the temperature of the atmosphere and the oceans, the state of the ozone layer, air pollution, and changes in sea ice and land ice through observational technology [41]. As of 2007, and NASA had 17 space missions collecting climate data. NASA's activities provide critical information for climate science that can be used to understand societal impacts and consequences of climate change. The Global Climate Change and Human Health project, supported by NASA's Global Climate Change in Education program, provides an opportunity for the Institute for the Application of Geospatial Technology (IAGT) and the Center for International Earth Science Information Network to engage educators to explore climate change in the classroom through the use of innovative technologies. This project developed from existing organizational collaborations to address the need to connect science, research, and resources used by scientists to classrooms and educational environments for youth [42].

NASA's Applied Earth Sciences Program also supports research related to climate change and human health through the activities of the Health and Air Quality Program [41]. The program focuses on themes of Air Quality Planning, Forecasting and Compliance, and the crosscutting themes of Climate and Emissions Inventories while also addressing problems in infectious disease, emergency preparedness and response, and environmental impacts. The program's goal is to help determine how air quality and other key environmental factors correlate with human health for the overall goal of improving our nation's health and safety.

National Science Foundation

The NSF programs address global change issues through investments that advance frontiers of knowledge, provide state-of-the-art instrumentation and facilities, develop new analytical methods, and enable cross-disciplinary collaborations while

also cultivating a diverse highly trained workforce. As the primary funder for basic natural, social, economic, and behavioral science research in U.S. academic institutions, NSF global change programs support the research and related activities to advance fundamental understanding of physical, chemical, biological, and human systems and the interactions among them. NSF regularly collaborates with other USGCRP agencies to provide support for a range of multidisciplinary research projects and is actively engaged in a number of international partnerships.

Examples of NSF's climate change and health research efforts include the Decision Making Under Uncertainty (DMUU) Centers and the Dynamics of Coupled Human and Natural Systems Program. The NSF funds three DMUU Centers and two interdisciplinary research teams to provide government leaders, the business community, and the public with tools to make decisions about climate change with uncertain science, information gaps, and complex variability in outcomes and future projections. The Dynamics of Coupled Human and Natural Systems Program is designed to explore climate change's effects on the interactions between humans and local ecosystems. The Dynamics of Coupled Human and Natural Systems Program is a multidisciplinary program to support teams of researchers focused on the social, natural, and physical science researching the connections between human and natural systems in the context of climate change.

The NSF, in collaboration with the NIH, co-funds the Ecology and Evolution of Infectious Diseases Initiative (EEID). This multidisciplinary program supports research to understand the underlying ecological and biological mechanisms that govern relationships between human-induced environmental changes and the emergence and transmission of infectious diseases. The highly interdisciplinary research projects funded under this program apply both ecological and biomedical methods and study how environmental events such as habitat alteration, biological invasion, climate change, and pollution alter the risks of emergence and transmission of viral, parasitic, and bacterial diseases in humans and other animals. Projects are encouraged to consider how integrated environmental and biomedical approaches to infectious diseases may enhance our ability to predict and control them [46].

U.S. Agency for International Development

The U.S. Agency for International Development (USAID) Climate Change and Development Strategy, released in January 2012, calls for the agency to support countries to accelerate their transition to climate-resilient, low-emission sustainable economic development through direct programming and integrating climate change adaptation and mitigation objectives across the Agency's development portfolio including health [47]. USAID has committed to conducting internal training to promote climate change integration and foster an understanding of climate change as a crosscutting theme across its programs. As a part of these efforts, USAID is developing guidance for development practitioners from diverse sectors to understand how climate change could affect their efforts [48]. In the health sector, this includes

information to anticipate climate change impacts on health-care delivery systems and prevalence of infectious diseases such as malaria, community health, and the health of vulnerable populations [48]. USAID also supports health adaptation by building on priorities identified by least developed and small island states in their National Adaptation Programs of Action (NAPAs). As of 2010, 95 % of NAPAs identified health as a sector likely to be impacted by climate [49]. USAID's engagement and expertise in agriculture, biodiversity, infrastructure, and other critical climate sensitive sectors provide an opportunity to implement and support innovative cross-sectoral climate change programs in partner countries. USAID has long-standing relationships with host country governments that enable it to work in partnership rather than together to develop shared priorities and implementation plans. In addition, USAID supports several collaborative programs that enable decision makers to apply high-quality climate information to decision making to promote climate resilience and adaptation.

Tool: Famine Early Warning System Network

USAID's Famine Early Warning System Network (FEWS NET) monitors environmental, socioeconomic factors, and relevant hazards to detect and predict current and future food insecurity. FEWS NET provides information for 25 countries in partnership with a private contractor, USDA, U.S. Geological Survey (USGS), NOAA, and NASA. FEWS NET has expanded its activities to better monitor weather and agricultural conditions to predict potential national level climate change trends' impacts on regional food security. The recent incorporation of the NASA Land Information System allows for better use of the sparse hydroclimatic data available in many food-insecure regions [53]. FEWS NET information can be used to predict and respond to food shortages and design climate-resilient food security programs. FEWS NET data have supported a number of peer-reviewed journal articles on promoting food security in food-insecure regions of Africa that are heavily affected by climate change [52].

Tool: SERVIR

USAID and NASA co-support SERVIR, a Regional Visualization and Monitoring System that integrates satellite and ground observation data and forecast models to help developing country governments and other stakeholders prepare for and respond to environmental changes including climate change [50]. These data provide crucial information for diverse set of activities including climate change adaptation, public health, water resource management, agricultural development, and disaster response. SERVIR also offers training on the uses of geospatial data for environmental decision making [51]. SERVIR operates in collaboration with numerous U.S. and international agencies and as well as host country governments, universities, and nongovernmental organizations. As a part of SERVIR, USAID and its

partners developed the Climate Mapper tool to make the results of climate models and historical weather information available to a broad range of users. This tool provides climate change projections and landscape information into the 2050s [52]. Currently, SERVIR has three regional hubs: SERVIR-Mesoamerica, SERVIR-East Africa, and SERVIR-Himalaya [50]. Plans to expand to the SERVIR model to other regions are under development.

U.S. Department of Agriculture

The U.S. Department of Agriculture's (USDA) Global Change Research Program (GCRP) aims to empower land managers, policy makers, and federal agencies with science-based knowledge to manage the risks, challenges, and opportunities posed by climate change; reduce greenhouse gas emissions; and enhance carbon sequestration. USDA's GCRP includes contributions from the Agricultural Research Service, the National Institute of Food and Agriculture (NIFA), the Forest Service, Natural Resources Conservation Service (NRCS), National Agricultural Statistics Service (NASS), and Economic Research Service. USDA draws upon this diversity to identify climate change challenges and priorities in continuing to meet the needs of its stakeholders, decision makers, and collaborators.

The USDA conducts in-house research and extramural investigations focused on understanding climate change effects on natural and managed ecosystems, developing tools to promote adaptation, enhancing mitigation of atmospheric greenhouse gases, and providing science-based information for decision support. USDA conducts assessments and projections of climate change impacts on agricultural and natural systems and develops greenhouse gas inventories. USDA develops cultivars, cropping systems, and management practices to improve drought tolerance and build resilience to climate variability. Conservation systems promoted by the USDA integrate USGCRP research findings into farm and natural resource management and help build resiliency to climate change on both private and public lands.

U.S. Department of Commerce

National Oceanic and Atmospheric Administration

The National Oceanic and Atmospheric Administration (NOAA), under the DOC, has a strategic climate goal of "an informed society anticipating and responding to climate and its impacts" [35]. NOAA's climate change activities aim to create a predictive understanding of the changing climate system and its impacts and to communicate climate information so that people can make more informed decisions in their lives, businesses, and communities. To support this objective, NOAA

provides climate predictions and services related to climate change adaptation and mitigation to a number of federal partners including Environmental Protection Agency (EPA), U.S. Departments of Energy (DOE), U.S. Department of State (DOS), U.S. Department of Agriculture (USDA), U.S. Department of Transportation (DOT), U.S. Department of the Interior (DOI), U.S. Department of Health and U.S. Department of Human Services (HHS), U.S. Department of Homeland Security (DHS), U.S. Department of Defense (DOD), and the National Aeronautics and Space Administration (NASA). These collaborations help to identify climate risks and vulnerabilities, deliver climate-relevant information for decision making, and better inform society about climate variability, change, and their impacts [36]. NOAA implements a global observing system, focused research to understand key climate processes, improved modeling capabilities, and the development and delivery of climate educational programs and information services to support climate goals including health adaptation and mitigation.

NOAA's capabilities in linking ocean and human health as well as the agency's monitoring and prediction tools and climate science activities provide critical expertise to efforts to understand the health effects of climate change [30]. NOAA leads the OHHI, which aims to improve understanding and management of the oceans, coasts, and Great Lakes to enhance benefits to human health and reduce public health risks. As the nation's lead ocean agency, NOAA's OHHI investigates the relationship between environmental stressors, coastal conditions, and human health to maximize health benefits from the ocean, improve the safety of seafood and drinking waters, reduce beach closures, and detect emerging health threats [37]. Many of the OHHI-supported initiatives have a climate focus, including 2 of the 3 OHHI Centers of Excellence (collaboration with NSF & NIEHS), 3 of 27 external grants, and 3 of 5 consortia for Graduate Training Programs [38].

National Oceanic and Atmospheric Administration and Centers for Disease Control Memorandum of Understanding

In October 2011, NOAA and CDC/Agency for Toxic Substances and Disease Registry (ATSDR) signed a Memorandum of Understanding (MOU) to strengthen collaboration in support of science and services to understand, communicate, and reduce environmental and public health impacts. Recognizing the mutual interests of NOAA and CDC, the MOU's cooperative framework supports a "One Health" approach to collaboration that will provide critical information and activities to inform decision and policy making, reduce public health threats of global change, and support climate change adaptation [39]. Activities will utilize shared technologies and infrastructure to enhance the accuracy, timeliness, and integrated application of climate, water, weather, oceanographic, ocean-related marine animal, human health, and ecosystem resource data to address public health issues.

Specific areas of collaboration include (1) scientific research; (2) services (diagnostic testing, prediction, and forecasting); (3) communication and information

dissemination; (4) integrated data and surveillance; (5) education, training, and capacity building; (6) workshops and meetings; (7) pilot projects and joint field projects; and (8) global health and local capacity development. These activities are designed to promote better integration of earth observation and surveillance data and the use of atmospheric, oceanographic, and hydrographic data to map, model, predict, and communicate public health impacts. The MOU, which runs through September 2017, will allow NOAA and CDC to better exchange, integrate, and leverage expertise to address existing and emerging public health threats including climate change.

National Institute of Standards and Technology

The DOC's National Institute of Standards and Technology (NIST) partners with industry, government, and academia to improve environmental science tools and measurement capability with climate and health applications. Relevant activities include calibrating the sensors of climate-mapping satellites and detecting quantities of toxins and pollutants in the air, soil, and water with more precision [40].

U.S. Department of Health and Human Services

The mission of the U.S. Department of Health and Human Services (HHS) is to enhance the health and well-being of all Americans by providing for effective health and human services and by fostering sound, sustained advances in the sciences underlying medicine, public health, and social services. HHS supports a broad portfolio of research and decision support initiatives related to environmental health and the health effects of global climate change. The NIH and the CDC provide the focus for this effort.

HHS supports all four goal areas of the GCRP: Advance Science, Inform Decisions, Conduct Sustained Assessments, and Communicate and Educate. By conducting fundamental and applied research on the linkages between climate change and health, translating scientific advances into decision support tools for public health professionals, conducting ongoing monitoring and surveillance of climate-related health outcomes, and disseminating scientific information and engaging the public health community in two-way communication, HHS provides a model of the "end to end" science paradigm the GCRP seeks to achieve.

The NIH's National Institute of Environmental Health Sciences (NIEHS) and CDC cochair (along with NOAA) the USGCRP's Climate Change and Human Health Interagency Working Group of the USGCRP. In addition, both NIEHS and CDC support the NCA, which seeks to provide the scientific information that can be used by communities around the country to effectively plan for adaptation and mitigation.

Centers for Disease Control

Through interdisciplinary work with local and state health departments, communities, research institutions, and other federal agencies, the Centers for Disease Control (CDC) identifies vulnerable populations, prevents and adapts to current and future health impacts, and supports the creation of detection systems to inform and respond to current and emerging health threats. These efforts are led by the CDC Climate and Health program, established in 2006, to prevent and adapt to the anticipated health impacts associated with climate change. The program's three core functions include (1) translating climate change science to inform states, communities, and local public health departments; (2) creating decision support tools to increase local capacity to respond to climate change; and (3) serving as the leader for planning for climate change-related public health impacts [25].

CDC Climate-Ready States and Cities Initiative

The CDC Climate and Health Program's Climate-Ready States and Cities Initiative provides technical assistance to ten states and cities around the country to apply climate science to better predict, prepare for, and adapt to current and potential climate change health impacts [26]. This initiative brings cities and states together with local and national climate change scientists to understand their region's potential climate changes. Funding for the Climate-Ready States and Cities is divided into two categories: (1) Assessment and Planning to Develop Climate Change Programs and (2) Building Capacity to Implement Climate Change Programs and Adaptations. Recipients of the first funding-stream prepare needs assessments, gap analyses, and strategic plans to address climate change impacts on health in the short and long term using the ten Essential Public Health Services framework. The second funding-stream supports local health departments to conduct health impact assessments and to increase the capacity of state and local governments to respond to the human health impacts of climate change.

CDC National Environmental Public Health Tracking Network and Climate Change

Established in 2006, The CDC National Environmental Public Health Tracking Network (Tracking Network) integrates health, exposure, and hazard information from various national, state, and local sources [27]. The Network involves multidisciplinary collaborations to collect, integrate, analyze, and distribute information derived from environmental hazard monitoring, human exposure surveillance, and health effects surveillance. The Network tracks relevant climate change and health data to help scientists and decision makers understand the connections between environmental conditions (and changes) and health impacts [28]. The Network focuses its climate change indicators on extreme heat with the aim of evaluating the

number of heat-related deaths at the national level. Information on heat vulnerabilities, heat mortality, and temperature distribution can be used to identify patterns in extreme weather and their health effects [29]. For example, the Network can track the effects of a heat wave by aggregating and reporting the number of health conditions and reported deaths from local health departments and hospitals. These data can be used by federal and local policy makers to identify high-risk populations and communities, understand trends in heat-related deaths, and inform adaptation strategies. The information produced by the Tracking Network is used by national, state, and local public health agencies to make decisions to protect human health in a timely and accurate manner.

National Institutes of Health

The NIH supports a large research portfolio relevant to the human health impacts of climate change, including research related to direct health impacts of increased temperatures and extreme weather events, the health effects of air pollution and aeroallergens, water quality and quantity, ecosystem influences on infectious disease transmission, and potential health effects of materials used in new technologies to mitigate or adapt to climate change.

At the NIH, the National Institute of Environmental Health Sciences (NIEHS) and the Fogarty International Center (FIC) cochair the Trans-NIH Climate Change Workgroup. The Workgroup aims to coordinate and promote climate change and health research at NIH. Shortly after its founding in 2009, the Workgroup conducted a portfolio analysis of climate change research at NIH to identify gaps and prioritize research needs [30].

Members of the NIH Workgroup, in collaboration with interagency colleagues from NOAA, U.S. Environmental Protection Agency, CDC, U.S. Department of Agriculture, U.S. Department of State, U.S. GCRP, and the White House OSTP, drafted the 2010 report, “A Human Health Perspective on Climate Change” [31]. The interagency author team outlined the research priorities for climate change and human health related to 11 categories of health outcomes and exposures [32]:

- Asthma, Respiratory Allergies, and Airway Diseases
- Cancer
- Cardiovascular Disease and Stroke
- Foodborne Disease and Nutrition
- Heat-Related Morbidity and Mortality
- Human Developmental Effects
- Mental Health and Stress-Related Disorders
- Neurological Diseases and Disorders
- Vectorborne and Zoonotic Diseases
- Waterborne Diseases
- Weather-Related Morbidity and Mortality

National Institutes of Health Climate Change and Health Grant Program

Based on this analysis, the NIEHS, in collaboration with ten other NIH institutes and centers, funded the Climate Change and Health: Assessing and Modeling Population Vulnerability to Climate Change grant program in 2010 [33]. The Climate Change and Health Grant Program funds research to understand risk factors that increase health vulnerability to climate change and its manifestations, including changes in environmental exposures (air pollution, toxic substances), changing weather patterns (increasing frequency and severity of extreme weather events and rising average temperatures), as well as the impacts of climate change mitigation and adaptation strategies. The research also explores the effect of a changing climate on common diseases, including asthma and cardiovascular disease and stroke.

The program supports the development of tools, models, and methods to better predict the health consequences of climate change and to understand the dimensions populations both in the USA and globally which are most vulnerable to the negative health consequences of climate change. This research will provide better tools to decision makers involved in protecting the health of particularly vulnerable populations, including communities with low socioeconomic status, the elderly, pregnant women, and other populations with increased risk. Ultimately, the research from this program is intended to inform climate change adaptation strategies and guide public health interventions to reduce current and future harms to the most vulnerable communities. The first round of nine grantees was announced in October 2011 (Table 19.1) and two additional rounds of awards are planned through 2013 [34]. Funding for this program runs through 2014.

National Institute of Environmental Health Sciences Climate Change and Health Program

At NIEHS, the Climate Change and Human Health Program provides leadership for a variety of NIEHS-supported research and initiatives as well as trans-NIH and interdepartmental coordination of climate change and human health activities. The goals of the program include:

- Provide research on human health impacts related to climate change and adaptation
- Raise awareness and create new partnerships to advance key areas of health research and knowledge development on human health effects of climate change
- Serve as an authoritative source of information on human health effects of climate change for NIEHS stakeholders, including the public
- Represent NIEHS science in climate change research and policy activities at the NIH, HHS, federal government, and international levels

The program partners with other government agencies, the academic community, NGOs, and international organizations to identify research gaps, support ongoing investigations, and communicate health impacts of climate change to key

Table 19.1 NIH climate change and health: assessing and modeling population vulnerability to climate change-funded projects

Investigator	Institution	Research summary	Funding institute or center
Ralph Delfino, M.D., Ph.D.	University of California, Irvine	Identify populations of children with asthma most vulnerable to air pollutants that are expected to increase with climate change	NIEHS
Julia Gohlke, Ph.D.	University of Alabama at Birmingham	Determine whether significant differences in vulnerability to heat-related health impacts exist between urban and rural communities	NIEHS
Karen Levy, Ph.D.	Emory University, Atlanta	Examine the impact of current and projected climate variables on the incidence of gastrointestinal disease in Ecuador, for use as a model system to help determine the importance of social factors and infrastructure availability in preventing gastrointestinal disease globally	FIC
Jonathan Patz, M.D.	University of Wisconsin—Madison	Develop models that factor in climate, air quality, power plant emissions, and health models to determine which populations will be most exposed to air pollution-related health risks	NIEHS
Roger Peng, Ph.D.	Johns Hopkins University, Baltimore	Quantify the effects of biological, environmental, and socioeconomic factors that make people more vulnerable to extreme heat	NIEHS
Joel Schwartz, Ph.D.	Harvard University, Cambridge, MA	Examine the impact of changing weather patterns, such as temperature, humidity, and barometric pressure, on the elderly, as observed through changes in blood pressure, inflammation, lung function, and related health outcomes	NIA
		Identify medical and other individual characteristics that put people at increased risk of dying due to weather, and determine air pollution impacts that contribute to those risks	NIEHS
Antonella Zanobetti, Ph.D.	Harvard University	Define and forecast high-risk days given pollution and climatic conditions, to help determine how reduction in pollution or improvement in climatic conditions could improve cardiovascular and cerebrovascular health	NIEHS
Ying Zhou, Sc.D.	Emory University	Develop models to identify vulnerable geographical locations with increased health impacts due to heat waves and air pollution exposures	NIEHS

decision makers. At the international level, the program participates in activities related to the United Nations Framework Convention on Climate Change (UNFCCC) processes and Group on Earth Observations (GEO) Health and Environment Community of Practice.

In addition, the NIEHS Climate Change and Health Program uses education and outreach to communicate the health impacts of climate change and encourage dialogue among a diverse set of stakeholders. In June 2012, the program cohosted with the EPA a weeklong training for high school students in North Carolina about the science and impacts of climate change. During the 2011 American Public Health Association's 2011 annual meeting, the program led an Interagency Climate Communication Learning Institute to teach participants how to effectively communicate the human health impacts of climate change.

References

1. Public Law 95–367: The National Climate Act. (15 U.S.C. 2901, 2908), 9/17/78). Library of Congress Thomas. <http://thomas.loc.gov/cgi-bin/bdquery/z?d095:H.R.6669>. Accessed 29 Aug 2012.
2. Pielke RA. The policy history of the US Global Change Research Program: Part I. Administrative development. *Global environmental change*, Vol 10. 2000. p. 12. <http://foehn.colorado.edu/nome/HARC/Readings/Pielke2.pdf>. Accessed 1 May 2012.
3. The United States Global Change Research Program. http://www.nap.edu/openbook.php?record_id=12782&page=493. Accessed May 2012.
4. GAO (General Accounting Office). Administration approach cautious pending validation of threat. Report to the Chairman, Subcommittee on Oversight and Investigations. Committee on Energy and Commerce, House of Representatives, NSIAD-90-63, Jan, US GPO, WA, DC. p. 16. <http://www.gao.gov/assets/150/148577.pdf> (1990).
5. Pielke RA. The policy history of the US Global Change Research Program: Part II. Legislative process. *Global environmental change*, Vol 10. 2000. p. 136. <http://nome.colorado.edu/HARC/Readings/Pielke3.pdf>. Accessed 1 May 2012.
6. Pielke RA. The development of the U.S. Global Change Research Program: 1987 to 1994. Prepared for American meteorological society policy symposium. 3–12 June 2001. Washington, DC. p. 8. <http://www.ametsoc.org/atmospolicy/documents/AMSUSGCRPCase5-15-01bis.pdf>. Accessed 1 May 2012.
7. Committee on the Human Dimensions of Global Change and Committee on Global Change Research, National Research Council (NRC). *Human dimensions of global environmental change: research pathways for the next decade*. Washington, DC: The National Academies Press; 1999. http://www.nap.edu/openbook.php?record_id=9641&page=R2. Accessed 29 Aug 2012.
8. National Research Council. *Climate Change Science: An Analysis of Some Key Questions*. Washington, DC: The National Academies Press, 2001. p. 24. http://www.nap.edu/catalog.php?record_id=10139 Accessed 1 May 2012.
9. The US Global Change Research Program (USGCRP). *Our changing planet 2010*. Washington, DC; 2010. p. 7. *Our changing planet 2010*. Accessed 1 May 2012.
10. Committee on the Human Dimensions of Global Change. *Science priorities for the human dimensions of global change*. Washington, DC: The National Academies Press; 1994. p. 3: http://www.nap.edu/openbook.php?record_id=9175&page=3. Accessed 1 May 2012.
11. The US Global Change Research Program (USGCRP). *Our changing planet 2010*. Washington, DC; 2010. p. 4. *Our changing planet 2010*. <http://downloads.globalchange.gov/ocp/ocp2010/ocp2010.pdf>. Accessed 1 May 2012.

