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Human Health Benefits

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Key Findings

Key findings of this chapter include the following:

Modernity and Public Health

- Fossil fuels improved human well-being and safety by powering labor-saving and life-protecting technologies such as cars and trucks, plastics, and modern medicine.
- Fossil fuels play a key and indispensable role in the global increase in life expectancy.

Mortality Rates

- Cold weather kills more people than warm weather. A warmer world would see a net decrease in temperature-related mortality in virtually all parts of the world, even those with tropical climates.
- Weather is less extreme in a warmer world, resulting in fewer injuries and deaths due to storms, hurricanes, flooding, etc.

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Cardiovascular Diseases

- Higher surface temperatures would reduce the incidence of fatal coronary events related to low temperatures and wintry weather by a greater degree than they would increase the incidence associated with high temperatures and summer heat waves.
- Non-fatal myocardial infarction is also less frequent during unseasonably warm periods than during unseasonably cold periods.

Respiratory Disease

- Climate change is not increasing the incidence of death, hospital visits, or loss of work or school time due to respiratory disease.
- Low minimum temperatures are a greater risk factor than high temperatures for outpatient visits for respiratory diseases.

Stroke

- Higher surface temperatures would reduce the incidence of death due to stroke in many parts of the world, including Africa, Asia, Australia, the Caribbean, Europe, Japan, Korea, Latin America, and Russia.
- Low minimum temperatures are a greater risk factor than high temperatures for stroke incidence and hospitalization.

Insect-borne Diseases

- Higher surface temperatures are not leading to increases in mosquito-transmitted and tick-borne diseases such as malaria, yellow fever, viral encephalitis, and dengue fever.
- Extensive scientific information and experimental research contradict the claim that malaria will expand across the globe and intensify as a result of CO₂-induced warming.
- Concerns over large increases in dengue fever as a result of rising temperatures are unfounded and

unsupported by the scientific literature, as climatic indices are poor predictors for dengue fever.

- Climate change has not been the most significant factor driving recent changes in the distribution or incidence of tick-borne diseases.

Conclusion

- Fossil fuels directly benefit human health and longevity by powering labor-saving and life-protecting technologies and perhaps indirectly by contributing to a warmer world.

Introduction

Fossil fuels directly benefit human health and longevity by powering labor-saving and life-protecting technologies such as cars and trucks, plastics, and modern medicine. They may also indirectly benefit human health by contributing to some part of the increase in surface temperatures that the United Nations' Intergovernmental Panel on Climate Change (IPCC) claims occurred during the twentieth and early twenty-first centuries and may continue for the rest of the twenty-first century and beyond. How much warming has occurred and will occur, and how much can be attributed to the combustion of fossil fuels, are unsolved scientific puzzles, as explained in Chapter 2. Whereas the IPCC predicts a global temperature increase of between 2°C and 4°C by 2100 (compared to the 1850–1900 average) (IPCC, 2013, p. 20), the Nongovernmental International Panel on Climate Change (NIPCC) says its best-guess forecast is of ~0.3 to 1.1°C (NIPCC, 2013).

In the Working Group II contribution to its Fifth Assessment Report, the IPCC admits “at present the world-wide burden of human ill-health from climate change is relatively small compared with effects of other stressors and is not well quantified,” but it also claims “impacts from recent climate-related extremes, such as heat waves, droughts, floods, cyclones, and wildfires, reveal significant vulnerability and exposure of some ecosystems and many human systems to current climate variability (*very high confidence*)” (IPCC, 2014, p. 6). It further claims to have “high confidence” that climate change will contribute to eight “risk factors” including “risk

of severe ill-health” and “mortality and morbidity during periods of extreme heat” (*Ibid.*, p. 13).

As has been common throughout its history, the IPCC’s claims have been repeated by legacy news media, politicians, environmental activists, and subsidy-seekers in the renewable energy industry, while its more cautious findings, qualifications, and admissions of uncertainty are unreported or even hidden. This has led to widespread fear of the health effects of global warming (Schulte, 2008) and even political attack ads claiming people are dying of “carbon pollution” (WMC, 2015).

Independent researchers who should know better have also overlooked the IPCC’s errors and admissions of uncertainties. This failure is illustrated by an otherwise commendable effort to quantify the health and other effects of global warming by Richard S.J. Tol, a professor of economics at the University of Sussex and professor of the economics of climate change at the Vrije Universiteit Amsterdam who is otherwise an outspoken critic of the IPCC (Tol, 2013). Tol developed the Climate Framework for Uncertainty, Negotiation, and Distribution (FUND) model, which he said “is a fully integrated model, including scenarios of population, economy, energy use, and emissions; a carbon cycle and simple climate model; and a range of impact models” (Tol, 2011, p. 4).

Tol’s model forecasts the decline in the number of deaths due to cold temperatures would exceed the increase in the number of deaths due to warm temperatures in the year 2055 and beyond, a finding supported by extensive research summarized in this chapter. But Tol also contends “Climate change has caused the premature deaths of a substantial number of people over the 20th century – on average 7.5 per million per year. In 2000, according to FUND, 90,000 people died because of climate change” (p. 13). These numbers, drawn from the IPCC and public

health advocacy groups, are not empirical data and should not be treated as though they were. They are derived from computer models that assume more local warming than actually occurred, assume causation when medical evidence says otherwise, and contradict actual public health data showing falling numbers of deaths due to respiratory and cardiovascular diseases and insect-borne diseases such as malaria.

Chapter 3 already explained some of the human health benefits produced by the prosperity made possible by fossil fuels. In this chapter, those benefits are explained in greater detail. Section 4.1 documents the direct human health benefits due to the prosperity and technologies made possible by fossil fuels. Section 4.2 documents how medical science and observational research in Asia, Europe, and North America confirm that global warming is associated with lower, not higher, temperature-related mortality rates. Sections 4.3, 4.4, and 4.5 report research showing warmer temperatures lead to decreases in premature deaths due to cardiovascular and respiratory disease and stroke occurrences. Section 4.6 finds global warming has little if any influence on mosquito-borne diseases such as malaria and dengue fever or tick-borne diseases. Section 4.7 is a brief summary and conclusion.

The health benefits of climate change were the subject of Chapter 7 of a previous volume in the *Climate Change Reconsidered II* series, subtitled *Biological Effects* (NIPCC, 2014). The authors of that chapter provided a more comprehensive survey of the literature than is presented here, including older studies excluded from this review and detailed summaries of the methodologies utilized by the authors. A summary of that chapter appears below as Figure 4.1. This new chapter features research published as recently as 2018.

Figure 4.1 Impacts on human health

- Warmer temperatures lead to a decrease in temperature-related mortality, including deaths associated with cardiovascular disease, respiratory disease, and strokes. The evidence of this benefit comes from research conducted in every major country of the world.
- In the United States the average person who died because of cold temperature exposure lost in excess of 10 years of potential life, whereas the average person who died because of hot temperature exposure likely lost no more than a few days or weeks of life.

- In the United States, some 4,600 deaths are delayed each year as people move from cold northeastern states to warm southwestern states. Between 3% and 7% of the gains in longevity experienced over the past three decades was due simply to people moving to warmer states.
- Cold-related deaths are far more numerous than heat-related deaths in the United States, Europe, and almost all countries outside the tropics. Coronary and cerebral thrombosis account for about half of all cold-related mortality.
- Warmer temperatures are reducing the incidence of cardiovascular diseases related to low temperatures and wintry weather by a much greater degree than they increase the incidence of cardiovascular diseases associated with high temperatures and summer heat waves.
- A large body of scientific examination and research contradicts the claim that malaria will expand across the globe and intensify as a result of CO₂-induced warming.
- Concerns over large increases in vector-borne diseases such as dengue as a result of rising temperatures are unfounded and unsupported by the scientific literature, as climatic indices are poor predictors for dengue disease.
- Whereas temperature and climate largely determine the geographical distribution of ticks, they are not among the significant factors determining the incidence of tick-borne diseases.

Source: Chapter 7. "Human Health," *Climate Change Reconsidered II: Biological Impacts* (Chicago, IL: The Heartland Institute, 2014).

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4.1 Modernity and Public Health

Fossil fuels improved public health in developed countries around the world by making electrification, safe transportation, plastics, and modern medicine possible. None of these technological advances would have occurred without fossil fuels providing the abundant, convenient, and affordable energy needed to power them or the critical feedstock used to create them. Proof of fossil fuels' success in advancing public health can be seen in the rising lifespans of people living all around the world, but especially those living with the most abundant supplies of energy.

4.1.1 Technology and Health

Fossil fuels improved human well-being and safety by powering labor-saving and life-protecting technologies such as cars and trucks, plastics, and modern medicine.

Fossil fuels produce the quantity and quality of energy needed to fuel technologies that produce enormous human health benefits. Electricity, whose widespread use is possible only with fossil fuels, made and continues to make the biggest contributions to public health. Electricity makes food safer, more plentiful, and more affordable by making possible refrigeration from fields to grocery stores and freezers for long-term storage of food, and powering the modern canning industry, dramatically reducing waste. Electricity promotes good health by greatly facilitating the sharing of information, allowing people in need of help to call family members or emergency medical services, patients to communicate with doctors and other medical professionals between visits to hospitals and clinics, and doctors to share insights and discoveries with one another across long distances. Electricity continues to revolutionize health care with electronic medical records, robotic surgery, and remote diagnosis.

Safe and clean transportation is a second example of health benefits made possible by fossil fuels. The development and widespread use of cars and trucks averted a public health crisis. According to Smith (1990), “a horse produces approximately 45 pounds of manure each day. In high-density urban environments, massive tonnages accumulated, requiring constant collection and disposal. Flies, dried dung dust, and the smell of urine filled the air, spreading disease and irritating the lungs. On rainy days, one walked through puddles of liquid wastes. Occupational diseases in horse-related industries were common” (p. 25). According to Smith, New York City was disposing of 15,000 dead horses every year in the 1890s, a task that posed major threats to public health. Tenner (1997) offered a sobering reprise of the bad old days of horses in the streets causing diseases and deaths by accidents, and the public health revolution made possible by automobiles:

Less remembered today than the sanitary problems caused by horses were the safety hazards they posed. Horses and horse-drawn vehicles were dangerous, killing more riders, passengers, and pedestrians than is generally

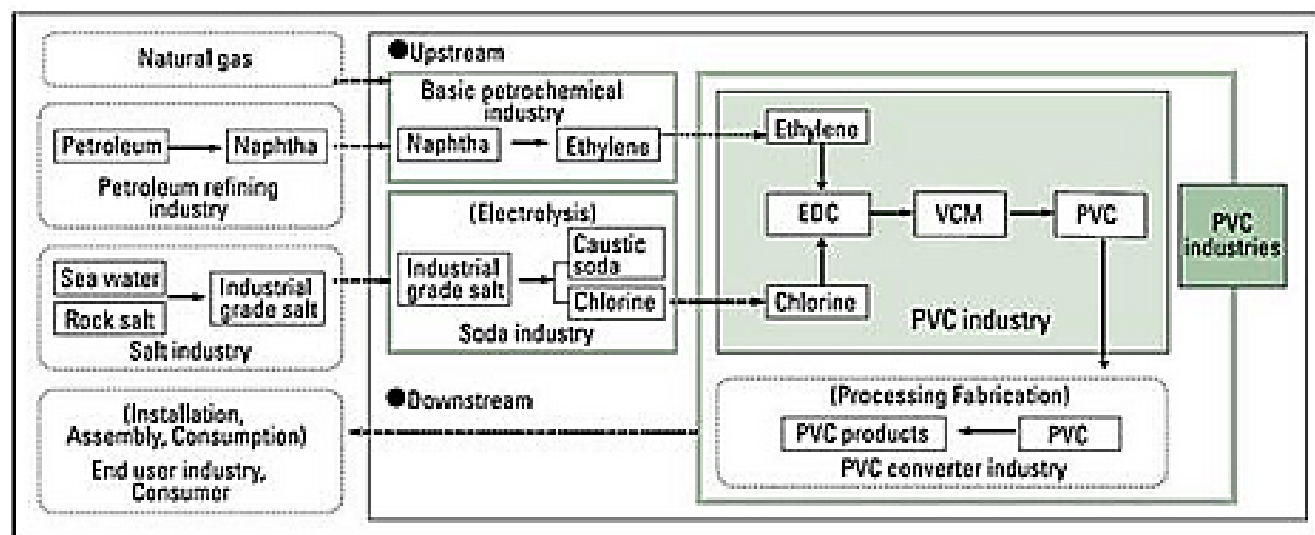
appreciated. Horses panicked. In frequent urban traffic snarls, they bit and kicked some who crossed their path. Horse-related accidents were an important part of surgical practice in Victorian England and no doubt in North America as well. In the 1890s in New York, per capita deaths from wagons and carriage accidents nearly doubled. By the end of the century they stood at nearly six per hundred thousand of population. Added to the five or so streetcar deaths, the rate of about 110 per million is close to the rates of motor vehicle deaths in many industrial countries in the 1980s. On the eve of motorization, the urban world was not such a gentle place.

The automobile was an answer to disease and danger. In fact, private internal-combustion transportation was almost utopian (pp. 333–4).

A third way fossil fuels-enabled technology has contributed to human health is by allowing the use of plastic products instead of metal, glass, or wooden products (Avery, 2000; North and Halden, 2013). Natural gas is the primary feedstock of plastic products made in the United States while oil is used by most European and Asian producers. See, for example, the representation of the production process of polyvinyl chloride (PVC) in Figure 4.1.1.1. According to the American Chemistry Council (ACC, 2015), falling prices of natural gas and oil in the United States made possible by the development of shale oil produced “a flood of new investment in U.S. plastics capacity announced since 2010. New factories are being built to produce more plastic resins, about half of that new resin production will be exported. In addition, with newly-available supplies of low-cost resin, producers of plastic products are building new production facilities” (p. 3).

While it is fashionable to minimize the benefits plastics bring to society, they are undeniably immense. Andrady and Neal (2009) write, “Plastics deliver unparalleled design versatility over a wide range of operating temperatures. They have a high strength-to-weight ratio, stiffness and toughness, ductility, corrosion resistance, bio-inertness, high thermal/electrical insulation, non-toxicity and outstanding durability at a relatively low lifetime cost compared with competing materials; hence plastics are very resource efficient.”

Figure 4.1.1.1
Production process of polyvinyl chloride (PVC) in the United States



Source: PVC.org, n.d.

Plastic components make up a growing percentage of the total composition of airplanes, cars, ships, buildings, and many other important technologies of the modern age. PVC is a maintenance-free and non-combustible material that is now essential for buildings, furniture, piping, and upholstery. Computers, cellphones, and the Internet itself would be impossible without plastics. A new technology – 3D printing – is using plastics to further revolutionize the manufacturing of consumer products.

Plastics contribute directly to improving public health in many ways. Plastic film helps protect food and other products in inventory and during shipping while dramatically reducing weight and cost relative to other types of containers. Plastic containers, plates, and cups replace glass containers that can cause injuries when broken. Plastic bottles with childproof caps reduce instances of accidental poisoning. Plastic airbags and seatbelts save lives every day by protecting passengers of cars and trucks from impact during accidents. Plastic insulation of electric wiring dramatically reduces the incidence of home and business fires and death and injury by electrocution.

Plastics have extensive applications in medicine, including disposable surgical gloves, masks, gowns, syringes, and petri dishes; flexible tubing and bags for plasma; tamper-resistant packaging for drugs; and

parts for innumerable medical devices. Plastic contact lenses restore vision to millions of people without the inconvenience of glasses, and plastic makes glasses lighter, break-resistant, and more affordable. Plastic prostheses are lighter, last longer, cost less than alternatives, and look more life-like. North and Halden (2013) write, “Plastics are cost-effective, require little energy to produce, and are lightweight and biocompatible. This makes them an ideal material for single-use disposable devices, which currently comprise 85% of medical equipment. Plastics can also be soft, transparent, flexible, or biodegradable and many different types of plastics function as innovative materials for use in engineered tissues, absorbable sutures, prosthetics, and other medical applications.”

Plastics infused with antibiotics, called antimicrobial plastic, can help stop the spread of diseases in hospitals, a major global public health threat. Surfaces containing antimicrobial plastic repel or kill bacteria on surfaces that doctors and patients touch, such as furniture in emergency and examination rooms. Sterile plastics can replace glass and steel containers used to store medicines and medical waste. Plastics speed the invention and wider use of new medical devices by making prototypes dramatically less expensive to create and modify. Plastic joints are typically longer-lasting than metal

joints, thereby reducing pain and the need for repeat surgeries. Because plastic devices are cheaper to produce than the metal or wood products they replace, they reduce the cost of many steps in the patient care cycle, thereby lowering the cost of and increasing public access to health care.

A fourth way fossil fuels contribute to human health is by enabling the world's farmers to increase their output faster than population, resulting in less hunger and starvation around the world. In 2015, the Food and Agriculture Organization of the United Nations (FAO) reported "the number of hungry people in the world has dropped to 795 million – 216 million fewer than in 1990–92 – or around one person out of every nine" (FAO, 2015). In developing countries, the share of the population that is undernourished (having insufficient food to live an active and healthy life) fell from 23.3 percent 25 years earlier to 12.9 percent. A majority of the 129 countries monitored by the FAO reduced undernourishment by half or more since 1996 (*Ibid.*).

Chapter 3, Section 3.3.1, explained how fossil fuels created and sustain the use of fertilizers and machines that make possible the Green Revolution and the more recent "Gene Revolution"; documented how rising levels of atmospheric CO₂ promote plant growth, increasing agricultural yields beyond levels farmers would otherwise achieve; and estimated the current and future value of aerial fertilization. Despite all this good news, concern has been expressed that increases in the *quantity* of food produced has come at a cost in *lower quality* food. There is some evidence that while aerial fertilization promotes crop yields, it may lower the level of key nutrients relative to total plant mass, making crops less nutritious. What does the latest science say about this?

It is possible to contrive growing conditions in which something other than CO₂ limits plant growth and health, or in which a shortage of some soil nutrient causes better crop yields to be accompanied by reduced levels of some nutrient, but such contrived conditions are easily avoided through normal agricultural fertilization practices. In real-world greenhouses, additional CO₂ is dramatically beneficial for agriculture at levels far beyond what are likely to be reached in the outdoor atmosphere, and the nutrient value of such crops grown with extra CO₂ is not significantly different from other crops.

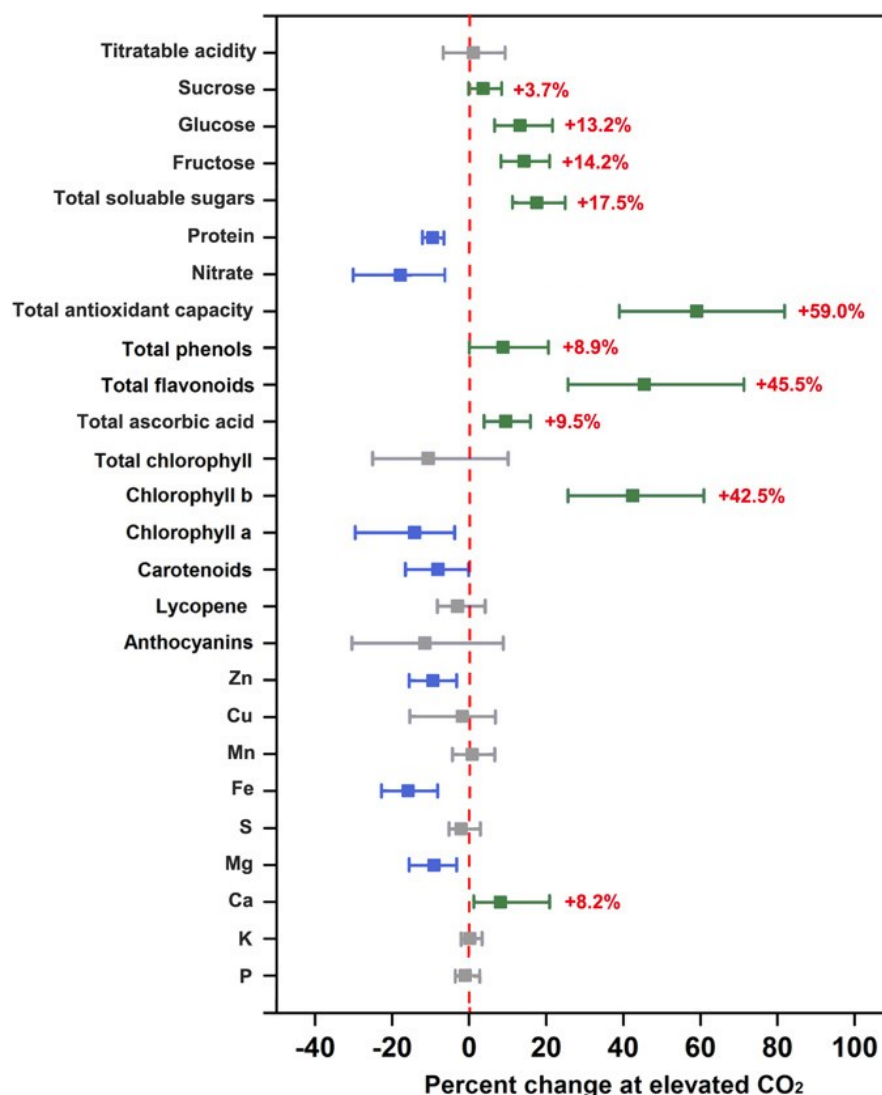
Dong *et al.* (2018) write, "a comprehensive review of recent studies explaining and targeting the key role of the effect of elevated CO₂ on vegetable quality is lacking." To remedy this knowledge gap,

the team of five researchers performed a meta-analysis of existing studies on the topic. In all, they examined 57 published works, which included CO₂ enrichment studies on root vegetables (carrot, radish, sugar beet, and turnip), stem vegetables (broccoli, celery, Chinese kale, ginger, onion, potato, and scallion), leafy vegetables (cabbage, Chinese cabbage, chives, fenugreek, Hongfengcat, lettuce, oily sowthistle, palak, and spinach) and fruit vegetables (cucumber, hot pepper, strawberry, sweet pepper, and tomato). The specific focus of their analysis was to examine measurements of nutritional quality on the vegetables, including measurements of soluble sugars, organic acids, protein, nitrates, antioxidants, and minerals.

The results of the analysis, shown in Figure 4.1.1.2, reveal elevated CO₂ "increased the concentrations of fructose, glucose, total soluble sugar, total antioxidant capacity, total phenols, total flavonoids, ascorbic acid, and calcium in the edible part of vegetables by 14.2%, 13.2%, 17.5%, 59.0%, 8.9%, 45.5%, 9.5%, and 8.2%, respectively, but [that it] decreased the concentrations of protein, nitrate, magnesium, iron, and zinc by 9.5%, 18.0%, 9.2%, 16.0%, and 9.4%. The concentrations of titratable acidity, total chlorophyll, carotenoids, lycopene, anthocyanins, phosphorus, potassium, sulfur, copper, and manganese were not affected."

In commenting on their findings, Dong *et al.* say that "overall, elevated CO₂ promotes the accumulation of antioxidants in vegetables, thus improving vegetable quality," while adding that the CO₂-induced stimulation of total antioxidant capacity, total phenols, total flavonoids, ascorbic acid, and chlorophyll b indicate "an improvement of beneficial compounds in vegetables."

For those concerned about the decreases in protein, nitrate, magnesium, iron, and zinc that were also observed in the meta-analysis, these slight declines can be reduced, if not reversed, through the application of several management approaches that were investigated and discussed by the authors, including "(1) selecting vegetable species or cultivars that possess greater ability in carbon fixation and synthesis of required quality-related compounds; (2) optimizing other environmental factors (e.g., moderate CO₂ concentrations, moderate light intensity, increased N availability, or increased fertilization of Fe or Zn) to promote carbon fixation and nutrient uptake interactively when growing plants under elevated CO₂; (3) harvesting vegetable products earlier in cases of over maturity and reduced benefit of elevated CO₂ to vegetative growth; and (4)

Figure 4.1.1.2**Effects of elevated CO₂ on the concentrations of various plant compounds and minerals in vegetables**

Data are means of percent change (relative to ambient CO₂) with 95% confidence intervals. Green squares and error bars represent positive changes, blue negative and grey indicate no significant change. *Source:* Adapted from Dong *et al.*, 2018.

combining elevated CO₂ with mild environmental stress (e.g., ultraviolet-B radiation or salinity) in instances when this enhances vegetable quality and might counteract the dilution effect or direct metabolic pathways toward the synthesis of health-beneficial compounds.”

The findings of Dong *et al.* clearly show that CO₂-induced plant nutritional enhancements outweigh CO₂-induced plant nutritional declines.

Thus, it can reasonably be concluded that rising atmospheric CO₂ concentrations will yield future health benefits to both human and animal plant consumers.

A fifth contribution of fossil fuels to human health, ironic in light of current debates, is to protect mankind from the climate. As Goklany wrote, “these technologies, by lowering humanity’s reliance on living nature, inevitably ensured that human well-

being is much less subject to whims of nature (as expressed through the weather, climate, disease, and other natural disasters)” (Goklany, 2012, p. 1). Writing in 2011, Goklany noted,

[E]xtreme weather events ... now contribute only 0.07% to global mortality. Mortality from extreme weather events has declined even as all-cause mortality has increased, indicating that humanity is coping better with extreme weather events than it is with far more important health and safety problems. The decreases in the numbers of deaths and death rates reflect a remarkable improvement in society’s adaptive capacity, likely due to greater wealth and better technology, enabled in part by use of hydrocarbon fuels. Imposing additional restrictions on the use of hydrocarbon fuels may slow the rate of improvement of this adaptive capacity and thereby worsen any negative impact of climate change. At the very least, the potential for such an adverse outcome should be weighed against any putative benefit arising from such restrictions (p. 4.).

Epstein (2014) pointed out, “Climate is no longer a major cause of death, thanks in large part to fossil fuels” (p. 126). “Historically, drought is the *number-one climate related cause of death*. Worldwide it has gone down by 99.98% in the last eighty years for many energy-related reasons: oil-powered drought-relief convoys, more food in general because of more prolific, fossil-fuel-based agriculture, and irrigation systems” (*Ibid.*). Environmentalists, Epstein wrote, have the issue backward: “[W]e don’t take a safe climate and make it dangerous; we take a dangerous climate and make it safe. High-energy civilization, not climate, is the driver of climate livability. No matter what, climate will always be naturally hazardous – and the key question will always be whether we have the adaptability to handle it, or better yet, master it” (*Ibid.*).

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4.1.2 Public Health Trends

Fossil fuels play a key and indispensable role in the global increase in life expectancy.

Historically, humankind was besieged by epidemics and other disasters that caused frequent widespread deaths and kept the average lifespan to less than 35 years (Omran, 1971). The average lifespan among the ancient Greeks was apparently just 18 years, and among the Romans, 22 years (Bryce, 2014 p. 59, citing Steckel and Rose, 2002). The discovery of uses for fossil fuels in the late-eighteenth and early-nineteenth century dramatically changed the world, a story told in Chapter 3.

The evidence of progress in public health in the United States and other developed countries in the twentieth and early-twenty-first centuries is overwhelming (Lehr, 1992; Lomborg, 2001). Economist Julian Simon edited a series of volumes (Simon, 1981, 1995, 1998; Simon and Kahn, 1984) and coauthored a book published posthumously (Moore and Simon, 2000) providing extensive data showing long-term trends for everything from mortality and longevity to food supplies, air and water quality, and the affordability of housing. Nearly every trend showed dramatic improvement over time. As Simon wrote in the introduction to his 1995 book, “Most important, fewer people are dying young. And life expectancy in the rich countries has increased most sharply in the older age cohorts, among which many thought there was no improvement. Perhaps most exciting, the quantities of education that people obtain all over the world are sharply increasing, which means less tragic waste of human talent and ambition” (Simon, 1995, p. 2). The trend Simon observed in 1995 continues today. According to the U.S. Census Bureau:

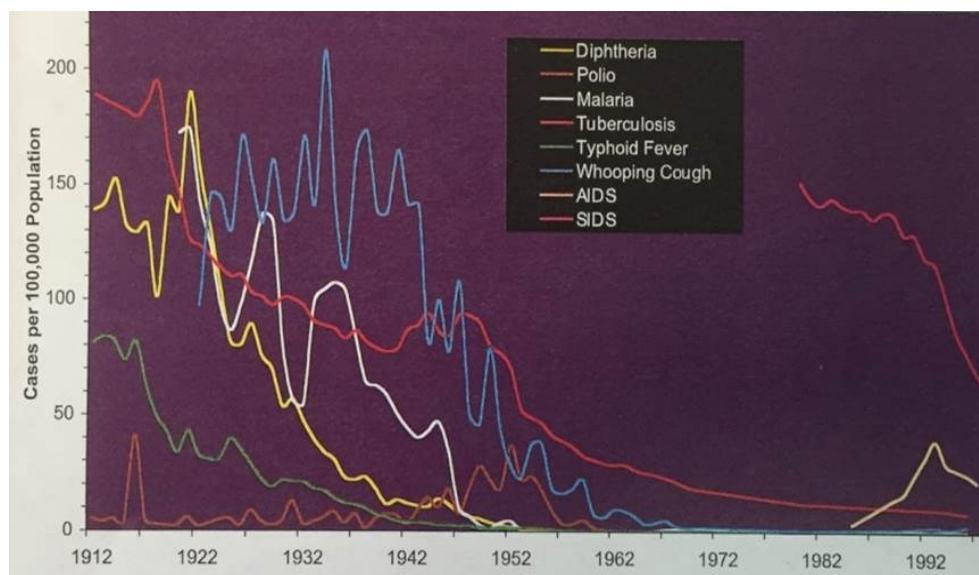
- “The world average age of death has increased by 35 years since 1970, with declines in death rates in all age groups, including those aged 60 and older (Institute for Health Metrics and

Evaluation, 2013; Mathers *et al.*, 2015). From 1970 to 2010, the average age of death increased by 30 years in East Asia and 32 years in tropical Latin America, and in contrast, by less than 10 years in western, southern, and central Sub-Saharan Africa (Institute for Health Metrics and Evaluation, 2013; Figure 4-1) ;

- “In the mean age at death between 1970 and 2010 across different WHO regions, all regions have had increases in mean age at death, particularly East Asia and tropical Latin America;
- “Global life expectancy at birth reached 68.6 years in 2015. A female born today is expected to live 70.7 years on average and a male 66.6 years. The global life expectancy at birth is projected to increase almost 8 years, reaching 76.2 years in 2050” (U.S. Census Bureau, 2015, pp. 31–33).

The U.S. historical record reveals the close correlation between prosperity and public health and longevity. As the nation grew richer thanks to fossil fuels, the incidence of nearly every disease in the United States fell dramatically, as shown in Figure 4.1.2.1.

Figure 4.1.2.1
Incidence of selected diseases in the United States, 1912–1997



Source: Moore and Simon, 2000, p. 35.

As Moore and Simon write, “Before 1900, major killers included such infectious diseases as tuberculosis, smallpox, diphtheria, polio, influenza, and bronchitis. Just three infectious diseases – tuberculosis, pneumonia, and diarrhea – accounted for almost half of all deaths in 1900. Now few Americans die from these diseases, and many diseases have been completely eradicated due to a medley of modern medicines” (Moore and Simon, 2000, p. 34).

The economic growth created by abundant and affordable energy, documented in Chapter 3, Section 3.6.1, is closely correlated with better health and longevity. Brenner (1984), in a study for the Joint Economic Committee of the U.S. Congress, found a one-percentage-point increase in the unemployment rate (e.g., from 5% to 6%) would lead to a 2% increase in the age-adjusted mortality rate. The growth of real income per capita also showed a significant correlation to decreases in mortality rates (except for suicide and homicide), hospitalization for mental illness, and property crimes. The European Commission has supported similar research showing comparable results throughout the European Union (Brenner, 2000, 2003).

Both in the United States and Europe, Brenner found that changes to the economic status of individuals result in changes to their health and lifespan, with decreased real income per capita and increased unemployment leading to increased mortality. Econometric analyses of time-series data have measured the relationship between changes in the economy and changes in health outcomes, and studies have determined declines in real income per capita and increases in unemployment led to elevated mortality rates over a subsequent period of six years (Brenner, 2005). The loss of disposable income also reduces the amount families can spend on critical health care, especially among the poorest and least healthy (Keeney, 1990; Lutter and Morrall, 1994; Viscusi, 1994; Viscusi and Zeckhauser, 1994; Hjalte *et al.*, 2003).

The U.S. Environmental Protection Agency (EPA) has acknowledged “People’s wealth and health status, as measured by mortality, morbidity, and other metrics, are positively correlated. Hence, those who bear a regulation’s compliance costs may also suffer a decline in their health status, and if the costs are large enough, these increased risks might be greater than the direct risk-reduction benefits of the regulation” (EPA, 1995). The U.S. Office of Management and Budget, Food and Drug Administration, and Occupational Safety and Health

Administration use methodologies similar to the EPA’s to assess the degree to which their regulations induce premature death among those who bear the costs of federal mandates (OMB, 1993).

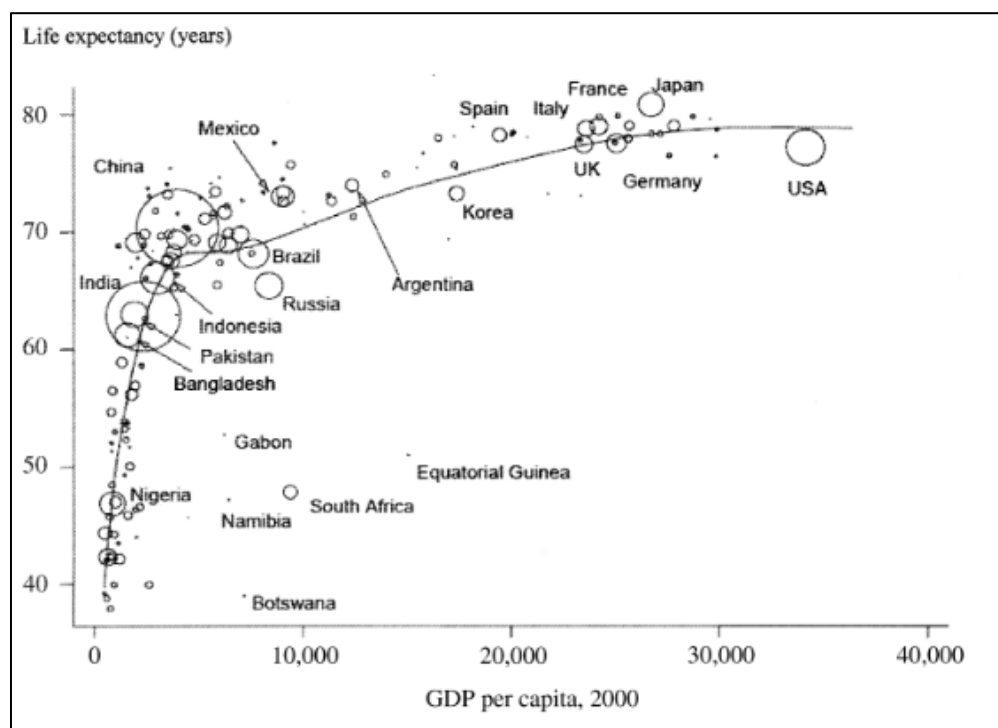
The global correlation between prosperity and life expectancy is represented by the “Preston curve” shown in Figure 4.1.2.2. It has been closely studied since the 1970s (Preston, 1975, 2007; Deaton, 2003, 2004; Bloom and Canning, 2007). The data make very clear that the people who live in impoverished societies live shorter lives, with the relationship strongest at per-capita GDP levels of less than \$30,000. At that stage of development investments in nutrition, clean water and sanitation, and public safety have their greatest impact on public health. The flattening of the curve at higher income levels reveals how other factors then affect lifespan, with the leading factors being educational attainment, spending on health care services, and personal savings. All of these factors contributing to longevity – nutrition, clean water and sanitation, public safety, education, health care services, and personal income – are positively impacted by abundant and affordable energy, impacts documented at length in Chapter 3 and earlier in this chapter. Fossil fuels played a key role in increasing global wealth and longevity.

It took eight millennia for the average global life expectancy to rise from 20 years to the high 20s. Since the discovery of fossil fuels, life expectancy soared to 75 years and longer in developed countries. Life expectancy increased for all age groups, from infancy to old age, as the three Industrial Revolutions brought improved nutrition, cleaner air and water, and safer work conditions to virtually every person in developed countries and to many in developing countries.

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Figure 4.1.2.2
The Millenium Preston Curve
Life expectancy and per-capita GDP in 2000



Dots and circles represent countries. Circles have diameter proportional to population size. GDP per capita is in purchasing power parity dollars. Source: Deaton, 2004.

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4.2 Mortality Rates

Cold weather kills more people than warm weather. A warmer world would see a net decrease in temperature-related mortality in

virtually all parts of the world, even those with tropical climates.

As stated in the introduction to this chapter, the United Nations' Intergovernmental Panel on Climate Change (IPCC) claims the carbon dioxide (CO₂) produced during the combustion of fossil fuels affects human health *indirectly* by causing an increase in surface temperatures that creates a “risk of severe ill-health” and “mortality and morbidity during periods of extreme heat” (IPCC, 2014, p. 13). Section 4.2.1 provides the basis in medical science for why warmer temperatures can be expected to *reduce* rather than increase mortality rates. Sections 4.2.2 through 4.2.6 summarize observational research conducted globally and specifically in Asia, Australia, Europe, and North America confirming the hypothesis.

Reference

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4.2.1 Medical Science

Carbon dioxide (CO₂) is invisible, odorless, and nontoxic, and it does not seriously affect human health until the CO₂ content of the air reaches approximately 15,000 ppm, more than 37 times greater than the current concentration of atmospheric CO₂ (Luft *et al.*, 1974). A long-term rise in the atmosphere's CO₂ content, in the recent past or in the future, will have no direct adverse human health consequences. Even extreme model projections do not indicate anthropogenic activities will raise the air's CO₂ concentration above 1,000 to 2,000 ppm.

The medical literature shows, overwhelmingly, that warmer temperatures and a smaller difference between daily high and low temperatures, as occurred during the twentieth and early twenty-first centuries, reduce mortality rates (the subject of this section) as well as illness and mortality due to cardiovascular and respiratory disease and stroke occurrence (the subject of later sections). Humans are increasingly adapting to changes in temperature (and weather generally) thanks to the spread of technologies such as air conditioning, more efficient home heating,

better insulation, and improvements in clothing and transportation. See, for example, the studies by Matzarakis *et al.* (2011) and Matthies and Menne (2009) and the additional sources they cite. This means over time, even the relatively small number of deaths caused by exposure to heat waves is declining, making the small temperature increase that may occur during the coming century highly unlikely to cause any deaths.

Medical science explains why colder temperatures often cause diseases and sometimes fatalities whereas warmer temperatures are associated with health benefits. Keatinge and Donaldson (2001) explain that “cold causes mortality mainly from arterial thrombosis and respiratory disease, attributable in turn to cold-induced hemoconcentration and hypertension [in the first case] and respiratory infections [in the second case].” McGregor (2005) notes “anomalous cold stress can increase blood viscosity and blood pressure due to the activation of the sympathetic nervous system which accelerates the heart rate and increases vascular resistance (Collins *et al.*, 1985; Jehn *et al.*, 2002; Healy, 2003; Keatinge *et al.*, 1984; Mercer, 2003; Woodhouse *et al.*, 1993)” due to vasoconstriction to reduce blood flow and heat loss at the surface, adding, “anomalously cold winters may also increase other risk factors for heart disease such as blood clotting or fibrinogen concentration, red blood cell count per volume and plasma cholesterol.”

Wang *et al.* (2013) write, “a large change in temperature within one day may cause a sudden change in the heart rate and circulation of elderly people, which all may act to increase the risk of cardiopulmonary and other diseases, even leading to fatal consequences.” This is significant for the climate change debate because, as Wang *et al.* also observe, “it has been shown that a rise of the minimum temperature has occurred at a rate three times that of the maximum temperature during the twentieth century over most parts of the world, which has led to a decrease of the diurnal temperature range (Karl *et al.*, 1984, 1991).” Robeson (2002) demonstrated, based on a study of 50 years of daily temperatures at more than 1,000 U.S. weather stations, that temperature variability declines with greenhouse warming, and at a very substantial rate, so this aspect of a warmer world would lead to a reduction in temperature-related deaths. Braganza *et al.* (2004) reported, “observed reductions in DTR over the last century are large.” Alexander *et al.* (2006) found a global trend toward warmer nights and a much smaller trend toward warmer days for the

period 1951–2003, concluding “these results agree with earlier global studies ... which imply that rather than viewing the world as getting hotter it might be more accurate to view it as getting less cold.” See also Easterling *et al.* (1997) and Seldenrich (2015).

Keatinge and Donaldson (2004) report coronary and cerebral thrombosis account for about half of all cold-related deaths, and respiratory diseases account for approximately half of the rest. They say cold stress causes an increase in arterial thrombosis “because the blood becomes more concentrated, and so more liable to clot during exposure to cold.” As they describe it, “the body’s first adjustment to cold stress is to shut down blood flow to the skin to conserve body heat,” which “produces an excess of blood in central parts of the body,” and to correct for this effect, “salt and water are moved out from the blood into tissue spaces,” leaving behind “increased levels of red cells, white cells, platelets and fibrinogen” that lead to increased viscosity of the blood and a greater risk of clotting. The British scientists report the infections that cause respiratory-related deaths spread more readily in cold weather because people “crowd together in poorly ventilated spaces when it is cold.” In addition, they say “breathing of cold air stimulates coughing and running of the nose, and this helps to spread respiratory viruses and bacteria.” The “train of events leading to respiratory deaths,” they continue, “often starts with a cold or some other minor infection of the upper airways,” which “spreads to the bronchi and to the lungs,” whereupon “secondary infection often follows and can lead to pneumonia.” They also note cold stress “tends to suppress immune responses to infections,” and respiratory infections typically “increase the plasma level of fibrinogen, and this contributes to the rise in arterial thrombosis in winter.”

Keatinge and Donaldson also note “cold spells are closely associated with sharp increases in mortality rates,” and “deaths continue for many days after a cold spell ends.” On the other hand, they report, “increased deaths during a few days of hot weather are followed by a lower than normal mortality rate,” because “many of those dying in the heat are already seriously ill and even without heat stress would have died within the next 2 or 3 weeks.” With respect to the implications of global warming for human mortality, Keatinge and Donaldson state, “since heat-related deaths are generally much fewer than cold-related deaths, the overall effect of global warming on health can be expected to be a beneficial one.” They report, “the rise in temperature of 3.6°F

expected over the next 50 years would increase heat-related deaths in Britain by about 2,000 but reduce cold-related deaths by about 20,000.”

Keatinge and Donaldson’s (2004) reference to deaths that typically would have occurred shortly even without excess heat is a phenomenon researchers call “displacement” or “harvesting.” A study from Germany found “cold spells lead to excess mortality to a relatively small degree, which lasts for weeks,” while “the mortality increase during heat waves is more pronounced, but is followed by lower than average values in subsequent weeks” (Laschewski and Jendritzky, 2002). The authors say the latter observation suggests people who died from short-term exposure to heat possibly “would have died in the short term anyway.” They found the mean duration of above-normal mortality for the 51 heat episodes that occurred from 1968 to 1997 was 10 days, with a mean increase in mortality of 3.9%, after which there was a mean decrease in mortality of 2.3% for 19 days. The net effect of the two perturbations was an overall decrease in mortality of 0.2% over the full 29-day period.