12. The US Global Change Program Website. <http://www.globalchange.gov/>. Accessed 1 May 2012.
13. The US Global Change Research Program (USGCRP). USGCRP strategic plan. Washington, DC; 2012. p. xiv. <http://downloads.globalchange.gov/strategic-plan/2012/usgcrp-strategic-plan-2012.pdf>. Accessed 1 May 2012.
14. ***The US Global Change Program Website Program History. <http://globalchange.gov/about/program-structure/program-history>. Accessed 1 May 2012.
15. The US Global Change Research Program (USGCRP). USGCRP strategic plan. Washington, DC; 2012. p. 102. <http://downloads.globalchange.gov/strategic-plan/2012/usgcrp-strategic-plan-2012.pdf>. Accessed 1 May 2012.
16. Committee on the Human Dimensions of Global Change. Science priorities for the Human dimensions of global change. Washington, DC: The National Academies Press; 1994. p. 496. http://www.nap.edu/openbook.php?record_id=12782&page=496. Accessed 1 May 2012.
17. The US Global Change Program Website. <http://globalchange.gov/publications/our-changing-planet-ocp>. Accessed 1 June 2012.
18. The US Global Change Program Website. What we do—interagency crosscutting group on climate and health. <http://www.globalchange.gov/what-we-do/climate-change-health>. Accessed 1 May 2012.
19. The US Global Change Research Program (USGCRP). Our changing planet 2010. Washington, DC; 2010. p. 38. Our changing planet 2010. <http://downloads.globalchange.gov/ocp/ocp2010/ocp2010.pdf>. Accessed 1 May 2012.
20. The US Global Change Research Program (USGCRP). USGCRP strategic plan. Washington, DC; 2012. p. 38, 40 & 69. <http://downloads.globalchange.gov/strategic-plan/2012/usgcrp-strategic-plan-2012.pdf>. Accessed 1 May 2012.
21. GCRP Newsletter. Paul Schramm, CDC/ONDIEH/NCEH.
22. <http://www.whitehouse.gov/administration/eop/ceq/initiatives/adaptation>
23. Executive Order 13514. 52122 Federal Register, Vol. 74, No. 194, Thursday, 8 Oct 2009/ Presidential documents. <http://www.gpo.gov/fdsys/pkg/FR-2009-10-08/pdf/E9-24518.pdf>. Accessed 31 May 2012.
24. Program activity descriptions adapted from The US Global Change Research Program (USGCRP) 2012 strategic plan. The US Global Change Research Program (USGCRP). USGCRP strategic plan. Washington, DC; 2012. <http://downloads.globalchange.gov/strategic-plan/2012/usgcrp-strategic-plan-2012.pdf>. Accessed 1 May 2012.
25. The CDC's Climate and Health Program, About our Program. <http://www.cdc.gov/climatechange/about.htm>. Accessed 1 May 2012.
26. The CDC's Climate Ready States and Cities Program. http://www.cdc.gov/climatechange/climate_ready.htm. Accessed 1 May 2012.
27. The CDC. National Environmental Public Health Tracking Network. <http://ephtracking.cdc.gov/showHome.action>. Accessed 1 May 2012.
28. The CDC Tracking Climate Change. <http://ephtracking.cdc.gov/showClimateChangeTracking.action>. Accessed 1 May 2012.
29. The CDC. Climate Change Indicators Available on the Environmental Public Health Tracking Network. <http://ephtracking.cdc.gov/showClimateChangeIndicators.action>. Accessed 1 June 2012.
30. Jessup CM, Balbus JM, Christian C, Haque E, Howe SE, Newton SA, Reid BC, Roberts L, Wilhelm E, Rosenthal JP. Climate change, human health, and biomedical research: analysis of the National Institutes of Health research portfolio. *Environ Health Perspect*. 2013 Apr;121(4):399-404. doi: 10.1289/ehp.1104518. Epub 2013 Jan 17. PubMed PMID: 23552460; PubMed Central PMCID: PMC3620768.
31. NOAA Website. "Vital New Roadmap" Underscores Need to Study Climate Change, Human Health Links. http://www.noanews.noaa.gov/stories2010/20100422_climatehealth.html. Accessed 29 Aug 2012.
32. <http://www.cdc.gov/climateandhealth/effects/default.htm>
33. Ball E. NIH announces climate change and health funding. *Environmental factor*. NIEHS. 2010. <http://niehs.nih.gov/news/newsletter/2010/september/spotlight-nih.cfm>. Accessed 1 May 2012.

34. NIEHS Grantees Human Health Impacts of Climate Change. <http://www.niehs.nih.gov/research/supported/programs/climate/grantees/Grantees>. Accessed 1 May 2012.
35. The US Global Change Research Program (USGCRP). USGCRP strategic plan. Washington, DC; 2012. p. 110. <http://downloads.globalchange.gov/strategic-plan/2012/usgcrp-strategic-plan-2012.pdf>. Accessed 1 May 2012.
36. NOAA Office of Program Planning and Integration Website. <http://www.ppi.noaa.gov/goals/>. Accessed 29 Aug 2012.
37. NOAA Oceans and Human Health Initiative (OHHI). <http://oceansandhumanhealth.noaa.gov/about/>. Accessed 1 May 2012.
38. NOAA's Role Perspective on Climate Change and Health Presentation. <http://iom.edu/~media/Files/Activity%20Files/Environment/EnvironmentalHealthRT/RowlesHealthResearchAgendaForClimateChange.pdf>. Accessed 1 May 2012.
39. Memorandum of Understanding Between the U.S. Department of Commerce National Oceanic and Atmospheric Administration and the Department of Health and Human Services Centers for Disease Control and Prevention for Environment and Public Health Impacts. NOS Agreement Code: MOA-2001-069/8371. Oct 2011.
40. NIST Website. Environment and climate portal overview portal. <http://www.nist.gov/environment-climate-portal.cfm>. Accessed 29 Aug 2012.
41. NASA Global Climate Change Website. <http://climate.nasa.gov/NasaRole/>. Accessed 30 Aug 2012.
42. NASA Climate Change and Human Health Project. <http://www.climatechangehumanhealth.org/aboutus/>. Accessed 1 May 2012.
43. NASA Applied Science Program Website. <http://appliedsciences.nasa.gov/health-air.html>. Accessed 23 Sept 2012.
44. EPA Environmental Benefits Mapping and Analysis Program. <http://www.epa.gov/air/ben-map/>. Accessed 1 May 2012.
45. Voorhees AS, et al. Climate change-related temperature impacts on warm season heat mortality: a proof-of-concept methodology using BenMAP. *Environ Sci Technol*. 2011;45(4):1450–7.
46. Fogarty International Center Ecology and Infectious Disease Program. <http://www.fic.nih.gov/Programs/Pages/ecology-infectious-diseases.aspx>. Accessed 1 May 2012.
47. USAID Climate and Development Strategy. 2012. <http://www.usaid.gov/content/global-climate-change/usaid-global-climate-change-and-development-strategy>. Accessed 29 Aug 2012.
48. USAID Website. Global climate change adaptation sectors. <http://www.usaid.gov/what-we-do/environment-and-global-climate-change/global-climate-change-adaptation/global-climate-change-adaptation-sectors>. Accessed 30 Aug 2012.
49. Manga L, Bagayoko M, Meredith T, Neira M. Overview of health considerations within National Adaptation Programmes of Action for climate change in least developed countries and small island states. Geneva: World Health Organization, Department of Public Health and Environment. 2010. http://www.who.int/phe/Health_in_NAPAs_final.pdf. Accessed 27 Aug 2012.
50. NASA Earth Science Applied Sciences Division. 2011 annual report. p. 37. 2012. <http://appliedsciences.nasa.gov/pdf/AppliedSciences2011AnnualReport.pdf>. Accessed 27 Aug 2012.
51. NASA Earth Science Applied Sciences Division. 2011 annual report. p. 38. Aug 2012. <http://appliedsciences.nasa.gov/pdf/AppliedSciences2011AnnualReport.pdf>. Accessed 27 Aug 2012.
52. USAID Climate Change Pillar: Adaptation. http://www.usaid.gov/our_work/environment/climate/policies_prog/adaptation.html. Accessed 1 May 2012.
53. NASA Earth Science Applied Sciences Division. 2011 annual report. p. 30. Aug 2012. <http://appliedsciences.nasa.gov/pdf/AppliedSciences2011AnnualReport.pdf>. Accessed 27 Aug 2012.

Chapter 20

Management of Climate Change Adaptation at the United States Centers for Disease Control and Prevention

Jeremy J. Hess, Gino Marinucci, Paul J. Schramm, Arie Manangan, and George Luber

Abstract As the nation's public health agency, CDC recognizes that climate change poses a multifaceted and potentially significant threat to domestic public health. To facilitate climate change preparedness in public health, the agency developed the Climate and Health Program, which is housed in the National Center for Environmental Health. The Program's mission is to translate science for public health partners, develop decision support tools to facilitate climate change adaptation in public health, and to serve as a credible leader in planning for the human health impacts of a changing climate. Since its formation, the Program has worked to articulate a public health approach to climate change and integrate science from public health and other sectors to facilitate public health adaptation efforts. The Program has developed an adaptive management framework for public health, the BRACE framework, and is working cooperatively with several state and local health departments to pursue an evidence-based approach to climate change adaptation. As public health's expertise and experience grows, the Climate and Health Program will work to continue disseminating relevant information for the increasing number of public health practitioners focused on reducing the adverse health effects of climate change.

Keywords Climate change and the CDC • CDC policies on climate change adaptation • Public health policy on climate change • Climate change adaptation and public policy • Climate and Health Program

J.J. Hess, M.D., M.P.H., F.A.C.E.P. (✉)
Emory Schools of Medicine and Public Health, Atlanta, GA, USA

Climate and Health Program, NCEH, CDC, Atlanta, GA, USA
e-mail: jhess@emory.edu

G. Marinucci, M.P.H. • P.J. Schramm, M.S., M.P.H. • A. Manangan, M.A. • G. Luber, Ph.D.
Climate and Health Program, Division of Environmental Hazards and Health Effects,
National Center for Environmental Health, Centers for Disease Control and Prevention,
Atlanta, GA, USA

The Centers for Disease Control and Prevention (CDC) is the nation's public health agency. A part of the Department for Health and Human Services, the CDC's mission is "[c]ollaboration to create the expertise, information, and tools that people and communities need to protect their health—through health promotion, prevention of disease, injury and disability, and preparedness for new health threats" [1]. In recent years, climate change has emerged as a significant potential public health threat, and the CDC has initiated a range of efforts to facilitate adaptation to climate change in the public health sector. Climate change is expected to have a wide range of health impacts [2–4], and a range of public health expertise will be required to adapt to it [5]. To facilitate leadership on the issue, CDC's climate change efforts have been housed primarily in the Climate and Health Program in the National Center for Environmental Health (NCEH), though the Program collaborates closely with several intramural and extramural partners. In general the Program has focused primarily on domestic efforts, in keeping with the CDC's general focus on supporting state and local public health partners. Here we provide an overview of CDC's efforts, including an overview of the Climate and Health Program, an outline of CDC's conceptual approach to the integration of climate change adaptation into public health programming, its adaptation framework Building Resilience Against Climate Effects (BRACE), and activities it has supported through its Climate-Ready States and Cities Initiative (CRSCI), including recent advances in climate and health science presented at the CDC's annual science symposium on climate and health. We close with brief consideration of future adaptation needs and CDC's plans for addressing ongoing needs.

Overview of CDC'S Climate and Health Program

The CDC's Climate and Health Program (the Program) serves as the primary hub of climate change adaptation activities at CDC. The Program, which is housed within the NCEH, was formed in 2006 and began receiving specific Congressional appropriations in 2009. The Program seeks to identify populations most vulnerable to the impacts of climate change, anticipate future climate and associated disease trends, assure that systems are in place to detect and respond to emerging health threats, and take steps to assure that these health risks can be managed now and in the future.

In pursuit of these goals, the Program serves three core functions in support of public health adaptation:

1. Translating climate science to inform public health practitioners
2. Developing decision support tools to enhance preparedness
3. Serving as a credible leader in planning for the human health impacts of a changing climate

The Program works with other parts of CDC to track data on environmental conditions, disease risks, and disease occurrence related to climate change. The Program also collaborates with other Federal agencies such as National Oceanic and

Atmospheric Association (NOAA) and National Aeronautics and Space Administration (NASA) and has participated in both the United States National Climate Assessment (NCA) and the Intergovernmental Panel on Climate Change (IPCC). Finally, the bulk of the Program's efforts go to supporting state and local governments in support of their climate change adaptation activities, principally through the CRSCI.

Like other efforts to anticipate and address the public health effects of climate change, the CDC Climate and Health Program was initially faced with the challenge of determining how public health should approach the problem given its broad set of projected impacts, varying time scales, and impact on complex systems, many of which are outside public health's direct control (e.g., agricultural systems and systems for maintaining critical infrastructure). From its inception the Program has thus invested in efforts to clarify and define the public health threats associated with climate change and to integrate adaptation activities within existing public health programming. This investment led to a landmark publication, "Climate Change: The Public Health Response," which outlined adaptation needs using the Ten Essential Public Health Services (EPHS) framework [5], as well as a paper on using adaptive management, an iterative, modeling-based approach, to guide adaptation efforts [6]. Next we will examine the issue of adaptive management and its role in climate change adaptation in greater detail.

Adaptive Management and Its Role

Adaptive management is an iterative, cyclic approach to designing, implementing, and evaluating interventions in complex adaptive systems [7]. Such systems are typically incompletely understood and exhibit some unexpected behaviors in response to management interventions; ecosystems are a frequently cited example. Importantly, an important aspect of managing these systems is the ongoing need to learn about their behavior, particularly in response to management interventions and shifting stressors over time. Evidence indicates that adaptive management better accommodates these needs than other approaches which tend not to actively address the dynamic nature of such systems. It relies heavily on systems modeling and explicitly emphasizes learning at each stage of the process.

As codified by the National Research Council in 2004, settings in which adaptive management may be a useful approach have six major elements:

1. Management objectives that are regularly revisited and revised
2. A model of the system(s) being managed
3. A range of management choices
4. Monitoring and evaluation of outcomes
5. Mechanisms for incorporating learning into future decisions
6. A collaborative structure for stakeholder participation and learning [8]

Increasingly, adaptive management has been touted as a useful approach for managing the health effects of climate change [6,9,10]. Climate change is impacting

a wide range of sectors and associated systems, from natural ecosystems upon which native peoples rely for food [11] to intensively managed socio-ecosystems such as urban environments in which people can be exposed to a range of climatic hazards, from heat to air pollution [12]. While many of these systems are not directly under the purview of the public health sector, in all cases public health can be considered a stakeholder (e.g., the electrical power grid, on which people rely heavily to power mechanical air conditioning, is highly pertinent to public health but managed by electrical utilities and their regulators, and emissions from fossil fuel combustion to generate electricity have significant public health impacts), and many of these systems satisfy all the criteria listed above.

Because climate change is likely to amplify stresses on certain systems essential to maintaining public health, it will be increasingly important for public health organizations to have the capacity to manage these systems as both the systems, the stressors, and management objectives evolve. In an effort to develop adaptive management expertise among its state and local public health partners, the CDC has developed a flexible approach that public health partners could choose to adapt in order to facilitate local public health adaptation to climate change entitled BRACE: Building Resilience Against Climate Effects.

Building Resilience Against Climate Effects (BRACE)

The changing climate presents a novel type of public health challenge in which assumptions based on historical climatic and meteorologic patterns and their impacts on risks for climate-sensitive health outcomes must be, at the very least, revisited. In the United States, with its federalist structure and decentralized public health system, there is a diverse arrangement of public health organizations at the state and local level, and much public health programming is locally developed and implemented. Risk assessment using anticipated future disease burden, particularly formal assessment involving projections of climate-sensitive health outcomes, is not a familiar exercise for many local public health agencies [13], and many health departments feel unready to meet the related challenges with their existing resources [14]. In a 2008 survey, health departments also indicated concern that the CDC did not have adequate expertise to facilitate their climate change preparedness efforts [13].

To address this gap in domestic public health preparedness for the health impacts of climate change, CDC has built up its climate and health expertise and initiated several programs to support state and local public health partners in building their capacity and pursuing their adaptation efforts. To ensure that states had adequate available guidance regarding climate change adaptation, CDC developed a framework entitled BRACE [15]. The BRACE framework incorporates vulnerability assessment using climate projections, modeling of projected health impacts, evidence-based evaluation of intervention options, intervention implementation, and systematic evaluation of all activities in an iterative framework that incorporates the principles of adaptive management. Once several states have implemented

BRACE, the results of implementation on adaptation activities will also be evaluated.

BRACE is a five-step process that enables a health department to incorporate the best available atmospheric science into climate-health impact projections for its jurisdiction. The BRACE framework involves health departments coupling retrospectively derived response functions describing associations between weather variables and health outcomes—preferably response functions derived from data on populations within their jurisdiction—with projected atmospheric data from global circulation models. These projections are then coupled with the response functions to project future disease burdens which can be used to facilitate planning and preparedness activities.

There are already frameworks for performing vulnerability assessments related to climate change and health [16] and comparative risk assessments [17]. BRACE is not designed to supplant or supersede this guidance. Instead, BRACE was designed to present these concepts in a structure that is relatively familiar for US health departments to emphasize that the underlying process of risk assessment, identification of appropriate interventions, intervention implementation, and evaluation is similar to that used successfully in public health for decades. The main departures from a more conventional approach are in the use of climate change impact projections for risk assessment and the strong emphasis on broad stakeholder engagement, learning, modeling, and iterative decision making that are hallmarks of adaptive management.

The Five Steps of BRACE

There are five sequential steps in the BRACE Framework:

Step 1: *Anticipating climate impacts and assessing vulnerabilities*, in which a health department identifies the scope of the most likely climate impacts, the potential health outcomes associated with those climatic changes, and the populations and locations vulnerable to these health impacts within its jurisdiction.

Step 2: *Projecting the disease burden*, in which a health department, as best as possible, estimates or quantifies the additional burden of health outcomes due to climate change—to support prioritization and decision making.

Step 3: *Assessing public health interventions*, in which a health department seeks to identify the most suitable health interventions for the health impacts of greatest concern.

Step 4: *Developing and implementing a climate and health adaptation plan*, in which a health department develops and implements a health adaptation plan for climate change that addresses health impacts and gaps in critical public health functions and services, and prepares a jurisdiction to enhance its adaptive capacity.

Step 5: *Evaluating impact and improving quality of activities*, in which a health department can evaluate the processes it has used, determine the value of utilizing the framework, and the value of climate and health activities undertaken. This step

is also important for quality improvement and for incorporating refined inputs such as updated data or new information, an essential component of adaptive management.

There are some key points to consider in the implementation of the BRACE framework. First, stakeholder engagement is very important throughout the process. A targeted selection of stakeholders can add significant value to the process overall, and specific stakeholders may be particularly important at specific points in the process. For example, in step 1, where much of the emphasis is understanding climate projections, a health department may profit significantly from engagement with their state climatologist and others in the climate science community, whereas in step 3, where an assessment is being made of the appropriateness of different public health interventions, it may be appropriate to solicit input from the larger public health practitioner and affected communities.

The second key consideration is that, while the BRACE framework lays out a comprehensive, sequential approach, it is flexible in that it allows the integration of prior analysis. Steps 1 through 3 focus on providing new or enhanced information that can aid a health department when making decisions on investments and program or operational changes. At any point from step 1 to step 3, a jurisdiction may have sufficient information based on prior analyses to make decisions without undertaking parts of the step. While the BRACE framework allows for the application of prior analyses and information, it is paramount that these inputs be vetted as providing the most up-to-date, available information regarding climate-related risk.

We have laid out each step in depth below. To frame the activities a health department would undertake in each step, we start each section with framing questions that highlight the lines of inquiry driving that specific step in the process. Table 20.1 illustrates how the first three steps of BRACE have been applied to the issue of extreme heat vulnerability by the New York City Department of Health and Mental Hygiene.

Step 1: Anticipating Climate Impacts and Assessing Vulnerabilities

In general terms, what will the climate look like in my jurisdiction in 10, 25, and 50 years?

How are the population profile and the profile of public health challenges likely to change in my jurisdiction at these intervals?

How might the anticipated changes in climate interact with these demographic and other challenges to shift population health risk and place vulnerabilities?

The goal of this first step in BRACE is to identify the range of climate impacts, associated potential health outcomes, vulnerable populations, and locations of potentially vulnerable populations within a health department's jurisdiction. In step 1 a health department works toward establishing a functional understanding of how the climate is changing in its jurisdiction, the likely associated effects on health, and the populations and systems most vulnerable to these changes. To carry out this step, health department personnel will rely on public health and medical literature,

Table 20.1 How the first three steps of BRACE have been applied to the issue of extreme heat vulnerability by the New York City Department of Health and Mental Hygiene

1. The initial step of BRACE is to assess public health vulnerabilities to climate change. To accomplish this, NYC Health utilized information in the NYC Panel on Climate Change Report (2009) detailing current and future trends in heat waves and other hazardous weather-related events. NYC Health conducted an epidemiologic analysis using vital statistics to identify subpopulations at the greatest risk for heat stroke and then mapped the distribution of heat vulnerability in the city's boroughs
2. Step 2 of BRACE involves projecting the burden of disease in a changing climate. NYC Health conducted a retrospective analysis to determine the relationship between temperature and mortality and then used global circulation model outputs to project future heat-related mortality in 2020. Their analysis showed that, all things being equal, there would be an increase in heat-related deaths
3. Step 3 of the BRACE frameworks is an assessment to determine the most effective and suitable public health interventions. NYC Health conducted a heat-health behavior survey to determine air conditioning (AC) prevalence and usage, assess behaviors of high-risk groups during hot weather, and gauge public awareness of heat warnings. They found approximately 700,000 New Yorkers were without functioning AC and approximately 550,000 were particularly vulnerable to heat illness (i.e., no functioning AC, age >65 years old, and living with underlying chronic health conditions). About half of this population stayed at home during hot weather. Survey findings suggested that the most vulnerable populations may not understand their true risk and outreach should focus on conveying the importance of AC use and the potential lethality of both outdoor and indoor heat exposure
4. Step 4 of BRACE is the development and implementation of a climate and health adaptation plan, which is a set of public health interventions aimed to reduce the adverse health effects resulting from climate-related hazards. In an effort to prevent heat-related illness, NYC Health prepared public health messaging and materials to better convey the risk of heat stress and improved active outreach to those most vulnerable. Additionally, they implemented plans to increase access to AC to specific vulnerable areas. The health interventions put forth by NYC Health will be included in the overarching climate adaption plan for city of New York to ensure that health is an essential component
5. Step 5 of BRACE is the evaluation of the effectiveness of the climate adaptation plan and specific interventions. NYC Health is currently in the process of conducting process, outcome, and impact evaluations of its climate change adaptation planning efforts

New York City's Department of Health and Mental Hygiene (NYC Health) is a CDC Climate-Ready States and Cities Initiative (CRSCI) grantee. NYC Health has used the grant support to assess the potential increase in heat-related illness resulting from climate change. Its efforts serve as an excellent case study on how a health department can build resilience against the health effects of climate, outlined here in terms of the Building Resilience Against Climate Effects (BRACE) framework developed by the CDC's National Center for Environmental Health (NCEH)

expert experience, and academic and or governmental partners with expertise in atmospheric science and modeling to gain an understanding of relevant climate-health burdens and projected climatic shifts.

Step 1 is both an exploratory exercise and scoping activity. It is exploratory in the sense that health departments must first work with partners (e.g., the state climatologist) to understand how climate and health have been and are likely to be related in their jurisdictions. This entails developing an understanding of how climate and weather have historically affected population health in the health department's jurisdiction, how the climate in the region has changed to date and how it is likely to

change in the future, and finally what factors have driven population vulnerability to climate-health impacts in the past. The climate-health literature, which is expanding rapidly, will likely provide insight into some of the most climate-sensitive diseases and health outcomes in a particular region as well as important factors affecting vulnerability to particular hazards and is generally where a health department should start to explore relevant climate-health relationships. The literature may not have much specific information relevant to the health department's locale, however, so health departments will also need to solicit inputs from local partners to supplement their literature search.

Step 1 is also a scoping exercise, in that health departments must make determinations about the geographic and temporal scope of their assessments based on the intended application of climate and associated health projection information and the availability and robustness of relevant climate and health data. For example, if a jurisdiction plans to use the assessment to help inform city planning and guide decisions regarding hard infrastructure with a lifespan of at least 50 years, planners would like to avail themselves of climatic projections going at least 50 years into the future to coincide with the infrastructure lifespan, and their analysis is likely to focus in particular on historical and future extremes that may test infrastructure capacity. In contrast, assessing how vector-borne disease patterns may shift is likely to be done on a shorter time span and to focus less on extremes than on changes in means and the effect of interannual variability of temperature and precipitation on ecological conditions associated with increased disease risk.

The outcome of step 1 is typically a Climate and Health Profile Report. Such a report lays out the findings of the exercise, including the geographic and temporal scope, a summary of prevalent health concerns in the area, a list of major climate-sensitive health outcomes in the region, factors affecting vulnerability historically, and an overview of how climate change is likely to affect exposures relevant to health in the region over the specified time frame. The report should also identify health impacts that may already be apparent and identify points at which other impacts are likely to manifest and highlight projected shifts in demographics that may affect population vulnerability and expected impacts on population health. Finally, the report should identify relevant infrastructure—from that in the health sector (clinics, hospitals, emergency medical services, etc.) to that in other sectors that is key to maintain public health (power plants, the electricity grid, sewage treatment, agriculture, transportation, etc.)—that may be vulnerable as the climate shifts.

Population and place vulnerability should be a theme throughout the Report. Population vulnerability is relatively familiar in public health and focuses on factors that increase a population's exposure to environmental hazards or amplify an exposure's health impacts. Age, chronic health conditions, and low socioeconomic status are examples. Place vulnerability focuses on factors associated with a specific place that can increase inhabitants' vulnerability to climatic hazards, from geographic fixtures to reliance on local ecosystem services (e.g., for food and employment), as well as strong cultural place connections that could lead to adverse health impacts if ruptured. For both types of vulnerability, vulnerability factors are likely to differ by health outcome and location—age may be a significant factor for some diseases,

while socioeconomic status is likely to be a major factor in others—and these vulnerability factors are not uniformly distributed.

To better characterize the distribution of vulnerability factors, health departments can include representations of their distribution in the Report. One particularly useful approach entails using geographic information systems (GIS) and non-GIS-based vulnerability mapping, which incorporating demographic, risk factor, and health trend data to identify populations and locations within a jurisdiction where vulnerability is particularly high. Further analysis can be undertaken to assess infrastructure, systems, and physical features in vulnerable areas which, if compromised, may compound risk. Infrastructure and system considerations can include factors such as combined sewer systems, location of critical infrastructure such as hospitals and clinics, or vulnerability of the power grid. Physical features can include factors that can amplify exposure such as low elevation, intensity of the urban heat island, and proximity to high-traffic areas with relatively poor air quality.

Once step 1 is complete, a jurisdiction will have a rich sense of how weather and climate have historically affected population health in its area, including a sense of which populations and places are most vulnerable, and how this vulnerability and the associated health burdens are likely to shift as the climate changes. This knowledge is fundamental to the next step in BRACE: projecting future disease burdens.

Step 2: Projecting the Disease Burden

What is the relationship between the exposure(s) of interest and health outcomes in the recent past?

What specific exposure shifts are expected as a result of climate change based on the most recent global circulation model projections?

Putting together these exposure-outcome associations (health response functions) with projected climatic shifts, what is the projected burden of disease secondary to climate change in the next 10, 25, and 50 years?

Through step 1, health departments identify the climate-sensitive health outcomes of greatest concern in their jurisdictions and consider how climate change may affect associated disease burdens over time and potential implications for the health department doing the analysis. In step 2, health departments take the next step and examine these shifting burdens more closely in an attempt to project and quantify shifting burdens associated with a changing climate.

While step 2 can be done qualitatively to yield a general impression of how climate change may affect the risk for certain outcomes, a quantitative effort is likely to be of the greater utility. A qualitative approach would, at the least, capture general trends in climatic exposures, population vulnerability, and identify associated trends in impacts to the extent possible. For instance, a region with significant projected warming and an aging population might note that extreme heat events in the region are expected to triple by 2050, that the proportion of the population over age 65 will double by that time, and that absolute risk of heat-related adverse health effects for older adults in the jurisdiction is likely to increase severalfold.

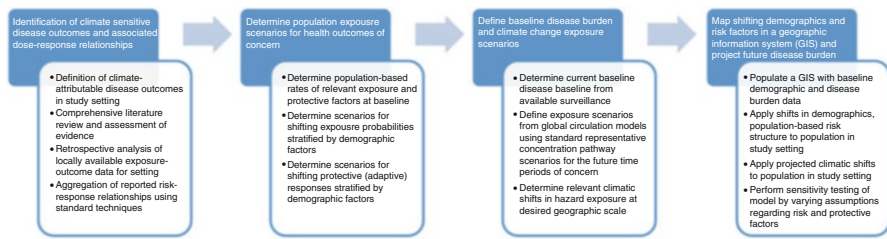


Fig. 20.1 Major steps in quantitative projection of climate-associated disease burdens

A quantitative approach entails a closer analysis of disease risks, vulnerability factors and their contributions to adverse health outcomes, changes in exposure, and relevant demographic shifts and has the potential to identify important aspects of shifting risks that might be missed in a more cursory qualitative analysis. This process has several major components, as noted in Fig. 20.1.

A detailed discussion of this process is outside the scope of this chapter, and there are several different studies that detail relevant methods [18–20]. Regardless of the specific approach taken, the first step is definition of the health outcomes of interest (which will have been identified in step 1 of BRACE) and the climate-health exposure pathway(s) of concern. Heat is the most commonly studied, but a wide range of outcomes are climate sensitive and may be important to study depending on the baseline burden of disease in a particular location. As noted in step 1, the chosen health outcome(s) should be relevant to the jurisdiction being studied based either on current or anticipated future disease burden, and baseline data on disease prevalence, preferably stratified by relevant demographic factors, should be available.

Several different methods have been used to project disease impacts. There is as yet no consensus regarding the most appropriate specific methods for disease projection and reporting of results, even for commonly studied exposures such as heat [21]. In general, the most commonly applied is the delta method, in which changes in the relevant climatic exposure are determined by comparing projected climatic variables (e.g., temperature, humidity, and precipitation) with historical baselines to determine the relevant shift in exposures averaged over a given period of time (e.g., an average increase of 0.7 °C in maximum temperature over June, July, and August in 2035 compared with the baseline period of 1980–2010). The shifted exposure is associated with relative risks (typically expressed as a change in relative risk per some fixed interval change in an environmental variable, e.g., an increase of emergency department visits for heat illness of 1.06 per 1 °C change in temperature above a particular threshold) derived from a comprehensive literature search and/or from retrospective analysis of locally available data for the jurisdiction. If novel associations are being evaluated, the question of whether the observed associations are indeed causal should be addressed. If possible, these exposure-outcome associations should be stratified by relevant demographic variables, e.g., age and socioeconomic status. Other strata may be relevant: for a hydrometeorological hazard such as flooding, for instance, dwelling elevation may also be a predictor of associated illness or death.

Table 20.2 Common data sources used in public health climate change impact projections

Category of data required	Common data sources
Baseline disease prevalence	Public health surveillance, regional and national datasets (e.g., National Hospital Ambulatory Medical Care Survey, Healthcare Cost and Utilization Project, Nationwide Emergency Department Sample, and Behavioral Risk Factor Surveillance System)
Exposure-outcome associations	Published literature, retrospective analysis of local health outcome datasets merged with local weather and climate data from National Climatic Data Center, CDC National Environmental Public Health Tracking Network
Demographic projections	Demographic projections are available from the United States Census for the country as a whole and for individual states via the Federal-State Cooperative for Population Projections
Global circulation model projections	There are a number of climate models worldwide, and certain outputs have been made publicly available; one commonly used source is the Coupled Model Intercomparison Project (CMIP), which issues ensemble model runs for various scenarios (e.g., CMIP3, CMIP5) that are available for download

At a minimum, data required to apply the delta method include baseline disease prevalence, exposure-outcome associations for relevant climatic hazards, demographic projections for the study region, and global circulation model projections of shifts in climatic hazards in the study region for the study period. Considerations regarding data sources for projections in step 2 are listed in Table 20.2. Of note, one of the data sources listed in the table, the CDC's National Environmental Public Health Tracking Network, is discussed briefly later in this chapter.

Adaptation, i.e., activity that reduces the adverse impacts of climate change, is also important to consider, as adaptation activities in public health have the potential to limit adverse impacts significantly, though many barriers have been identified [22]. Depending on the length of study period (i.e., how far into the future health impacts are projected), projections of likely adaptations—active and passive, planned and unplanned—will be more or less important. If adaptations are not considered the projected disease burdens will be systematic overestimates, perhaps dramatically so if the projections are far into the future when adaptations may be widespread. There are many different adaptations to climatic exposures, some of which are passive (e.g., physiologic adaptation to heat exposure) and some of which are active (e.g., purchase, installation, and usage of mechanical air conditioning) that should be considered as part of the BRACE framework. The degree to which various adaptations may protect against exposure or dampen its impacts is not always well known but can be estimated in cases where there is no specific estimate available in the literature. In some cases, physiologic adaptation to the exposure of concern has been incorporated into exposure-outcome response functions [19]. In other cases, adaptation has been accounted for by systematically discounting estimates of future impacts [23].

Projecting disease burden is a potentially data-intensive exercise. However, once models for projecting disease burden are developed, these models can be used to guide several different types of decisions over time and can be used to engage with various stakeholders relevant to risk management decisions affecting public health. As additional information regarding exposures, adaptation options, and trends in demographics and disease burdens becomes available, the models can be updated to provide more precise estimates regarding likely future disease burdens and the cost-effectiveness of specific risk management interventions. Models can also be coupled with other efforts, such as health impact assessments aimed at characterizing climate change mitigation opportunities and associated health co-benefits (e.g., reduced emergency department visits for asthma exacerbations as a result of a shift to renewable energy sources for power generation) [24].

Step 3: Assessing Public Health Interventions

What are the most suitable adaptations and interventions that can be implemented to prevent or reduce anticipated increases in morbidity and mortality?

What types of evidence do we have supporting particular interventions?

How much morbidity and mortality might an early warning system for severe weather reliably avoid?

Following the development of a Climate-Health Profile Report and a model for projecting the health burdens of climate change in a given jurisdiction, the next step in the BRACE framework is to identify and assess possible interventions that might be deployed to limit these anticipated impacts. This is an exercise in the evidence-based practice of public health (EBPH). While much has been written about EBPH in general, there is very little literature on EBPH and public health adaptation to climate change specifically apart from a recent publication surveying policy-relevant scientific literature in the field [25].

In general, EBPH entails problem assessment, systematic review of the public health literature to identify relevant interventions, and assessment of the identified literature to identify the interventions that have the strongest evidence of desired impacts [26,27]. While there is abundant literature regarding the likely public health impacts of climate change (i.e., problem assessment), there is relatively little published on specific adaptations and interventions that may avoid or limit these projected impacts, even when potential exposures are considered outside the context of climate change (e.g., when strategies to protect against heat illness are considered outside of the climate change context). For instance, a recent structured review of population level interventions to reduce the impacts of extreme heat identified only 14 studies, all of which were cross-sectional or retrospective, and the authors were unable to generate a specific impact estimate [28].

BRACE steps 1 and 2 ensure that adequate attention is paid to problem assessment, but do not provide for systematic assessment of relevant interventions. For this, a systematic literature review and accepted approach to evaluation of evidence is required. The methods for conducting systematic literature reviews and

combining estimates of effect are relatively well established (see, for instance, guidelines on Preferred Reporting Items for Systematic Reviews and Meta-analyses [PRISMA] [29]), though there is not yet complete consensus regarding evaluation of evidence in public health, where experimental evidence (e.g., randomized controlled trials) is rare and it is not entirely clear when additional high-level evidence may be required [30]. In practice, public health organizations have taken an inclusive approach to evidence for public health interventions, as demonstrated by the CDC Guide to Community Preventive Services.

While experimental evidence can be particularly useful to justify more costly interventions and determine whether an outcome is causally related to the intervention, observational evidence is frequently very important in guiding day-to-day decisions that many public health officials encounter in the course of their activities. In addition to evidence available in the literature, some locales may decide it is more appropriate for them to supplement with their own evidence through analysis of locally available data to assess problems and guide interventions, a well-established approach (see Table 20.1). CDC's public health partners have also frequently cited the importance of anecdotal evidence conveyed through informal professional networks in making ad hoc decisions when little studied issues arise, such as strategies for promoting the use of cooling centers and making decisions about when to issue heat-health warnings. While considered expert opinion, such evidence is nevertheless important when formal studies have not been done and the potential harm associated with the interventions is low.

Evidence may also not be available for certain potential risks, particularly those associated with cascading failures of risk management like electrical blackouts or sewage treatment failures after extreme precipitation events. In such cases, public health officials may need to access literature outside of public health in order to identify strategies for promoting resilience across a range of linked systems upon which public health relies.

Overall, while systematic review of the literature and identification of efficacious interventions is of paramount importance, it is also clear that other forms of evidence such as observational evidence and expert opinion will also enter into deliberations regarding the interventions to pursue. As the field matures and various interventions are implemented, public health practitioners can prioritize reporting of these interventions and their effects using relevant guidelines already in the literature.

Step 4: Developing and Implementing a Climate and Health Adaptation Plan

What resources are required to implement the adaptations and interventions deemed suitable and feasible within the jurisdiction?

How will these resources be used to implement these adaptations and interventions?

Who and what needs to be mobilized to implement these adaptations and interventions?

Having characterized climate change vulnerability in their jurisdictions, projected likely health impacts associated with climate change, and assessed the effectiveness and suitability of interventions for each of the prioritized health impacts or risk factors, health departments will be in a good position to pursue step 4, development and implementation of a climate change adaptation plan. These plans identify changes to health system functions and programs that are needed to prevent or reduce the anticipated impacts of climate change in the jurisdiction and outline steps for implementing the identified interventions.

The BRACE framework holds that plans should be comprehensive, cutting across all the essential public health functions from surveillance to regulation to outreach and education [5]. As such, the plans must be developed via both a comprehensive inward looking assessment at the health department's activities and with an outward looking engagement of stakeholders and partners to identify priorities, opportunities, and gaps in climate-sensitive disease prevention and health promotion. The planning horizon should be at least several years long, and the scope should be intersectoral with a focus on public health and the health department's role.

Climate change adaptation plans for public health are also both internal and external communication documents. To clarify internal priorities and activities, the intervention plan should clearly outline the resources required to pursue these activities, how existing activities should be modified to account for shifting risks associated with climate change, and who should be responsible for implementation. If key responsibilities lie in partnerships with other agencies, these agencies should be included, and the nature of the working partnership should be outlined explicitly. For external partners, the climate action plan should provide a vision regarding health protection in the jurisdiction and serve as an educational tool regarding ways in which partners can contribute to the overall health protection strategy.

When complete, the plan should be widely disseminated both internally and externally to all stakeholders that may have a role in executing elements of the plan. It should also identify how stakeholders can integrate adaptations into their existing functions and highlight how interventions will be evaluated and make clear the health department's commitments to communicating evaluating findings and updates to stakeholders as the adaptation plan is implemented.

Step 5: Evaluating Impact and Improving Quality of Activities

Did the process used to assess relevant risks, develop interventions, and engage stakeholders result in the outcomes we anticipated?

Did interventions have an impact on population health outcomes?

What lessons were learned from this iteration of the process?

The final step in the BRACE framework relates to evaluating the processes from a process, outcome, and impact perspective. From a process standpoint, this step is useful for determining whether the appropriate stakeholders were involved and whether the methods of engagement resulted in the desired participation and identified the desired inputs. From an outcome standpoint, step 5 should identify the various

programmatic outcomes that resulted from the activity, i.e., stronger relationships with particular stakeholders, model-building skills, increased awareness of synergies across programs, and appreciation of needed shifts in surveillance activities. From an impact standpoint, the evaluation should attempt to determine whether the interventions identified and implemented had the desired impacts on population health.

Each of these different types of evaluation—process, outcome, and impact—uses different methods and different indicators are measured to assess progress or lack thereof. Again, a comprehensive discussion of evaluation methods is outside the scope of this chapter. Health departments will have more or less resources to devote to evaluation activities and may not be able to engage each type of evaluation equally. Regardless, health departments using BRACE should have the capacity to answer the following questions at the end of their evaluation efforts:

1. Has the health department developed a reasonable estimate of future climate change health impacts?
2. Have the BRACE process enabled prioritization of health impacts and interventions?
3. Did the process result in a health department climate change adaptation plan?
4. Is climate change being considered in public health planning and implementation activities?
5. Is public health being considered in climate change planning and implementation activities?
6. Are there specific population health impact indicators that are being tracked to evaluate the interventions identified and implemented as a part of the BRACE process?
7. What aspects of the process can be improved in the next iteration?
8. What are the three top institutional learning priorities in the next round?

While evaluation is located in step 5, this is largely for ease of discussion and communication. Evaluation is in fact a central concern from the beginning of the process and is fundamental to the process of learning so central to adaptive management. Public health has a long tradition of institutional learning in response to novel threats. If the field maintains its commitment to learning it will be able to overcome many of the potential constraints and barriers to climate change adaptation in public health [22].

CDC National Environmental Public Health Tracking Network

Analysis of surveillance data is an important component of learning in public health. The CDC Environmental Public Health Tracking Network (Tracking Network) was established in 2002 to facilitate such learning in environmental health. The Tracking Network is administered by the NCEH and integrates health, exposure, and hazard information from various national, state, and local sources into a dynamic web-based tool that can be used to track and report environmental hazards and health

problems related to them. Interested parties can query to analyze health impacts associated with environmental exposures [31]. The Network involves multidisciplinary collaborations to collect, integrate, analyze, and distribute information derived from environmental hazard monitoring, human exposure surveillance, and health effects surveillance.

Among other exposures, the Network tracks relevant climate and health data to help scientists and decision makers understand the connections between environmental conditions (and changes) and health impacts [32]. At this point the Network focuses its climate change indicators on extreme heat with the aim of evaluating the number of heat-related deaths at the national level. Information on heat vulnerabilities, heat mortality, and temperature distribution can be used to identify patterns in extreme weather and their health effects [33]. For example, the Network can track the effects of a heat wave by aggregating and reporting the number of health conditions and reported deaths from local health departments and hospitals. These data can be used by policymakers at all levels to identify high-risk populations and communities, understand trends in heat-related deaths, and inform adaptation strategies.

The information produced by the Tracking Network is used by national, state, and local public health agencies to make decisions to protect human health in a timely and accurate manner hazards. For health departments interested in assessing the health effects of heat in their jurisdictions and generating exposure-outcome response functions for various heat-related exposures, the Tracking portal is the leading available tool.

Climate-Ready States and Cities Initiative

The Climate and Health Program has developed the BRACE framework as an option for all state and local health departments, and its guidance is available for all interested public health partners. To provide intensive assistance with public health adaptation to climate change in several locales, the Program has developed the CRSCI. The CRSCI aims to build resilience against climate effects in communities by strengthening the capabilities of state and local health departments to deal with the challenges associated with climate change. The CRSCI is working toward this goal by providing multiple cohorts of health departments with funding and technical support. In total, health departments in 16 states and two cities have been funded through the CRSCI as illustrated in Fig. 20.2.

Funding for the Climate-Ready States and Cities is divided into two categories: (1) Assessment and Planning to Develop Climate Change Programs and (2) Building Capacity to Implement Climate Change Programs and Adaptations. Recipients of the first funding stream prepare needs assessments, gap analyses, and strategic plans to address climate change impacts on health in the short and long term using the ten EPHS framework. The second funding stream supports local health departments to implement the BRACE framework. In 2009, the first round of funding for the CRSCI provided support to eight states and two cities. Arizona, Massachusetts,

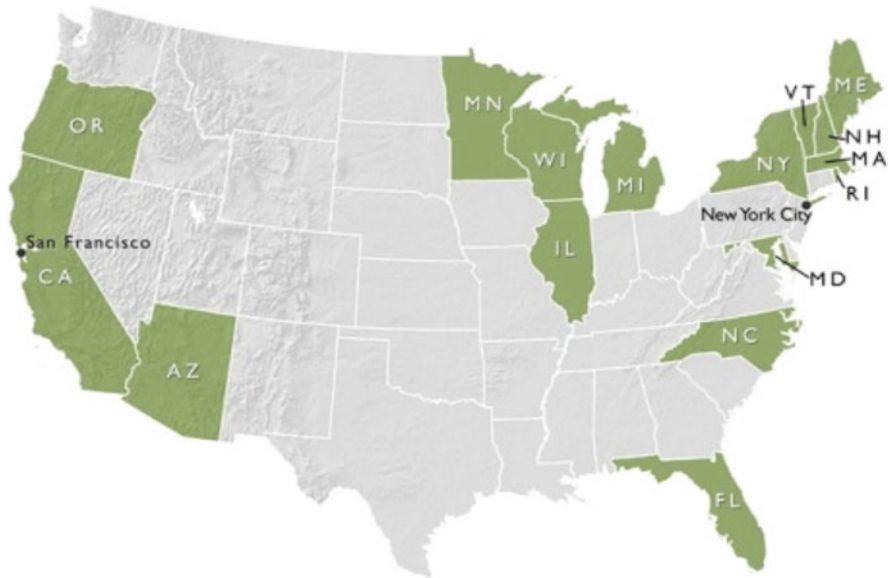


Fig. 20.2 Climate-Ready States and Cities Initiative (CRSCI) funded locations

New York State, North Carolina, and San Francisco received funding to assess jurisdictional capabilities and weaknesses and to plan climate change programs. Michigan, Minnesota, New York City, Oregon, and Maine received funding to build capacity to implement climate change programs and adaptations. In 2012, eight additional states received this multi-year funding: California, Wisconsin, Illinois, Vermont, New Hampshire, Rhode Island, Maryland, and Florida. All 16 of these health departments will apply the BRACE framework with appropriate amendments for each state and city, in order to determine and plan for the regionally specific effects of climate change on human health and vulnerable populations.

The CRSCI is funded via a cooperative agreement mechanism, and the state and local health departments collaborate with CDC to collectively develop a knowledge base regarding public health adaptation to climate change. To facilitate this process, the CDC Climate and Health Program and the grantees have regular sessions to report on progress and share findings. One such meeting is an Annual Science Symposium, where grantees and CDC scientists come together to discuss pressing public health issues related to climate change and health.

CDC Science Symposium on Climate and Health

The Climate and Health Program hosted the first Science Symposium on Climate and Health in 2011. The Symposium brought together scientists from CDC working on topics related to the health impacts of climate change. In 2012, the Symposium

was expanded and co-hosted with the NOAA with participation from Health Canada and the Public Health Agency of Canada. The 2-day symposium also included scientific presentations by academic institutions and state health departments.

The purpose of these symposia is to facilitate information exchange on the state of science related to climate change and to identify data, tools, and partnerships that support improved climate-related public health decision making. Presentations address the current and anticipated impact of climate, weather, and water patterns; impacts of climate patterns on marine, animal, human, and ecosystem health and safety; and climatic influence on ecological and epidemiologic factors that influence disease incidence and distribution. As CRSCI grantees move through the BRACE framework, it is expected that they will present on their progress, the models they develop to project relevant climate impacts, and their processes for identifying and implementing public health interventions to avoid and reduce the adverse health impacts of climate change.

Conclusion

As the nation's public health agency, CDC recognizes that climate change poses a multifaceted and potentially significant threat to domestic and global public health. To facilitate climate change preparedness in public health, the agency developed the Climate and Health Program, which is housed in the NCEH. The Program's mission is to translate science for public health partners, develop decision support tools to facilitate climate change adaptation in public health, and to serve as a credible leader in planning for the human health impacts of a changing climate. Since its formation, the Program has worked to articulate a public health approach to climate change and integrate science from public health and other sectors to facilitate public health adaptation efforts. The Program has developed an adaptive management framework for public health, the BRACE framework, and is working cooperatively with several state and local health departments to pursue an evidence-based approach to climate change adaptation. As public health's expertise and experience grows, the Climate and Health Program will work to continue disseminating relevant information for the increasing number of public health practitioners focused on reducing the adverse health effects of climate change.

References

1. Centers for Disease Control and Prevention. Vision, mission, core values, and pledge. Atlanta, GA. 2011. <http://www.cdc.gov/about/organization/mission.htm>. Accessed 1 Feb 2012.
2. Longstreth J. Anticipated public health consequences of global climate change. *Environ Health Perspect.* 1991;96:139–44.
3. Patz JA, Kovats RS, Patz JA, Kovats RS. Hotspots in climate change and human health. *BMJ.* 2002;325(7372):1094–8.