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4.2.2 Global

Gasparrini *et al.* (2015a) analyzed more than 74 million deaths in 384 locations across 13 countries between 1985 and 2012, finding 20 times more people die from cold-related rather than heat-related weather events, and extreme cold weather is much deadlier. They write, “Our findings show that temperature is responsible for advancing a substantial fraction of deaths, corresponding to 7.71% of mortality in the selected countries within the study period. Most of this mortality burden was caused by days colder than the optimum temperature (7.29%), compared with days warmer than the optimum temperature (0.42%). Furthermore, most deaths were caused by exposure to moderately hot and cold temperatures, and the contribution of extreme days was comparatively low, despite increased RRs [relative risks].” They also found “the optimum temperature at which the risk is lowest was well above the median, and seemed to be increased in cold regions.” A figure illustrating their findings is reproduced below as Figure 4.2.2.1.

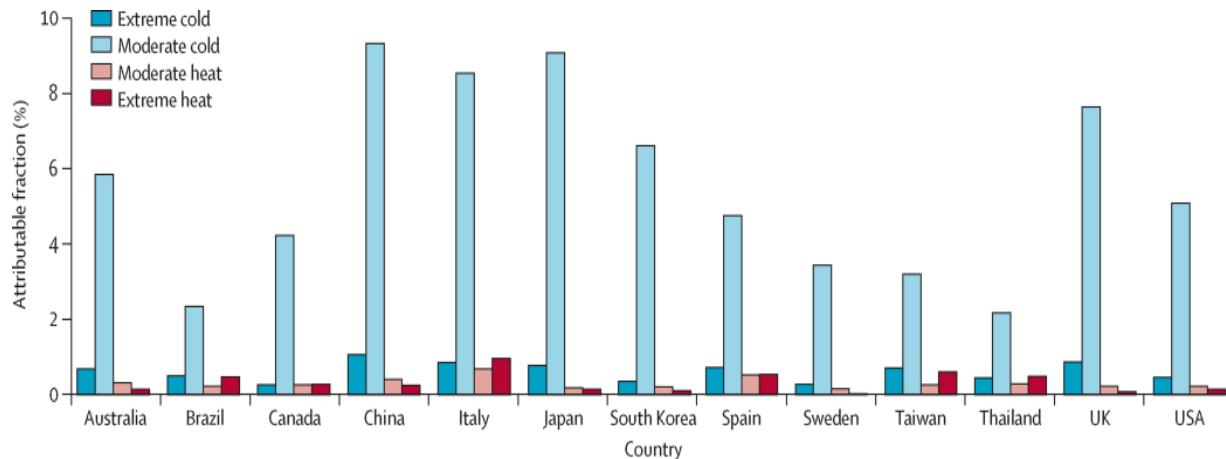
In a second paper, Gasparrini *et al.* (2015b) collected data for more than 20.2 million heat-related deaths that occurred in Australia, Canada, Japan, South Korea, Spain, the United Kingdom, and the United States during the summer months between 1985 and 2012. They report “mortality risk due to heat appeared to decrease over time in several countries, with relative risks associated with high temperatures significantly lower in 2006 compared with 1993 in the United States, Japan and Spain”; there was “a non-significant decrease in Canada”; “temporal changes were difficult to assess in Australia and South Korea due to low statistical power”; and they “found little evidence of variation in the United Kingdom,” while “in the United States, the risk seemed to be completely abated in 2006 for summer temperatures below their 99th percentile.”

They concluded there was “a statistically significant decrease in the relative risk for heat-related mortality in 2006 compared with 1993 in the majority of countries included in the analysis.

Seltenrich (2015) writes, “while isolated heat waves pose a major health risk and grab headlines when they occur, recent research has uncovered a more complex and perhaps unexpected relationship between temperature and public health,” which is, as he continues, that “on the whole, far more deaths occur in cold weather than in hot.” Seltenrich reports that “an analysis by the Centers for Disease Control and Prevention of U.S. temperature-related deaths between 2006 and 2010 showed that 63% were attributable to cold exposure, while only 31% were attributable to heat exposure,” citing *National Health Statistics Report No. 76* of the National Center for Health Statistics of the U.S. Centers for Disease Control and Prevention. “In Australia and the United Kingdom, cold-related mortality between 1993 and 2006 exceeded heat-related mortality by an even greater margin, and is likely to do so through at least the end of the century,” he writes, citing Vardoulakis *et al.* (2014).

Arbuthnott *et al.* (2016) examined “variations in temperature related mortality risks over the 20th and 21st centuries [to] determine whether population adaptation to heat and/or cold has occurred.” A search of 9,183 titles and abstracts dealing with the subject returned 11 studies examining the effects of ambient temperature over time and six studies comparing the effect of heatwaves at specific points in time. Of the first 11 studies, with respect to the hot end of the temperature spectrum, Arbuthnott *et al.* report “all except one found some evidence of decreasing susceptibility.” At the cold end of the temperature spectrum, they say “there is little consistent evidence for decreasing cold related mortality, especially over the latter part of the last century.” With respect to the impacts of specific heatwave events on human health, Arbuthnott *et al.* state that four of the six papers included in this portion of their analysis revealed “a decrease in expected mortality,” again signaling there has been a decrease in the vulnerability of the human populations studied over time. As for the cause(s) of the observed temperature-induced mortality declines, the authors acknowledge their methods are incapable of making that determination. However, they opine that it may, in part, be related to physiological acclimatization (human adaptation) to temperature.

Figure 4.2.2.1
Deaths caused by cold vs. heat



Source: Gasparrini *et al.*, 2015a, p.369.

Son *et al.* (2016) examined how mortality in Sao Paulo, Brazil, was affected by extremes of heat and cold over the 14.5-year period from 1996 to 2010, using “over-dispersed generalized linear modeling and Bayesian hierarchical modeling.” They found “cold effects on mortality appeared higher than heat effects in this subtropical city with moderate climatic conditions.”

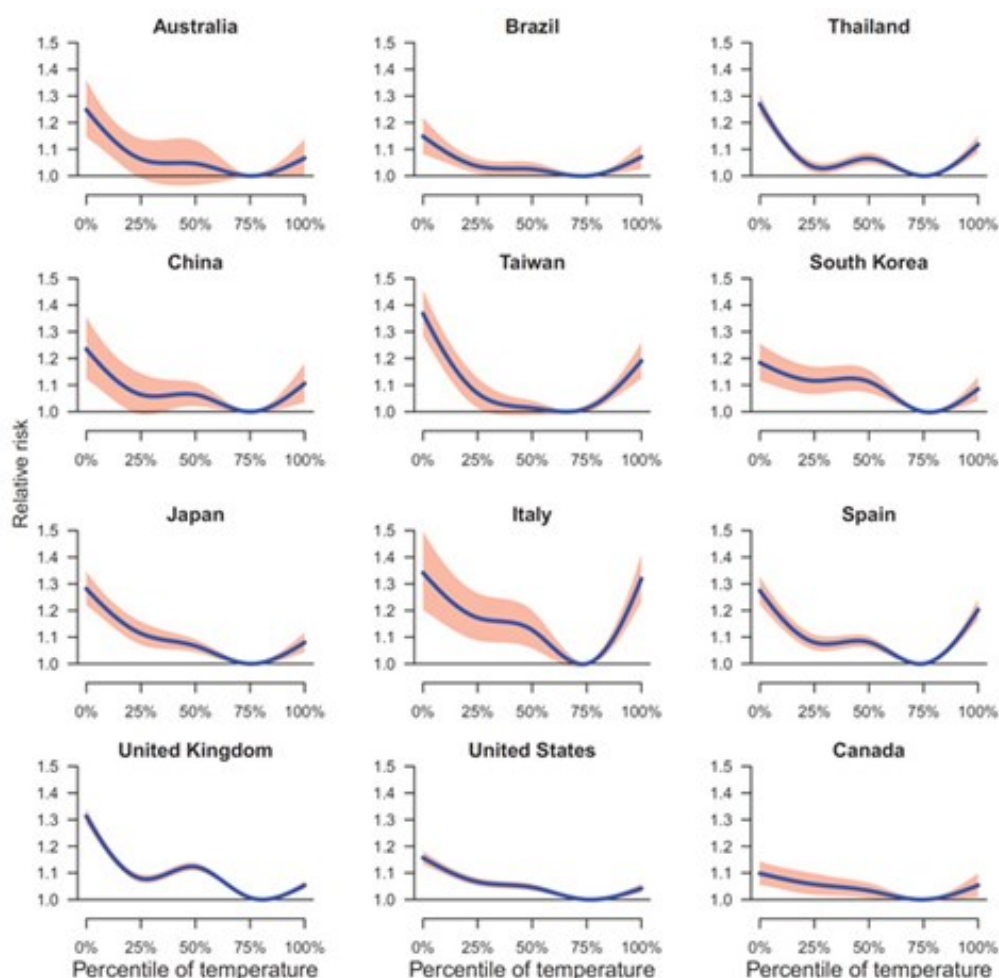
Guo *et al.* (2014) obtained daily temperature and mortality data from 306 communities located in 12 countries (Australia, Brazil, Canada, China, Italy, Japan, Korea, Spain, Taiwan, Thailand, the United Kingdom, and the United States) within the time period 1972–2011. In order to “obtain an easily interpretable estimate of the effects of cold and hot temperatures on mortality,” they “calculated the overall cumulative relative risks of death associated with cold temperatures (1st percentile) and with hot temperatures (99th percentile), both relative to the minimum-mortality temperature [75th percentile]” (see Figure 4.2.2.2). Despite the “widely ranging climates” they encountered, they report “the minimum-mortality temperatures were close to the 75th percentile of temperature in all 12 countries, suggesting that people have adapted to some extent to their local climates.”

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Figure 4.2.2.2

The pooled overall cumulative relation between temperature and deaths over lags of 0–21 days in 12 countries/regions



Source: Adapted from Guo *et al.*, 2014.

mortality in the United Kingdom and Australia. *Environmental Health Perspectives* **122**: 1285–92.

4.2.3 Asia

Behar (2000) studied sudden cardiac death (SCD) and acute myocardial infarction (AMI) in Israel, concentrating on the role temperature may play in the incidence of these health problems. Behar notes “most of the recent papers on this topic have concluded that a peak of SCD, AMI and other

cardiovascular conditions is usually observed in low temperature weather during winter.” He cites an Israeli study by Green *et al.* (1994), which reported between 1976 and 1985 “mortality from cardiovascular disease was higher by 50% in mid-winter than in mid-summer, both in men and women and in different age groups,” even though summer temperatures in the Negev, where much of the work was conducted, often exceed 30°C (86°F) and winter temperatures typically do not drop below 10°C (50°F). Behar concludes these results “are reassuring for populations living in hot countries.”

Kan *et al.* (2003) investigated the association between temperature and mortality in Shanghai, China, finding a V-like relationship between total mortality and temperature that had a minimum mortality risk at 26.7°C. Above this optimum temperature, they observed that total mortality increased by 0.73% for each degree Celsius increase, while for temperatures below the optimum value, total mortality increased by 1.21% for each degree Celsius decrease. The net effect of a warming in Shanghai, China, therefore, would likely be decreased mortality on the order of 0.5% per degree Celsius increase in temperature, or perhaps more.

Kan *et al.* (2007) examined the association between diurnal temperature range (DTR, defined as daily maximum temperature minus daily minimum temperature) and human mortality, using daily weather and mortality data from Shanghai over the period January 1, 2001 to December 31, 2004. They say their data suggest “even a slight increase in DTR is associated with a substantial increase in mortality.” Their results suggest that in addition to the reduction in human mortality typically provided by the increase in daily mean temperature, the accompanying decrease in DTR also should have been tending to reduce human mortality.

Ma *et al.* (2011) investigated the impact of heat waves and cold spells on hospital admissions in Shanghai, China. The four researchers report the number of excess (above normal) hospital admissions during an eight-day heat wave was 352 whereas during a 10-day cold spell there were 3,725 excess admissions. Ma *et al.* conclude “the cold spell seemed to have a larger impact on hospital admission than the heat wave in Shanghai.”

Cheng and Kan (2012) analyzed mortality, air pollution, temperature, and covariate data over the period January 1, 2001 through December 31, 2004 in Shanghai. They report they “did not find a significant interaction between air pollution and higher temperature [>85 th percentile days],” but “the interaction between PM₁₀ [particulate matter, 10 micrometers or smaller] and extreme low temperature [<15 th percentile days] was statistically significant for both total and cause-specific mortality.” Compared to normal temperature days (15th to 85th percentile), they found a 10- $\mu\text{g}/\text{m}^3$ increase in PM₁₀ on extreme low temperature days led to all-cause mortality rising from 0.17% to 0.40%. They add, “the interaction pattern of O₃ with low temperature was similar,” noting their finding of “a stronger association between air pollution and daily mortality on extremely cold days confirms those of three

earlier seasonal analyses in Hong Kong, Shanghai and Athens,” citing Touloumi *et al.* (1996), Wong *et al.* (1999, 2001), and Zhang *et al.* (2006).

Guo *et al.* (2012) examine the nonlinear and delayed effects of temperature on cause-specific and age-specific mortality employing data from 1999 to 2008 for Chiang Mai, Thailand, with a population of 1.6 million people. Controlling for season, humidity, ozone, and particulate matter (PM₁₀) pollution, the three researchers found “both hot and cold temperatures resulted in an immediate increase in all mortality types and age groups,” but “the hot effects on all mortality types and age groups were short-term, while the cold effects lasted longer.” The cold effects were greater, with more people dying from them than from the effects of heat.

Lindeboom *et al.* (2012) used daily mortality and weather data for the period 1983–2009 pertaining to Matlab, Bangladesh to measure lagged effects of weather on mortality, controlling for time trends and seasonal patterns. The four researchers report “mortality in the Matlab surveillance area shows overall weak associations with rainfall, and stronger negative association with temperature.” They determined there was “a 1.4% increase in mortality with every 1°C decrease in mean temperature at temperatures below 29.2°C,” but only “a 0.2% increase in mortality with every 1°C increase in mean temperature.”

Wang *et al.* (2013) evaluated the short-term effect of DTR on emergency room (ER) admissions among elderly adults in Beijing. The nine researchers report “significant associations were found between DTR and four major causes of daily ER admissions among elderly adults in Beijing.” They state “a 1°C increase in the 8-day moving average of DTR (lag 07) corresponded to an increase of 2.08% in respiratory ER admissions and 2.14% in digestive ER admissions,” and “a 1°C increase in the 3-day and 6-day moving average of DTR (lag 02 and lag 05) corresponded to a 0.76% increase in cardiovascular ER admissions, and a 1.81% increase in genitourinary ER admissions, respectively.”

Wu *et al.* (2013) assessed the health effects of temperature on mortality in four subtropical cities of China (Changsha, Guangzhou, Kunming, and Zhuhai). The 11 researchers report a U-shaped relationship between temperature and mortality was found in the four cities, indicating “mortality is usually lowest around a certain temperature and higher at lower or higher temperatures.” Although “both low and high temperatures were associated with increased mortality in the four subtropical

Chinese cities,” Wu *et al.* state the “cold effect was more durable and pronounced than the hot effect.”

Yang *et al.* (2013) examined the effects of DTR on human mortality rates using daily meteorological data for the period January 1, 2003 through December 31, 2010 from a single station located in the heart of the urban area of Guangzhou City (the largest metropolis in Southern China). They found “a linear DTR-mortality relationship, with evidence of increasing mortality with DTR increase,” where “the effect of DTR occurred immediately and lasted for four days,” such that over that time period, a 1°C increase in DTR was associated with a 0.47% increase in non-accidental mortality. In addition, they report there was a joint adverse effect with temperature “when mean temperature was below 22°C [71.6°F], indicating that high DTR enhanced cold-related mortality.” In light of their findings, the eight researchers speculate the expected “decrease in DTR in future climate scenarios might lead to two benefits: one from decreasing the adverse effects of DTR [which is reduced due to greater warming at night than during the day], and the other from decreasing the interaction effect with temperature [which is expected to rise with greenhouse warming].”

Onozuka and Hagihara (2015) acquired data on daily emergency ambulance dispatches in Japan’s 47 prefectures from 2007 to 2010, which they used to determine relationships between medical emergency transport and temperature. They found the fraction of ambulance dispatches attributable to low temperatures was 6.94% for all causes, while that attributable to high temperatures was 1.01% for all causes. They report “the majority of temperature-related emergency transport burden was attributable to lower temperature,” which burden was almost seven times greater than that attributable to higher temperatures.

Huang *et al.* (2015) analyzed community-specific daily mortality data for the period January 1, 2006 to December 31, 2011, obtained from the Chinese Center for Disease Control and Prevention, together with community-specific daily meteorological data for the same period, obtained from the China National Weather Data Sharing System. They found temperature-mortality relationships were “approximately V-shaped or U-shaped, with a minimum mortality temperature (MMT),” above and below which human mortality increased. For each of the 66 communities they studied, they calculated “the change in mortality risk for a 1°C decrease in temperature below the MMT (cold effect) and for a

1°C temperature increase above the MMT (heat effect).” This work revealed that a 1°C temperature increase above the MMT resulted in a mean increase of 1.04% in human mortality for the 66 communities, while a 1°C temperature decrease below the MMT resulted in a mean increase of 3.44% in human mortality, demonstrating that cooling below the minimum mortality temperature was 3.31 times more deadly than was warming above it.

Chau and Woo (2015) examined summer (June–August) versus winter (December–February) excess mortality trends among the older population (65 years and older) of Hong Kong citizens over the 35-year period 1976–2010. They performed statistical analyses that searched for relationships between various measures of extreme meteorological data and recorded deaths due to cardiovascular and respiratory-related causes. They report there was an average rise in mean temperature of “0.15°C per decade in 1947–2013 and an increase of 0.20°C per decade in 1984–2013.” They also note that over the 35-year period of their analysis “winter became less stressful,” with fewer extreme cold spells. Summers, on the other hand, became “more stressful as the number of Hot Nights in summer increased by 0.3 days per year and the number of summer days with very high humidity (daily relative humidity over 93%) increased by 0.1 days per year.” Given such observations, it would be expected under the global warming hypothesis that cold-related deaths should have declined and heat-related deaths should have increased across the length of the record. As shown in Figure 4.2.3.1, cold-related death rates did indeed decline (by 49.3%), from approximately 21 deaths per 1,000 persons in 1976 to 10.6 deaths in 2010. Heat-related death rates, however, did not increase. Rather, they too declined, from 13.2 per 1,000 persons in 1976 to 8.10 in 2010 (a decrease of 38.8%). Thus, both cold- and heat-related death rates declined over the 35-year period of study. The authors concluded, “Hong Kong has not observed an increase in heat-related deaths as predicted in the Western literature.”

Ma *et al.* (2015) used a distributed lag non-linear model to determine the community-specific effects of extreme hot and cold temperatures on non-accidental mortality during 2006–2011 in 66 Chinese cities, after which they conducted a multivariate meta-analysis that enabled them to pool the individual estimates of community-specific effects. They found a U-shaped relationship whereby both daily maximum and minimum temperatures were associated with increased mortality risk compared to

that of the overall mean temperature, but the relative risk (RR) at the mean daily minimum temperature was significantly greater than the RR at the mean daily maximum temperature. Typically experienced extreme cold throughout China, they conclude, is much more deadly than is typically experienced extreme heat, which the 14 researchers note “is consistent with previous studies,” including Guo *et al.* (2011), Guo *et al.* (2013), Chen *et al.* (2013), Wu *et al.* (2013), and Xie *et al.* (2013).

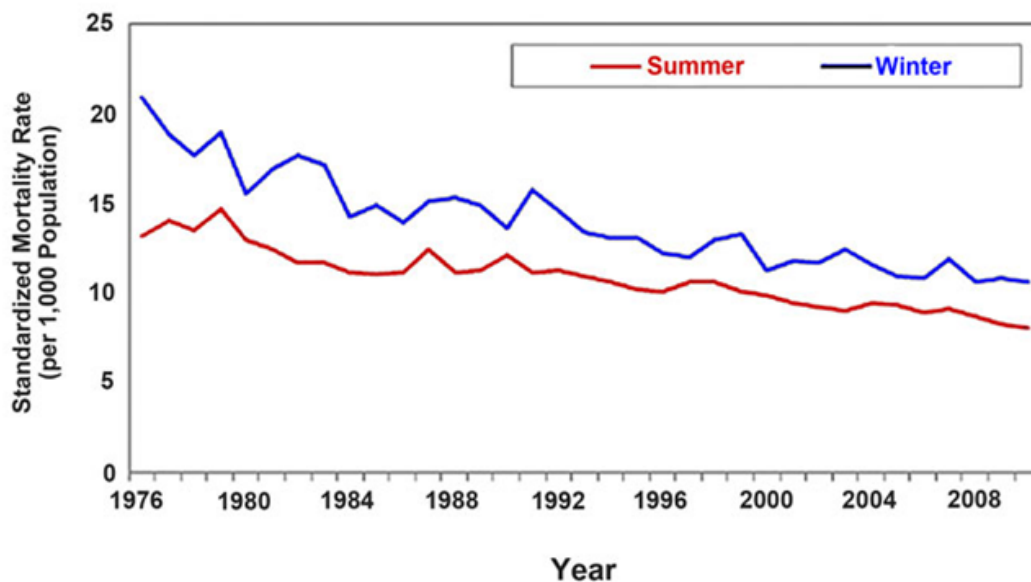
Ng *et al.* (2016) analyzed “daily total [from natural causes], cardiovascular and respiratory disease mortality and temperature data from 1972 to 2010 for 47 prefectures.” They report their data “show a general decrease in excess heat-related mortality over the past 39 years despite increasing temperatures [of approximately 1°C],” demonstrating, in their words, “that some form of adaptation to extreme temperatures has occurred in Japan.” More specifically, their data revealed a national reduction of 20, 21, and 46 cases of deaths per 1,000 due to natural, cardiovascular, and respiratory causes, corresponding to respective drops of 69, 66, and 81%. Ng *et al.* write, an “increase of AC [air conditioning] prevalence was not associated

with a reduction of excess mortality over time,” yet they note “prefectures and populations with improved economic status documented a larger decline of excess mortality,” adding that “healthcare resources were associated with fewer heat-related deaths in the 1970s, but the associations did not persist in the more recent period (i.e., 2006–2010).”