4. Haines A, Patz JA. Health effects of climate change. *JAMA*. 2004;291(1):99–103.
5. Frumkin H, Hess J.J, Luber G, Malilay J, McGeehin M. Climate change: the public health response. *Am J Public Health*. 2008;98(3):435–45.
6. Hess J, McDowell J, Luber G. Integrating climate change adaptation into public health practice: using adaptive management to increase adaptive capacity and build resilience. *Environ Health Perspect*. 2012;120(2):171–9.
7. Holling C. *Adaptive environmental assessment and management*. New York: Wiley; 1978.
8. National Research Council. *Adaptive management for water resources project planning*. Washington, DC: National Academy Press; 2004.
9. Ebi K. Adaptive management to the health risks of climate change. In: Ford J, Berrang-Ford L, editors. *Climate change adaptation in developed nations*. New York: Springer; 2011.
10. Ebi K. Climate change and health risks: assessing and responding to them through ‘adaptive management’. *Health Aff*. 2011;30:924–30.
11. Brubaker M, Berner J, Chavan R, Warren J. Climate change and health effects in Northwest Alaska. *Glob Health Action*. 2011;4.
12. Kahn ME. Urban growth and climate change. *Annu Rev Resour Econom*. 2009;1(1):333–50.
13. Maibach EW, Chadwick A, McBride D, Chuk M, Ebi KL, Balbus J. Climate change and local public health in the United States: preparedness, programs and perceptions of local public health department directors. *PLoS One*. 2008;3(7):e2838.
14. Balbus J, Ebi K, Finzer L, Malina C, Chadwick A, McBride D, Chuk M, Maibach E. Are we ready? Preparing for the public health challenges of climate change. Washington, DC: Environmental Defense Fund, National Association of County and City Health Officials, Center of Excellence in Climate Change Communication Research at George Mason University; 2008.
15. Marinucci G, Luber G. Bracing for impact: preparing a comprehensive approach to tackling climate change for public health agencies. *American Public Health Association Annual Conference 2011*; 11 Feb 2011; Washington, DC; 2011.
16. Ebi K, Kovats R, Menne B. An approach for assessing human health vulnerability and public health interventions to adapt to climate change. *Environ Health Perspect*. 2006;114(12):1930–4.
17. Campbell-Lendrum D, Woodruff R. Comparative risk assessment of the burden of disease from climate change. *Environ Health Perspect*. 2006;114(12):1935–41.
18. Peng R, Bobb J, Tebaldi c, McDaniel L, Bell M, Dominici F. Toward a quantitative estimate of future heat wave mortality under global climate change. *Environ Health Perspect*. 2011;119(5):701–6.
19. Knowlton K, Lynn B, Goldberg RA, Rosenzweig C, Hogrefe C, Rosenthal JK, Kinney PL. Projecting heat-related mortality impacts under a changing climate in the New York City region. *Am J Public Health*. 2007;97(11):2028–34.
20. Sheffield PE, Knowlton K, Carr JL, Kinney PL. Modeling of regional climate change effects on ground-level ozone and childhood asthma. *Am J Prev Med*. 2011;41(3):251–7.
21. Huang C, Barnett AG, Wang X, Vaneckova P, FitzGerald G, Tong S. Projecting future heat-related mortality under climate change scenarios: a systematic review. *Environ Health Perspect*. 2011;119(12):1681–90.
22. Huang C, Vaneckova P, Wang X, FitzGerald G, Guo Y, Tong S. Constraints and barriers to public health adaptation to climate change. *Am J Prev Med*. 2011;40(2):183–90.
23. Sheridan SC, Allen MJ, Lee CC, Kalkstein LS. Future heat vulnerability in California, Part II: projecting future heat-related mortality. *Clim Change*. 2012:1–16.
24. Patz J, Campbell-Lendrum D, Gibbs H, Woodruff R. Health impact assessment of global climate change: expanding on comparative risk assessment approaches for policy making. *Annu Rev Public Health*. 2008;29(1):27–39.
25. Hosking J, Campbell-Lendrum D. How well does climate change and human health research match the demands of policymakers? A scoping review. *Environ Health Perspect*. 2012;120(8):1076–82.

26. Eriksson C. Learning and knowledge-production for public health: a review of approaches to evidence-based public health. *Scand J Public Health*. 2000 Dec;28(4):298–308.
27. Brownson RC, Fielding JE, Maylahn CM. Evidence-based public health: a fundamental concept for public health practice. *Annu Rev Public Health*. 2009;30:175–201.
28. Bassil KL, Cole DC. Effectiveness of public health interventions in reducing morbidity and mortality during heat episodes: a structured review. *Int J Environ Res Public Health*. 2010;7(3):991–1001.
29. Moher D, Liberati A, Tetzlaff J, Altman DG. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLoS Med*. 2009;6(7):e1000097.
30. Petticrew M, Chalabi Z, Jones DR. To RCT or not to RCT: deciding when ‘more evidence is needed’ for public health policy and practice. *J Epidemiol Community Health*. 2012;66(5):391–6.
31. Prevention CfDca. National environmental public health tracking network. 2012. <http://ephtracking.cdc.gov/showHome.action>. Accessed 5 Jan 2012.
32. Prevention CfDca. Tracking climate change. 2012. <http://ephtracking.cdc.gov/showClimateChangeTracking.action>. Accessed 5 Jan 2012.
33. Prevention CfDca. Climate change indicators available on the environmental public health tracking network. 2012. <http://ephtracking.cdc.gov/showClimateChangeIndicators.action>. Accessed 6 Jan 2012.

Chapter 21

Public Health and Climate Programs at the U.S. Environmental Protection Agency

Erika N. Sasser and C. Andrew Miller

Abstract This chapter explores several key aspects of EPA's climate change program related to public health: (1) the Agency's findings regarding the risks posed by greenhouse gases; (2) the federal regulations developed to reduce greenhouse gas emissions; (3) EPA's efforts on short-lived climate pollutants such as black carbon, methane, and ozone; and (4) the EPA's intramural and extramural research programs related to climate and public health.

Keywords EPA climate programs • Health and climate change • Global consequences for the United States • Greenhouse gas emissions • Emissions reporting requirements • Combined air quality and climate impact

The mission of the U.S. Environmental Protection Agency (EPA) is to protect human health and the environment. This includes ensuring that all Americans are protected from significant risks to human health and the environment where they live, learn, and work and that national efforts to reduce environmental risk are based on the best available scientific information. The challenge of protecting public health in the United States is heightened by the threat of climate change. Although the United States has relatively well-developed public health systems, climate change will still likely affect many Americans. In addition, the impacts of climate change on public health around the globe could have important consequences for the United States.

E.N. Sasser, Ph.D. (✉)

Health and Environmental Impacts Division, Office of Air Quality Planning and Standards,
Office of Air and Radiation, U.S. EPA, Washington, DC, USA
e-mail: sasser.erika@epa.gov

C.A. Miller, Ph.D.

Air, Climate, and Energy Research Program, Office of Research and Development,
U.S. EPA, Washington, DC, USA

The impacts of climate change on public health will depend on many factors. These factors include the effectiveness of a community's public health and safety systems to address or prepare for the risk and the behavior, age, gender, and economic status of individuals affected. Impacts will likely vary by region, the sensitivity of populations, the extent and length of exposure to climate change impacts, and society's ability to adapt to change. EPA's ongoing research programs are investigating these factors, and the Agency is working to identify opportunities to limit public health impacts through emissions mitigation and adaptation efforts. In addition, EPA has recently issued its first regulations under the Clean Air Act (CAA) to begin controlling US emissions of greenhouse gases (GHGs). The Agency has also invested resources in studying the linkages between climate and air quality, including the role that conventional air pollutants such as black carbon particles play in climate change.

This chapter explores several key aspects of EPA's climate change program related to public health: (1) the Agency's findings regarding the risks posed by GHGs; (2) the federal regulations developed to reduce GHG emissions; (3) EPA's efforts on short-lived climate pollutants such as black carbon, methane, and ozone; and (4) the EPA's intramural and extramural research programs related to climate and public health.

Defining EPA's Obligations to Control Greenhouse Gas Emissions

Supreme Court Massachusetts v. EPA Ruling (2007)

On October 20, 1999, the International Center for Technology Assessment and 18 other environmental and renewable energy industry organizations filed a petition seeking the regulation of GHG emissions from on-road vehicles under the CAA (International Center for Technology Assessment [1999], Petition for Rulemaking and Collateral Relief Seeking the Regulation of Greenhouse Gas Emissions from New Motor Vehicles Under [Section] 202 of the CAA). These petitioners argued that EPA had a mandatory duty to regulate long-lived GHGs such as CO₂, methane (CH₄), nitrous oxide (N₂O), and hydrofluorocarbons (HFCs), since these pollutants met the definition of an air pollutant under the CAA, and the cumulative science (including work by the EPA as well as international bodies such as the United Nations Intergovernmental Panel on Climate Change [IPCC]) amounted to a finding that these pollutants are reasonably anticipated to endanger public health and welfare. EPA denied the petition without deciding whether GHGs endanger public health or welfare.

On April 2, 2007, after years of litigation related to the petition, the Supreme Court ruled in *Massachusetts v. EPA*, 549 U.S. 497 (2007) that GHGs are air pollutants covered by the CAA. The Court held that the Administrator must determine whether or not emissions of GHGs from new motor vehicles cause or contribute to

air pollution, which may reasonably be anticipated to endanger public health or welfare, or whether the science is too uncertain to make a reasoned decision. In making these decisions, the Court stated that the Administrator was required to make the science-based judgment called for by the language of section 202(a) of the CAA, and not rely on policy judgments unrelated to the issue of endangerment from GHG air pollution. In light of the Court's decision, EPA took a voluntary remand of another case which concerned GHG emissions from utilities (*New York v. EPA*, No. 06–1322, D.C. Cir., September 24, 2007). This case rested on similar grounds but was still pending at the time of the *Massachusetts v. EPA* decision. EPA later agreed to a settlement with the state and environmental petitioners to issue GHG standards for utilities; these standards for new utilities were proposed in March 2012.

The Agency proceeded carefully in responding to the Supreme Court's decision. EPA reviewed the accumulated science and considered the statutory requirements of the CAA. On January 31, 2008, EPA Administrator Stephen Johnson transmitted a letter to President Bush noting that the decision in *Massachusetts v. EPA* "combined with the latest science of climate change requires the Agency to propose a positive endangerment finding." Work continued over the next several months to develop the scientific and technical support documents necessary to support such a finding, but it was not until the following year that EPA actually moved forward with a finding that GHGs do indeed endanger public health and welfare.

Endangerment Finding: The Health Effects Associated with Climate Change

On April 17, 2009, new EPA Administrator Lisa P. Jackson proposed both an endangerment finding and a finding that motor vehicles contribute to the GHG air pollution that endangers public health and welfare. The two findings were finalized in December 2009, under CAA section 202(a). Each action addressed separate set of issues:

- *Endangerment finding*: In this action, the Administrator found that the current and projected atmospheric concentrations of the mix of six long-lived and directly emitted GHGs—CO₂, CH₄, N₂O, HFCs, PFCs, and SF₆ (referred to as "well-mixed GHGs" in the endangerment finding)—are reasonably anticipated to endanger the public health and welfare of current and future generations.
- *Cause or contribute finding*: In this second finding, the Administrator found that the aggregate emissions of these six well-mixed GHGs from new motor vehicles and new motor vehicle engines contribute to the GHG air pollution that threatens public health and welfare.

These findings, which were published in the *Federal Register* on December 15, 2009 (74 FR 66496), do not themselves impose any requirements on industry or other entities. However, they were a prerequisite to the first regulations on GHGs, the emissions standards for light-duty vehicles described below.

Administrator Jackson determined that the body of scientific evidence compellingly supported an endangerment finding. Major assessments by the US Global Change Research Program (USGCRP), the IPCC, and the National Research Council (NRC) served as the primary scientific basis supporting the Administrator's endangerment finding. In the endangerment finding, EPA pointed to a host of climate-induced impacts that supported a finding of endangerment to public health:

The Administrator has considered how elevated concentrations of the well-mixed GHGs and associated climate change affect public health by evaluating the risks associated with changes in air quality, increases in temperatures, changes in extreme weather events, increases in food- and water-borne pathogens, and changes in aeroallergens. The evidence concerning adverse air quality impacts provides strong and clear support for an endangerment finding. Increases in ambient ozone are expected to occur over broad areas of the country, and they are expected to increase serious adverse health effects in large population areas that are and may continue to be in nonattainment. The evaluation of the potential risks associated with increases in ozone in attainment areas also supports such a finding.

The impact on mortality and morbidity associated with increases in average temperatures, which increase the likelihood of heat waves, also provides support for a public health endangerment finding. There are uncertainties over the net health impacts of a temperature increase due to decreases in cold-related mortality, but some recent evidence suggests that the net impact on mortality is more likely to be adverse, in a context where heat is already the leading cause of weather-related deaths in the United States.

The evidence concerning how human-induced climate change may alter extreme weather events also clearly supports a finding of endangerment, given the serious adverse impacts that can result from such events and the increase in risk, even if small, of the occurrence and intensity of events such as hurricanes and floods. Additionally, public health is expected to be adversely affected by an increase in the severity of coastal storm events due to rising sea levels.

There is some evidence that elevated carbon dioxide concentrations and climate changes can lead to changes in aeroallergens that could increase the potential for allergenic illnesses. The evidence on pathogen borne disease vectors provides directional support for an endangerment finding. The Administrator acknowledges the many uncertainties in these areas. Although these adverse effects provide some support for an endangerment finding, the Administrator is not placing primary weight on these factors.

Finally, the Administrator places weight on the fact that certain groups, including children, the elderly, and the poor, are most vulnerable to these climate-related health effects. (74 FR 66496, 66497–8 [December 15, 2009])

Administrator Jackson found that this body of evidence, in particular the evidence related to the impacts of climate on air quality, supported a finding of endangerment for public health. She also considered the impacts on public welfare, comprehensively reviewing the evidence of impacts and risks over several sectors of society. The Administrator found that

The Administrator has considered how elevated concentrations of the well mixed GHGs and associated climate change affect public welfare by evaluating numerous and far-ranging risks to food production and agriculture, forestry, water resources, sea level rise and coastal areas, energy, infrastructure, and settlements, and ecosystems and wildlife. For each of these sectors, the evidence provides support for a finding of endangerment to public welfare. The evidence concerning adverse impacts in the areas of water resources and sea level rise and coastal areas provides the clearest and strongest support for an endangerment finding, both for current and future generations. Strong support is also found in the evidence concerning infrastructure and settlements, as well ecosystems and wildlife. Across the sectors, the potential serious adverse impacts of extreme events, such as wildfires, flooding, drought, and extreme weather conditions, provide strong support for such a finding. (74 FR 66498 [December 15, 2009])

The finding on endangerment to public health and welfare paved the way for regulations limiting GHG emissions to minimize such adverse health and welfare impacts.

EPA quickly received ten petitions challenging the endangerment finding. The petitions to reconsider EPA's finding claimed that the underlying climate science could not be trusted and asserted a conspiracy that called into question the findings of the IPCC, the U.S. National Academy of Sciences, and the USGCRP. After months of serious consideration of the petitions and of the state of climate change science, EPA documented its responses to all issues raised by these petitions and found no evidence to support these claims that the underlying science was either not robust or not properly interpreted by EPA. On July 29, 2010, EPA denied these petitions, precipitating numerous lawsuits.

As the lawsuits challenging the Endangerment Finding percolated through the court system, EPA continued moving forward with a number of actions. Many of these involved laying the groundwork for future GHG regulations and mitigation efforts. These actions included establishing a reporting system for GHG emissions nationwide; laying out a phased-in approach under which the largest stationary sources of GHG emissions would be the first ones subject to the mandatory CAA permitting requirements; and determining how state and federal actions (e.g., motor vehicle standards) could work together effectively to avoid conflicting requirements. In addition, specific regulations were developed to control GHG emissions from motor vehicles (including both light-duty and heavy-duty on-road vehicles) and to reduce emissions from large stationary sources such as power plants. The Agency's regulatory efforts in each of these key areas are described further below.

Overall, more than 60 lawsuits by industry and states had been filed against the Agency's actions on GHGs by 2012. Other parties, including environmental groups and some states, intervened in support of EPA's actions. While not all of these suits have been resolved, the Agency won a major victory on June 26, 2012, when the U.S. Court of Appeals for the D.C. Circuit upheld EPA's Endangerment Finding and GHG regulations for passenger vehicles and dismissed challenges to regulations defining the scope of GHG permitting requirements for stationary sources. EPA Administrator Lisa P. Jackson welcomed this ruling, noting that it confirms that "EPA followed both the science and the law in taking common-sense, reasonable actions to address the very real threat of climate change by limiting greenhouse gas pollution from the largest sources." The Agency is continuing to investigate other measures that could contribute to climate change mitigation.

Regulatory Underpinnings: Emissions Reporting and Permitting Requirements

EPA regulation of GHGs is relatively recent and is evolving rapidly as the Agency grapples with the requirements of the CAA and legal challenges. These regulations include certain basic provisions such as reporting requirements for GHG emissions

and also cover some of the largest sources of GHG emissions such as motor vehicles and power plants.

Prior to issuing any regulations, in July 2008, EPA issued a broad Advance Notice of Proposed Rulemaking (ANPR), regarding approaches for regulating GHGs under the CAA. (For more information on the ANPR, see <http://www.epa.gov/climatechange/anpr/>.) The ANPR focused on describing key provisions and programs in the CAA, advantages, disadvantages, and limitations of regulating GHGs under those provisions and ways in which those provisions might be interpreted and implemented. It also included an extensive discussion of whether and how regulating GHG emissions under one section of the CAA could or would lead to regulation of GHG emissions under other sections of the Act, including sections establishing permitting requirements for major stationary sources of air pollutants. In addition, it considered the potential for overlap between possible future congressional legislation on climate and regulation under the existing CAA. The Agency received thousands of comments on the ANPR, and it was not until EPA had thoroughly considered these comments that the Agency moved forward with the first regulations on GHGs under the CAA.

Some of the first steps EPA took to address GHGs under the CAA involved dealing with some of the basic underlying requirements of the CAA that affect a number of different types of emission sources. These early regulations did not impose control requirements to limit GHG emissions from particular source categories, but rather involved establishing basic rules for reporting emissions and obtaining permits for major sources of GHG emissions. The CAA is structured in such a way that these steps were important predicates for other regulatory actions. These efforts either collected specific information about GHG emissions or constituted provisions that had to be in place in order to avoid regulatory gaps triggered by the complex, nested requirements of the CAA.

Mandatory Reporting Rule

On October 30, 2009, in response to the FY2008 Consolidated Appropriations Act (H.R. 2764; Public Law 110–161), EPA published a rule for the mandatory reporting of GHG emissions from large sources in the United States. This program, known as the Greenhouse Gas Reporting Program (GHGRP), requires reporting by approximately 13,000 facilities in 41 industrial categories representing approximately 85–90 % of the total US GHG emissions. The rule applies to direct GHG emitters, fossil fuel suppliers, industrial gas suppliers, and facilities that inject CO₂ underground for sequestration or other reasons. In general, facilities must report if they emit 25,000 metric tons or more of CO₂ equivalent (CO₂e) per year. This excludes most small businesses, which generally fall below the 25,000-metric-ton threshold. Reporting is generally done at the facility level, with reports submitted electronically each year by the end of March with data for the previous calendar year.

Reporting began with the year 2010, and as data accumulates this will be a valuable resource for understanding US GHG emissions. These comprehensive,

nationwide emissions data will provide a better understanding of the sources of GHGs and can be used to guide development of the policies and programs to reduce emissions. The data are publically available, which allows facilities and other interested parties to track emissions, compare them to similar facilities, and identify cost-effective opportunities to reduce emissions in the future.

Stationary Source Permitting: The “Tailoring” Rule

By law, major sources must obtain permits under the Prevention of Significant Deterioration (PSD) and Title V Operating Permit program for their emissions of any pollutant(s) regulated under the CAA. The CAA requires stationary sources of air pollution to get permits before they start construction. For GHGs, this includes operating permits (Title V permits) and PSD permits. Operating permits are legally enforceable documents that permitting authorities issue to air pollution sources after the source has begun to operate. These permits, which are required by Title V of the CAA, clarify what facilities (sources) must do to control air pollution. They are issued to all large sources (“major” sources) and a limited number of smaller sources (called “area” sources, “minor” sources, or “nonmajor” sources). In addition to Title V permits, PSD permits are required for new major sources or a major source making a major modification in an area which has “clean” air quality in terms of conventional air pollutants (i.e., areas designated as being in attainment with standards for pollutants such as ozone and particulate matter). The PSD permitting program is designed to ensure that air quality is not significantly degraded from the addition of new and modified industrial sources and that these sources will be as clean as possible. This means that as soon as a new pollutant is regulated under any provision of the Act, new and modifying major stationary sources like power plants and refineries have to apply for new or modified permits covering their emissions of that pollutant. The Endangerment and Cause or Contribute Findings did not by themselves make GHGs regulated pollutants; these findings merely laid the groundwork for the Agency to issue regulations for GHGs on the grounds that (a) those pollutants did indeed endanger public health and welfare and that (b) mobile sources contributed to the emissions causing endangerment. However, as soon as EPA set standards for GHGs—even from mobile sources—GHGs would become a “regulated pollutant” and the stationary source permitting requirements for GHGs would be activated. Therefore, as EPA moved forward with plans to issue GHG regulations for motor vehicles, the Agency also had to anticipate what new permitting requirements would apply to stationary sources. The first regulations on mobile sources became effective on January 2, 2011, and it was imperative that EPA has the permitting program in place prior to that date.

Normally, permitting requirements apply to any sources above specific thresholds as defined in the CAA. But the thresholds established in the Act for determining when emissions of pollutants make a source subject to these permitting programs, 100 and 250 tons per year, were based on traditional air pollutants and were not designed to be applied to GHGs. Applying these thresholds to sources of

GHG emissions would capture not just large sources like power plants but also very small sources such as restaurants and commercial facilities, which was not the intent of the CAA permitting provisions. Applying these thresholds to GHGs would lead to dramatic increases in the number of required permits—tens of thousands of PSD permits and millions of Title V permits. Under these circumstances, state and local permitting authorities would be overwhelmed, the programs' abilities to manage air quality would be severely impaired, and many small sources would be burdened by regulatory requirements.

Therefore, EPA issued a permitting rule known as the GHG Tailoring Rule in May 2010 which effectively "tailored" the requirements of the CAA permitting programs to phase in which facilities will be required to obtain PSD and Title V permits for GHG emissions. The rule focused GHG permitting initially on the largest industrial sources, while shielding millions of small businesses that make up the vast majority of the US economy. The Tailoring Rule set thresholds for GHG emissions that define when PSD and Title V operating permits are required for new and existing industrial facilities and established a stepwise approach for phasing in these requirements. To date, the Agency has issued rulemakings covering Steps 1, 2, and 3.

- Under Step 1, PSD permitting requirements applied to sources' GHG emissions if the sources were subject to PSD anyway due to their non-GHG-regulated air pollutants ("anyway" sources), and emit or had the potential to emit at least 75,000 tpy CO₂e if the source is a new major source, or increases emissions by this amount if the source is an existing source that proposes to undertake a modification. For Title V, existing sources with, or new sources obtaining, Title V permits are required to address GHG emissions in those permit as necessary.
- Under Step 2, PSD applied to the largest GHG-emitting sources that emit or have the potential to emit at least 100/250 tpy of GHGs on a mass basis and that are either new sources that emit at least 100,000 tpy CO₂e or existing sources that emit at that level and that undertake modifications that increase emissions by at least 75,000 tpy CO₂e. In addition, under Step 2, Title V applied to sources that emit or have the potential to emit 100 tpy GHG on a mass basis and emit or have the potential to emit 100,000 tpy CO₂e. These Step 2 applicability thresholds went into effect on July 1, 2011.
- Under Step 3, EPA retained GHG permitting thresholds at the levels established in Step 1 and 2 after determining that state permitting authorities had not had sufficient time to develop the necessary permitting infrastructure and to increase their GHG permitting expertise and capacity, or to develop streamlined approaches. Therefore, the Agency determined it was not yet appropriate to extend the PSD and Title V permitting requirements to smaller sources of GHG emissions. This decision was issued in June 2012.

Facilities responsible for nearly 70 % of the national GHG emissions from stationary sources are subject to these permitting requirements. This includes the nation's largest GHG emitters, such as power plants, refineries, and cement production facilities.

Regulations Affecting Mobile Sources

The first regulations that actually limited the GHGs emissions from a particular source category affected light-duty on-road vehicles (cars and light-duty trucks) and became effective on January 2, 2011, for model year 2012–2016 vehicles. GHG emissions from motor vehicles were the focus of the 1999 lawsuit which culminated in the Supreme Court’s 2007 *Massachusetts v. EPA* decision. Once EPA had issued the Endangerment Finding and the Cause and Contribute Finding for Motor Vehicles in 2009, the next step was to define emissions limitations for that source category. Here too, however, the complexities of the CAA added a few wrinkles to the decision-making process.

California Waiver

The first challenge facing EPA was a pending request from California for a waiver of the CAA’s requirement prohibiting states from enacting emission standards for new motor vehicles. The CAA allows EPA to waive this prohibition for the state of California, which traditionally has had very strict standards for motor vehicles that help to shape the emission controls designed into newer model year vehicles by manufacturers. If EPA grants a waiver, California can enforce its own standards, and other states can also adopt California standards in lieu of federal standards if they choose to do so. In the absence of federal GHG standards for vehicles, however, California’s waiver request for GHGs was subject to intense scrutiny from a wide array of interested stakeholders.

The California Air Resources Board (CARB) originally requested this waiver in December 2005. That request was denied by EPA in 2008. CARB requested that EPA reconsider this denial, and on January 26, 2009, President Obama signed a Presidential Memorandum directing EPA to assess whether denial of the waiver based on California’s application was appropriate in light of the CAA. Less than 6 months later, on June 30, 2009, EPA granted the waiver of CAA preemption to California for its greenhouse gas emission standards for motor vehicles beginning with the 2009 model year.

Light-Duty Vehicle Rules

Meanwhile, the Administration was busy moving forward with new GHG emissions and fuel economy standards for motor vehicles at the federal level. On April 1, 2010, EPA and the Department of Transportation’s National Highway Traffic Safety Administration (NHTSA) jointly finalized a rule that establishes new standards for light-duty vehicles to reduce GHG emissions and improve fuel economy. EPA

finalized the national GHG emission standards under the CAA, and NHTSA finalized the Corporate Average Fuel Economy (CAFE) standards under the Energy Policy and Conservation Act. The standards apply to new passenger cars, light-duty trucks, and medium-duty passenger vehicles, starting with model year 2012 and increasing in stringency through model year 2016. The EPA GHG standards are projected to result in an estimated combined (fleet-wide) average emissions level of 250 g of CO₂ per mile for model year 2016 vehicles. The standards are a fleet average for each manufacturer, based on a footprint attribute curve, meaning that the actual target for a vehicle will vary depending on the size of the vehicle. Under the footprint-based standards, each manufacturer will have a GHG standard unique to its fleet, depending on the footprints of the vehicle models produced by that manufacturer. A manufacturer will have separate footprint-based standards for cars and for trucks.

On August 28, 2012, EPA and NHTSA issued a final rulemaking to extend the national program to reduce GHGs and improve fuel economy from light-duty vehicles by establishing new standards for later model year vehicles. The final standards apply to passenger cars, light-duty trucks, and medium-duty passenger vehicles, covering model years 2017–2025. The standards are projected to result in an average industry fleet-wide basis—that is all passenger cars, light-duty trucks, and medium duty passenger vehicles (including all SUVs)—163 g/mile of CO₂ in model year 2025, which is equivalent to 54.5 miles per gallon (mpg) when vehicles meet this CO₂ level all through fuel economy improvements.

Medium- and Heavy-Duty Vehicle Rule

On August 9, 2011, EPA and NHTSA also announced new standards to reduce GHG emissions and improve the fuel efficiency of heavy-duty trucks and buses. The agencies estimate that the final combined standards of the Heavy-Duty National Program will reduce CO₂ emissions by about 270 million metric tons and save about 530 million barrels of oil over the life of vehicles built for the 2014–2018 model years. The heavy-duty sector addressed in the EPA and NHTSA rules (including the largest pickup trucks and vans, semi trucks, and all types and sizes of work trucks and buses in between) accounted for nearly 6 % of all US GHG emissions and 20 % of transportation emissions in 2007. The standards were developed in response to President Obama's 2010 request to jointly establish greenhouse gas emissions and fuel efficiency standards for the medium- and heavy-duty highway vehicle sector.

Regulations on GHG Emissions from Stationary Sources

Other than the permitting requirements contained in the Tailoring Rule, regulations on GHG emissions from stationary sources have lagged slightly behind, in part because of the complexity and expense associated with reducing GHG emissions

from the large existing fleet of stationary sources in the United States. Under CAA section 111, which defines performance standards for industrial sources, the requirements for new sources are separate from standards for existing sources, largely in recognition of the design differences between older facilities and newer ones, and the large capital requirements often associated with retrofitting existing sources. It is generally easier to build emissions limitations and efficiency requirements into the design specifications of new sources than to require that such changes be incorporated into existing sources. However, sources undergoing major modifications are generally subject to the same requirements as new facilities, since they have an opportunity to incorporate advanced (efficient) equipment during the modification process. Under the CAA, these differences between new and modified sources on the one hand, and existing sources on the other, are captured in the different statutory provisions in CAA section 111, with section 111(b) defining performance standards for new/modified sources and section 111(d) requiring EPA to establish regulations for existing sources (which the Agency has done via emissions guidelines under 40 CFR Part 60).

The first step for stationary sources, therefore, is generally to establish New Source Performance Standards (NSPS) that limit the amount of pollution new facilities may emit. The Act allows flexible and innovative approaches that take into account cost, health and environmental impacts, and energy requirements. EPA must also periodically update these standards to reflect improvements in control technologies.

For GHGs, EPA has focused on the largest industrial pollution sources. Several states, local governments, and environmental organizations had sued EPA over the agency's failure to update the NSPS for fossil fuel power plants and petroleum refineries, two of the largest source categories of GHG pollution in the United States. On December 23, 2010, EPA proposed a schedule for establishing GHG NSPS for these two source categories, which make up nearly 40 % of the nation's GHG emissions.

On March 27, 2012, EPA proposed a carbon pollution standard for new power plants. The proposed rule would limit CO₂ emissions from power plants built in the future. EPA's proposed standard reflects the ongoing trend in the power sector to build cleaner plants that take advantage of American-made technologies, including new, clean-burning, efficient natural gas generation, which is already the technology of choice for new and planned power plants. The proposed standard would require that new power plants meet an emissions rate standard equivalent to new combined-cycle gas-fired units; new coal-fired power plants could meet the standard through installation of carbon capture and storage (CCS) systems. EPA has proposed an alternative compliance pathway, whereby units implementing CCS could comply by meeting the standard on average over the course of a 30-year period. Under this option, a company could build a coal-fired plant and add CCS later, or a company that installs and operates CCS from the outset would have the flexibility to emit more CO₂ in the early years as it optimizes the controls over time. EPA has not yet issued standards for other categories of stationary sources.

Short-Lived Climate Pollutants: Black Carbon and Methane

Another major area of activity for EPA in the last several years has been investigating the role that conventional air pollutants play in climate change. In particular, EPA has focused on black carbon and methane, two pollutants that adversely affect both air quality and climate by contributing to ambient levels of particulate matter and ozone, respectively. Because they remain in the atmosphere for much shorter periods than CO₂ and other long-lived GHGs (which have atmospheric lifetimes of hundreds or even thousands of years), black carbon and methane are often referred to as “short-lived climate pollutants.” Methane has an atmospheric lifetime of approximately 12 years, which is significantly shorter than the lifetime of CO₂ (50–200 years) but long enough to ensure methane becomes well mixed in the atmosphere. Thus, methane was included in the basket of six directly emitted GHGs addressed by the endangerment finding, although its lifetime is still shorter than many of the other GHGs.

For air pollutants like these that also serve as climate pollutants, EPA’s existing regulatory programs have accomplished a great deal in terms of emissions reductions. Regulations on particulate matter (PM) help to control black carbon, for example, just as regulations on ozone precursors may lead to methane reductions. The health benefits of PM and ozone reductions are well documented in EPA’s air quality regulatory programs. However, new scientific evidence linking black carbon in particular to climate change has brought new attention to the role that “short-lived” climate pollutants may play in near-term climate change mitigation strategies.

Black carbon is a component of fine particle (PM_{2.5}) pollution and is emitted from a wide variety of sources including vehicles, fires, and industrial sources. Exposures to PM_{2.5} are associated with a broad range of adverse human health effects, including premature mortality, increased hospital admissions and emergency department visits for cardiovascular and respiratory diseases, and development of chronic respiratory disease [1]. As a short-lived climate pollutant, black carbon is also associated with an array of adverse climate impacts, including increases in global temperature and disruptions in precipitation. Black carbon also has impacts on ice and snow (including earlier spring melting, reduced snowpack and glacial retreat) that are especially significant in sensitive regions such as the Arctic, the Himalayas, and the Western United States.

The United States currently accounts for about 8 % of global emissions of black carbon, and transportation sources (especially mobile diesel engines) account for more than half of US emissions. However, US emissions have been declining, in large part due to EPA regulations. EPA has taken a number of steps to reduce PM_{2.5} emissions, including stringent air quality standards, regulations on more than 40 different types of stationary sources, and tight emissions standards for new mobile source engines including heavy-duty diesel trucks, nonroad diesel engines (such as those used in the construction industry and agriculture), locomotives, and commercial marine. In fact, between 2005 and 2030, existing regulations will help cut 86 % of black carbon emissions from the transportation sector [2].

Elsewhere, black carbon emissions have been rising. By 2000, India and China together accounted for over 35 % of the global inventory. While transportation remains a big (and growing) source in many countries, there are a variety of other sources that are important too. These include open biomass burning, residential cookstoves used in developing countries, and industrial processes such as brick kilns and coke ovens. Black carbon emissions from these sources can be mitigated using existing technologies.

Methane is a precursor to tropospheric ozone, which contributes to adverse health and environmental effects. Specifically, methane forms ozone via reactions with nitrogen oxides (NO_x). Because methane is relatively slow-reacting compared to other ozone precursors, methane contributes mainly to “background” levels of tropospheric ozone, raising the baseline level of ozone gradually on a global scale. Importantly, methane emissions can lead to ozone formation far from the original source due to the fact that methane remains in the atmosphere for 12 years on average and becomes fairly well mixed in the Northern Hemisphere. The effects of tropospheric ozone on health and the environment are well established. Exposure to ozone is linked to respiratory health problems ranging from decreased lung function and aggravated asthma to increased emergency department visits, hospital admissions, and premature death. In addition, tropospheric ozone has been shown to have significant adverse effects on crop yields, pasture and forest growth, and species composition. Elevated ozone levels are linked to visible leaf injury, reduced growth and productivity, and changes in nutrient levels across a number of agricultural, forest, and grassland species [3–5]. These effects can have serious implications for natural ecosystems and agricultural productivity.

EPA’s air quality standards for tropospheric ozone and emissions standards for ozone precursors have traditionally focused on controlling NO_x and volatile organic compounds (VOCs) rather than methane. This is largely because of the greater reactivity of these compounds, which make them more important for limiting ozone formation at the local or regional scale. Methane reductions in the United States have largely been achieved as a byproduct of regulations on VOC emissions. Rules on sources such as landfills and oil and gas wells require control or capture of VOC emissions, and these same measures reduce methane. For example, oil and natural gas production and processing is the single largest methane source nationwide, accounting for approximately 40 % of US methane emissions. A 2012 rule requiring “green completions” for new oil and gas wells is expected to reduce methane emissions from these sources by 9–15 % (i.e., by 1.0–1.7 million tons of methane, which is about 19–33 million metric tons of CO_2 equivalent) at full implementation. Also, standards for mobile source exhaust VOC emissions substantially reduce methane.

EPA researchers estimate that the global health and environmental burden of black carbon (as part of $\text{PM}_{2.5}$) and ozone pollution is substantial. For example, a study designed to investigate the total global mortality burden associated with anthropogenic $\text{PM}_{2.5}$ and ozone estimated that these pollutants lead to 3.7 million and 700,000 premature deaths worldwide each year, respectively [6]. These impacts occur in developed as well as developing countries. In the United States alone,

despite significant improvements in air quality over the past several decades, a recent study estimated that 130,000 PM_{2.5}-related deaths and 4,700 ozone-related deaths resulted from 2005 air quality levels [7]. This study also estimated nearly 1.1 million life years lost from PM_{2.5} exposure and approximately 36,000 life years lost from ozone exposure among populations aged 65–99. These studies do not account for the additional burden to society of the illnesses attributable to exposure to ozone and PM_{2.5}, which lead to large numbers of missed school and work days, hospital and emergency department visits, and additional doctor visits for respiratory and cardiovascular effects.

The Environmental Protection Agency's Role in Efforts to Reduce Short-Lived Climate Pollutants

As the scientific evidence has increased regarding the health and climate impacts of black carbon and methane, EPA has led or supported a number of research and policy efforts to reduce emissions. Chief among these efforts is a 2012 report to the US Congress on the role of black carbon in climate change, work under the Arctic Council and the Convention on Long-Range Transboundary Air Pollution (LRTAP) to spur countries to adopt mitigation measures, and the launch in 2012 of the international Climate and Clean Air Coalition, whose goal is to reduce short-lived climate pollutants (including black carbon, methane, and HFCs). The simultaneous air quality and climate benefits that can be achieved provide a strong incentive to take actions to reduce emissions of these pollutants.

On March 30, 2012, EPA issued a comprehensive *Report to Congress on Black Carbon* (available online at <http://www.epa.gov/blackcarbon>) to clarify the role of black carbon in climate change and identify cost-effective options for reducing these harmful emissions. The report evaluated both domestic and international opportunities. EPA concluded that because of black carbon's strong warming potential and the short amount of time—days to weeks—it stays in the atmosphere, targeted strategies to reduce black carbon emissions can be expected to provide climate benefits within the next several decades. These benefits may be especially significant for certain sensitive regions, such as the Arctic and the Himalayas. Because black carbon is a regional pollutant, the best mitigation strategies for black carbon depend on the local sources and meteorology.

In the United States, mobile diesel sources represent a key opportunity. As mentioned earlier, implementation of the EPA mobile source standards for particulate matter emissions from new diesel engines (the on-road standards effective for model year 2007, the nonroad diesel standards effective beginning in 2012, and the commercial marine/locomotive standards effective beginning in 2014/2015) will be especially important for reducing US black carbon emissions. Also, there are presently 11 million in-use diesel engines produced before these stringent standards became effective. Control of in-use diesel emissions on a state/local level such as is being done in California, as well as through the EPA National Clean

Diesel Program, will be important. Internationally, the implementation of strict diesel emission standards will be important as well. Some countries have already implemented such standards, especially for on-road diesel engines, but more remains to be done.

The report to Congress highlighted the strength of US emissions inventories and ambient data on black carbon, the array of mitigation technologies and strategies available, and the human health benefits of reducing emissions. The size of these potential benefits is remarkable: recent work by EPA, UN Environment Programme (UNEP), and the World Meteorological Organization (WMO) indicates that black carbon reduction strategies implemented at the global scale could potentially result in hundreds of thousands of avoided premature deaths each year [6, 8–10].

In addition to the report to Congress, EPA has been involved in a number of international efforts to investigate and mitigate short-lived climate pollutants. Since 2009, for example, EPA has co-chaired the Task Force on Short-Lived Climate Forcers under the Arctic Council. The Arctic Council is a high-level intergovernmental forum to provide a means for promoting cooperation, coordination, and interaction among the Arctic States. The Council includes Canada, Denmark, Finland, Iceland, Norway, Russia, Sweden, and the United States, as well as a group of permanent participants such as Arctic Indigenous communities and other Arctic inhabitants. This Task Force focuses on identifying measures to reduce emissions of short-lived climate pollutants and recommending immediate actions that Arctic Council countries could undertake. The initial phase of work, which focused on black carbon, was completed in May 2011 (available online at: http://arctic-council.npolar.no/accms/export/sites/default/en/meetings/2011-nuuk-ministerial/docs/3-0a_TF_SPM_recommendations_2May11_final.pdf) and provided recommendations regarding black carbon measures that could provide both health and climate benefits. The Task Force then expanded its work to include methane and provided additional recommendations to Ministers in May 2013 (report forthcoming).

EPA has also been involved in recent changes to the Gothenburg Protocol under the LRTAP Convention. EPA cochaired an Ad Hoc Expert Group on Black Carbon in 2011, which recommended that BC mitigation efforts could reduce black carbon impacts on snow and ice and provide public health benefits. In May 2012, the LRTAP Executive Body formally adopted black carbon provisions as part of the new PM requirements added to the Gothenburg Protocol, Europe's main transboundary air pollution accord.

International momentum to address the health and climate impacts of black carbon and methane is growing. On February 16, 2012, Secretary of State Hillary Rodham Clinton and EPA Administrator Lisa Jackson announced the Climate and Clean Air Coalition to Reduce Short-Lived Climate Pollutants, a new global initiative to seize the opportunity of realizing concrete benefits for climate, public health, food, and energy resulting from reducing black carbon, HFCs, and methane. The founding partner countries included Bangladesh, Canada, Ghana, Mexico, Sweden, and the United States, together with the UNEP. The Coalition has continued to expand rapidly, including more than 66 partners within the first 18 months, and has agreed on numerous fast-action initiatives, including initiatives focused on key

emissions sectors such as mobile diesel engines, brick kilns, cookstoves, landfills, oil and gas, and agriculture, and one initiative focused on encouraging alternatives to HFCs. The Coalition will also focus on providing financing for mitigation measures and promoting national action plans for partner countries.

All of these efforts on short-lived climate pollutants will promote public health improvements. EPA is very aware of the interconnections between air quality planning and climate goals and seeks to promote win-win solutions that maximize public health benefits. Especially in light of the increased public health burden that may result from climate change due to increases in tropospheric ozone (as emphasized in EPA's endangerment finding), measures focusing on reducing black carbon and methane seem like an opportunity to make progress toward both climate and public health goals.

EPA'S Research Programs on Climate and Public Health

American communities face serious health and environmental challenges from air pollution and the growing effects of climate change, both of which are intricately linked with current and future energy options. Improving air quality, reducing GHG emissions, and developing adaptation strategies to address climate change are central to the EPA's mission to protect public health and the environment. To achieve these goals, it is necessary to more fully understand the interplay between air quality, climate change, and the changing energy landscape. Climate change impacts and our responses to climate change (adaptation and mitigation) will impact air quality and human health, often in ways that we have not yet experienced to any significant extent.

Major Areas of Research

There are three major lines of research in EPA that address links between climate change and health associated with air quality and temperature. These are: evaluating the interactions between climate change, air quality, and health; investigating pollutant emissions that impact both air quality and climate; and developing air quality models that account for changes in atmospheric conditions driven by a changing climate. There are other areas of EPA research that also touch upon the links between climate change and health, such as the potential reductions in water quality due to climate change and climate-driven extreme weather events and the spread of waterborne diseases as water bodies increase in temperature. The focus of the discussion in this chapter, however, will be on the three areas noted above. Information about these areas of research, along with other climate-related activities within the Agency, is available on EPA's website on Climate Change Research (<http://epa.gov/research/climatescience/>).

Climate Change, Air Quality, and Health Interactions

EPA has long recognized that air quality is intrinsically linked with climate. Research funded by the Agency over the last several decades indicates that climate change has the potential to cause significant air quality degradation, for example, by changing atmospheric chemical reaction rates and gas solubility; atmospheric transport, mixing, and deposition; and emissions from the biosphere, wildfires, and dust. These changes to air quality due to climate change have the potential to have important consequences for human health and ecosystems. Conversely, changes in air pollutant concentrations also have important implications for climate. For example, trace gases other than carbon dioxide (CO₂) have contributed significantly to anthropogenic climate change in recent decades; air pollution emissions affect concentrations of the hydroxyl radical (OH), thereby influencing the lifetimes of reactive GHGs such as methane (CH₄), HFCs, and hydrochlorofluorocarbons (HCFCs); and aerosol particles strongly affect climate by scattering and absorbing radiation and through their impacts on clouds. As discussed in other chapters in this volume, changes in weather as a result of climate change will have health consequences as well, either directly, as from heat, or indirectly, as from increased exposure to wildfires. In addition, physiological responses to air pollutants may be impacted by the additional stressor of elevated temperature or humidity. Information on specific projects related to climate-air quality interactions is available through EPA's Science Inventory website, which houses a searchable database of EPA science activities and scientific and technical products conducted by EPA and through EPA-funded assistance agreements (see <http://cfpub.epa.gov/si/index.cfm>).

The nature of the interactions between climate, air quality, and health are complex and still not fully understood. Improving our understanding of interactions between climate change and air quality at global, regional, and personal scales is needed to inform decision-making. There is a growing need for scientifically credible, policy-relevant, and timely information on these interactions and their practical consequences for air quality management and climate policy, to maximize the benefits of management and assess tradeoffs between human health and climate change mitigation goals. Simultaneously, understanding the multiple, complex interactions between air quality and climate change, and the associated implications for human health, is a grand challenge for the scientific community, requiring integration across a large number of physical, chemical, and biological processes, many of which are themselves poorly understood.

Combined air quality and climate impacts. One example of a research effort underway to address this need is the development of EPA's GLIMPSE (GEOS-Chem LIDORT Integrated with MARKAL for the Purpose of Scenario Exploration), a decision-support tool to explore US policy scenarios that simultaneously improve air quality and human health, reduce impacts to ecosystems, and mitigate climate change [11]. It is designed to provide results rapidly and to allow decision-makers to comprehensively investigate a range of options to avoid unintended consequences. GLIMPSE couples an energy system model to understand the impact of policy actions on emissions and an atmospheric chemistry-transport model to

understand the impact of emissions on short-lived climate forcers, GHGs, and human health costs of air pollutants. Because the calculations are efficient, the uncertainties can be evaluated, key assumptions uncovered, and scenarios with a high effectiveness identified. As this work is further developed, international collaborators will have the opportunity to employ GLIMPSE for policy decisions that extend beyond the United States.

Ozone and particulate matter. It has long been recognized that tropospheric ozone concentrations generally increase with ambient temperatures. Temperature increases driven by climate change can therefore result in higher ambient O₃ concentrations, and in areas where O₃ levels are already near or above ambient air quality standards, the effect of climate change may lead to O₃ levels that exceed those considered to be protective of public health. The link between climate change, short-term meteorological variability, and O₃ formation is complex, however [12]. Increased exposure to ambient O₃ can also be associated with a longer O₃ season as a consequence of climate change [13]. A growing body of evidence from research conducted and supported by EPA is indicating that it will likely be more difficult to achieve and maintain protective air pollution standards set under the National Ambient Air Quality Standards (NAAQS) program as the effects of climate change become more pronounced [14–16].

The science of climate-air quality interactions is still in its relative infancy, and many critical knowledge gaps remain. This is true for O₃, and especially PM. EPA is beginning efforts to better understand how climate change influences meteorology, particularly those aspects of importance to formation and transport of air pollutants. Such aspects include the role of long-term changes in clouds and precipitation, the impacts of isoprene nitrate recycling, and the relative roles of climate and emissions changes on regional air quality. Additional efforts are designed to develop improved understanding of the behavior and complexities of the global-to-regional climate change-air quality system, including the potential range of impacts of climate change on air quality, the complex interplay between air quality and climatic and meteorological drivers, feedbacks from changes in atmospheric chemistry and air pollution on climate, and implications of climate policies for air quality.

This effort will draw from the growing body of research on climate-air quality interactions, and is intended to address these issues from a perspective of the impacts to NAAQS for O₃ and PM. This connection to the NAAQS ensures that the increased knowledge is presented in the context of health-protective standards.

Emissions That Affect Both Air Quality and Climate Change

As mentioned earlier in this chapter, a number of traditional air pollutants—including black carbon, ozone, and methane—have recently come under focus for their climate effects, in addition to their already well-known health effects. EPA continues to maintain an active research program investigating these pollutants and their effects on climate and air quality. Tropospheric ozone and methane are short-lived GHGs.

In regions subject to high O₃ concentrations, O₃ serves to enhance greenhouse warming. Methane functions similarly and with greater effectiveness per molecule than CO₂. Methane further contributes to warming by serving as a precursor for O₃. Particulate matter (PM) is already regulated as an air pollutant due to its adverse human health effects, including respiratory and cardiovascular disease. From the climate perspective, PM has mixed effects depending upon chemical composition, among other variables. Particles composed of inorganic sulfate salt, for example, scatter incoming solar radiation, promoting cooling. In contrast, black carbon absorbs incoming solar radiation, heating the atmosphere and the surfaces upon which black carbon has deposited. The effect of deposition is particularly pronounced in Arctic areas where deposition of black carbon on snow and ice accelerates ice loss, which, in turn, decreases the Earth's albedo. Furthermore, there is concern that warming in the Arctic could lead to destabilization of permafrost-bound methane hydrates, resulting in large emissions of methane and still greater warming.