Wang *et al.* (2016) collected daily mortality and meteorological data from 66 communities across China over the period 2006–2011. They analyzed the data to discern relationships between cold spell characteristics and human mortality, finding cold spells significantly increased human mortality risk in China. They found the combined cumulative excess mortality risk (CER) for all of China when defining cold spells with a 5th and 2.5th percentile temperature intensity threshold was 28.5 and 39.7%, respectively. However, there were notable geographic differences: CER was tempered and near zero in the colder/higher latitudes, but increased to 58.7 and 92.9% at the corresponding 5th and 2.5th percentile temperature intensity thresholds for the warmest and most southern latitude. Such geographic differences in mortality risk, according to the authors, are likely the product of better physiological and behavioral

Figure 4.2.3.1

Summer and winter age-standardized mortality rate (per 1,000 population) for adults age 65 and older in Hong Kong over the period 1976–2010



Source: Adapted from Chau and Woo, 2015.

acclimatization of the northerly populations to cold weather.

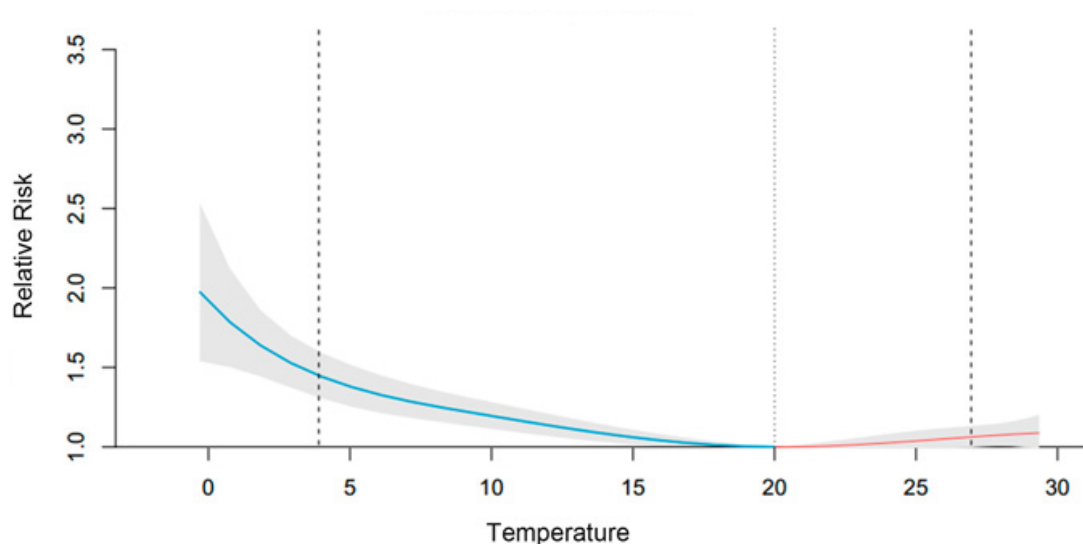
Wang *et al.* also report that the strength of the temperature/mortality relationship was modified by cold spell characteristics and human-specific factors, such that there was a significant increase in non-accident mortality during cold spells that were longer, stronger, and/or earlier in the season. In addition, mortality rates were found to increase by age and decrease by education level; the older and less educated tended to experience the greatest risk of death. The health status of an individual was also a factor. Those with respiratory illnesses had higher CER rates than those suffering from cardiovascular or cerebrovascular diseases.

Cui *et al.* (2016) set out to examine which end of the temperature spectrum (hot or cold weather) exerts a greater deleterious effect on human health. They focused their analysis on the largest city within the Sichuan Province of China, Chengdu, with a population of 14.65 million. They gathered daily meteorological and death record data for the city for the period January 1, 2011 through December 31, 2014 and estimated the relationship between daily mortality and ambient temperature using a

distribution lag model with a quasi-Poisson regression, controlling for long-time trends and day of the week. They also calculated the relative risk of mortality, defined as the risk of death attributable to heat or cold above or below the optimum temperature at which minimum mortality occurred. They found the “total fraction of deaths caused by both heat and cold was 10.93%.” However, they note “the effect of cold was significant and was responsible for most of the burden,” whereas “the effect of heat was small and non-significant.” The effect of cold temperatures was calculated to be ten times larger than that of warm temperatures (9.96% vs. 0.97%). Figure 4.2.3.2 illustrates the far greater impact of cold temperatures.

Chung *et al.* (2017) note “understanding how the temperature-mortality association worldwide changes over time is crucial to addressing questions of human adaptation under climate change.” They investigated the temporal change in this relationship for 15 cities in three countries from Northeast Asia (Japan, Korea, and Taiwan) over the past four decades, during which time temperatures increased in all cities. They utilized a generalized linear model with splines, allowing them to investigate a nonlinear association between temperature and mortality, as well as a non-

Figure 4.2.3.2
Exposure-response relationship of temperature and non-accidental deaths in Chengdu, China (2011–2014)



The blue part of the curve is the exposure-response association (with 95% empirical confidence interval, shaded grey) of cold, and the red one presents the heat. The dotted line at 20°C is minimum mortality temperature and the dashed lines are the 2.5th and 97.5th percentile. *Source:* Adapted from Cui *et al.*, 2016.

linear change in this association over time. Additionally, their analysis was stratified by cause-specific mortality (from cardiovascular, respiratory, and non-cardiorespiratory) and age group (under age 65, 65–75 years, and greater than 75 years of age).

Their analysis revealed that cold-related mortality risk remained relatively constant over time, with only one of the 15 locations exhibiting a trend that was statistically significant. In contrast, all of the study locations revealed declines in heat-related mortality (weighted average of approximately -16% change), only three of which declines were not statistically significant. Chung *et al.* also report the temporal pattern of decreasing heat-related mortality differed by age and cause of death, where the oldest segment of the population and respiratory-caused deaths experienced the largest decreases.

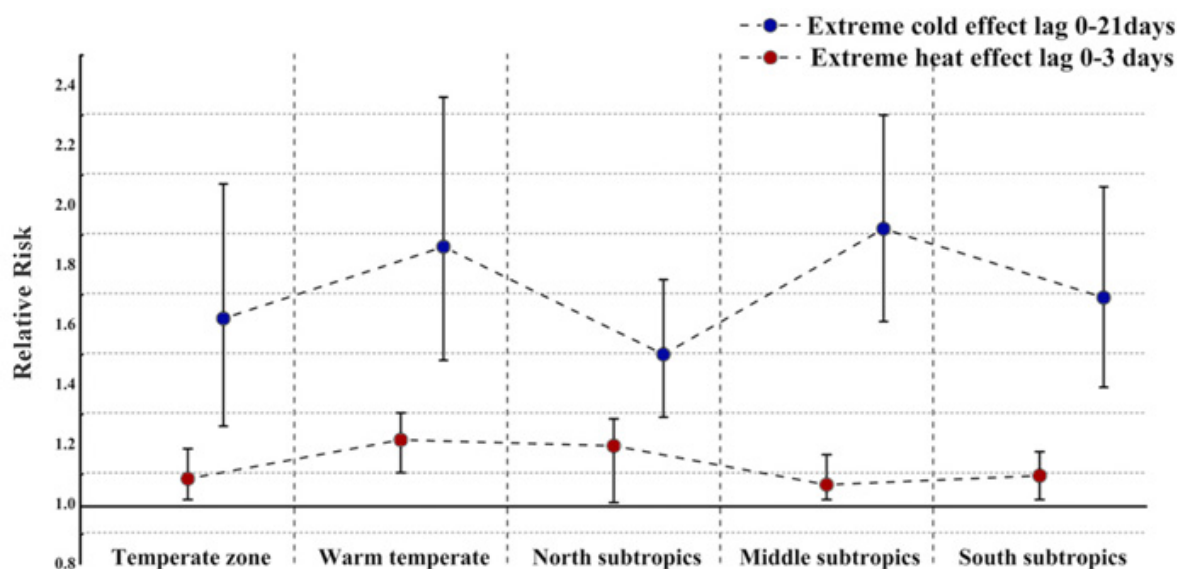
Chung *et al.*'s findings dispel two claims: that global warming will enhance heat-related deaths and that the elderly population will suffer the most. In direct contradiction of these assertions, the results of this study clearly demonstrate that populations are adapting to warmer temperatures much better than to colder temperatures (by a factor of about 10), as evidenced by declining trends in mortality risk over time, and the elderly are not suffering a disproportion ate number of heat-related deaths. Whereas trends in heat-related deaths were higher in the older populations at the beginning of the records, they have disproportionately declined and, Chung *et al.* report, “converged to become similar among the three age groups in later years.” Wang *et al.* (2017) studied 122 communities across mainland China, using daily non-accidental mortality and meteorological data for the period January 1, 2007 through December 31, 2012 and a quasi-Poisson regression with a distributed lag nonlinear model to estimate the relationship between daily mean temperature and mortality in each of the communities they studied. They pooled their data into one of five temperature zones from which they analyzed the temperature-mortality relationship at the regional and national level. They report that both high and low temperatures increase the risk of mortality, but that the risk is higher and lasts longer at the cold edge of the temperature spectrum. Qualitatively, the relative risk of mortality due to cold was 1.63 versus 1.15 for heat.

To further illustrate the greater danger of extreme cold, the average relative risk of mortality due to extreme cold and heat for each of the five temperature regions are presented in Figure 4.2.3.3.

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Figure 4.2.3.3
Pooled mortality risks of extreme cold and heat for five temperature zones



Source: Wang *et al.*, 2017.

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4.2.4 Australia

Bennett *et al.* (2014) studied the ratio of summer to winter deaths against a background of rising average annual temperatures over a period of four decades in Australia, finding this summer/winter “death ratio” had increased from a value of 0.71 to 0.86 since 1968, due to summer deaths rising faster than winter deaths.

Bennett *et al.* also note “the change [the increase in summer/winter death ratio] has so far been driven more by reduced winter mortality [due to reductions in extreme cold] than by increased summer mortality [due to increases in extreme warmth],” as well as the fact that the greater number of typical winter-season deaths “is largely explained,” in their words, “by infectious disease transmission peaks during winter and the exacerbation of chronic diseases, especially cardiovascular and respiratory conditions,” citing Cameron *et al.* (1985).

In a study of Adelaide, Brisbane, Melbourne, Perth, and Sydney, Australia, Huang *et al.* (2015) split “seasonal patterns in temperature, humidity and mortality into their stationary (seasonal) and non-stationary (unseasonal) parts,” where “a stationary seasonal pattern is consistent from year-to-year, and a non-stationary pattern varies from year-to-year,” with the aim to determine “how unseasonal patterns in temperature and humidity in winter and summer were associated with unseasonal patterns in death.” Working with mortality data for more than 1.5 million deaths from January 1, 1988 to December 31, 2009, the researchers found there were “far more deaths in winter,” such that “death rates were 20–30% higher in a winter than a summer” (see Figure 4.2.4.1). They note “this seasonal pattern is consistent across much of the world, and many countries suffer 10% to 30% excess deaths in winter,” citing the work of Healy (2003) and Falagas *et al.* (2009). They also report that winters that were colder or drier than a typical winter had significantly increased death risks, whereas “summers that were warmer or more humid than average showed no increase in death risks.”

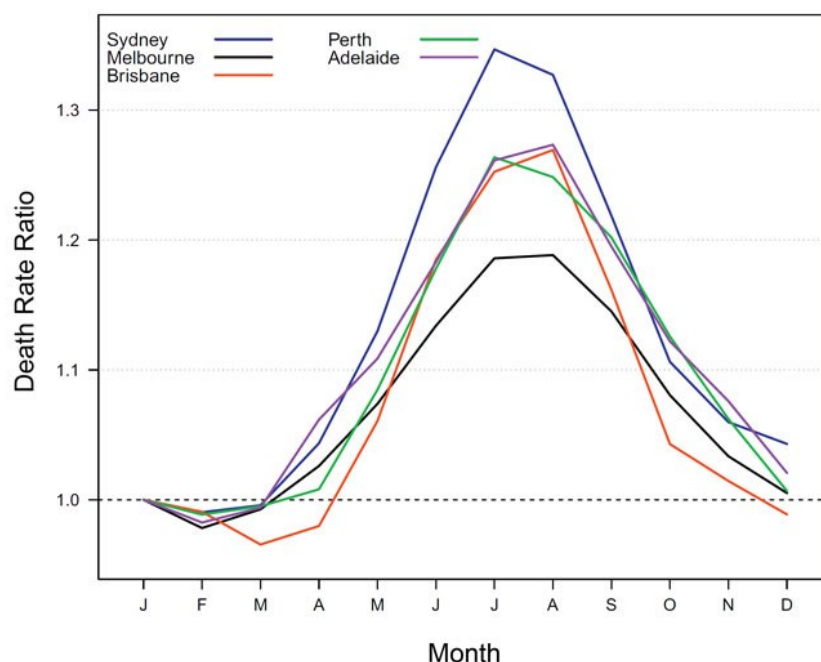
Utilizing a database of natural hazard event impacts known as PerilAUS, produced by Risk Frontiers, an independent research center sponsored by the insurance industry and located at Australia’s Macquarie University, Coates *et al.* (2014) derived “a lower-bound estimate of heat-associated deaths in Australia since European settlement.” The estimate for “extreme heat events,” also often referred to as “heat waves,” from the time of European settlement in 1844 to 2010 was at least 5,332, while from 1900 to 2010 it was 4,555.

The five researchers also determined “both deaths and death rates (per unit of population) fluctuate widely but show an overall decrease with time.” In South Australia, for example, where the death rate has been the highest, they report “the decadal death rate has fallen from 1.69 deaths per 100,000 population in the 1910s to 0.26 in the 2000s,” a decline of nearly 85%. Although “the elderly are significantly more vulnerable to the risk of heat-associated death than the general population, and this vulnerability increases with age,” they find “death rates amongst seniors also show a decrease with time.” That finding is in harmony with Bobb *et al.* (2014), who found much the same thing for the elderly in the United States, where between 1987 and 2005, the decline in death rate due to heat “was largest among those ≥ 75 years of age.”

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Figure 4.2.4.1
Stationary seasonal patterns of mortality (standardized to January) in five Australian cities (1988–2009)



Australian spring/summer is September to February; fall/winter is March to August. *Source:* Adapted from Huang *et al.*, 2015.

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4.2.5 Europe

Keatinge and Donaldson (2001) analyzed the effects on human mortality of temperature, wind, rain, humidity, and sunshine during high pollution days in the greater London area over the period 1976–1995. They observed simple plots of mortality rate versus daily air temperature revealed a linear increase as temperatures fell from 15°C (59°F) to near 0°C (32°F). Mortality rates at temperatures above 15°C, however, were “grossly alinear,” as they describe it, showing no trend. Only low temperatures were found to have a significant effect on immediate and long-term mortality. They conclude, “the large, delayed

increase in mortality after low temperature is specifically associated with cold and is not due to associated patterns of wind, rain, humidity, sunshine, SO₂, CO₂ or smoke.”

Kysely and Huth (2004) calculated deviations of the observed number of deaths from the expected number of deaths for each day of the year in the Czech Republic for the period 1992–2000. They found “the distribution of days with the highest excess mortality in a year is clearly bimodal, showing a main peak in late winter and a secondary one in summer.” Regarding the smaller number of summer heat-wave-induced deaths, they also found “a large portion of the mortality increase is associated with the harvesting effect, which consists in short-term shifts in mortality and leads to a decline in the number of deaths after hot periods (e.g. Rooney *et al.*, 1998; Braga *et al.*, 2002; Laschewski and Jendritzky, 2002).” For the Czech Republic, they report, “the mortality displacement effect in the severe 1994 heat waves can be estimated to account for about 50% of the total number of victims.” As

they describe it, “people who would have died in the short term even in the absence of oppressive weather conditions made up about half of the total number of deaths.”

Diaz *et al.* (2005) examined the effect of extreme winter temperature on mortality in Madrid, Spain for people older than 65, using data from 1,815 winter days over the period 1986–1997, during which time 133,000 deaths occurred. They found that as maximum daily temperature dropped below 6°C (42.8°F), which they describe as an unusually cold day (UCD), “the impact on mortality also increased significantly.” They also found the impact of UCDs increased as the winter progressed, with the first UCD of the season producing an average of 102 deaths/day at a lag of eight days and the sixth UCD producing an average of 123 deaths/day at a lag of eight days.

Laaidi *et al.* (2006) conducted an observational population study in six regions of France between 1991 and 1995 to assess the relationship between temperature and mortality in areas of widely varying climatic conditions and lifestyles. In all cases they found “more evidence was collected showing that cold weather was more deadly than hot weather.” These findings, the researchers say, are “broadly consistent with those found in earlier studies conducted elsewhere in Europe (Kunst *et al.*, 1993; Ballester *et al.*, 1997; Eurowinter Group, 1997; Keatinge *et al.*, 2000a, 2000b; Beniston, 2002; Muggeo and Vigotti, 2002), the United States (Curriero *et al.*, 2002) and South America (Gouveia *et al.*, 2003).” They also say their findings “give grounds for confidence in the near future,” stating even a 2°C warming over the next half century “would not increase annual mortality rates.”

Analitis *et al.* (2008) analyzed short-term effects of cold weather on mortality in 15 major European cities using data from 1990–2000 and found “a 1°C decrease in temperature was associated with a 1.35% increase in the daily number of total natural deaths and a 1.72%, 3.30% and 1.25% increase in cardiovascular, respiratory, and cerebro-vascular deaths, respectively.” In addition, they report “the increase was greater for the older age groups” and the cold effect “persisted up to 23 days, with no evidence of mortality displacement.” They conclude their results “add evidence that cold-related mortality is an important public health problem across Europe and should not be overlooked by public health authorities because of the recent focus on heat-wave episodes.”

Christidis *et al.* (2010) compiled the numbers of daily deaths from all causes for men and women 50

years of age or older in England and Wales for the period 1976–2005, and then compared the death results with surface air temperature data. As expected, during the hottest portion of the year, warming led to increases in death rates, whereas during the coldest portion of the year warming led to decreases in death rates. The three scientists report there were only 0.7 death per million people per year due to warming in the hottest part of the year, but a decrease of fully 85 deaths per million people per year due to warming in the coldest part of the year, for a phenomenal lives-saved to lives-lost ratio of 121.4.

Fernandez-Raga *et al.* (2010) obtained data from weather stations situated in eight of the provincial capitals in the Castile-Leon in Spain for the period 1980–1998, and they obtained contemporary mortality data for deaths associated with cardiovascular, respiratory, and digestive system diseases. For all three of the disease types studied, they found “the death rate is about 15% higher on a winter’s day than on a summer’s day,” which they describe as “a result often found in previous studies,” citing Fleming *et al.* (2000), Verlato *et al.* (2002), Grech *et al.* (2002), Law *et al.* (2002), and Eccles (2002). Their data, plotted in Figure 4.2.5.1, clearly demonstrate the people of the Castile-Leon region of Spain are much more likely to die from a cardiovascular disease in the extreme cold of winter than in the extreme heat of summer. The same holds true with respect to dying from respiratory and digestive system diseases.

Wichmann *et al.* (2011) investigated the association between the daily three-hour maximum apparent temperature (which reflects the physiological experience of combined exposure to humidity and temperature) and deaths due to cardiovascular disease (CVD), cerebrovascular disease (CBD), and respiratory disease (RD) in Copenhagen over the period 1999–2006. During the warm half of the year (April–September), they found a rise in temperature had an inverse or protective effect with respect to CVD mortality (a 1% decrease in death in response to a 1°C increase in apparent temperature). This finding is unusual but also has been observed in Dublin, Ireland, as reported by Baccini *et al.* (2008, 2011). Wichmann *et al.* found no association with RD and CBD mortality. At the other end of the thermal spectrum, during the cold half of the year, all three associations were inverse or protective. This finding, according to the researchers, is “consistent with other studies (Eurowinter Group, 1997; Nafstad *et al.*, 2001; Braga *et al.*, 2002;

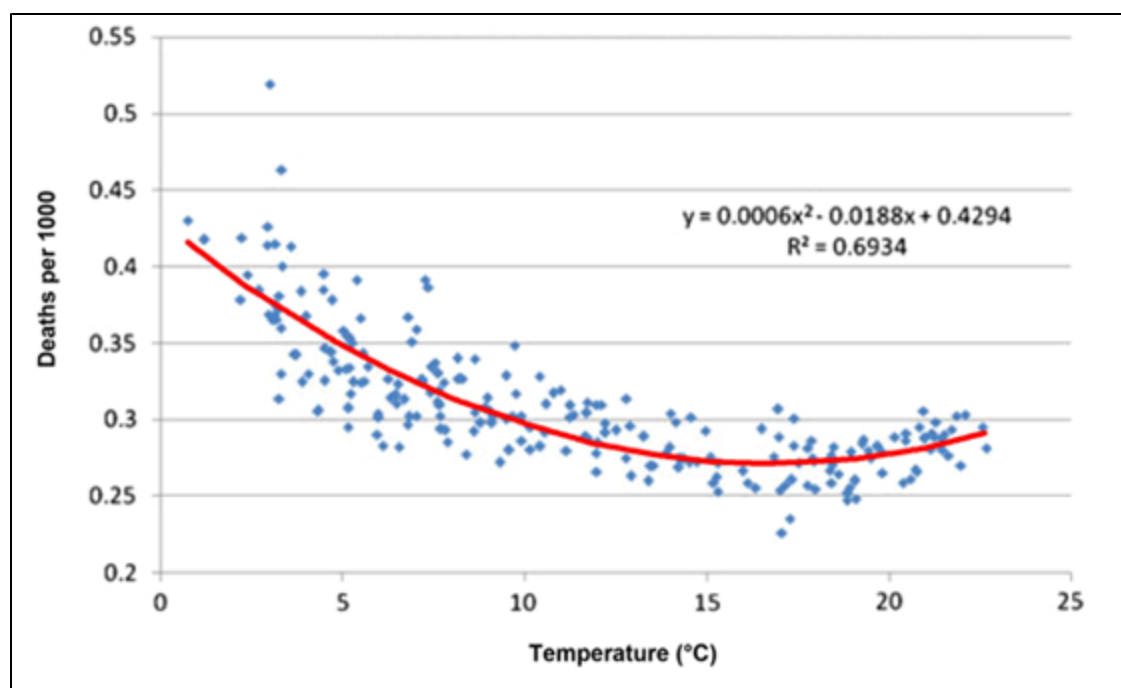
O'Neill *et al.*, 2003; Analitis *et al.*, 2008)."