Cookstoves. As discussed in an earlier chapter in this volume, almost half of the world's population cooks with rudimentary stoves burning solid fuels, exposing primarily women and children to high concentrations of pollutants. The World Health Organization (WHO) estimates that 1.9 million people die prematurely due to exposure to cookstove pollution, more deaths than are caused by malaria. In addition to the significant human health effects, cookstoves also contribute to concentrations of pollutants in the overall atmosphere, including carbon dioxide, methane, and black carbon.

EPA is actively researching ways to address the health and climate impacts associated with cookstove use. This research includes efforts to identify and quantify emissions, exposures, health effects, and other environmental parameters of concern associated with existing and new cookstoves and fuels. Emissions are being measured from multiple stove/fuel combinations in controlled laboratory settings to provide a consistent baseline against which field test results can be compared. Improved protocols and standards are being evaluated in the laboratory studies to improve uniformity in the way emissions and performance of new stoves are characterized and to enable better correlation between laboratory and field test results [17, 18].

EPA's cookstove-related health research will examine the health benefits associated with reduced exposure to cookstove smoke. The work focuses on developing dose-response relationships for key acute and chronic health effects at the extremely high smoke exposures associated with cookstoves. These dose-response relationships can then be coupled with indoor pollutant concentrations measured with traditional and new cookstove designs to quantify the benefits of improved design and operation.

EPA's Science to Achieve Results (STAR) program is in the process of evaluating external proposals to evaluate the impacts on air quality and climate from residential cooking, heating, or lighting, with a focus on the developing world and on Indian tribes and Alaska Native groups. This research will quantify the extent to which interventions for cleaner cooking, heating, or lighting can impact air quality and climate, which in turn affect human health and welfare.

Emissions from production and use of energy. The production and use of energy generate substantial emissions of GHGs and air pollutants such as PM, NO_x, and VOCs, the last two of which are precursors to formation of tropospheric ozone. As energy technologies and environmental policies evolve, it is likely that the types and amounts of emissions from energy production and use will also change. Policies designed to achieve air quality goals can influence GHG emissions, and conversely, policies focused on GHG reductions can influence emissions of other air pollutants.

To better understand these interactions, and to inform decision makers of the co-benefits and possible adverse impacts of different policy approaches, EPA researchers have applied the MARKAL (MARKet ALlocation) model to examine a range of possible scenarios for the US energy system. Databases developed by EPA researchers for national and regional energy production and use include technology-specific GHG and air pollutant emissions for a broad range of technologies, from passenger vehicles to large power generating stations to renewable energy systems. The MARKAL model can investigate how changes in policies and the mix of energy technologies (e.g., coal, natural gas, renewable energy, gasoline-hybrid vehicles) may impact emissions of pollutants. These results can be used in air quality models to evaluate how emission changes impact air quality, with implications regarding potential changes in associated health risks [19]. More broadly, EPA has supported efforts to develop scenario development methodologies to ensure that modeling scenarios reflect internally consistent storylines and assumptions [20].

Coupled Air Quality and Regional Climate Models

Designing robust policy options that simultaneously help achieve both air quality and climate mitigation goals requires the development of a comprehensive modeling framework that can represent the complex interactions between physical, chemical, and dynamical processes at local to global scales. Such tools must also be able to rapidly screen large numbers of scenarios to uncover alternative policy options, isolate determining assumptions, and present trade-offs to decision makers.

Air quality modeling has long been a critical tool for evaluating changes in air quality associated with policy design and implementation. In the context of a changing climate, such models must be extended to incorporate global-scale changes in atmospheric conditions that can influence the formation and transport of pollutants such as O₃ and PM, which have significant health impacts. Conversely, emissions of aerosols such as black carbon and sulfates will impact radiative forcing.

To address these complex interactions, EPA has placed considerable emphasis on developing air quality models that can represent the global-scale changes in the atmosphere due to climate change in regional-scale air quality models. Because of the atmospheric interactions between regional and global scales, the ideal is to have models that accurately represent the feedback mechanisms between these scales. EPA's research has made considerable progress in developing the capability of its major air quality model, the Community Multiscale Air Quality (CMAQ) model to account for these complex interactions [21–24]. EPA has also supported additional

work to develop other climate-air quality modeling approaches that enable new approaches to evaluating climate-air quality interactions [25–27]. These efforts will provide public health agencies and researchers with improved information about potential adverse impacts to air quality, and ultimately health, associated with a changing climate.

Conclusion

The links between climate and public health are likely to remain a major focus of EPA's evolving climate change programs. As the scientific evidence related to climate change and its impacts on exposures, responses, and health outcomes increases, EPA will need to incorporate these key findings into regulatory decisions and future research plans. Furthermore, it will be important to communicate the linkages between climate and public health outside the Agency in public outreach activities, in part to foster dialogue between public health organizations and potentially vulnerable populations about the altered or additional risks that may result from climate change. EPA has already begun to adjust its programs and policies to incorporate climate change considerations, as evidenced by the Endangerment Finding and the subsequent regulations affecting major emitting sources, such as motor vehicles. These efforts, combined with current and future investments in research related to climate and health, will help ensure that the Agency is able to fulfill its critical mission of protecting public health and the environment for many years to come.

References

1. U.S. EPA. Integrated science assessment for particulate matter (Final report). Washington, DC: U.S. Environmental Protection Agency; 2009. EPA/600/R-08/139F.
2. U.S. EPA. Report to congress on black carbon. Research Triangle Park, NC: Office of Air and Radiation, Office of Air Quality Planning and Standards; 2012 Mar. EPA-450/R-12-001.
3. U.S. EPA. Integrated science assessment of ozone and related photochemical oxidants (Final report). Washington, DC: U.S. Environmental Protection Agency; 2013 Feb. EPA/600/R-10/076C.
4. Emberson LD, Ashmore MR, Murray F. Air pollution impacts on crops and forests: a global assessment. London: Imperial College Press; 2003.
5. Ashmore MR. Assessing the future global impacts of ozone on vegetation. *Plant Cell Environ.* 2005;28:949–64.
6. Anenberg SC, Horowitz LW, Tong DQ, West JJ. An estimate of the global burden of anthropogenic ozone and fine particulate matter on premature human mortality using atmospheric modeling. *Environ Health Perspect.* 2010;118:1189–95.
7. Fann N, Lamson AD, Anenberg SC, Wesson K, Risley D, Hubbell BJ. Estimating the national public health burden associated with exposure to ambient PM_{2.5} and ozone. *Risk Anal.* 2011;32(1):81–95.

8. Anenberg SC, Talgo K, Arunachalam S, Dolwick P, Jang C, West JJ. Impacts of global, regional, and sectoral black carbon emission reductions on surface air quality and human mortality. *Atmos Chem Phys*. 2011;11:7253–67.
9. UNEP, WMO. Integrated assessment of black carbon and tropospheric ozone. Geneva: United Nations Environment Programme and World Meteorological Organization; 2011. http://www.unep.org/dewa/Portals/67/pdf/BlackCarbon_report.pdf
10. Shindell D, Kuylensstierna JCI, Vignati E, et al. Simultaneously mitigating near-term climate change and improving human health and food security. *Science*. 2012;335:183–9.
11. Henze DK, Akhtar F, Pinder RW, Loughlin D, Spurr R. Linking BC direct radiative forcing to source regions and sectors as a constraint on future emissions mitigations strategies. San Francisco, CA: American Geophysical Union, Fall Meeting; 2010.
12. Weaver CP, Liang X-Z, Zhu J, et al. A preliminary synthesis of modeled climate change impacts on U.S. regional ozone concentrations. *Bull Am Meteorol Soc*. 2009;90:1843–63.
13. Bloomer BJ, Vinnikov KY, Dickerson RR. Changes in seasonal and diurnal cycles of ozone and temperature in the eastern U.S. *Atmos Environ*. 2010;44(21–22):2543–51. doi:10.1016/j.atmosenv.2010.04.031.
14. Wu SL, Mickley LJ, Leibensperger EM, Jacob DJ, Rind D, Streets DG. Effects of 2000–2050 global change on ozone air quality in the United States. *J Geophys Res*. 2008;108, D06302. doi:10.1029/2007JD008917.
15. 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, L09803. doi:10.1029/2009GL037308.
16. Jacob DJ, Winner DA. Effect of climate change on air quality. *Atmos Environ*. 2009;43:51–63.
17. Jetter JJ, Kariher P. Solid-fuel household cook stoves: characterization of performance and emissions. *Biomass Bioenergy*. 2009;33:294–305.
18. DeCarlo PF, Jetter J, Khan B, Zhao Y, Yelverton T, Hays MD. Characterization of cookstove emissions from various stoves, fuels, and cycles; intervention pathways and implications for climate. San Francisco, CA: American Geophysical Union, Fall Meeting; 2011.
19. Loughlin DH, Benjey WG, Nolte CG. ESP v1.0: methodology for exploring emission impacts of future scenarios in the United States. *Geosci Model Dev*. 2011;4:287–97.
20. Smith SJ, West JJ, Kyle P. Economically consistent long-term scenarios for air pollutant emissions. *Clim Change*. 2011;108:619–27.
21. Nolte C, Gilliland A, et al. Linking global and regional models to simulate U.S. air quality in the year 2050. In: Borrego C, Miranda AI, editors. *Air pollution modeling and its application XIX*. Netherlands: Springer; 2008. p. 559–67.
22. Bowden JH, Otte TL, Nolte CG, Otte MJ. Examining interior grid nudging techniques using two-way nesting in the WRF model for regional climate modeling. *J Climate*. 2011;25:2805–23.
23. Alapaty K, Mathur R, Pleim J, Hogrefe C, Rao ST, Ramaswamy V, Galmarini S, Schaap M, Makar P, Vautard R, Baklanov A, Kallos G, Vogel B, Sokhi R. New directions: understanding interactions of air quality and climate change at regional scales. *Atmos Environ*. 2012;49:419–21.
24. Otte TL, Nolte CG, Otte MJ, Bowden JH. Does nudging squelch the extremes in regional climate modeling? *J Climate*. 2012;25:7046–66.
25. Jacobson MZ. Short-term effects of controlling fossil-fuel soot, biofuel soot and gases, and methane on climate, Arctic ice, and air pollution health. *J Geophys Res*. 2010;115(D14).
26. Jacobson MZ. The enhancement of local air pollution by urban CO₂ domes. *Environ Sci Technol*. 2010;44:2497–502.
27. Jacobson MZ, Ginnebaugh DL. The global-through-urban nested 3-D simulation of air pollution with a 13,600-reaction photochemical mechanism. *J Geophys Res*. 2010; 115(D14).

Chapter 22

California's Cap-and-Trade Program

John R. Balmes

Abstract A market-based mechanism to incentivize investment in cleaner and more efficient technologies is recognized by most economists as an important tool to reduce GHG emissions. While economists frequently disagree about which market-based mechanism should be used, a cap-and-trade program or a carbon tax, CARB has chosen the former because of its firm limit on emissions, flexibility for businesses, and political feasibility. In California, such a mechanism is only one component of the overall effort to mitigate climate change under AB 32, with direct regulations accounting for a much greater reduction in GHG emissions. California has long been an international leader in policies to improve air quality and in recent years has added climate change policy to its leadership role. The design and implementation of the California cap-and-trade program have presented many challenges, but CARB is committed to the eventual success of the program.

Keywords California's cap-and-trade program • Cap-and-trade program in California • Climate change and cap and trade • Greenhouse gas emissions • California Global Warming Solutions Act

The enormity of the problem of climate change and the rapidity with which greenhouse gas (GHG) emissions are increasing requires that strategies to reduce emissions need to be highly effective and relatively easily implemented. Both short-term and long-term strategies are required to prevent a climate change tipping point. California has long been a leader in developing policies to prevent environmental

J.R. Balmes, M.D. (✉)

Division of Occupational and Environmental Medicine, Department of Medicine,
University of California, San Francisco, CA, USA

Division of Environmental Health Sciences, School of Public Health,
University of California, Berkeley, CA, USA
e-mail: john.balmes@ucsf.edu

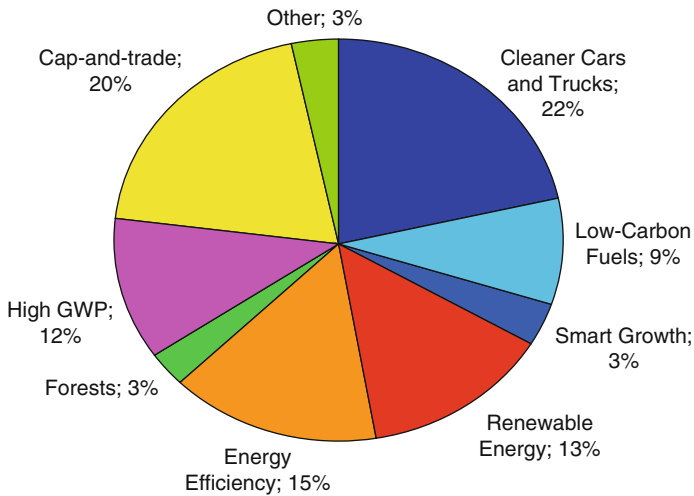


Fig. 22.1 Percentages of greenhouse gas emission reductions from components of California's AB 32 scoping plan

degradation, especially in the area of air quality, so it should come as no surprise that the California legislature passed and then Governor Schwarzenegger signed a landmark bill to mitigate climate change, Assembly Bill 32 (AB 32), the California Global Warming Solutions Act, in 2006 [1]. The California Air Resources Board (CARB), the state agency with authority to control air quality, was given the responsibility for implementing AB 32.

The primary goal of AB 32 was to reduce GHG emissions to 1990 levels by 2020—a reduction of approximately 30 %, followed by an 80 % reduction below 1990 levels by 2050. To achieve this goal, CARB developed a “scoping plan” that included several strategies to reduce GHG emissions [2]. The main strategies are the following: promulgate direct regulations, provide monetary and non-monetary incentives, encourage voluntary actions, and develop market-based mechanisms such as a cap-and-trade system.

As CO₂ is the most abundant long-lived GHG, CARB's overall approach has been to take policy actions to reduce CO₂ emissions. The source of most CO₂ emissions is fossil fuel combustion that provides power for our motor vehicles and generates electricity for our economy. Thus, CARB has been implementing policies to require or incentivize motor vehicle manufacturers, electric utilities, oil companies, and other industries to move away from the current reliance on fossil fuel combustion toward cleaner or more efficient ways to meet our energy needs.

Although the greatest attention has been directed to the cap-and-trade system that CARB has been implementing, this system accounts for only a fraction (20 %) of the GHG reduction to be achieved under the AB 32 scoping plan (Fig. 22.1). Direct regulations, a so-called command and control strategy, actually account for a far greater proportion of GHG emission reductions [2]. These regulations include the Pavley (AB 1493) requirements for reduced GHG automotive tailpipe emissions

(22 %), the Low-Carbon Fuel standard (9 %), and the Renewables Portfolio standard that requires California utilities to generate 33 % of their power from renewable sources by 2020 (13 %). Other important components of the overall strategy to reduce CO₂ emissions are Regional Transportation-Related GHG Targets required by Senate Bill 375, the Sustainable Communities and Climate Protection Act of 2008 [3], and energy efficiency in building and motor vehicle design.

The language of AB 32 specifically authorized CARB to “adopt a regulation that establishes a system of market-based declining annual aggregate emission limits for sources or categories of sources that emit greenhouse gases, applicable from January 1, 2012, to December 31, 2020” [1]. The Board has determined that a cap-and-trade program is the market-based mechanism that is most feasible to implement in California at the present time. Before describing the specific features of the California cap-and-trade system, a discussion of alternative approaches to incentivize industry to reduce CO₂ emissions and the pros and cons of each approach is necessary.

Approaches to Incentivize the Reduction of CO₂ Emissions Across the Economy

To achieve the AB 32 mandate of developing a market-based mechanism to reduce GHG emissions requires placing additional costs on fossil fuel combustion, i.e., a price on carbon. Any approach that places a price on carbon must discourage the use of oil, coal, and natural gas and encourage the development of renewable sources of energy. Several approaches have been proposed. One is a cap-and-trade system, which would place progressively stricter limits on fossil fuel use; require power plants, industries, and other major sources of greenhouse gases to purchase permits to discharge CO₂; and establish a market for trading (buying and selling) those permits. Another is a tax on fossil fuels, a so-called carbon tax. Although the economic hardship created by higher energy prices by either of these approaches could be offset by rebates, a third approach that has been proposed is a so-called cap and dividend, where most if not all revenue generated from sales of CO₂ emission permits would be distributed to the public.

A cap-and-trade system has several strengths [4]. It sets a steadily declining ceiling on carbon emissions, and, by creating a market that rewards companies for slashing CO₂ (corporations that reduce emissions below their allotment can sell them on the open market), it uses the free enterprise system to wean the state off fossil fuels and onto renewable energy. Proponents of a cap-and-trade approach also argue that it is more politically feasible in the current US political climate than a carbon tax [5]. There is some experience with the cap-and-trade approach. The U.S. EPA's Acid Rain Program (a component of the 1990 Clean Air Act) has been successful at reducing sulfur emissions from power plants [6]. Given that climate change is a global problem, a market for trading CO₂ allowances could be expanded to include existing programs—the European Union has had a comprehensive

program in place since 2005 [7], and nine Northeastern states have adopted a cap-and-trade program for the energy sector (the Regional Greenhouse Gas Initiative or RGGI) [8].

A carbon tax has the overall strength of simplicity. By imposing a predictable price on fossil fuels, a carbon tax would drive development of alternative sources of energy. Because a carbon tax does not cap emissions, the actual tax would likely have to be calibrated to ensure that the price signal actually led to significant reductions in CO₂. It is likely that the tax would have to be steadily increased over time. British Columbia enacted a carbon tax in 2008 [9]. The tax started at \$10 per ton of carbon in its first year and rose by \$5 per ton each year thereafter. That translates into a roughly 9-cent tax on a gallon of gasoline, rising 5 cents per gallon each year. Although in its relative infancy, the program does seem to be at least modestly successful because the province has reduced its CO₂ emissions at a modestly faster rate than the rest of Canada [10]. The British Columbia tax is revenue neutral, meaning every dollar generated by the tax is returned to provincial citizens through reductions in both income and business taxes.

A third approach to placing a price on carbon combines features of both cap-and-trade and a carbon tax, a cap-and-dividend system. This approach is seen by some as politically attractive because the revenue generated from the sale of emission permits would be distributed to the public. Cap-and-dividend legislation has been introduced in the US Congress that would place an upstream cap on the first sellers of fossil fuels (e.g., oil, coal mining, and natural gas extraction companies), mandate 100 % auctioning of permits, and return of all or most auction revenue to citizens on a per capita basis [11]. The plan is modeled after the Alaska Permanent Fund, which pays equal dividends to Alaskan residents from the proceeds generated from state oil leases. Like British Columbia's revenue-neutral carbon tax, such a cap-and-dividend system has the features of simplicity, economy-wide coverage, and protection of households from the impact of rising energy prices. Moreover, like a traditional cap-and-trade system, it places a limit on CO₂ emissions and, if trading of permits is allowed, would provide the flexibility that many industry leaders advocate.

The basic difference between a cap-and-trade approach and a carbon tax is that the former provides certainty on emissions reduction but allows uncertainty about the price of carbon, while the latter fixes the price of carbon but does not provide certainty about reductions in CO₂ emissions. Because of its simplicity, a carbon tax has been said to be both easier to implement and harder to game. Based on decades of experience with tax legislation in the USA, however, special interests often are able to influence how tax laws are written with the usual result that some economic sectors are favored over others. Special interest lobbying and infighting also tends to complicate and often prolong the tax legislative process.

In principle, both a carbon tax and a cap-and-trade program can achieve cost-effective reductions. To achieve continued progress in reducing CO₂ emissions over time, a tax would have to be progressively increased, which could be very difficult to realize given political pressures, especially during times of economic downturn. Such political pressures would most likely lead to exemptions of

certain economic sectors, which reduces environmental effectiveness and drives up costs, as some low-cost emission reduction opportunities would be left off the table. Political pressures on a cap-and-trade system would lead to different allocations of allowances, which affect distribution, but not environmental impact and cost-effectiveness. Advocates of a carbon tax suggest that political pressures under a cap-and-trade system would unfairly compensate certain sectors through free allowance allocations, but a carbon tax is sensitive to the same political pressures and could be modified in ways that lead to lower reductions in emissions and potentially higher costs [5].

Environmental integrity and flexibility are two critical features of a cap-and-trade system that helped motivate CARB to select this approach as the market-based mechanism to implement under AB 32. The flexibility afforded by emissions trading markets helps identify where emission reductions can be achieved most cost-effectively, while providing some protection for businesses that need more time to reduce emissions. The main driver of the CARB decision to go with a cap-and-trade system was the political reality that a new tax during a time of economic hardship was simply not going to be approved by the required two-thirds majorities of both houses of the California legislature.

California's Cap-and-Trade Program

The development of California's cap-and-trade program was initiated in November 2009 [12], mandatory monitoring and reporting of GHG emissions began in January 2010, and the first auction of emission permits ("allowances") was held in November 2012. Businesses and governmental agencies such as utilities, refineries, cement plants, and manufacturers in California that emit more than 25,000 metric tons of CO₂ per year ("covered entities") are required to obtain allowances to emit greenhouse gases. The program covers around 350–400 entities. The cap, the overall amount of emissions CARB allows, progressively shrinks over time, as will the number of allowances for sale. That means covered entities will need to reduce their GHG emissions or buy allowances.

A sizeable portion of these allowances is distributed for free to many covered entities. Some utilities, for example, receive all their allowances at no cost. Covered entities that have to purchase a portion of their allowances must buy them either at auctions held by CARB or in the marketplace from other companies. Companies that generate less can opt into the program, and some of them have. Companies generally opt in when their business plans indicate they can receive free allowances by following the cap-and-trade regulations—monitoring and reporting emissions. The advantage to them is that they do their calculations and determine that they will receive enough allowances under the program that they can sell some. Covered entities can also meet the cap by purchasing a limited amount of credits from projects being implemented elsewhere that reduce GHG emissions according to several specific protocols ("offsets"). The provision of offset credits adds greater flexibility to

the program, especially for companies that will have difficulty meeting their emission reduction targets in the initial compliance period.

The first compliance period began on January 1, 2013. The initial 1-year cap was 162.8 million metric tons of CO₂. The initial auction participants were primarily refineries, utilities, cement manufacturers, glassmakers, and some of the larger food processing facilities. The auctions are closed to registered participants and blind (bidding entities cannot see each other's bids). The initial floor price for each allowance, a so-called reserve price, was \$10. Covered entities can purchase no more than 15 % of the available allowances and third parties no more than 4 %. CARB has no involvement in the secondary market, other than that all trades or sales must be registered with the agency.

Allowances are allocated based on the products that businesses in the different sectors produce. There is a sector-based benchmark process, and the benchmark is essentially an average of the carbon intensity required for the manufacture of the finished products in each sector. If a covered entity emits CO₂ significantly below the benchmark in its sector, that entity ends up with a surplus of allowances, which it can bank or sell. If that entity emits at or above the benchmark, it will likely have to purchase allowances from an entity with a surplus or at auction.

For the first 2 years of the program, all of the industrial entities get 90 % free allowances, so they only have to account for 10 % of their emissions in terms of actually buying allowances or reducing emissions. If they can reduce their emissions by that 10 %, they need not purchase any allowances. The utilities got all of their allowances free for the duration of the program, but the investor-owned utilities and some of the public ones are required to sell all of their free allowances at auction. The proceeds from the auction of these free allowances will be used in a process being developed by the California Public Utilities Commission to return some value to the utilities' customers, either by controlling rates or funding more renewable sources.