Matzarakis *et al.* (2011) studied the relationship between heat stress and all-cause mortality in the densely populated city of Vienna, Austria. Based on data from 1970–2007, and after adjusting the long-term mortality rate to account for temporal variations in the size of the population of Vienna, temporal changes in life expectancy, and the changing age structure of Vienna's population, the three researchers found a significant relationship between heat stress and mortality. However, over this 38-year period, "some significant decreases of the sensitivity were found, especially in the medium heat stress levels," they report. These decreases in sensitivity, they write, "could indicate active processes of long-term adaptation to the increasing heat stress." In the discussion section of their paper, they write such sensitivity changes "were also found for other regions," citing Davis *et al.* (2003b), Koppe (2005), Tan *et al.* (2007), and Donaldson and Keatinge (2008). In the conclusion of their paper, they refer to these changes as "positive developments."

Kysely and Plavcova (2012) write, "there is much concern that climate change may be associated with large increases in heat-related mortality," but "growing evidence has been emerging that the relationships between temperature extremes and mortality impacts are nonstationary," and "most of these studies point to declining heat-related mortality in developed countries, including the US, Australia, the UK, the Netherlands and France (Davis *et al.*, 2002, 2003a, 2003b; Bi and Walker, 2001; Donaldson *et al.*, 2001; Garssen *et al.*, 2005; Carson *et al.*, 2006; Fouillet *et al.*, 2008; Sheridan *et al.*, 2009)." This is true, they note, despite "aging populations and prevailing rising trends in temperature extremes."

Kysely and Plavcova then examined "temporal changes in mortality associated with spells of large positive temperature anomalies (hot spells) in extended summer season in the population of the Czech Republic (Central Europe) during 1986–2009." They found declining mortality trends in spite of rising temperature trends, just the opposite of what the IPCC claims will occur in response to global

Figure 4.2.5.1
Monthly deaths in the Castile-Leon Region of Spain attributable to cardiovascular disease



Source: Adapted from Fernandez-Raga *et al.*, 2010.

warming. The Czech scientists add, “the finding on reduced vulnerability of the population remains unchanged if possible confounding effects of within-season acclimatization and mortality displacement are taken into account,” and “neither does it depend on the changing age structure of the population, since similar (and slightly more pronounced) declines in the mortality impacts are found in the elderly (age group 70+ years) when examined separately.”

Carmona *et al.* (2016) determined the impact of daily minimum temperatures on mortality in each of Spain’s 52 provincial capitals; they report this effort revealed relative cold-induced mortality increases of 1.13 due to natural causes, 1.18 due to circulatory causes, and 1.24 due to respiratory causes, all of which they found to be “slightly greater than those obtained to date for heat.” They note, “from a public health standpoint, there is a need for specific cold wave prevention plans at a regional level which would enable mortality attributable to low temperatures to be reduced.”

Minimum mortality temperature (MMT) is defined as the temperature at which the lowest mortality rate for a given location occurs over a given time period of examination. Above and below this value mortality rates increase as temperatures rise and fall, respectively. Todd and Valleron (2015) note MMT values in comparative studies from different latitudes are repeatedly shown to be higher in locations where the mean summer temperature is higher. Such observations, according to the two French researchers, have led to the interpretation that humans are capable of adapting to local climatic conditions. However, they additionally note that “drawing conclusions from this geographic observation about the possible adaptability of human populations to future climate change requires observing that, similarly, MMT at a given location changes over time when climate changes.” With this caveat in mind, Todd and Valleron investigated whether MMT for a given location does indeed change over time as climate changes.

Todd and Valleron examined the change in MMT in France over the 42-year period from 1968–2009 and over three 14-year subsets: 1968–1981, 1982–1995, and 1996–2009. Their data included 228 0.5 x 0.5 degree latitude/longitude grid squares of daily mean temperature and individual death certificate information for persons > 65 years old who died in France over the 42-year period of examination (approximately 16.5 million persons).

Their results indicate MMT was strongly correlated with, and had a positive linear relationship

with, mean summer temperature over the entire period. They also determined that mean MMT increased from 17.5°C in the first 14-year period (1968–1981) to 17.8°C and 18.2°C in the second (1982–1995) and third (1996–2009) time periods examined. Todd and Valleron conclude their “spatiotemporal analysis indicated some human adaptation to climate change, even in rural areas.”

Ballester *et al.* (2016) analyzed a host of climate variables against daily regional counts of mortality from 16 European countries over the period 1998–2005. They report their analyses “highlight the strong association between year-to-year fluctuations in winter mean temperature and mortality, with higher seasonal cases during harsh winters.” Exceptions were noted for Belgium, the Netherlands, and the United Kingdom, which lack of correlation was likely explained by socioeconomic factors (e.g., higher housing efficiency, better health care, reduced economic and fuel poverty, etc.). Upon further analysis, Ballester *et al.* determined that, despite the lack of mortality association in those three countries, “it can be concluded that warmer winters will contribute to the decrease in winter mortality everywhere in Europe.”

Citing the IPCC, Díaz *et al.* (2018) note climate models predict heat waves will become more frequent and intense in the future. However, they say the impact of such events on human health “is not so clear,” as human adaptation, improved health services, and the implementation of advance warning systems can minimize the impacts of heat waves on health. They investigated whether there has been a temporal change in the relative risk of human mortality in response to these and other mitigating factors.

Díaz *et al.* examined the relationship between temperature and mortality for three time periods (1983–1992, 1993–2003, and 2004–2013) using data from ten Spanish provinces, carried out for the summer period only (June–September) in each year. They found “there has been a sharp decrease in mortality attributable to heat over the past 10 years” in Spain. More specifically, they found an identical relative risk of mortality due to heat of 1.15 across the first (1983–1992) and second (1993–2003) time periods, which thereafter experienced a statistically significant decline to 1.01 during the third period (2004–2013).

Díaz *et al.* write their work shows “a drastic decrease in the impact of heat, with a decline in attributable risk per degree of $T_{\text{threshold}}$ values from 14% to 1%; a decrease of around 93%,” which they

note “is similar to the decline in the heat-related mortality rate found in Australia (Coates *et al.*, 2014) of 85% and above, though also around 70% in the U.S. (Barreca *et al.*, 2016).”

The temporal variation in relative risk observed by Díaz *et al.* calls into question the results of numerous model-based studies that project the future impact of heat on mortality to be constant over time. As observed by Díaz *et al.* and others in different parts of the world (Coates *et al.*, 2014; Petkova *et al.*, 2014; Gasparrini *et al.*, 2015; Åström *et al.*, 2016; Barreca *et al.*, 2016) such projections are likely to overestimate the true impact of heat on human mortality. If results observed for Spain apply to the rest of the world, humankind has little to fear, for its relative risk of mortality declined to such a point in the most recent period (1.01; 95% confidence interval of 1.00 to 1.01) that there was no significant heat-related mortality risk.

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4.2.6 North America

Goklany and Straja (2000) examined trends in United States death rates over the period 1979–1997 due to excessive hot and cold weather. They report there were no trends in deaths due to either extreme heat or extreme cold in the entire population or in the older, more-susceptible age groups, those aged 65 and over, 75 and over, and 85 and over. Deaths due to extreme cold in these older age groups exceeded those due to extreme heat by as much as 80 to 125%. With respect to the absence of trends in death rates attributable to either extreme heat or cold, Goklany and Straja say this “suggests that adaptation and technological change may be just as important determinants of such

trends as more obvious meteorological and demographic factors.”

Davis *et al.* (2002) studied changes in the impact of high temperatures on daily mortality rates over a period of four decades in six major metropolitan areas (Atlanta, Boston, Charlotte, Miami, New York City, and Philadelphia) along a north-south transect in the eastern United States. They found few significant weather-mortality relationships for any decade or demographic group in the three southernmost cities examined, where warmer weather is commonplace. In the three northernmost cities, however, there were statistically significant decreases in population-adjusted mortality rates during hot and humid weather between 1964 and 1994. The authors write, “these statistically significant reductions in hot-weather mortality rates suggest that the populace in cities that were weather-sensitive in the 1960s and 1970s have become less impacted by extreme conditions over time because of improved medical care, increased access to air conditioning, and biophysical and infrastructural adaptations.” They further note, “this analysis counters the paradigm of increased heat-related mortality rates in the eastern US predicted to result from future climate warming.”

Davis *et al.* (2003) evaluated “annual excess mortality on days when apparent temperatures – an index that combines air temperature and humidity – exceeded a threshold value for 28 major metropolitan areas in the United States from 1964 through 1998.” They found “for the 28-city average, there were 41.0 ± 4.8 excess heat-related deaths per year (per standard million) in the 1960s and 1970s, 17.3 ± 2.7 in the 1980s, and 10.5 ± 2.0 in the 1990s,” a remarkable decline. They conclude, “heat-related mortality in the United States seems to be largely preventable at present.”

Davis *et al.* (2004) examined the seasonality of mortality due to all causes, using monthly data for 28 major U.S. cities from 1964 to 1998, then calculated the consequences of a future 1°C warming of the conglomerate of those cities. At all locations studied, they report “warmer months have significantly lower mortality rates than colder months.” They calculate “a uniform 1°C warming results in a net mortality *decline* of 2.65 deaths (per standard million) per metropolitan statistical area” (*italics added*). The primary implication of Davis *et al.*’s findings, in their words, “is that the seasonal mortality pattern in US cities is largely independent of the climate and thus insensitive to climate fluctuations, including changes related to increasing greenhouse gases.”

O'Neill *et al.* (2005) assessed the influence of air pollution and respiratory epidemics on empirical associations between apparent temperature, which “represents an individual’s perceived air temperature,” and daily mortality in Mexico’s largest and third-largest cities: Mexico City and Monterrey, respectively. They found “in Mexico City, the 7-day temperature mortality association has a hockey stick shape with essentially no effect of higher temperatures,” whereas in Monterrey the function they fit to the data “shows a U-shape,” with “a higher mortality risk at both ends of the distribution,” although the effect is much weaker at the high-temperature end of the plot than at the low-temperature end, and the absolute value of the slope of the mortality vs. temperature relationship is smaller across the high-temperature range of the data.

Interestingly, the researchers also found that “failure to control for respiratory epidemics and air pollution resulted in an overestimate of the impact of hot days by 50%,” whereas “control for these factors had little impact on the estimates of effect of cold days.” They note “most previous assessments of effects of heat waves on hot days have not controlled for air pollution or epidemics.” Stedman (2004) made a similar claim after analyzing the impact of air pollutants present during a 2003 heat wave in the United Kingdom, claiming to have found 21% to 38% of the total excess deaths claimed to be due to high temperatures were actually the result of elevated concentrations of ozone and PM₁₀ (particulate matter of diameter less than 10µm). Likewise, Fischer *et al.* (2004) claimed 33% to 50% of the deaths attributed to the same heat wave in the Netherlands were caused by concurrent high ozone and PM₁₀ concentrations.

O'Neill *et al.*, Stedman, and Fischer *et al.* are correct in pointing to factors other than temperature contributing to deaths during periods of very warm or very cold temperatures. However, attributing fatalities to air pollution in developed countries is a dubious exercise at best. As explained in detail in Chapter 6, epidemiological studies purporting to show such attribution are easily manipulated, cannot prove causation, and often do not support a hypothesis of toxicity with the small associations in uncontrolled observational studies. Exaggeration of effects and certainty has been the rule in that field, just as has been in the climate debate.

Deschenes and Moretti (2009) analyzed the relationship between weather and mortality, based on “data that include the universe of deaths in the United States over the period 1972–1988,” in which they “match each death to weather conditions on the day

of death and in the county of occurrence.” They discovered “hot temperature shocks are indeed associated with a large and immediate spike in mortality in the days of the heat wave,” but “almost all of this excess mortality is explained by near-term displacement.” As a result, “in the weeks that follow a heat wave, we find a marked decline in mortality hazard, which completely offsets the increase during the days of the heat wave,” so “there is virtually no lasting impact of heat waves on mortality.” In the case of cold temperature days, they also found “an immediate spike in mortality” but “there is no offsetting decline in the weeks that follow,” so “the cumulative effect of one day of extreme cold temperature during a thirty-day window is an increase in daily mortality by as much as 10%.”

Vuteovici *et al.* (2014) studied the impact of variations of diurnal temperature on daily mortality of residents of Montreal aged 65 years and older during the period 1984–2007, finding “a 5.12% increase in the cumulative effects on mortality for an increase of the diurnal temperature range from 6°C to 11°C.” When the diurnal temperature range increased from 11°C to 17.5°C, they found an 11.27% increase in mortality.

Petkova *et al.* (2014) “examined adaptation patterns by analyzing daily temperature and mortality data spanning more than a century in New York City,” where using a distributed-lag nonlinear model they analyzed the heat-mortality relationship in people 15 years of age or older during two periods – 1900–1948 and 1973–2006 – in order to “quantify population adaptation to high temperatures over time.” The three researchers report that “during the first half of the century, the decade-specific relative risk of mortality at 29°C vs. 22°C ranged from 1.30 in the 1910s to 1.43 in the 1900s.” Since 1973, however, they found “there was a gradual and substantial decline in the relative risk, from 1.26 in the 1970s to 1.09 in the 2000s.” In addition, they say “age-specific analyses indicated a greater risk for people of age 65 years and older in the first part of the century,” but “less evidence for enhanced risk among this older age group in more recent decades.” Petkova *et al.*’s discovery that the excess mortality originally experienced at high temperatures fell substantially over the course of the century they studied is indicative, in their words, of “population adaptation to heat in recent decades,” which they attribute primarily to “the rapid spread and widespread availability of air conditioning.”

Bobb *et al.* (2014) note increasing temperatures are anticipated to have health impacts but “little is

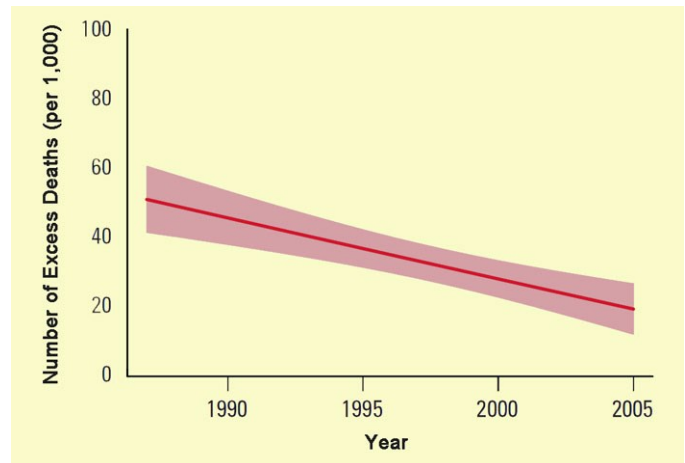
known about the extent to which the population may be adapting.” They examined “the hypothesis that if adaptation is occurring, then heat-related mortality would be decreasing over time,” using “a national database of daily weather, air pollution, and age-stratified mortality rates for 105 U.S. cities (covering 106 million people) during the summers of 1987–2005.” They found, “on average across cities, the number of deaths (per 1,000 deaths) attributable to each 10°F increase in same-day temperature decreased from 51 in 1987 to 19 in 2005” (see Figure 4.2.6.1). They report “this decline was largest among those ≥ 75 years of age, in northern regions, and in cities with cooler climates.” In addition, they write that “although central air conditioning (AC) prevalence has increased, we did not find statistically significant evidence of larger temporal declines among cities with larger increases in AC prevalence.” They conclude the U.S. population has “become more resilient to heat over time.”

White (2017) used daily hospital visit and meteorological data to examine the dynamic relationship between temperature and morbidity in California over the period 2005–2014. He determined that the 31-day cumulative impact of a cold day with a mean temperature under 40°F (4.5°C) resulted in an 11% net increase in total morbidity (defined by the number of emergency department visits), which value rose to 17% when the cumulative impact was extended another month. (See Figure 4.2.6.2.)

The most influential disease category driving cumulative cold temperature morbidity was respiratory disease (including influenza and pneumonia), which far exceeded any other cause and amounted to approximately 6 of the 8.5 increased hospital visits per 100,000 persons that were due to cold temperatures. Stratifying the effect of cold temperatures by age group, White further reports the greatest risk of morbidity fell within the youngest age group (children under 5 years of age), which group experienced a 27.7% increase in hospital visits above the mean daily visit rate – a value four times as large as that observed for the least affected group (non-elderly adults aged 25–64).

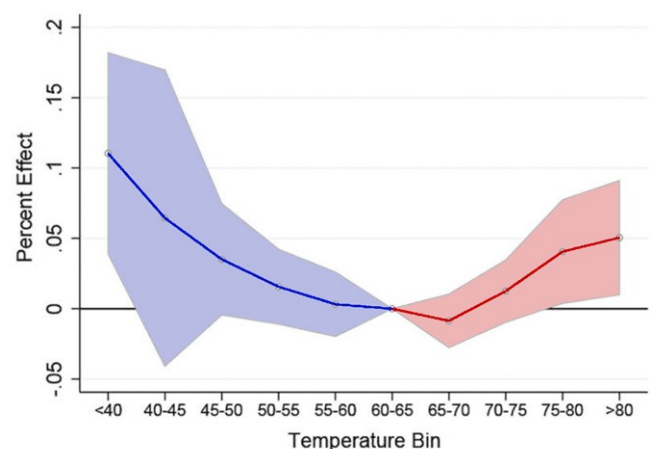
With respect to the warm end of the temperature spectrum, White found the 31-day cumulative impact of a hot day (with mean temperature above 80°F (26.7°C) was to increase human morbidity by 5.1%. Several disease categories contributed to this overall relationship, including injuries, nervous system, and

Figure 4.2.6.1
Excess U.S. deaths (per 1,000) attributable to each 10°F increase in the same day’s summer temperature, 1987–2005



Shaded region represents 95% confidence intervals.
Source: Adapted from Bobb *et al.*, 2014.

Figure 4.2.6.2
Cumulative (one month) effect of temperature (°F) on emergency department visits in California (2005–2014)



Shaded regions represent 95% confidence intervals.
Source: White, 2017.

genitourinary problems. As with cold weather, hot weather morbidity was also influenced by age, and the effect was more pronounced in the youngest age categories, where morbidity risk was 8.6% and 9.5% for the under 5 and 5–14 age groups, respectively. White also reported finding evidence of an early harvesting effect (an initial morbidity increase that is offset by a later decrease) on the elderly during hot days. The over 64 age group, for example, experienced an initial contemporaneous increase of 3.7% in morbidity on days where the mean temperature was above 80°F. However, the cumulative morbidity effect fell to 2.7% at the end of 31 days.

White also calculated the health care cost impacts associated with both hot and cold morbidity, reporting that a day over 80°F mean temperature is associated with an approximate \$8,000 increase in hospital costs per 100,000 persons, whereas cost impacts from a mean daily temperature below 40°F amount to a much larger \$12,000 per 100,000 people. Lastly, White also estimated the potential effects of climate change on human morbidity and its associated health care costs using future temperature projections derived from climate model output produced under the IPCC's RCP8.5 greenhouse gas emission scenario. Results of that exercise revealed “the effects of climate change on hospital visits and costs in California to be negligible.”

Allen and Sheridan (2018) investigated the relationship between all-cause mortality and extreme temperature events for 50 metropolitan areas in the United States with populations from 1.0 million to 19.5 million for the period 1975–2004. They assessed mortality impacts in response to three-day means of daily apparent temperature for a cumulative 14-day period following hot and cold events. They also subdivided their calculations to discern such effects from both heat and cold events (95th and 5th percentiles) and extreme heat and cold events (97.5th and 2.5th percentiles) that occurred both early and late in the summer and winter seasons, respectively. They also determined the impact on mortality of the length of heat and cold spells (short = events lasting two days or less, long = events lasting for three or more days).

Allen and Sheridan found the highest relative risk of mortality is for extreme cold events that occur early in the winter season and last for two days or less, which risk is double that observed for extreme heat events that occur in the summer. They also found the cumulative relative risk of mortality values from both short and long temperature events decline

over the course of the winter and summer seasons, suggesting there is a seasonal human adaptation to extreme weather events occurring at both ends of the temperature spectrum.

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4.3 Cardiovascular Disease

Higher surface temperatures would reduce the incidence of fatal coronary events related to low temperatures and wintry weather by a greater degree than they would increase the incidence associated with high temperatures and summer heat waves.

Non-fatal myocardial infarction is also less frequent during unseasonably warm periods than during unseasonably cold periods.

Cardiovascular diseases (CVDs) affect the heart or blood vessels. They include arrhythmia, arteriosclerosis, congenital heart disease, coronary artery disease, diseases of the aorta and its branches, disorders of the peripheral vascular system, endocarditis, heart valve disease, hypertension, orthostatic hypotension, and shock. According to the Working Group II contribution to the Intergovernmental Panel on Climate Change (IPCC) Fifth Assessment Report, “Numerous studies of temperature-related morbidity, based on hospital admissions or emergency presentations, have reported increases in events due to cardiovascular, respiratory, and kidney diseases (Hansen *et al.*, 2008; Knowlton *et al.*, 2009; Lin and Chan, 2009) and the impact has been related to the duration and intensity of heat (Nitschke *et al.*, 2011) (IPCC, 2014, p. 721). The IPCC overlooks the fact that cooler temperatures cause an even larger number of premature deaths, with the result that a warmer world would experience fewer deaths in total.