The CARB cap-and-trade regulation permits entities to purchase offset credits to meet up to 8 % of their triennial compliance obligation. Each credit is equal to one allowance (i.e., 1 metric ton of CO₂ equivalent) and can be issued by CARB for achieving emissions reductions through implementation of an offset project pursuant to one of CARB's approved compliance protocols. The initially approved protocols were for livestock projects, ozone depleting substances projects, urban forest projects, and US forest projects. These protocols were developed by the Climate Action Reserve [13], and other offset protocols will be developed in the future.

A concern about CARB's cap-and-trade program is that there may not be sufficient allowances and credits to cover the needs of covered entities, thereby driving up the price per allowance. To prevent an economically untenable spike in the cost of an allowance, CARB has set aside a strategic price containment reserve of 123 million 1-ton allowances for sale at a price of \$40 per ton. The agency's economic modeling of the impact of the cap-and-trade program suggests that it is highly unlikely that the price reserve will be needed.

Critics of CARB's cap-and-trade program also are concerned about the issue of "leakage," the loss of jobs or business when companies decide to move out of the

state because of the increased cost of business due to the program. What CARB is doing to prevent leakage is to distribute some free allowances to companies in sectors where significant leakage threat can be documented. A good example of leakage prevention involves the cement industry. The process of manufacturing hydraulic cement, the kind of cement that will stay hard underwater, is extremely GHG intensive, and currently there is no alternative technology for making it. To prevent California manufacturers of hydraulic cement from going out of business, CARB gave them a modification to their allowance budgets that takes this into consideration. CARB has pledged to intermittently review evidence of leakage to prevent undue economic hardship. In some cases, additional free allowances may be distributed. In other cases, later entry into the program is the solution, which was granted to combined heat and power facilities.

To some extent California's cap-and-trade program is modeled after the RGGI in the Northeast and the European Union's emissions trading system. However, CARB has endeavored to avoid mistakes made when those programs were established. For example, there were too many allowances distributed when the RGGI program was initiated such that a persistent surplus has driven down the price of an allowance, and windfall profits were generated when the EU program's initial allowances were issued free, rather than auctioned. The CARB program started with a mandatory monitoring and reporting period to ensure that the correct emissions cap and allowance number were established. As noted above, CARB also required auction of some allowances from the outset of the program.

Not surprisingly, the impact of the CARB cap-and-trade regulation on the California economy has been a topic of considerable controversy. The economic impact analysis of AB 32 implementation, including the cap-and-trade program, conducted by the agency predicts that there will be no negative impact on the growth of the California economy through 2020 compared to a business-as-usual scenario, and increases in productivity, jobs, and per capita income will result [14]. Investment in clean technologies and increased energy efficiency are the main reasons why a positive impact is projected. A review of the economic impact of the first 3 years of the RGGI indicates that 16,000 new jobs were created for these reasons [15]. Of course, not all economic impact analyses of the California program have been so rosy, with some suggesting that increased energy costs will hurt both businesses and homeowners, leading to business closures and job losses [16]. The analyses of several environmental groups support that of CARB [17].

The revenues generated from the auction of allowances may be substantial. How these revenues are allocated will say a lot about California as a society. CARB has no authority to appropriate funds so decisions will have to be made by the legislature. Legislation has been enacted to lay out a framework for how cap-and-trade revenue will be spent [18]. One bill requires that the revenues be spent on environmental purposes, with an emphasis on improving air quality. Another bill requires that at least 25 % of the money be spent on projects that help "identified disadvantaged communities"—mainly poorer communities that tend to suffer the worst air pollution; at least 10 % must be spent on projects specifically within those communities.

International Linkage

Climate change is a global problem that requires global solutions. The establishment of the California cap-and-trade program is, therefore, just one step among many that are required to significantly mitigate climate change. During the design of the cap-and-trade regulation, CARB has never expected it to be a stand-alone system for the long-term future. Since its inception in 2007, California has participated in the Western Climate Initiative (WCI) [19]. The WCI is a collaboration of independent jurisdictions working together to identify, evaluate, and implement emissions trading policies to tackle climate change at a regional level. At one point the WCI included seven US states and four Canadian provinces, and the major goal was to develop the framework for a multi-sector, market-based program to reduce GHG emissions across the jurisdictions. Because of the economic impacts of the Great Recession, as well as political pressure from those that do not accept the scientific consensus about climate change, the other US states aside from California decided in 2010 to either delay participation in or pull out of the WCI. As of late 2012, only California and Quebec were actively working to link their carbon-trading systems [20]. The two jurisdictions drafted guidelines to ensure that California and Québec carbon allowances are interchangeable at auction and can be used for compliance purposes in one another's programs. The guidelines also provide joint, enforceable standards for development and use of carbon offsets, as well as a range of steps to ensure the security of the market.

The proposed linkage of California and Quebec cap-and-trade programs is an example of the increasingly large number of international efforts to reduce GHG emissions trading programs. In addition to the already established EU system, Norway, Iceland, Switzerland, Australia, New Zealand, and South Korea have announced plans for developing carbon-trading schemes. Harmonization of such schemes will be challenging, but is necessary for significant progress in mitigating climate change.

Conclusions

A market-based mechanism to incentivize investment in cleaner and more efficient technologies is recognized by most economists as an important tool to reduce GHG emissions. While economists frequently disagree about which market-based mechanism should be used, a cap-and-trade program or a carbon tax, CARB has chosen the former because of its firm limit on emissions, flexibility for businesses, and political feasibility. In California, such a mechanism is only one component of the overall effort to mitigate climate change under AB 32, with direct regulations accounting for a much greater reduction in GHG emissions.

California has long been an international leader in policies to improve air quality and in recent years has added climate change policy to its leadership role. The design and implementation of the California cap-and-trade program have presented many challenges, but CARB is committed to the eventual success of the program.

References

1. <http://www.arb.ca.gov/cc/factsheets/ab32factsheet.pdf>, <http://www.arb.ca.gov/cc/ab32/ab32.htm>
2. <http://www.arb.ca.gov/cc/scopingplan/scopingplan.htm>
3. <http://www.arb.ca.gov/cc/sb375/sb375.htm>
4. <http://www.c2es.org/docUploads/climate101-captrade.pdf>
5. http://e360.yale.edu/feature/putting_a_price_on_carbon_an_emissions_cap_or_a_tax/2148/
6. <http://www.epa.gov/airmarkets/progsregs/arp/>
7. http://ec.europa.eu/clima/policies/ets/index_en.htm
8. <http://www.rggi.org>
9. http://www.fin.gov.bc.ca/tbs/tp/climate/carbon_tax.htm
10. <http://www.sustainableprosperity.ca/dl872&display>
11. <http://cantwell.senate.gov/issues/CLEAR%20Act%20Overview%20Memo.pdf>
12. <http://www.arb.ca.gov/cc/capandtrade/capandtrade.htm>
13. <http://www.climateactionreserve.org>
14. <http://www.arb.ca.gov/cc/scopingplan/economics-sp/economics-sp.htm>
15. http://www.analysisgroup.com/uploadedFiles/Publishing/Articles/Economic_Impact_RGGI_Report.pdf
16. <http://ab32ig.com/documents/Tanton%20Study%20FINAL.pdf>
17. <http://www.edf.org/sites/default/files/EDF-Cap-and-Trade-FAQ-January-2012.pdf>
18. <http://www.sacbee.com/2012/10/02/4872539/new-california-laws-set-up-framework.html>
19. <http://www.westernclimateinitiative.org>
20. <http://www.arb.ca.gov/newsrel/newsrelease.php?id=300>

Index

A

- AB 32. *See* Assembly bill 32 (AB 32)
- AC. *See* Air conditioning (AC)
- Acute lower respiratory tract infections (ALRI), 241, 263
- Advance notice of proposed rulemaking (ANPR), 366
- Aedes aegypti* life cycle and climatic factors
relative humidity, 171–172
temperature, 170–171
- Aeroallergens
anthropogenic climate change, 163
asthma and ozone study, 163
earth's temperature, 163
Hubbard Glacier Alaska, 163, 164
reactive oxygen species (ROS), 162
urban air pollution, 163
- Airborne allergens, 122
- Air conditioning (AC)
apparent temperature-hospitalization, 77
racial disparities, 72
- Air pollution and children's health
long-term effects, 276
short-term effects, 275–276
- Air pollution and health, 143, 147
- Air pollution effects on mothers' and children's health
child health (*see* Air pollution and children's health)
children's health
lower birth weight and prematurity, 275
risk, obesity, hypertension and cardiovascular disease, 275
- Air quality and climate change
air pollution co-benefit estimation, 146, 147
changes, ground-level ozone, 38, 39
cost of illness (COI) method, 148
costs and benefits, 147–148
estimated potential human health benefits, 147, 148
European Environmental Agency, 149
GHG, 138, 149, 150
health consequences, 145, 146
health effects, PM (*see* Particulate matter (PM) time series studies)
implications, 150
mortality risk reductions, 149
pollution and health relationships, 149
public health changes, 150
quality-adjusted life year (QALY)
approaches, 148
stagnant weather patterns, 36
tropospheric ozone (*see* Ozone exposures)
willingness to pay (WTP) methods, 148
- Algae. *See* Dinoflagellates
- Allergic rhinitis, 130, 133
- Allergies and climate change. *See* Pollen and climate change
- Allergy medication
OTC, 131–133
pollen (*see* Pollen forecasting)
tree pollen peak dates, 131
urban area, France, 133
- ALRI. *See* Acute lower respiratory tract infections (ALRI)
- Amazon climate change
Anopheles, 196
coupled mathematical nonlinear models, 195
ENSO, 194
forest, savannah-like conditions, 195
global climate models (GCMs), 195

- Amazon climate change (*cont.*)
 IPCC project, 195
 sea surface temperatures (SST), 194
 temperatures, 194–195
 and vector-borne disease
 (*see* Vector-borne disease in the Amazon)
- ANPR. *See* Advance notice of proposed rulemaking (ANPR)
- Anthropogenic-induced changes, 31
- Anthropogenic sources, burning oil and natural gas, 2
- AR4. *See* Assessment report 4 (AR4)
- Architecture, house and biomass smoke exposure
 agricultural crop residues, cooking medium indoors, 261, 262
 assessments, 260
 chimney and ventilatory measures, 260
 climatic and cultural variations, 260
 crop residues, cooking medium outdoors, 260, 261
 fire wood, cooking medium indoors, 261, 262
 outdoor area measurements, 263
 preparation, cow dung cakes, 260, 261
- Assembly bill 32 (AB 32), 384
- Assessment report 4 (AR4), 280
- Asthma and climate change
 and allergies, 158
 geo-climate effects, 160
 ozone, 163
 pollen, 161
- Atmospheric temperatures, climate changes
 AR4, 280
 IPCC, 280, 281
 island air temperatures, 281
 ocean surface, 281
 SRES models, 280
 tropical small island states, 281
- B**
- BenMAP, 327–328
- Biodiversity, 300–301
- Biomass
 and coal, 238
 COPD and exposure, 243
 exposure, 244
 fuels, 238
 lung cancer, 242
 ocular disorders, 242
- Biomass fuels
 architecture (*see* Architecture, house and biomass smoke exposure)
- coal, 258
 in India (*see* India experience with biomass fuel use)
 indoor air pollution, 259
 LPG and natural gas, 258
 lung diseases (*see* Lung disease and biomass fuel use)
 reduction strategy, 269
 source, domestic energy, 258
- Biomass stove and exposure determination,
 lung disease
 benzopyrene, 260
 burning, 259–260
 carbon monoxide, 260
 formaldehyde, 260
 human carcinogens, 260
 PAHs, 260
 pathological effect, 260
 physical and chemical products, 259
 solid particles, 260
- Biosphere consequences
 changes, Arctic regions, 7
 glaciers rate of change, global warming, 7, 8
 Greenland ice sheet melted, summer, 5, 6
 health of Carysfort Reef, 9, 10
 Larsen B ice shelf breakup, Antarctic Peninsula, 5, 6
 sea-level rise, mechanisms, 9
 temperate glaciers, 5, 7
- BRACE. *See* Building resilience against climate effects (BRACE)
- Building resilience against climate effects (BRACE)
 anticipation, climate impacts and assessing vulnerabilities, 346–349
 climate and health adaptation plan, 353–354
 development, quality of activities, 354–355
 disease burden, 349–352
 five-step process, 345–346
 framework, 344–345
 impacts, climate-sensitive health outcomes, 344
 public health interventions, 352–353
 risk assessment, 344
 vulnerability, 345
- C**
- CAA. *See* Clean Air Act (CAA)
- California
 biologic mechanisms, 80
 global warming impacts, 71
 heat warning systems, 81
 morbidity and temperature studies, 77–79

- projections, climate and mortality, 80
- public health impacts, 80
- temperature and mortality studies
 - (*see* Temperature and mortality studies, epidemiology)
- California Air Resources Board (CARB)
 - AB 32, 384, 385
 - cap-and-trade system, 384, 390
 - protocols, 388
 - scoping plan, 384
- California Global Warming Solutions Act, 284
- California's cap and trade program
 - AB 32, 384
 - allowances, 388
 - benchmark process, 388
 - CARB, 384, 387
 - CO₂ emissions (*see* CO₂ emissions, reduction)
 - command and control strategy, 384
 - covered entities, 387
 - development, 387
 - economic impact analysis, 389
 - enormity, climate changes, 383
 - GHG emissions, 383, 384
 - legislation, 389
 - manufacturing hydraulic cement, 389
 - reserve price, 388
 - RGGI program, 389
 - scoping plan, 384
 - WCI, 390
- California waiver, 369
- Cap and trade program in California.
 - See* California's cap and trade program
- CARB. *See* California Air Resources Board (CARB)
- Carbon capture and storage (CCS), 371
- Caribbean and global warming. *See* Small island states and climate change
- CCAS. *See* Climate change adaptation strategies (CCAS)
- CCHHG. *See* Climate change and human health group (CCHHG)
- CCMS. *See* Climate change mitigation strategies (CCMS)
- CCS. *See* Carbon capture and storage (CCS)
- CDC National Environmental Public Health Tracking Network, 355–356
- CDC policies on climate change adaptation.
 - See* Climate change and the CDC
- Centers for Disease Control and Prevention (CDC), 342
- Challenges of traditional risk assessment
 - adverse health outcomes, 308–309
 - agent, 308
 - assumptions, 308, 309
 - climate-sensitive health outcomes, 309
 - data sets analysis, 310
 - exposure-response relationships, 308
 - global warming, 310
 - morbidity and mortality, health outcomes, 309
 - multiple weather variables, 309
 - Vibrio parahaemolyticus*, Alaska, 309
 - weather variables, 309
- Change in surface temperature
 - differences, annual global temperature, 32, 33
 - global trend, maximum and minimum, 32, 34
 - NOAA, 32, 33
 - observed and projected increase, 35, 37
 - precise rise, greenhouse gas concentrations, 35, 38
 - projected changes, US, 35, 36
 - trend, annual average surface, 32, 33
- Changing distribution of conifers, 42
- Children's health and climate change, 275
- Chronic obstructive pulmonary disease (COPD)
 - biomass, 243
 - risk, 266
 - women, 263
- Clean Air Act (CAA)
 - air pollutant, 362
 - decision making process, 369
 - operating permits, 367
 - thresholds, 367
- Climate and Health Program
 - adaptation activities, CDC, 342
 - control, 343
 - framework, EPHS, 343
 - functions, public health adaptation, 342
 - health risks, 342
 - NASA, NCA, and IPCC, 343
 - NCEH, 342
 - NOAA, 342–343
 - symposium, 357–358
- Climate change
 - airborne allergens, 122
 - air pollution, 122
 - and air quality (*see* Air quality and climate change)
 - Amazon (*see* Vector-borne disease in the Amazon)
 - and asthma (*see* Asthma and climate change)
 - Bali Road Map, 60–61
 - black carbon and ozone, 250

- Climate change (*cont.*)
- changes, surface temperature, 32–36
 - China, 62
 - consequences, biosphere
 - (*see* Biosphere consequences)
 - cookstove project, 250–251
 - Copenhagen Accord, 61
 - COP17 reflection, Durban Platform, 63–65
 - CO₂ temperature, 156
 - desertification (*see* Desertification)
 - disastrous effects, black carbon and ozone, 250
 - disease, 159–162
 - estimation, IPPC, 297
 - and federal programs
 - (*see* Federal programs in climate change and health research)
 - finance, response measures, 62
 - and forest health (*see* Forest health)
 - global environmental change, 123
 - global warming, Himalayan-Tibetan region, 249–250
 - greenhouse gas emissions, 294–295
 - growth, water use and phenological phases, 158
 - impacts, 294
 - India, 62
 - indoor and outdoor air pollution, 251
 - Kyoto Protocol, 60
 - middle east countries, 294
 - non-allergenic air pollution, aeroallergens, 162–164
 - ozone (O₃) (*see* Ozone (O₃))
 - participation, COP15, 65–67
 - planet, peril, 241
 - plant and pollen distribution, 157–158
 - pollen seasons, 156, 157
 - Post COP15, 63
 - potency/allergenicity, 156
 - precipitation, Aegean and Mediterranean coasts, 296
 - rainfall, 296–297
 - risk, 238
 - short-lived climate forces, 251
 - summer temperatures, Turkey, 295–296
 - technology, Copenhagen Accord, 63
 - thriving global market, 241
 - UNEP, 294
 - UNFCCC, 60
 - United States, 62
 - and variability, Climate variability and change
 - worsening asthma and allergies, 158
- Climate change adaptation
- and public policy. *See* Climate change and the CDC
- Climate change adaptation strategies (CCAS), 87
- Climate change and air quality. *See* Air quality and climate change
- Climate change and dengue fever. *See* Dengue fever and climate change
- Climate change and effects on mothers and children
- child health, 275
 - disease and air pollution, 273
 - and food safety, 275
 - health impacts, 273
 - maternal health, 274
- Climate change and food and water. *See* Water and food-borne diseases
- Climate change and health risks
- determinants, 310–311
 - impacts, 307
 - infrastructure, 308
 - IPCC assessment (*see* Intergovernmental Panel on Climate Change (IPCC) assessment)
 - literature, 315
 - migration, 308
 - patterns, weather, 307
 - traditional risk assessment (*see* Challenges of traditional risk assessment)
 - weather events, 308
- Climate change and human health group (CCHHG)
- adaptation task force, 325–326
 - agencies, 323
 - creation, GCRP, 324
 - description, charter, 323–324
 - federal agency adaptation plans, 326
 - identificatin, priority workstreams, 324
 - implications, 323
 - report, National Research Council (NRC), 323
 - workstream activity, 325
- Climate change and the CDC
- adaptive management and functions, 343–344
 - BRACE (*see* Building resilience against climate effects (BRACE))
 - CRSCI, 356–357
 - description, 342
 - and health program, 342–343
 - integration, 342
 - NCEH, 355–356
 - science symposium, 357–358

- Climate change, cap and trade
 - carbon tax, 385
 - GHG emissions, 383
 - harmonization, 390
 - Climate change in the Amazon. *See* Amazon climate change
 - Climate change in the desert. *See* Desertification
 - Climate change mitigation strategies (CCMS), 87
 - Climate data
 - assessment, partners, 29–30
 - changes, climate, 24
 - data culture, 22, 23
 - early warning, 25
 - forecasts, 25
 - global warming, 24
 - in situ* data, 27
 - local/regional climate, 24
 - NOAA seamless suite, forecasts, 23
 - prediction, 25
 - products, 27–28
 - projections, 26, 28–29
 - reanalysis, 28
 - satellites, 27
 - scale, 26–27
 - weather, 23
 - Climate-induced forest mortality, 41–42
 - Climate-Ready States and Cities Initiative (CRSCI), 356–357
 - Climate-sensitive health outcomes
 - determinants, 311
 - traditional risk assessment, 309–310
 - Climate variability and change
 - biofuel production, 228
 - “co-benefits,” 226–227
 - environmental public health researchers, 230
 - ethical considerations, 229
 - extreme weather events and health (*see* Weather events and health)
 - financial considerations, 229–230
 - fisheries, 217
 - food production and drought, 215–217
 - health co-benefits, greenhouse gas mitigation, 227–228
 - instrument records, 211
 - land-use effects, weather and health (*see* Land-use effects, weather and health)
 - mitigation and adaptation, 226
 - net effect, food production, 214
 - and sea level rise and extremes (*see* Sea level rise in climate change)
 - temperature changes, 212
 - vulnerable geographic regions, 214
 - water and food-borne diseases (*see* Water and food-borne diseases)
 - Climate variability and health, 21, 24
 - CO₂ emissions, reduction
 - cap-and-trade system, 385–387
 - carbon tax, 385, 386
 - cost-effective, 386
 - environmental integrity and flexibility, 387
 - fossil fuels, 385, 386
 - GHG emissions, 385
 - political pressures, 387
 - tax legislative process, 386
 - Combined air quality and climate impact
 - air pollutant concentrations, 377
 - cookstoves, 379
 - decision-making, 377
 - emissions, production and energy, 380
 - greenhouse warming, 379
 - human health and ecosystems, 377
 - NAAQS, 378
 - ozone and particulate matter, 378
 - tropospheric ozone, 378
 - WHO, 379
 - CO₂ measurements
 - air bubbles, Antarctic ice, 3
 - and annual averages, 2
 - Complexity of health risk evaluation, 309
 - Conferences of the Parties (COP), 15, 16
 - Cooking fires
 - adoption, fuel technology, 239
 - evolution, 238
 - health and environmental risks, 238
 - poor house, 238
 - social and cultural changes, 239
 - solid fuels (*see* Solid fuels and cooking solutions)
 - women activities, 238–239
 - COP. *See* Conferences of the Parties (COP)
 - COPD. *See* Chronic obstructive pulmonary disease (COPD)
 - Coral bleaching, 9, 10
 - Cox proportional hazard model, 11
 - CRSCI. *See* Climate-Ready States and Cities Initiative (CRSCI)
- D**
- Dengue disease, 168–169
 - Dengue fever and climate change
 - adaptive immune response, 168
 - description, 167–168
 - DHF, 168–169
 - indirect effects (*see* Regional implications, dengue fever)
 - non-climatic risk factors, 176
 - vectors and transmission, 169–170

Dengue fever and climate change (*cont.*)
 and viral parameters, transmission cycle
 (*see* Viral parameters, dengue
 transmission cycle)
 virus, 168
 Dengue hemorrhagic fever (DHF), 168–169
 Desertification
 annual and winter precipitation, 297–298
 biodiversity, 300–301
 dust storms, 299
 estimation, Middle East region, 298
 human health, 301–303
 sandstorms, 299
 water usage, 299–300
 Determinants of health risk, 310–311
 DHF. *See* Dengue hemorrhagic fever (DHF)
 Dinoflagellates, 9
 Disease burden and climate change, 345,
 349–352
 Disease exacerbation in small island states, 280
 Diurnal temperature range (DTR), 32
 DTR. *See* Diurnal temperature range (DTR)
 Dust storms, 299

E

“Earth summit,” 15
 El Niño Southern Oscillation (ENSO),
 174–175, 194
 Emissions reporting requirements
 ANPR, 366
 CAA, 365–368
 EPA regulation, GHGs, 365
 GHG, 368
 GHG emissions, 366
 mandatory reporting rule, 366–367
 operating permits, 367, 368
 permitting requirements, 368
 PSD, 367, 368
 stationary sources, 368
 tailoring rule, 367–368
 thresholds, 367, 368
 ENSO. *See* El Niño Southern Oscillation
 (ENSO)
 Environmental health multiple-determinant
 model
 HRI and HRMM, 92
 MDM, 90
 stages, 90, 91
 vulnerability factors, 91
 Environmental Protection Agency (EPA)
 adaptation and vulnerability, 327
 BenMAP, 327–328
 and CAA, 362

California waiver, 369
 description, 327
 emissions reporting requirements
 (*see* Emissions reporting requirements)
 functions, 327
 GHG emissions (*see* Greenhouse gas
 (GHG) emissions)
 health and climate change (*see* Health and
 climate change)
 human health and the environment, 361
 impacts, climate change, 361, 362
 light-duty vehicle rules, 369–370
 vs. Massachusetts, 369
 medium and heavy-duty vehicle rule, 370
 regulatory standards, outdoor pollution,
 242–243
 research activities, 327
 short-lived climate pollutants, 372–376
 solid fuels and cooking solutions, HAP, 239
 statutory requirements, 327
 EPA. *See* Environmental protection
 agency (EPA)

F

Famine Early Warning System Network
 (FEWS NET), 330
 Federal programs in climate change and health
 research
 adaptation task force, 325–326
 CCHHG and workstream activity, 324–325
 EPA, 327–328
 federal agency adaptation plans, 326
 GCRA and GCRP, 321–323
 HHS, US, 333–338
 NASA, 328
 NIST, 333
 NOAA, 331–333
 NSF programs, 328–329
 prior to 1990, 320–321
 USAID, 329–331
 USDA, 331
 FEWS NET. *See* Famine Early Warning
 System Network (FEWS NET)
 Food security
 Caribbean’s agricultural model, 285
 impacts, climate change, 286
 ocean acidification, 286
 SIDS, 285
 Forest health and climate change
 “aggressive” bark beetles, 42–43
 conditions, seasonally dry forests, 40, 41
 conservative scenarios, 41
 distribution, coniferous vegetation, 42

increased wildfire activity, 43
 loss, whitebark pine (*P. albicaulis*), 44, 45
 natural disturbances, European forests, 40
 phytophagous insects, 42, 43
 western pine beetle, 43, 44

G

GCMs. *See* General circulation models (GCMs)
 GCRP. *See* Global Change Research Program (GCRP)

GDP. *See* Gross domestic product (GDP)

General circulation models (GCMs), 29

GHCN dataset. *See* Global Historical Climate Network (GHCN) dataset

GHG emissions. *See* Greenhouse gas (GHG) emissions

Global burden of disease (GBD), 241–242

Global Change Research Act of 1990 (GCRA), 321

Global Change Research Program (GCRP) activities, 322

areas, 322

earth's climate, 321

economic and social impacts, 321

elements, 322

federal research and observation, global environmental change, 321

and GCRA, 321

interagency CCHHG (*see* Climate change and human health group (CCHHG))

and NRC assessment, 321–322

report, IPCC, 322–323

2012 strategic plan, 322

Global consequences for U.S., 372

Global Historical Climate Network (GHCN) dataset, 32, 33, 34

Global Ocean Observing System (GOOS), 28

Global public health, climate data.