Nafstad *et al.* (2001) examined the effects of temperature on mortality due to all forms of cardiovascular disease for citizens of New South Wales, Australia, over the period 1990 to 1995. Their analysis showed the average daily number of cardiovascular-related deaths was 15% higher in the winter months (October–March) than in the summer months (April–September), leading them to conclude

“a milder climate would lead to a substantial reduction in average daily number of deaths.” This confirmed an earlier finding by Enquesselassie *et al.* (1993) of the Hunter region of New South Wales which covered July 1, 1985 to June 30, 1990 and found “fatal coronary events and non-fatal definite myocardial infarction were 20–40% more common in winter and spring than at other times of year.” With respect to daily temperature effects, they found “rate ratios for deaths were significantly higher for low temperatures,” noting “on cold days coronary deaths were up to 40% more likely to occur than at moderate temperatures.”

Hajat and Haines (2002) analyzed data obtained between January 1992 and September 1995 for registered patients aged 65 and older from several medical practices in London, England. They found the number of general practitioner consultations was higher in the cool-season months (October–March) than in the warm-season months (April–September) for all CVDs.

Braga *et al.* (2002) determined both the acute effects and lagged influence of temperature on cardiovascular-related deaths in a study of both “hot” and “cold” cities in the United States: Atlanta, Georgia; Birmingham, Alabama; and Houston, Texas comprised the “hot” group, and Canton, Ohio; Chicago, Illinois; Colorado Springs, Colorado; Detroit, Michigan; Minneapolis-St. Paul, Minnesota; New Haven, Connecticut; Pittsburgh, Pennsylvania; and Seattle and Spokane, Washington comprised the “cold” group. They found in the hot cities neither hot nor cold temperatures had much impact on mortality related to CVDs. In the cold cities, on the other hand, both high and low temperatures were associated with increased CVD deaths. The effect of cold temperatures persisted for days, whereas the effect of high temperatures was restricted to the day of the death or the day before. For all CVD deaths, the hot-day effect was five times smaller than the cold-day effect. In addition, the hot-day effect included some “harvesting,” where Braga *et al.* observed a deficit of deaths a few days later, which they did not observe for the cold-day effect.

Gouveia *et al.* (2003) determined the number of cardiovascular-related deaths in adults aged 15–64 in Sao Paulo, Brazil over the period 1991–1994 increased by 2.6% for each 1°C decrease in temperature below 20°C (68°F), while they found no evidence for heat-induced deaths due to temperatures rising above 20°C. In the elderly (65 years of age and above), a 1°C warming above 20°C led to a 2% increase in deaths, but a 1°C cooling below 20°C led

to a 6.3% increase in deaths, more than three times as many cardiovascular-related deaths due to cooling than to warming in the elderly.

McGregor *et al.* (2004) obtained and analyzed data on ischaemic heart disease (IHD) and temperature for five English counties aligned on a north-south transect (Tyne and Wear, West Yorkshire, Greater Manchester, West Midlands, and Hampshire) for the period 1974–1999. They determined “the seasonal cycles of temperature and mortality are inversely related,” and “the first harmonic accounts for at least 85% (significant at the 0.01 level) of the variance of temperature and mortality at both the climatological and yearly time scales.” They also report “years with an exaggerated mortality peak are associated with years characterized by strong temperature seasonality,” and “the timing of the annual mortality peak is positively associated with the timing of the lowest temperatures.”

Chang *et al.* (2004) studied the effects of monthly mean temperature on rates of hospitalization for arterial stroke and acute myocardial infarction (AMI) among young women aged 15–49 from 17 countries in Africa, Asia, Europe, Latin America, and the Caribbean. These efforts revealed “among young women from 17 countries, the rate of hospitalized AMI, and to a lesser extent stroke, was higher with lower mean environmental air temperature.” They report, “on average, a 5°C reduction in mean air temperature was associated with a 7 and 12% increase in the expected hospitalization rates of stroke and AMI, respectively.” Finally, they note, “lagging the effects of temperature suggested that these effects were relatively acute, within a period of a month.”

Bartzokas *et al.* (2004) “examined the relationship between hospital admissions for cardiovascular (cardiac in general including heart attacks) and/or respiratory diseases (asthma etc.) in a major hospital in Athens [Greece] and meteorological parameters for an 8-year period.” Over the study period, “there was a dependence of admissions on temperature” and low temperatures were “responsible for a higher number of admissions,” they found. Specifically, “there was a decrease of cardiovascular or/and respiratory events from low to high values [of temperature], except for the highest temperature class in which a slight increase was recorded.”

Nakaji *et al.* (2004) evaluated seasonal trends in deaths in Japan from 1970 to 1999 and recorded mean monthly temperature. The nine researchers note Japan has “bitterly cold winters,” and their analysis

indicates the numbers of deaths due to infectious and parasitic diseases – including tuberculosis, respiratory diseases including pneumonia and influenza, diabetes, digestive diseases, and cerebrovascular and heart diseases – rise to a maximum during that cold time of year. Of the latter two categories, they found peak mortality rates due to heart disease and stroke were one-and-a-half to two times greater in winter (January) than in August and September, when mortality rates for those conditions are at their yearly minimums.

Sharovsky *et al.* (2004) investigated “associations between weather (temperature, humidity, and barometric pressure), air pollution (sulfur dioxide, carbon monoxide, and inhalable particulates), and the daily death counts attributed to myocardial infarction” in Sao Paulo, Brazil, where 12,007 deaths were observed from 1996 to 1998. As mean daily temperature dropped below 18°C, death rates rose in essentially linear fashion to attain a value at 12°C (the typical lower limit of observed temperature in Sao Paulo) more than 35% greater than the minimum baseline value registered between 21.6°C and 22.6°C. Sharovsky *et al.* say “myocardial infarction deaths peak in winter not only because of absolute low temperature but possibly secondary to a decrease relative to the average annual temperature.”

Kovats *et al.* (2004) analyzed patterns of temperature-related hospital admissions and deaths in Greater London during the mid-1990s. For the three-year period 1994–1996, cardiovascular-related deaths were approximately 50% greater during the coldest part of the winter than during the peak warmth of summer, whereas respiratory-related deaths were nearly 150% greater in the depths of winter cold than at the height of summer warmth. With respect to heat waves, the mortality impact of the notable heat wave of July 29 to August 3, 1995 was so tiny it could not be discerned among the random scatter of plots of three-year-average daily deaths from cardiovascular and respiratory problems versus day of year.

Carder *et al.* (2005) investigated the relationship between outside air temperature and deaths due to all non-accident causes in the three largest cities of Scotland (Glasgow, Edinburgh, and Aberdeen) between January 1981 and December 2001. They observed “an overall increase in mortality as temperature decreases,” which “appears to be steeper at lower temperatures than at warmer temperatures,” while “there is little evidence of an increase in mortality at the hot end of the temperature range.” The seven scientists found, for temperatures below 11°C, a 1°C drop in the daytime mean temperature on

any one day was associated with an increase in cardiovascular-caused mortality of 3.4% over the following month. At any season of the year a decline in air temperature in the major cities of Scotland leads to increases in deaths due to cardiovascular causes, whereas there is little or no such increase in mortality associated with heat waves.

Cagle and Hubbard (2005) examined the relationship between temperature and cardiac-related deaths in King County, Washington (USA) over the period 1980–2000. They determined there was an average of 2.86 cardiac-related deaths per day for all days when the maximum temperature fell within the broad range of 5–30°C. For days with maximum temperatures less than 5°C, the death rate rose by 15% to a mean value of 3.30, whereas on days with maximum temperatures greater than 30°C, death rates fell by 3% to a mean value of 2.78. In addition, “the observed association between temperature and death rate is not due to confounding by other meteorological variables,” and “temperature continues to be statistically significantly associated with death rate even at a 5-day time lag.”

Tam *et al.* (2009) employed daily mortality data in Hong Kong for the years 1997 to 2002 to examine the association between diurnal temperature range (DTR) and cardiovascular disease among the elderly (age 65 and older). They report “a 1.7% increase in mortality for an increase of 1°C in DTR at lag days 0–3” and describe these results as being “similar to those reported in Shanghai.” The four researchers state “a large fluctuation in the daily temperature – even in a tropical city like Hong Kong – has a significant impact on cardiovascular mortality among the elderly population.”

Cao *et al.* (2009) assessed the relationship between DTR and coronary heart disease (CHD) deaths of elderly people (66 years of age or older) occurring in Shanghai between January 1, 2001 and December 31, 2004. They found “a 1°C increase in DTR (lag = 2) corresponded to a 2.46% increase in CHD mortality on time-series analysis, a 3.21% increase on unidirectional case-crossover analysis, and a 2.13% increase on bidirectional case-crossover analysis,” and “the estimated effects of DTR on CHD mortality were similar in the warm and cool seasons.” The seven scientists conclude their “data suggest that even a small increase in DTR is associated with a substantial increase in deaths due to CHD.”

Bayentin *et al.* (2010) analyzed the standardized daily hospitalization rates for ischemic heart disease (IHD) and their relationship with climatic conditions up to two weeks prior to the day of admission to

determine the short-term effects of climate conditions on the incidence of IHD over the period 1989–2006 for 18 health regions of Quebec. The authors report “a decline in the effects of meteorological variables on IHD daily admission rates” that “can partly be explained by the changes in surface air temperature,” which they describe as warming “over the last few decades.”

Toro *et al.* (2010) used data on 7,450 cardiovascular-related deaths in Budapest, Hungary between 1995 and 2004 to find potential relationships between those deaths and daily maximum, minimum, and mean temperature, air humidity, air pressure, wind speed, and global radiation. The six Hungarian scientists report “on the days with four or more death cases, the daily maximum and minimum temperatures tend to be lower than on days without any cardiovascular death events,” “the largest frequency of cardiovascular death cases was detected in cold and cooling weather conditions,” and “we found a significant negative relationship between temperature and cardiovascular mortality.”

Bhaskaran *et al.* (2010) explored the short-term relationship between ambient temperature and risk of heart attacks (myocardial infarction) in England and Wales by analyzing 84,010 hospital admissions from 2003 to 2006. They found a broadly linear relationship between temperature and heart attacks that was well characterized by log-linear models without a temperature threshold, such that each 1°C reduction in daily mean temperature was associated with a 2.0% cumulative increase in risk of myocardial infarction over the current and following 28 days. They also report heat had no detrimental effect, as an increased risk of myocardial infarction at higher temperatures was not detected.

Kysely *et al.* (2011) used a database of daily mortality records in the Czech Republic that cover the 21-year period 1986–2006 – which, in their words, “encompasses seasons with the hottest summers on record (1992, 1994, 2003) as well as several very cold winters (1986/87, 1995/96, 2005/06)” – to compare the effects of hot and cold periods on cardiovascular mortality. The four Czech scientists report “both hot and cold spells are associated with significant excess cardiovascular mortality,” but “the effects of hot spells are more direct (unlagged) and typically concentrated in a few days of a hot spell, while cold spells are associated with indirect (lagged) mortality impacts persisting after a cold spell ends.” Although they report “the mortality peak is less pronounced for cold spells,” they determined “the cumulative magnitude of excess

mortality is larger for cold than hot spells.” They conclude, “in the context of climate change, substantial reductions in cold-related mortality are very likely in mid-latitudinal regions, particularly if the increasing adaptability of societies to weather is taken into account (cf. Christidis *et al.*, 2010),” and “it is probable that reductions in cold-related mortality will be more important than possible increases in heat-related mortality.”

Lim *et al.* (2012) assessed the effects of increasing DTR on hospital admissions for the most common cardiovascular and respiratory diseases in the four largest cities of Korea (Seoul, Incheon, Daegu, and Busan) for the period 2003–2006. According to the three South Korean researchers, the data showed “the area-combined effects of DTR on cardiac failure and asthma were statistically significant,” and the DTR effects on asthma admissions were greater for the elderly (75 years or older) than for the non-elderly group. “In particular,” they write, “the effects on cardiac failure and asthma were significant with the percentage change of hospital admissions per 1°C increment of DTR at 3.0% and 1.1%, respectively.”

Wanitschek *et al.* (2013), noting Austria’s 2005/2006 winter was very cold whereas the 2006/2007 winter was extraordinarily warm, studied the cases of patients who were suffering acute myocardial infarctions and had been referred for coronary angiography (CA). They compared the patients’ risk factors and in-hospital mortality rates between these two consecutive winters and found nearly identical numbers of CA cases (987 vs. 983), but 12.9% of the CA cases in the colder winter were acute, while 10.4% of the cases in the warmer winter were acute. They conclude, “the average temperature increase of 7.5°C from the cold to the warm winter was associated with a decrease in acute coronary angiographies ...”

Vasconcelos *et al.* (2013) studied the health-related effects of a daily human-biometeorological index known as the Physiologically Equivalent Temperature (PET), which is based on the input parameters of air temperature, humidity, mean radiant temperature, and wind speed, as employed by Burkart *et al.* (2011), Grigorieva and Matzarakis (2011), and Cohen *et al.* (2012), focusing their attention on Lisbon and Oporto Counties in Portugal over the period 2003–2007. The five Portuguese researchers report there was “a linear relationship between daily mean PET, during winter, and the risk of myocardial infarction, after adjustment for confounding factors,” thus confirming “the thermal

environment, during winter, is inversely associated with acute myocardial infarction morbidity in Portugal.” They observed “an increase of 2.2% of daily hospitalizations per degree fall of PET, during winter, for all ages.” In Portugal and many other countries where low winter temperatures “are generally under-rated compared to high temperatures during summer periods,” Vasconcelos *et al.* conclude cold weather is “an important environmental hazard” that is much more deadly than the heat of summer.

Hart (2015) writes, “warm temperatures are thought to be associated with increased death rates,” citing the works of Longstreth (1991) and Zanobettia *et al.* (2012). Rather than accepting the assumption at face value, Hart conducted a statistical analysis to test it. Using linear multiple regression analysis of data from all 67 counties in Alabama (USA), he analyzed the relationship between daily mean air temperature and land elevation (both as predictor variables) and death rates from cancer and heart disease (the two response variables) over the periods 2006–2010 and 2008–2010, respectively. Hart reports there was no “statistically significant adverse health effects for either predictor with these response variables” (see Figure 4.3.1). However, as evident in the figure, his analysis did reveal an inverse relationship between temperature and heart disease death rates, such that a one degree Fahrenheit rise in temperature would have the effect of reducing heart disease death rates by 12 persons per 100,000 over the temperature range analyzed in his study. The findings, Hart writes, “contradict dire predictions of adverse health consequences as a result of global warming,” yet are “consistent with a previous report that indicated a beneficial association between warmer temperatures and decreased mortality (Idso *et al.*, 2014).”

Ponjoan *et al.* (2017) analyzed the effects of both heat waves and cold spells on emergency hospitalizations due to cardiovascular diseases in Catalonia (a region of Spain in the Mediterranean basin) over the period 2006–2013. They used the self-controlled case series statistical methodology to assess the relative incidence rate ratios (IRRs) of hospitalizations during the hot and cold waves in comparison to reference time periods with normal temperature exposure. Heat waves were defined as a period of at least three days in July and August in which the daily maximum temperatures were higher than the 95th percentile of daily maximum temperature for those two months. Cold waves were similarly defined as periods of at least three days in January and February when daily minimum temperatures were lower than the 95th percentile of

daily minimum temperatures for those two months. IRRs were adjusted for age, time interval, and air pollution. The number of hospitalizations due to cardiovascular diseases during January and February over the period of study was 22,611, whereas there were only 17,017 during July and August.

Ponjoan *et al.* found the incidence of cardiovascular hospitalizations increases during cold spells by 20%, rising to 26 and 29% when a lag of three and seven days, respectively, are added to the cold spell. In contrast, Ponjoan *et al.* report, “the effect of heatwaves on overall cardiovascular hospitalizations was not significantly different from the null,” adding “no significant differences were observed when stratifying by sex, age or cardiovascular type categories” for either heat waves or cold spells. This latter finding challenges the oft-repeated concern that the elderly will disproportionately suffer more health maladies than younger people during such temperature departures (either hot or cold).

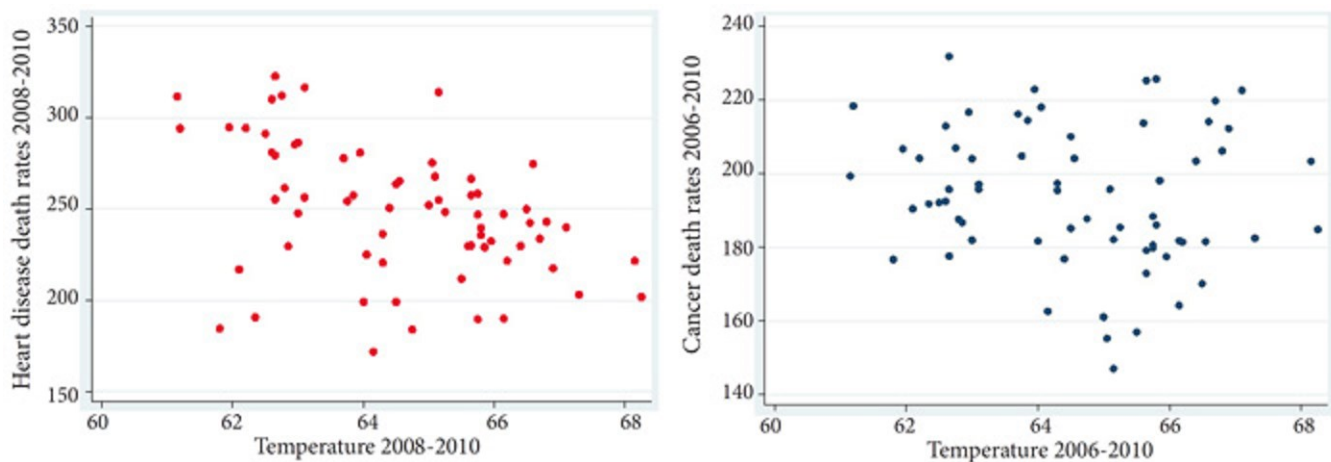
In a review of the research literature, Claeys *et al.* (2017) note “acute myocardial infarctions (AMIs) are the leading cause of mortality worldwide and are usually precipitated by coronary thrombosis, which is induced by a ruptured or eroded atherosclerotic plaque that leads to a sudden and critical reduction in

blood flow,” citing Davies and Thomas (1985), Nichols *et al.* (2013), and a 2014 report of the American Heart Association that was produced by a team of 44 researchers. Claeys *et al.* note, “the majority of the temperature-related mortality has been shown to be attributable much more to cold, when compared with extreme hot weather,” citing the work of Gasparrini *et al.* (2015) and reporting that “for each 10°C decrease in temperature, there was a 9% increase in the risk of AMI.”

Zhang *et al.* (2017) studied daily meteorological data and records of all registered deaths in Wuhan, central China, between 2009 and 2012, performing a series of statistical procedures to estimate the exposure-response impact of DTR on human mortality (including non-accidental deaths and those due to cardiorespiratory, cardiovascular, respiratory, stroke, and ischemic heart disease causes) and years of life lost (YLL). They found a 1°C increase in DTR at lag 0–1 days significantly increased daily non-accidental mortality by 0.65% and cardiovascular-specific mortality by 1.12%. The relationships between DTR and deaths due to other investigated causes (cardiorespiratory, respiratory, stroke, and ischemic heart disease) were not significant. Nor was there any significant relationship between DTR and YLL for any of the cause-specific mortalities

Figure 4.3.1

Scatter plots of heart disease death rates and mean temperature (left panel) and cancer death rates and mean temperature (right panel) for all 67 counties in Alabama



Source: Hart, 2015.

investigated. In stratifying their findings by subgroups for the two mortality categories that did have a significant relationship with DTR (non-accidental and cardiovascular deaths), Zhang *et al.* found that, “compared with males and younger persons, females and the elderly suffered more significantly and substantially from both increased mortality and YLL in relation to a high DTR.” They also found that those who had obtained a higher degree of education were more susceptible to increased mortality and YLL from a DTR increase than those who were less educated, an unusual finding for which they suggested further research would be required.

Multiple researchers have reported a decline in DTR in recent decades at locations all across the globe in conjunction with a rise in global temperatures. For China, Shen *et al.* (2014) recently calculated a country-wide DTR decrease at a mean rate of 0.157°C/decade based on an analysis of 479 weather stations over the period 1962–2011; they cited the works of a number of other authors who also determined that the “DTR decreased significantly in China over the past several decades, including Karl *et al.* (1991, 1993), Kukla and Karl (1993), Dai *et al.* (1997, 1999), Liu *et al.* (2004), Ye *et al.* (2010), Zhou and Ren (2011), Wang and Dickinson (2013), Xia (2013), and Wang *et al.* (2014). Consequently, it would appear that if global temperatures continue to rise and the DTR continues to decline, there will likely be fewer cases of non-accidental and cardiovascular deaths in the future.

Daisuke Onozuka and Akihito Hagihara (Onozuka and Hagihara, 2017), two researchers at the Kyushu University Graduate School of Medical Sciences in Fukuoka, Japan, state that out-of-hospital cardiac arrest (OHCA) is “an on-going public health issue with a high case fatality rate and associated with both patient and environmental factors,” including temperature. Recognizing the concern that exists over the potential impacts of climate change on human health, the two scientists investigated the population-attributable risk of OHCA in Japan due to temperature, and the relative contributions of low and high temperatures on that risk, for the period 2005–2014.

Onozuka and Hagihara obtained OHCA data on more than 650,000 cases in the 10-year period from all 47 Japanese prefectures. Using climate data acquired from the Japan Meteorological Agency, they conducted a series of statistical analyses to determine the temperature-related health risk of OHCA.

They found “temperature accounted for a substantial fraction of OHCA, and ... most of [the] morbidity burden was attributable to low temperatures.” Nearly 24% of all OHCA were attributable to non-optimal temperature, and low temperature was responsible for 23.64%. The fraction of OHCA attributed to high temperature was just 0.29% – a morbidity burden two orders of magnitude smaller than that due to low temperature.