See Climate data

Global warming

Arctic Ocean, 55

Arctic sea ice, 56

challenges of traditional risk assessment, 310

climate change (*see* Climate change)

climate education, 68

crossing Antarctica, 52, 53

expedition team, 56–57

Hall Peninsula, 57, 58

International Arctic Project expedition team, 55, 56

Larsen ice shelves, 54

“nature deficit disorder,” 69

North Pole Expedition team, 52

quality environmental education materials and educator support, 69

sea ice, 55

unusual ice and snow conditions, 58

Will Steger Foundation's mission, 67, 68

windblown snow, 52, 53

young explorers, ruins of Arctic Ocean summer ice melt, 59

Global warming and caribbean. *See* Small island states and climate change

Global warming, public's health

climate change consequences, biosphere Biosphere consequences)

consequences, 9–12

efforts, mediation and regulation, 15–17

greenhouse gases and temperature, 2–5

social stability, 13–15

GOOS. *See* Global Ocean Observing System (GOOS)

Greenhouse gas (GHG) emissions.

See also Human health and GHG;

Temperature and GHG

AB 32 scoping plan, 384

air pollutants, 362, 367

and CAA, 371

California, 379

CARB, 384

and CCS, 371

CO₂ emission, 295

covered entities, 387

economists, 390

endangerment finding, 373

energy consumption, 295

enormity, climate change, 383

fossil fuels, 385

GDP measures, 295

general energy and electricity, 294–295

health and climate change, 363–365

long-lived, 372

manufacturing hydraulic cement, 389

Massachusetts vs. EPA, Supreme Court, 362–363

and NSPS, 371

operating permits, 377

performance standards, 371

reductions, 384

short-lived, 378–379

solar energy, middle east countries, 295

stationary sources, 370–371

tailoring rule, 370

thresholds, 368

Turkey, 294

US emissions, 372

Gross domestic product (GDP), 295

H

- Hansen's approach, 17
- HAP. *See* Household air pollution (HAP)
- Hay fever and climate change, 160, 161
- Hay fever and pollen, 160
- Health and climate change
 - Agency's regulatory efforts, 365
 - air pollution, 366
 - air quality and temperature, 366
 - American communities, 366
 - assessments, 364
 - combined air quality and climate impact, 377–381
 - endangerment finding, 363–365
 - evidence, 364
 - federal register (FR), 363
 - GHG air pollution, 363
 - impacts, air quality, 364
 - lawsuits, 365
 - mortality and morbidity, impacts, 364
 - petitions, 365
- Health consequences of climate variability, 21–22
- Health effects of ecological changes, 274
- Health impacts, HAP
 - ALRI, 241
 - biological response, 243
 - biomass, 242
 - burns, 242
 - children, low socio-economic status, 243
 - chronic exposure, 242
 - COPD, 241
 - EPA regulatory standards, outdoor pollution, 242–243
 - epigenetics and pregnancy, 244
 - exposure-response data, 242
 - exposure-risk association, 244
 - GBD, 241–242
 - genetic expression, 243
 - host genetics, 243
 - human health and climate, 251–252
 - interventions, cookstoves/fuels, 247–249
 - reduction, climate impacts, 249–251
 - regulation, oxidative stress, 243
 - risks, 242
 - susceptible and vulnerable populations, 244
 - women and children, low socio-economic status, 243
- Health research and climate change
 - EPA, 327–328
 - HHS, 333–338
 - national aeronautics and space administration, 328
 - NIST, 333
 - NOAA, 331–333
 - NSF programs, 328–329
 - USAID, 329–331
 - USDA, 331
- Heat acclimatization, 87, 95–96
- Heat-health action plans
 - environmental and population data, 110–111
 - “extreme heat events” (EHE), 110
 - geospatial analysis, 111–112
 - health-protective measures, 113
 - home air conditioner, 114
 - HRMM, 115
 - local-scale population and environmental information, 111
 - older adults, 111
 - principles and core elements, 110, 111
 - public health messaging, 113, 114
 - social service and healthcare, 114
 - vital statistics death data, 112
 - Zip Code-level analysis, 113
- Heat-related illness (HRI)
 - cramps, edema, syncope and exhaustion, 97
 - ED-visit risk, 112
 - epidemiologic studies, 89
 - pathophysiology, 88, 92, 99–100
 - stroke, 98
 - thermoregulatory capacity
 - Thermoregulatory capacity)
- Heat-related morbidity/mortality (HRMM)
 - children, 101, 103
 - chronic diseases, 90
 - description, 87
 - “extreme heat events” (EHE), 106, 110
 - heat-health action plans Heat-health action plans)
 - and HRI, 107
 - potential exposures and ambient heat, 108–110
 - quality healthcare, 107–108
 - sex/gender, 103
 - “top-down” and “bottom-up” approach, 107, 115
- Heat stress
 - biological adaptations, 92
 - description, 87
 - and heat strain, 107, 109
- Heat stroke, 98, 99
- Heat waves and climate change
 - acclimatization and thermotolerance, 95–96
 - biological adaptations, heat stress, 92
 - environmental conditions, 86
 - epidemiologic studies, 89–90
 - “extreme heat events” (EHE), 86, 87
 - geographic patterns, 87

- global environmental and societal challenges, 105–106
 - HRI, 97
 - HRMM
- Heat-related morbidity/mortality (HRMM)
 - MDM (*see* Multiple determinant models (MDM))
 - pathophysiology, 99–100
 - protection, public health, 88
 - stroke, 98
 - thermoregulatory capacity (*see* Thermoregulatory capacity)
 - thermotolerance, acclimatization and thermoregulation, 93–94
- Household air pollution (HAP)
 - biomass fuels, 238
 - black carbon and short-lived climate forcers, 246–247
 - clean fuels uses, 252
 - climate change (*see* Climate change)
 - cooking fires (*see* Cooking fires)
 - emissions, 238
 - environmental degradation, 238
 - exposure-responses, 252
 - health impacts (*see* Health impacts, HAP)
 - interventions, cookstoves/fuels, 247–249
 - outdoor air pollution, 245–246
 - poverty, 252
 - regional environmental degradation, 244–245
 - solid fuels (*see* Solid fuels and cooking solutions)
 - stove testing, 241
- HRI. *See* Heat-related illness (HRI)
- HRMM. *See* Heat-related morbidity/mortality (HRMM)
- Human health
 - air pollutants, ozone, 302
 - direct and indirect effects, climate change, 301
 - heat waves, 302
 - impacts, climate change, 301–302
 - infectious diseases, warmer periods, 302
 - malnutrition and hunger, 303
 - Middle East countries, 301
 - water shortages, 303
- Human health and climate change. *See* Climate variability and change
- Human health and GHG
 - air pollution co-benefit estimation, 146
 - fossil-fuel combustion processes, 138
 - PM pollution, 147
 - public health costs and benefits, 149–150
- Human health consequences
 - heat waves, 10–12
 - vector-borne diseases, 12
- Hydrology and storm events
 - agriculture and food security, 283
 - freshwater resources management, 283
 - natural disasters, 282
 - SIDS, 282
 - small islands, 282
- I**
- ILD. *See* Interstitial lung diseases (ILD)
- Increased mortality, 73, 75–77, 80
- India experience with biomass fuel use
 - cow dung cakes, 258
 - domestic cooking, 258
 - solid fuels, 258
 - urban households cooking, 258
- Indoor air pollution
 - agricultural crop residues, 261, 262
 - fire wood, 261, 262
 - lung disease and biomass fuel use, 259
- Intergovernmental Panel on Climate Change (IPCC) assessment
 - establishment, 312
 - experts, 313, 314
 - government functions, 313
 - organizations, 313
 - peer-reviewed and non-peer-reviewed literature, 312
 - periodic, 311
 - policymakers and users, 313–314
 - principles and procedures, 313
 - process, fifth assessment report, 314–315
 - reports and guidance, 312
 - SPM, 313
 - working groups (WGs) and contribution, 313, 314
- Interstitial lung diseases (ILD), 267
- IPCC assessment. *See* Intergovernmental Panel on Climate Change (IPCC) assessment
- L**
- Land use and land cover change (LUCC), 193, 196
- Land-use effects, weather and health ecosystems, 225
 - urban heat island effect, 224, 225
- Liquified petroleum gas (LPG), 268
- LPG. *See* Liquified petroleum gas (LPG)

- LUCC. *See* Land use and land cover change (LUCC)
- Lung disease and biomass fuel use
 ALRI, children, 263
 biomass stove and exposure determination (*see* Biomass stove and exposure determination, lung disease)
 cancer, 265
 COPD, 263, 265
 exposure and lung functions, 268
 indoor air pollution, 259
 pneumoconiosis and ILD, 267
 risk, 264
 TB and asthma, 263, 265–267
 tuberculosis and asthma, 263, 265–267
- M**
- Malaria-climate change
Aedes aegypti, 205
 air temperature and *P. falciparum*, 203, 204
Anopheles mosquitoes, 199–200
 blood sample, 201
 case detection, treatment, and bednet distribution, 201
 larval and adult *Anopheles* habitat, 200
 Peruvian Amazon region, 200
P. falciparum and *P. vivax*, 202
 stages, 199
 transmission season expansion, 203
 treatment and development of immunity, 203
 VBZD, 199, 205
- Massachusetts vs. EPA, Supreme Court
 air pollutants, 362, 363
 CAA, 362
 endangerment finding, 363
 IPCC, 362
 state and environmental petitioners, 363
- MDM. *See* Multiple determinant models (MDM)
- Middle East
 biodiversity, 300, 301
 CO₂ emissions, 295
 consequences, climate change, 294
 global climate changes, 294
 human health, 301
 prediction, 294
 risk, malnutrition and hunger, 303
 sandstorms, 299
 solar energy, 295
 surface temperature and rainfall reduction, 297
 UNDP, 294
 water consumption, 300
 water-stressed regions, 300
- Morbidity and temperature studies, epidemiology
 adverse birth outcomes, 78–79
 biologic mechanisms, 80
 El Niño events, 79
 health outcomes, 72
 and hospitalizations, 77–78
- Mortality
 analysis, 124
 heat-related, 126, 128, 129
 ozone (O₃), 124, 126, 128, 129
- Mortality displacement
 apparent temperature, 77
 effects, 75
 harvesting, 75
 mean apparent temperature, 75, 76
 non-accidental mortality, 75, 76
- Mosquito-borne disease. *See* Dengue fever and climate change
- Multiple determinant models (MDM)
 heat-related morbidity and mortality, 91–92
 risk factors, 90
 vulnerability factors, 91
- N**
- NAAQS. *See* National Ambient Air Quality Standards (NAAQS)
- NAPAs. *See* National Adaptation Programs of Action (NAPAs)
- NASA. *See* National Aeronautics and Space Administration (NASA)
- National Adaptation Programs of Action (NAPAs), 330
- National Aeronautics and Space Administration (NASA)
 climate change and human health activities, 328
 climate data, 328
 education program, 328
 global change research, 328
 satellite observations and development, earth system, 328
- National Ambient Air Quality Standards (NAAQS), 378
- National Center for Environmental Health (NCEH), 342
- National Institute of Standards and Technology (NIST), 333
- National Institutes of Health (NIH), US
 climate change and health, 336, 337
 climate change grant program, 336
 health outcomes and exposures, 335
 interagency, 335
 and NIEHS, 335, 336, 338
 research, 335

National Oceanic and Atmospheric Administration (NOAA) and Centers for Disease Control (CDC), 332–333
 climate change activities, 331
 climate predictions and services, 331–332
 GHCN, 32, 33 34
 implementation, 332
 monitoring and prediction, 332
 OHHI-supported initiatives, 332
 RCCs, 29
 seamless suite, forecasts, 23
 National Science Foundation (NSF) programs, 328–329
 “Nature deficit disorder,” 69
 NCDs. *See* Non-communicable chronic diseases (NCDs)
 New source performance standards (NSPS), 371
 NGOs. *See* Nongovernmental organizations (NGOs)
 NIST. *See* National Institute of Standards and Technology (NIST)
 NOAA. *See* National Oceanic and Atmospheric Administration (NOAA)
 Non-communicable chronic diseases (NCDs), 288
 Nongovernmental organizations (NGOs), 66
 North Pole global warming changes, 52, 55, 56
 NSF programs. *See* National Science Foundation (NSF) programs
 NSPS. *See* New source performance standards (NSPS)

O

Outdoor air pollution, HAP, 245–246
 Ozone (O₃)
 A2 greenhouse gas emissions, 126, 128
 climate effect, 123
 comparison, mortality, 126, 129
 description, 122
 epidemiologic analysis, 124
 Goddard Institute for Space Studies (GISS), 125
 health impacts, 124, 126
 vs. heat, 126, 129
 implications, 127
 regional distribution, heat-related mortality, 126, 127
 and temperature, 126
 Ozone (O₃) exposures
 emissions, fossil fuel, 142
 long-term mortality effects, 144–145
 nitrogen oxides (NO_x), 142
 short-term effects, 142–144
 volatile organic compounds (VOCs), 142

P

Particulate matter (PM) time series studies
 health effects, 140–141
 long-term exposure effects, 139–140
 short-term exposure effects, 138–139
 Partnership for clean indoor air (PCIA), 239
 PCIA. *See* Partnership for clean indoor air (PCIA)
 Phytophagous insects, 42, 43
 Polar exploration and global warming, 65, 69
 Pollen and climate change
 allergic symptoms, 159
 asthma and allergic diseases, 160
 Europe over time, 159
 global warming, 160
 hay fever, 160
 inhalation, disease, 161, 162
 physical examination signs, 160, 161
 potency/allergenicity, 156
 and temperature, 156, 157
 Pollen forecasting
 allergenic pollen protein, 129–130
 allergic rhinitis, 130
 asthma, 130
 climate factors, 133
 CO₂ concentrations, 129, 130
 data and methods, 130–131
 estimated impacts, tree pollen peaks, 131, 132
 human health, 133
 OTC allergy medication, 131–133
 seasonal onset, 128
 Potential socioeconomic effects
 carbon tax, 283–284
 energy, water, and waste management, 284–285
 food security, 285–286
 fossil fuels, 284
 infrastructure and population displacement, 287
 renewable energy, 284
 SIDS, 284
 sustainable development, 284
 tourism, 286–287
 waste management, 285
 water resources management, 284
 Poverty and climate change, 252
 Precipitation
 Aegean and Mediterranean coasts, 296
 and aridity index series, 297
 drier conditions, annual, 297, 298
 reduction rate, 297
 temperature, 294
 winter, 294, 296, 297

- Prevention of significant deterioration (PSD), 367, 368
- Projection, 124, 125, 127
- PSD. *See* Prevention of significant deterioration (PSD)
- Public health burden, heat waves
health risks, 113
HRMM (*see* Heat-related morbidity/mortality (HRMM))
individual clinical, 111
- Public health in Caribbean
chronic respiratory diseases, 289
freshwater resources, 288
health impacts, 288
NCDs, 288
PM pollution, 289
rising temperatures, 288
- Public health policy on climate change, 352–353
- R**
- Radiative forcing, 3, 4, 380
- RCCs. *See* Regional Climate Centers (RCCs)
- Regional Climate Centers (RCCs), 29
- Regional greenhouse gas initiative (RGGI), 389
- Regional implications, dengue fever
Africa, 181–182
Asia, 177–178
Australia and New Zealand, 178–179
Europe, 182–183
Latin America, 183–184
The Middle East, 180
small island nations, 179–180
The United States, 185–186
- Research in climate change
CCHHG (*see* Climate Change and Human Health Group (CCHHG))
GCRP and GCRA (*see* Global Change Research Program (GCRP))
prior to 1990, 320–321
- RGGI. *See* Regional greenhouse gas initiative (RGGI)
- Rising temperatures in climate change.
see Heat waves and climate change
- S**
- Sandstorms, 299
- Sea level rise and ocean acidification
AR4, 281
concentration, carbon dioxide, 282
SIDS, 282
tectonic shifts, 281
thermal expansion, 281
- Sea level rise in climate change
sea ice record melting, 212, 213
surface temperatures and hurricanes, 212
- Short-lived climate pollutants
air pollutants, 372
black carbon, 372, 373
Clean Air Coalition, 374
EPA, 372
health and environmental effects, 373
methane, 372, 373
mobile diesel sources, 374
National Clean Diesel Program, 374–375
PM, 372
public health improvements, 376
task force, 375
tropospheric ozone, 373
VOC emissions, 373
WMO, 375
- SIDS. *See* Small island developing states (SIDS)
- Small island developing states (SIDS)
agriculture and food security, 283
ocean acidification, 282
water resources management, 284
- Small island states and climate change
atmospheric temperatures, 280–281
ecosystem structures, 283
human health, 279
hydrology and storm events, 282–283
potential socioeconomic effects, 283–287
public health in Caribbean, 288–290
sea level rise and ocean acidification, 281–282
tropical/subtropical, 280
- Social stability
climate change, 13
global warming, 13
US military, 14–15
weather-related disasters, 14
- Societal impact of climate change, 32, 226, 323, 328
- Solar energy, middle east countries, 295
- Solid fuels and cooking solutions
adverse impacts, 241
CO₂ emission, 240
cookstove efficiencies and affordability, 239, 240
development, cookstove programs, 239
exposures reduction, 240
Global Alliance, 241
human health and environment, 241
PCIA and EPA, 239
PM_{2.5} and CO, 240
three-stone fire, 239

Special Report on Emissions Scenarios (SRES), 280
 SRES. *See* Special Report on Emissions Scenarios (SRES)
 Steger eyewitness account in polar explorations. *See* Global warming
 Summary for policymakers (SPM), 313
 Syndromic surveillance, 133

T

TB. *See* Tuberculosis (TB)
 Temperature
 CO₂ concentrations, 122
 and ozone (O₃) (*see* Ozone (O₃))
 Temperature and GHG
 CO₂ concentration, 2
 decay, fossil fuel CO₂ emissions, 3
 IPCC, 5
 methane, 3–4
 Temperature and hospitalizations, morbidity exposure assessment, 77
 heat waves, 78
 impact, AC, 77
 mean apparent temperature, 77
 time-stratified case-crossover, 78
 Temperature and mortality studies,
 epidemiology
 air pollutants, 74, 75
 cardiorespiratory mortality, 72–73
 heat waves, 72, 76–77
 humidity, 73
 investigators, 72–74
 mean apparent temperature, 74, 75
 mortality displacement, 75–76
 regional analyses, 72
 time-stratified case-crossover design, 72, 73
 vulnerable subgroups, 75
 Thermoregulatory capacity
 genetics/epigenetics, 104
 infants and children, 101–103
 older adults, 100–101
 race/ethnicity, 104
 sex/gender, 103–104
 Tuberculosis (TB)
 agricultural crop residues, 265, 266
 fire wood, 265–267
 in India, 265
 risk, 265
 Turkey
 annual mean precipitation, 296
 biodiversity, 301
 desertification, 297, 298
 greenhouse gas emissions, 294, 295

infectious diseases, 302
 mean annual temperature, 296
 rainfall, 296–297
 sand and dust storms, 299
 summer temperatures, 294, 295
 UNEP, 294
 water consumption, 300
 winter precipitation, 294, 296

U

UNEP. *See* United Nations Environment Program (UNEP)
 UNFCCC. *See* United Nations Framework Convention on Climate Change (UNFCCC)
 United Nations Environment Program (UNEP), 294
 United Nations Framework Convention on Climate Change (UNFCCC), 60, 65, 66, 338
 Urbanization and dengue fever
 A. aegypti, 169
 A. albopictus, 170
 Africa, 181
 Asia, 178
 climate change, 177
 Latin America, 183, 184
 The Middle East, 180
 small island nations, 179
 transmission, 176
 The United States, 185
 U.S. Agency for International Development (USAID) climate change and development strategy
 description, 329
 FEWS NET, 330
 NAPAs, 330
 SERVIR, 330–331
 training and guidance, 329–330
 USDA. *See* U.S. Department of Agriculture (USDA)
 U.S. Department of Agriculture (USDA), 331
 U.S. Department of Health and Human Services (HHS)
 CDC Climate-Ready States and Cities Initiative, 333
 CDC National Environmental Public Health tracking Network and climate change, 334–335
 and GCRP, 333
 National Institutes of Health (NIH) (*see* National Institutes of Health (NIH), US)
 research, global climate change, 333

V

- VBZD. *See* Vector-borne and zoonotic diseases (VBZD)
- Vector-borne and zoonotic diseases (VBZD)
- climate change, 196
 - dengue, malaria and leishmaniasis disease, 197
 - geophysical and socio-demographic characteristics, 196
 - human mobility, urbanization and land cover, 199
 - land-climate coupling, 196
 - LUCC, 196
 - malaria, 196–197
 - weekly surveillance reports, malaria and dengue, 198
- Vector-borne disease in the Amazon
- climate change (*see* Amazon climate change)
 - coupled environment-climate impacts, VBZD, 196–199
 - designs and/or surveillance networks, 206
 - disease transmission, 205
 - environmental change, disease transmission, 194
 - epidemiology, 199–201
 - funding agencies, 206
 - LUCC, 193
 - malaria-climate change (*see* Malaria-climate change)
 - satellite imagery, 206
 - VBZD, 194
- Vector-borne diseases, 12
- Viral parameters, dengue transmission cycle *Aedes aegypti*, 170–172
- biting behavior and climatic factors, 172

- climate-based, mechanistic transmission models, 172–173
 - observational studies, 173–175
 - populations at risk, 175–176
- VOCs. *See* Volatile organic compounds (VOCs)
- Volatile organic compounds (VOCs), 373
- Vulnerable subgroups
- comorbidity, 81
 - temperature and mortality, California, 75

W

- Water and food-borne diseases
- changes, temperature/humidity, 224
 - cryptosporidiosis, 220
 - marine environments, 222–224
 - recreational waters, 221
 - relationship, precipitation and *E. coli* counts, 221
 - water quality, 220
- Water consumption, Turkey, 299–300
- WCI. *See* Western climate initiative (WCI)
- Weather events and health
- floods, 218
 - natural disasters, 217–218
 - sea level rise and health, 219
 - wildfires, 218–219
- Western climate initiative (WCI), 390
- WHO. *See* World Health Organization (WHO)
- WMO. *See* World Meteorological Organization (WMO)
- World Health Organization (WHO), 379
- World Meteorological Organization (WMO), 375