Onozuka and Hagihara also examined the impact of extreme versus moderate temperatures, as well as the effects of gender and age on OHCA risk. With respect to extreme versus moderate temperatures, the two scientists report “the effect of extreme temperatures was substantially less than that of moderate temperatures.” For gender, they determined the attributable risk of OHCA was higher for females (26.86%) than males (21.12%). For age, they found that the elderly (75–110 years old) had the highest risk at 28.39%, followed by the middle-aged (65–74 years old, 25.24% attributable risk), and then the youngest section of the population (18–64 years old, 17.93% attributable risk).

Onozuka and Hagihara’s analysis reveals moderately cold temperatures carry an inherently far greater risk for OHCA than moderately warm temperatures; extremely cold or extremely warm temperatures are responsible for only a small fraction of attributable risk of OHCA; and female and elderly portions of the population are more prone to the temperature-related effects on OHCA. It would appear modest global warming would yield great benefits for the Japanese by reducing OHCA risk/morbidity, particularly for women and the elderly.

These several studies clearly demonstrate global warming is beneficial to humanity, reducing the incidence of cardiovascular diseases related to low temperatures and wintry weather by a much greater degree than it increases the incidence of cardiovascular diseases associated with high temperatures and summer heat waves.

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4.4 Respiratory Disease

Climate change is not increasing the incidence of death, hospital visits, or loss of work or school time due to respiratory disease.

Respiratory diseases affect the organs and tissues that make gas exchange possible in humans and other higher organisms. They range from the common cold, allergies, and bronchiolitis to life-threatening conditions including asthma, chronic obstructive pulmonary disease (COPD), pneumonia, and lung cancer. Sudden acute respiratory disease (SARS) is a condition in which breathing becomes difficult and oxygen levels in the blood drop lower than normal.

Respiratory diseases are widespread. Non-fatal respiratory diseases impose enormous social costs due to days lost from work and school (Mourtzoukou and Falagas, 2007). Contrary to claims made by the IPCC, real-world data reveal unseasonable cold temperatures cause more deaths and hospital admissions due to respiratory disease than do unseasonable warm temperatures.

Before reviewing the literature on respiratory diseases generally, some background information on asthma may be useful. Childhood asthma affects more than 300 million people worldwide (Baena-Cagnani and Badellino, 2011) and has been increasing by 50% per decade in many countries (Beasley *et al.*, 2000). Like so many government bureaucracies, the U.S. Centers for Disease Control

and Prevention (CDC, 2014) has sought to associate a subject of public concern with climate change, but alternative explanations include increasing hygiene (Liu, 2007), antibiotic use (Kozyraskyj *et al.*, 2007), and the pasteurisation of cow's milk (Ewaschuk *et al.*, 2011; Loss *et al.*, 2017). Rising atmospheric CO₂ concentrations and temperatures may increase ragweed pollen numbers and perhaps other pollens associated with respiratory allergies (Wayne *et al.*, 2002), but ragweed pollen allergenicity can vary four-fold (Lee *et al.*, 1979) and pollen numbers are spatially and temporally highly variable (Weber, 2002). All of this suggests there is no simple relationship between climate or surface temperature and asthma, and therefore no reason to believe somehow slowing or stopping climate change would reduce the incidence of asthma. Asthma is discussed at greater length in Chapter 6.

Some of the studies cited earlier in this chapter on lower death rates due to warmer temperatures and cardiovascular disease also identified specific reductions in fatalities due to respiratory diseases, so their research also appears in this section. Keatinge and Donaldson (2001), for example, studied the effects of temperature on mortality in people over 50 years of age in the greater London area over the period 1976–1995. Simple plots of mortality rate versus daily air temperature revealed a linear increase in mortality as the air temperature fell from 15°C to near 0°C (59°F–32°F). Mortality rates at temperatures above 15°C, on the other hand, showed no trend. The authors say it is because “cold causes mortality mainly from arterial thrombosis and *respiratory disease*, attributable in turn to cold-induced hemo-concentration and hypertension and *respiratory infections*” (italics added).

Nafstad *et al.* (2001) studied the association between temperature and daily mortality in citizens of Oslo, Norway over the period 1990–1995. They found the mean daily number of respiratory-related deaths was considerably higher in winter (October–March) than in summer (April–September). Winter deaths associated with respiratory diseases were 47% more numerous than summer deaths. They conclude, “a milder climate would lead to a substantial reduction in average daily number of deaths.”

Hajat and Haines (2002) examined the relationship between cold temperatures and the number of visits by the elderly to general practitioners for asthma, lower respiratory diseases other than asthma, and upper respiratory diseases other than allergic rhinitis as obtained for registered patients aged 65 and older from several London

practices between January 1992 and September 1995. They found the mean number of consultations was higher in cool-season months (October–March) than in warm-season months (April–September) for all respiratory diseases. At mean temperatures below 5°C, the relationship between respiratory disease consultations and temperature was linear, and stronger at a time lag of six to 15 days. A 1°C decrease in mean temperature below 5°C was associated with a 10.5% increase in all respiratory disease consultations.

Braga *et al.* (2002) conducted a time-series analysis of both the acute and lagged influence of temperature and humidity on mortality rates in 12 U.S. cities, finding no clear evidence for a link between humidity and respiratory-related deaths. With respect to temperature, they found respiratory-related mortality increased in cities with more variable temperature. This phenomenon, they write, “suggests that increased temperature variability is the most relevant change in climate for the direct effects of weather on respiratory mortality.”

Gouveia *et al.* (2003) extracted daily counts of deaths from all causes, except violent deaths and neonatal deaths (up to one month of age), from Sao Paulo, Brazil’s mortality information system for the period 1991–1994 and analyzed them for effects of temperature. For respiratory-induced deaths, death rates due to a 1°C cooling were twice as great as death rates due to a 1°C warming in adults and 2.8 times greater in the elderly.

Nakaji *et al.* (2004) evaluated seasonal trends in deaths due to various diseases in Japan, using nationwide vital statistics from 1970 to 1999 and concurrent mean monthly air temperature data. They found the numbers of deaths due to respiratory diseases, including pneumonia and influenza, rise to a maximum during the coldest time of the year. The team of nine scientists concludes, “to reduce the overall mortality rate and to prolong life expectancy in Japan, measures must be taken to reduce those mortality rates associated with seasonal differences.”

Bartzokas *et al.* (2004) “examined the relationship between hospital admissions for cardiovascular (cardiac in general including heart attacks) and/or respiratory diseases (asthma etc.) in a major hospital in Athens [Greece] and meteorological parameters for an 8-year period.” Over the study period, they found, “there was a dependence of admissions on temperature,” and low temperatures were “responsible for a higher number of admissions.” Specifically, “there was a decrease of cardiovascular or/and respiratory events from low to

high values [of temperature], except for the highest temperature class in which a slight increase was recorded.”

Kovats *et al.* (2004) studied patterns of temperature-related hospital admissions and deaths in Greater London during the mid-1990s. For the three-year period 1994–1996, they found respiratory-related deaths were nearly 150% greater in the depth of winter cold than at the height of summer warmth. They also found the mortality impact of the heat wave of July 29 to August 3, 1995 (which boosted daily mortality by just over 10%) was so tiny it could not be discerned among the random scatter of plots of three-year-average daily deaths from cardiovascular and respiratory problems versus day of year. Similarly, in a study of temperature effects on mortality in three English counties (Hampshire, West Midlands, and West Yorkshire), McGregor (2005) found “the occurrence of influenza ... helps elevate winter mortality above that of summer.”

Carder *et al.* (2005) investigated the relationship between outside air temperature and deaths due to all non-accident causes in the three largest cities of Scotland (Glasgow, Edinburgh, and Aberdeen) between January 1981 and December 2001. The authors observed “an overall increase in mortality as temperature decreases,” which “appears to be steeper at lower temperatures than at warmer temperatures,” and “there is little evidence of an increase in mortality at the hot end of the temperature range.” Specifically regarding respiratory disease, they found “for temperatures below 11°C, a 1°C drop in the daytime mean temperature on any one day was associated with an increase in respiratory mortality of 4.8% over the following month.”

Donaldson (2006) studied the effect of annual mean daily air temperature on the length of the yearly season for respiratory syncytial virus (RSV), which causes bronchiolitis, in England and Wales for 1981–2004. Reporting “climate change may be shortening the RSV season,” Donaldson found “the seasons associated with laboratory isolation of respiratory syncytial virus (for 1981–2004) and RSV-related emergency department admissions (for 1990–2004) ended 3.1 and 2.5 weeks earlier, respectively, per 1°C increase in annual central England temperature ($P = 0.002$ and 0.043 , respectively).” Since “no relationship was observed between the start of each season and temperature,” he reports, “the RSV season has become shorter.” He concludes, “these findings imply a health benefit of global warming in England and Wales associated with a reduction in the duration

of the RSV season and its consequent impact on the health service.”

Frei and Gassner (2008) studied hay fever prevalence in Switzerland from 1926 to 1991, finding it rose from just under 1% of the country’s population to just over 14%, but from 1991 to 2000 it leveled off, fluctuating about a mean value on the order of 15%. The authors write, “several studies show that no further increase in asthma, hay fever and atopic sensitization in adolescents and adults has been observed during the 1990s and the beginning of the new century,” citing Braun-Fahrlander *et al.* (2004) and Grize *et al.* (2006). They write, “parallel to the increasing hay fever rate, the pollen amounts of birch and grass were increasing from 1969 to 1990,” but “subsequently, the pollen of these plant species decreased from 1991 to 2007.” They say this finding “is more or less consistent with the changes of the hay fever rate that no longer increased during this period and even showed a tendency to decrease slightly.” Nearly identical findings were presented a year later (Frei, 2009). Although some have claimed rising temperatures and atmospheric CO₂ concentrations will lead to more pollen and more hay fever (Wayne *et al.*, 2002), the analyses of Frei (2009) and Frei and Gassner (2008) suggest that is not true of Switzerland.

Jato *et al.* (2009) collected airborne samples of Poaceae pollen in four cities in Galicia (Northwest Spain) – Lugo, Ourense, Santiago, and Vigo – noting “the global climate change recorded over recent years may prompt changes in the atmospheric pollen season (APS).” The four researchers report “all four cities displayed a trend towards lower annual total Poaceae pollen counts, lower peak values and a smaller number of days on which counts exceeded 30, 50 and 100 pollen grains/m³.” The percentage decline in annual pollen grain counts between 1993 and 2007 in Lugo was approximately 75%, and in Santiago the decline was 80%, as best as can be determined from the graphs of the researchers’ data. In addition, they write, “the survey noted a trend towards delayed onset and shorter duration of the APS.” Thus, even though there was a “significant trend towards increasing temperatures over the months prior to the onset of the pollen season,” according to the Spanish scientists, Poaceae pollen became far less of a negative respiratory health factor in the four cities over the decade and a half of their study.

Miller *et al.* (2012) extracted from the U.S. National Health Interview Survey for 1998 to 2006 annual prevalence data for frequent otitis media

(defined as three or more ear infections per year), respiratory allergy, and non-respiratory seizures in children. They also obtained average annual temperatures for the same period from the U.S. Environmental Protection Agency. They found “annual temperature did not influence the prevalence of frequent otitis media,” “annual temperature did not influence prevalence of respiratory allergy,” and “annual temperature and sex did not influence seizure prevalence.” They conclude their findings “may demonstrate that average temperature is not likely to be the dominant cause of the increase in allergy burden or that larger changes in temperatures over a longer period are needed to observe this association.” They further conclude, “in the absence of more dramatic annual temperature changes, we do not expect prevalence of otitis media to change significantly as global warming may continue to affect our environment.”

Lin *et al.* (2013) used data on daily area-specific deaths from all causes, circulatory diseases, and respiratory diseases in Taiwan, developing relationships between each of these cause-of-death categories and a number of cold-temperature related parameters for 2000–2008. The five researchers discovered “mortality from [1] all causes and [2] circulatory diseases and [3] outpatient visits of respiratory diseases has a strong association with cold temperatures in the subtropical island, Taiwan.” In addition, they found “minimum temperature estimated the strongest risk associated with outpatient visits of respiratory diseases.”

Xu *et al.* (2013a) found that reported cases of childhood asthma increased 0–9 days after a diurnal temperature range (DTR) above 10°C, and also found a 31% increase in emergency department admissions per 5°C increment in DTR.

Xu *et al.* (2013b) found in Brisbane, Queensland, that “a 1°C increase in diurnal temperature range was associated with a 3% increase of Emergency Department Admissions for childhood diarrhea.” Their conclusion that “the incidence of childhood diarrhea may increase if climate variability increases as predicted” reveals gross ignorance of the science on greenhouse warming. This is another example of the statement on page 2 about “independent researchers who should know better.”

Liu *et al.* (2015) examined the association between temperature change and clinical visits for childhood respiratory tract infections (RTIs) in Guangzhou, China based on outpatient records of clinical visits for pediatric RTIs between January 1, 2012 and December 31, 2013, where temperature

change was defined as the difference between the mean temperatures of two consecutive days, and where a distributed lag non-linear model was employed to examine the impact of the observed temperature changes. Their analyses of the weather and hospital data revealed “a large temperature decrease was associated with a significant risk for an RTI, with the effect lasting for ~10 days.” In addition, they found that “children aged 0–2 years, and especially those aged <1 year, were particularly vulnerable to the effects of temperature drop,” noting an extreme temperature decrease “was significantly associated with increased pediatric outpatient visits for RTI’s in Guangzhou.”

Xie *et al.* (2017) investigated the relationship between acute bronchitis and diurnal temperature range (DTR), which they considered to be “a meteorological indicator closely associated with urbanization, global climate change and [reflective of] the stability of the weather.” They examined 14,055 cases of acute bronchitis among children aged 0–14 in Hefei, China over the winter months (December–February) of 2010 through 2013. Their analysis indicated the risk of bronchitis increased as the DTR increased, which relationship was greatest at a 3-day lag, where a 1°C increase in DTR led to a 1.0% increase in the number of daily cases for childhood bronchitis.

Song *et al.* (2018) analyzed daily meteorological data and daily emergency hospital visits in the Haidian district of Beijing, China between 2009 and 2012. They studied the relative risk of respiratory morbidity for both heat waves and cold spells of ≥ 2 , ≥ 3 , and ≥ 4 days of duration in which the average daily temperature fell within the 95th through 99th percentiles and 1st through 5th percentiles, respectively.

Song *et al.* found the relationship between ambient temperature and respiratory emergency department visits followed a U-shaped curve, where the minimum relative risk value of 1.0 was observed at a mean daily temperature of 21.5°C, a full 6.0°C warmer than the mean average temperature of the entire study period (15.5°C). Given that this minimum-morbidity temperature is much higher than the mean temperature over the period of study, some form of human adaptability or respiratory morbidity acclimation to warmer weather appears to be taking place.

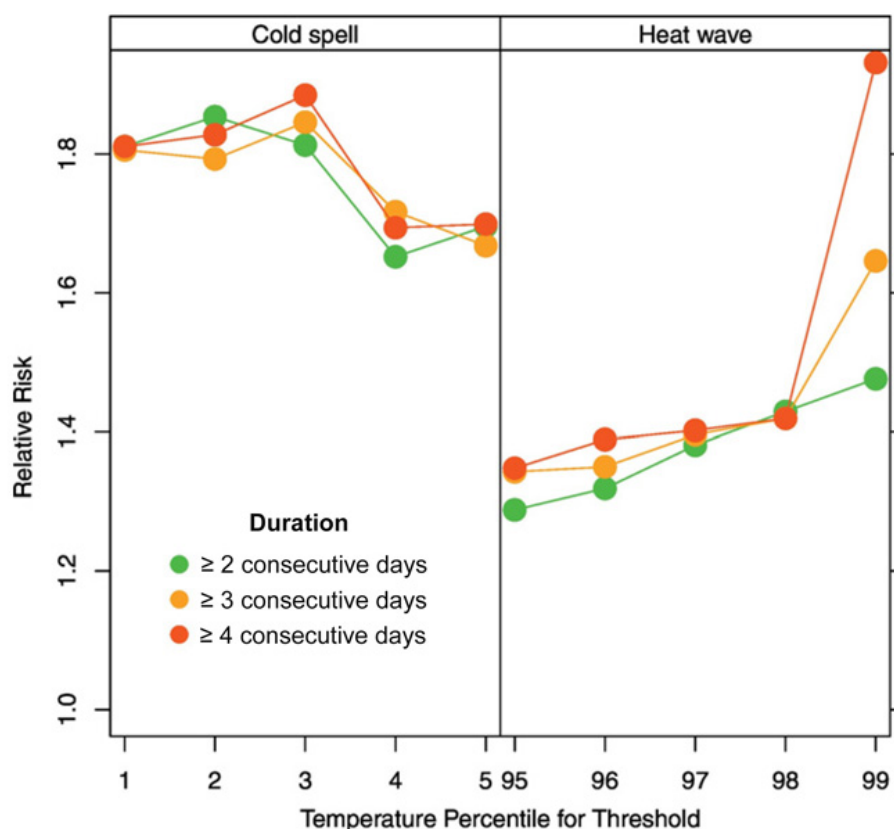
Song *et al.*’s work also revealed that the relative risk (RR) of daily respiratory morbidity due to cold spells is typically much greater (RR of ~1.7 vs RR of ~1.4) than that due to heat waves (see Figure 4.4.1).

The only exception is the RR for heat waves lasting four or more days at the 99th percentile threshold. That data point is represented by only one heat wave in the entire study and may be an aberration skewing the results, given the closer relationship in RR values observed at the 95th through 98th percentiles.

In light of these several findings, it would appear the most effective policies for reducing respiratory emergency department visits would be targeted towards the higher relative risks observed at the cold end of the temperature spectrum.

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Figure 4.4.1**Relative risk in daily respiratory morbidity due to cold spells and heat waves in Beijing, China**Source: Song *et al.*, 2018.

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4.5 Stroke

Higher surface temperatures would reduce the incidence of death due to stroke in many parts of the world, including Africa, Asia, Australia, the Caribbean, Europe, Japan, Korea, Latin America, and Russia.

Low minimum temperatures are a greater risk factor than high temperatures for stroke incidence and hospitalization.

Strokes are either ischemic or hemorrhagic. An ischemic stroke occurs when blood flow to part of the brain is cut off, due to a clot forming either on an atherosclerotic plaque (fatty deposit) in a cerebral artery or in the heart or blood vessels leading to the brain, breaking off and travelling to the brain. By contrast, the most common causes of hemorrhagic stroke are high blood pressure and brain aneurysms. An aneurysm is a dilated segment of artery due to a weakness or thinness in the blood vessel wall, which leads to excessive ballooning, leakage of blood, or rupture. The result is blood seeping into or around the brain tissue, causing damage to brain cells.

According to the IPCC, rising atmospheric CO₂ concentrations due to the combustion of fossil fuels cause surface temperatures to rise, which then causes increased deaths due to strokes. However, as was the case with cardiovascular disease and respiratory disease, examination of real-world data reveals unseasonably cold temperatures cause more deaths and hospital admissions due to stroke than do unseasonably warm temperatures.

Feigin *et al.* (2000) examined the relationship between the incidence of stroke and ambient temperatures over the period 1982–1993 in

Novosibirsk, Siberia, which has one of the highest stroke incidence rates in the world. Based on analyses of 2,208 patients with sex and age distributions similar to those of Russia as a whole, the researchers found a statistically significant association between stroke occurrence and low ambient temperature. In the case of ischemic stroke (IS), which accounted for 87% of all strokes, they determined “the risk of IS occurrence on days with low ambient temperature [was] 32% higher than that on days with high ambient temperature.” They conclude the “very high stroke incidence in Novosibirsk, Russia may partially be explained by the highly prevalent cold factor there.”

Hong *et al.* (2003) investigated the association between the onset of ischemic stroke and prior episodic decreases in temperature in 545 patients who suffered strokes in Incheon, Korea from January 1998 to December 2000. They report “decreased ambient temperature was associated with risk of acute ischemic stroke,” with the strongest effect being seen on the day after exposure to cold weather, further noting “even a moderate decrease in temperature can increase the risk of ischemic stroke.” They also found “risk estimates associated with decreased temperature were greater in winter than in the summer,” which suggests “low temperatures as well as temperature changes are associated with the onset of ischemic stroke.” Finally, they explain the reason for the 24- to 48-hour lag between exposure to cold and the onset of stroke “might be that it takes some time for the decreasing temperature to affect blood viscosity or coagulation.”

Nakaji *et al.* (2004) evaluated seasonal trends in deaths due to various diseases in Japan using nationwide vital statistics from 1970 to 1999 together with mean monthly temperature data. They found the peak mortality rate due to stroke was two times greater in winter (January) than at the time of its yearly minimum (August and September).

Chang *et al.* (2004) analyzed data from the World Health Organization (WHO) Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception (WHO, 1995) to determine the effects of monthly mean temperature on rates of hospitalization for arterial ischemic stroke and acute myocardial infarction among women aged 15 to 49 from 17 countries in Africa, Asia, the Caribbean, Europe, and Latin America. They found among these women, a 5°C reduction in mean air temperature was associated with a 7% increase in the expected hospitalization rate due to stroke, and this effect was

relatively acute, within a period of about a month, the scientists write.

Gill *et al.* (2012) write, “in the past two decades, several studies reported that meteorologic changes are associated with monthly and seasonal spikes in the incidence of aneurysmal subarachnoid hemorrhage (aSAH),” and “analysis of data from large regional databases in both hemispheres has revealed increased seasonal risk for aSAH in the fall, winter and spring,” citing among other sources Feigin *et al.* (2001), Abe *et al.* (2008), and Beseoglu *et al.* (2008). Gill *et al.* identified the medical records of 1,175 patients at the Johns Hopkins Hospital in Baltimore, Maryland (USA) who were admitted with a radiologically confirmed diagnosis of aSAH between January 1, 1991 and March 1, 2009. The six scientists report both “a one-day decrease in temperature and colder daily temperatures were associated with an increased risk of incident aSAH,” and “these variables appeared to act synergistically” and were “particularly predominant in the fall, when the transition from warmer to colder temperatures occurred.” Gill *et al.* add their study “is the first to report a direct relationship between a temperature decrease and an increased risk of aSAH,” and “it also confirms the observations of several reports of an increased risk of aSAH in cold weather or winter,” citing Nyquist *et al.* (2001) and other sources.

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4.6 Insect-borne Diseases

Higher surface temperatures are not leading to increases in mosquito-transmitted and tick-borne diseases such as malaria, yellow fever, viral encephalitis, and dengue fever.

The IPCC’s Fifth Assessment Report (AR5) backs down from previous predictions that global warming would facilitate the spread of mosquito-borne diseases including malaria and dengue fever and tick-borne diseases. The full report from Working Group II on the subject (IPCC, 2014a, Chapter 11, pp. 722–6) repeatedly admits there is no evidence that climate change has affected the range of vector-borne diseases. However, the *Summary for Policymakers* inexplicably warns, “Throughout the 21st century, climate change is expected to lead to increases in ill-health in many regions and especially in developing countries with low income, as compared to a baseline without climate change (*high confidence*).” Among the “examples” given is “vector-borne diseases (*medium confidence*)” (IPCC, 2014b, pp. 19–20).

In a research report in *Science*, Rogers and Randolph (2000) note “predictions of global climate change have stimulated forecasts that vector-borne diseases will spread into regions that are at present too cool for their persistence.” However, the effect of warmer temperatures on insect-borne diseases is complex, sometimes working in favor of and sometimes against the spread of a disease. For example, ambient temperature has historically not determined the range of insect-borne diseases and human adaptation to climate change overwhelms the role of climate. Even those who support the IPCC admit, “It’s a little bit tricky to make a solid prediction” (Irfan, 2011, quoting Marm Kilpatrick).

Gething *et al.* (2010), writing specifically about malaria, may have put it best when they said there has been “a decoupling of the geographical climate-malaria relationship over the twentieth century, indicating that non-climatic factors have profoundly confounded this relationship over time.” They note “non-climatic factors, primarily direct disease control and the indirect effects of a century of urbanization and economic development, although spatially and temporally variable, have exerted a substantially greater influence on the geographic extent and intensity of malaria worldwide during the twentieth century than have climatic factors.” As for the future, they conclude climate-induced effects “can be offset by moderate increases in coverage levels of currently available interventions.”

This section investigates the reliability of the IPCC’s claim with respect to the three main kinds of insect-borne diseases: malaria, dengue fever, and tick-borne diseases. According to scientific examination and research on this topic, there is little support for the claims appearing in the latest IPCC *Summary for Policymakers*.

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4.6.1 Malaria

Extensive scientific information and experimental research contradict the claim that malaria will expand across the globe and intensify as a result of CO₂-induced warming.

Jackson *et al.* (2010) say “malaria is one of the most devastating vector-borne parasitic diseases in the tropical and subtropical regions of the world,” noting it affects more than 100 countries. According to the World Health Organization (WHO, 2017), an estimated 216 million cases of malaria occurred worldwide in 2016, down from 237 million cases in 2010. Ninety-one countries reported indigenous malaria cases with Africa accounting for 90% of all cases. Approximately 445,000 people died from malaria that year.

According to Reiter (2000), claims that malaria will become more widespread due to CO₂-induced global warming ignore other important factors and disregard known facts. A historical analysis of malaria trends, for example, reveals this disease was an important cause of illness and death in England during a period of colder-than-present temperatures throughout the Little Ice Age. Its transmission began to decline only in the nineteenth century, during a warming phase when, according to Reiter, “temperatures were already much higher than in the Little Ice Age.” In short, malaria was prevalent in Europe during some of the coldest centuries of the past millennium, and it has only recently undergone widespread decline, when temperatures have been warming. Clearly, there are other factors at work that are more important than temperature. Such factors include the quality of public health services, public awareness, irrigation and agricultural activities, land use practices, civil strife, natural disasters, ecological

change, population change, use of insecticides, and the movement of people (Reiter, 2000; Reiter, 2001; Hay *et al.*, 2002a, 2002b).

Nevertheless, concerns have lingered about the possibility of widespread future increases in malaria due to global warming. These concerns are generally rooted in climate models that typically use only one, or at most two, climate variables in making their predictions of the future distribution of the disease over Earth, and they generally do not include any of the non-climatic factors listed in the preceding paragraph. When more variables are included, a less-worrisome future is projected. In one modeling study, for example, Rogers and Randolph (2000) employed five climate variables and obtained very different results. Briefly, they used the present-day distribution of malaria to determine the specific climatic constraints that best define that distribution, after which the multivariate relationship they derived from this exercise was applied to future climate scenarios derived from climate models in order to map potential future geographical distributions of the disease. Their study revealed very little change: a 0.84% increase in potential malaria exposure under the “medium-high” scenario of global warming and a 0.92% decrease under the “high” scenario. Rogers and Randolph explicitly state their quantitative model “contradicts prevailing forecasts of global malaria expansion” and “highlights the use of multivariate rather than univariate constraints in such applications.”

Hay *et al.* (2002a) investigated long-term trends in meteorological data at four East African highland sites that experienced significant increases in malaria cases over the past couple of decades, reporting “temperature, rainfall, vapour pressure and the number of months suitable for *P. falciparum* transmission have not changed significantly during the past century or during the period of reported malaria resurgence,” thus these factors could not be responsible for the observed increases in malaria cases. Likewise, Shanks *et al.* (2000) examined trends in temperature, precipitation, and malaria rates in western Kenya over the period 1965–1997, finding no linkages among the variables.

Small *et al.* (2003) examined trends in a climate-driven model of malaria transmission between 1911 and 1995, using a spatially and temporally extensive gridded climate dataset to identify locations in Africa where the malaria transmission climate suitability index had changed significantly over this time interval. They found “climate warming, expressed as a systematic temperature increase over the 85-year

period, does not appear to be responsible for an increase in malaria suitability over any region in Africa.” They conclude “research on the links between climate change and the recent resurgence of malaria across Africa would be best served through refinements in maps and models of precipitation patterns and through closer examination of the role of nonclimatic influences.”

Kuhn *et al.* (2003) analyzed the determinants of temporal trends in malaria deaths within England and Wales in 1840–1910 and found “a 1°C increase or decrease was responsible for an increase in malaria deaths of 8.3% or a decrease of 6.5%, respectively,” which explains “the malaria epidemics in the ‘unusually hot summers’ of 1848 and 1859.” Nevertheless, the long-term near-linear temporal decline in malaria deaths over the period of study, the researchers write, “was probably driven by nonclimatic factors,” among which they identify increasing livestock populations (which tend to divert mosquito biting away from humans), decreasing acreages of marsh wetlands (where mosquitoes breed), as well as “improved housing, better access to health care and medication, and improved nutrition, sanitation, and hygiene.” Kuhn *et al.* say “the projected increase in proportional risk is clearly insufficient to lead to the reestablishment of endemicity.”

Zhou *et al.* (2004) employed a nonlinear mixed-regression model study that focused on the numbers of monthly malaria outpatients of the past 10 to 20 years in seven East African highland sites and their relationships to the numbers of malaria outpatients during the previous time period, seasonality, and climate variability. They state, “for all seven study sites, we found highly significant nonlinear, synergistic effects of the interaction between rainfall and temperature on malaria incidence, indicating that the use of either temperature or rainfall alone is not sensitive enough for the detection of anomalies that are associated with malaria epidemics.” Githeko and Ndegwa (2001), Shanks *et al.* (2002), and Hay *et al.* (2002b) reached the same conclusion. In addition, climate variability – not just temperature or not just warming – contributed less than 20% of the temporal variance in the number of malaria outpatients, and at only two of the seven sites studied.

Rogers and Randolph (2006) conducted a major review of the potential impacts of global warming on vector-borne diseases, focusing on recent upsurges of malaria in Africa, asking, “Has climate change already had an impact?” They demonstrate “evidence for increasing malaria in many parts of Africa is

overwhelming, but the more likely causes for most of these changes to date include land-cover and land-use changes and, most importantly, drug resistance rather than any effect of climate,” noting “the recrudescence of malaria in the tea estates near Kericho, Kenya, in East Africa, where temperature has not changed significantly, shows all the signs of a disease that has escaped drug control following the evolution of chloroquine resistance by the malarial parasite.”

Childs *et al.* (2006) present a detailed analysis of malaria incidence in northern Thailand based on a quarter-century monthly time series (January 1977 through January 2002) of total malaria cases in the country’s 13 northern provinces. Over this period, when the IPCC claims the world warmed at a rate and to a level unprecedented over the prior one to two millennia, Childs *et al.* report there was an approximately constant rate of *decline* in total malaria incidence (from a mean monthly incidence in 1977 of 41.5 cases per hundred thousand people to 6.72 cases per hundred thousand people in 2001). Noting “there has been a steady reduction through time of total malaria incidence in northern Thailand, with an average decline of 6.45% per year,” they say this result “reflects changing agronomic practices and patterns of immigration, as well as the success of interventions such as vector control programs, improved availability of treatment and changing drug policies.”

Zell *et al.* (2008) conducted a similar review of the literature and determined “coupled ocean/atmosphere circulations and continuous anthropogenic disturbances (increased populations of humans and domestic animals, socioeconomic instability, armed conflicts, displaced populations, unbalanced ecosystems, dispersal of resistant pathogens etc.) appear to be the major drivers of disease variability,” and “global warming at best contributes.”

Reiter (2008) came to similar conclusions, writing, “simplistic reasoning on the future prevalence of malaria is ill-founded; malaria is not limited by climate in most temperate regions, nor in the tropics, and in nearly all cases, ‘new’ malaria at high altitudes is well below the maximum altitudinal limits for transmission.” He further states, “future changes in climate may alter the prevalence and incidence of the disease, but obsessive emphasis on ‘global warming’ as a dominant parameter is indefensible; the principal determinants are linked to ecological and societal change, politics and economics.”

Hulden and Hulden (2009) analyzed malaria statistics collected in Finland from 1750 to 2008 via correlation analyses between malaria frequency per million people and all variables that have been used in similar studies throughout other parts of Europe, including temperature data, animal husbandry, consolidation of land by redistribution, and household size. Over the entire period, “malaria frequency decreased from about 20,000–50,000 per 1,000,000 people to less than 1 per 1,000,000 people,” they report. The two Finnish researchers conclude, “indigenous malaria in Finland faded out evenly in the whole country during 200 years with limited or no counter measures or medication,” making that situation “one of the very few opportunities where natural malaria dynamics can be studied in detail.” Their study indicates “malaria in Finland basically was a sociological disease and that malaria trends were strongly linked to changes in the human household size and housing standard.”

Russell (2009) studied the “current or historic situations of the vectors and pathogens, and the complex ecologies that might be involved” regarding malaria, dengue fever, the arboviral arthritides (Ross River and Barmah Forest viruses), and the arboviral encephalitides (Murray Valley encephalitis and Kunjin viruses) in Australia and found “there might be some increases in mosquito-borne disease in Australia with a warming climate, but with which mosquitoes and which pathogens, and where and when, cannot be easily discerned.” He concludes, “of itself, climate change as currently projected, is not likely to provide great cause for public health concern with mosquito-borne disease in Australia.”

Nabi and Qader (2009) considered the climatic conditions that impact the spread of malaria – temperature, rainfall, and humidity – and the host of pertinent nonclimatic factors that play important roles in its epidemiology: the presence or absence of mosquito control programs, the availability or non-availability of malaria-fighting drugs, changing resistances to drugs, the quality of vector control, changes in land use, the availability of good health services, human population growth, human migrations, international travel, and standard of living. The two researchers report “global warming alone will not be of a great significance in the upsurge of malaria unless it is accompanied by a deterioration in other parameters like public health facilities, resistance to anti-malarial drugs, decreased mosquito control measures,” etc. They say “no accurate prediction about malaria can truly be made,”

because “it is very difficult to estimate what the other factors will be like in the future.”

Jackson *et al.* (2010) linked reported malaria cases and deaths from the years 1996 to 2006 for 10 countries in western Africa (Benin, Burkina Faso, Cote d’Ivoire, Gambia, Ghana, Liberia, Mali, Senegal, Sierra Leone, and Togo) with corresponding climate data from the U.S. National Oceanic and Atmospheric Administration’s National Climatic Data Center. They searched for transitive relationships between the weather variables and malaria rates via spatial regression analysis and tests for correlation. Jackson *et al.* report their analyses showed “very little correlation exists between rates of malaria prevalence and climate indicators in western Africa.” This result, as they describe it, “contradicts the prevailing theory that climate and malaria prevalence are closely linked and also negates the idea that climate change will increase malaria transmission in the region.”

Haque *et al.* (2010) analyzed monthly malaria case data for the malaria endemic district of Chittagong Hill Tracts in Bangladesh from January 1989 to December 2008, looking for potential relationships between malaria incidence and various climatic parameters (rainfall, temperature, humidity, sea surface temperature, and the El Niño-Southern Oscillation), as well as the normalized difference vegetation index (NDVI), a satellite-derived measure of surface vegetation greenness. The six scientists report, “after adjusting for potential mutual confounding between climatic factors there was no evidence for any association between the number of malaria cases and temperature, rainfall and humidity,” and “there was no evidence of an association between malaria cases and sea surface temperatures in the Bay of Bengal and [the El Niño-Southern Oscillation index for Niño Region 3].”

Gething *et al.* (2010) compared historical and contemporary maps of the range and incidence of malaria and found endemic/stable malaria is likely to have covered 58% of the world’s land surface around 1900 but only 30% by 2007. They report, “even more marked has been the decrease in prevalence within this greatly reduced range, with endemicity falling by one or more classes in over two-thirds of the current range of stable transmission.” They write, “widespread claims that rising mean temperatures have already led to increases in worldwide malaria morbidity and mortality are largely at odds with observed decreasing global trends in both its endemicity and geographic extent.” Rather, “the combined natural and anthropogenic forces acting on

the disease throughout the twentieth century have resulted in the great majority of locations undergoing a net reduction in transmission between one and three orders of magnitude larger than the maximum future increases proposed under temperature-based climate change scenarios.”

Stern *et al.* (2011) examined trends in temperature and malaria for the Highlands of East Africa, which span Rwanda, Burundi, and parts of Kenya, Tanzania, and Uganda, to resolve controversies over whether the area has warmed and malaria has become more prevalent. They report temperature has increased significantly in the region, yet “malaria in Kericho and many other areas of East Africa has decreased during periods of unambiguous warming.”

Nkurunziza and Pilz (2011) assessed the impact of an increase in temperature on malaria transmission in Burundi, a landlocked country in the African Great Lakes region of East Africa. They found “an increase in the maximum temperature will cause an increase in minimum temperature,” and “the increase in the latter will result in a decreasing maximum humidity, leading to a decrease in rainfall.” These results, they continue, “suggest that an increased temperature will result in a shortening of the life span of mosquitoes (due to decreasing humidity) and decrease in the capacity of larva production and maturation (due to decreasing rainfall).” Thus, “the increase in temperature will not result in an increased malaria transmission in Burundi,” which is “in good agreement with some previous works on the topic,” citing as examples WHO, WMO, UNEP (2003), Lieshout *et al.* (2004), and Thomas (2004). In a final statement on the matter, Nkurunziza and Pilz note that in regions with endemic malaria transmission, such as Burundi, “the increase in temperature may lead to unsuitable climate conditions for mosquitoes survival and, hence, probably to a decreasing malaria transmission.”

Béguin *et al.* (2011) estimated populations at risk of malaria (PAR) based on climatic variables, population growth, and GDP per capita (GDPpc). GDPpc is an approximation for per-capita income (“income” for short) for 1990, 2010, and 2050, based on sensitivity analyses for the following three scenarios: (1) a worst-case scenario, in which income declines to 50% of its 2010 values by 2050; (2) a “growth reduction” scenario, in which income declines by 25% in 2030 and 50% in 2050, relative to the A1B scenario (socioeconomic change plus climate change); and (3) a scenario in which income stays constant at 2010 values. The results are

presented in Figure 4.6.1.1. The authors observe, “under the A1B climate scenario, climate change has much weaker effects than GDPpc increase on the geographic distribution of malaria.” This result is consistent with the few studies that have considered the impact of climate change and socioeconomic factors on malaria. (See, e.g., Tol and Dowlatabadi, 2001; Bosello *et al.*, 2006). With respect to malaria, therefore, climate change is a relatively minor factor compared to economic development.

Paaajmans *et al.* (2012) examined the effects of temperature on the rodent malaria *Plasmodium yoelii* and the Asian malaria vector *Anopheles stephensi*. The three U.S. researchers found “vector competence (the maximum proportion of infectious mosquitoes, which implicitly includes parasite survival across the incubation period) tails off at higher temperatures, even though parasite development rate increases.” Moreover, “the standard measure of the parasite incubation period (i.e., time until the first mosquitoes within a cohort become infectious following an infected blood-meal) is incomplete because parasite development follows a cumulative distribution, which itself varies with temperature. Finally, “including these effects in a simple model dramatically alters estimates of transmission intensity and reduces the optimum temperature for transmission.” Therefore, in regard to “the possible effects of climate warming,” they conclude “increases in temperature need not simply lead to increases in transmission.”

Paaajmans *et al.* conclude their results “challenge current understanding of the effects of temperature on malaria transmission dynamics,” and they note their findings imply “control at higher temperatures might be more feasible than currently predicted.”

Using a high-resolution computer model that incorporates “climate-driven hydrology as a determinant of mosquito populations and malaria transmission,” Yamana *et al.* (2016) found the impact of future climate change on West African malaria transmission will be “negative at best, and positive but insignificant at worst,” confirming that “no major increases in the frequency or the severity of malaria outbreaks in West Africa are expected as a result of climate change.”

Murdock *et al.* (2016) investigated “how increases in temperature from optimal conditions (27°C to 30°C and 33°C) interact with realistic diurnal temperature ranges (DTR: \pm 0°C, 3°C and 4.5°C) to affect the ability of key vector species from Africa and Asia (*Anopheles gambiae* and *An. Stephensi*) to transmit the human malaria parasite,

Figure 4.6.1.1

Effects of climate change and socioeconomic factors on the projected future global distribution of malaria

Model type	Population at Risk 2030 [billions]	Population at Risk 2050 [billions]
Socioeconomic changes only (no climate change)	3.52	1.74
Socioeconomic and climatic changes (A1B scenario)	3.58 [3.55–3.60]	1.95 [1.93–1.96]
Socioeconomic changes and CC (slower growth scenario)	3.82 [3.39–3.84]	3.42 [3.28–3.45]
No growth scenario , only CC	4.61 [4.54–4.67]	5.20 [5.11–5.25]
Pessimistic growth scenario and CC	5.18 [5.07–5.30]	6.27 [6.19–6.32]

CC refers to climate change scenarios developed by the authors; mean temperature of the coldest month and mean precipitation of the wettest month were modeled. *Source: Béguin et al., 2011.*

Plasmodium falciparum.” They report “the effects of increasing temperature and DTR on parasite prevalence, parasite intensity, and mosquito mortality decreased overall vectorial capacity for both mosquito species” (see Figure 4.6.1.2). They also note “increases of 3°C from 27°C reduced vectorial capacity by 51–89% depending on species and DTR, with increases in DTR alone potentially halving transmission,” and “at 33°C, transmission potential was further reduced for *An. Stephensi* and blocked completely in *An. Gambiae*.” The researchers concluded that rather than *increasing* malaria transmission, any current or future warming should actually diminish malaria transmission potential in what are currently high transmission settings.

Zhao *et al.* (2016) quantified the impact of several factors that led to freeing Europe from endemic malaria transmission during the twentieth century. They analyzed spatial datasets representing climatic, land use, and socioeconomic factors thought to be associated with the decline of malaria in twentieth century Europe and integrated the data with historical malaria distribution maps in order to quantify changes and differences across the continent before, during, and after malaria elimination. Their goal was to understand which factors significantly influence malaria transmission and decline, as well as which factors continue to play a role in limiting the risks of its re-establishment.

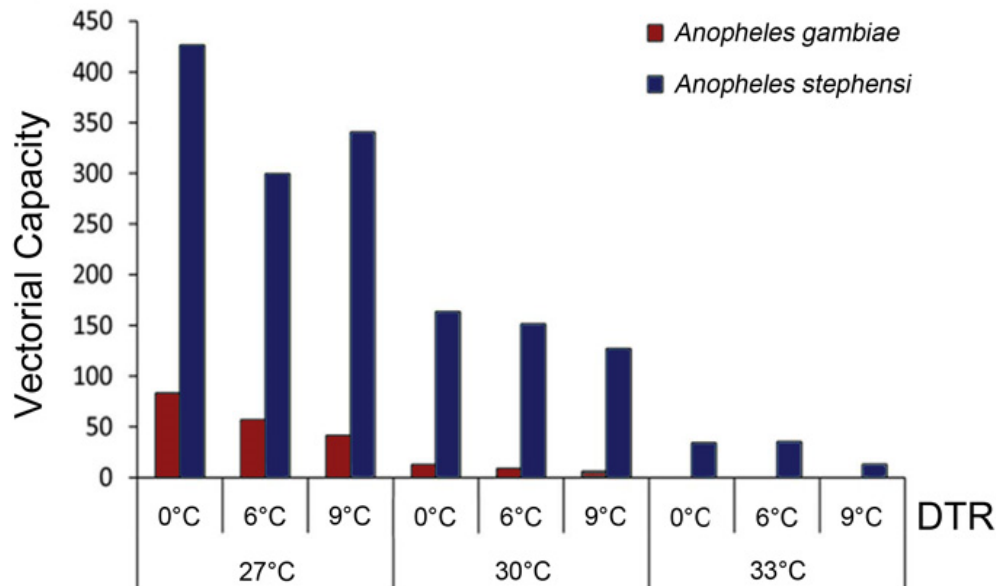
Of the nine factors analyzed by Zhao *et al.*, three of them were climate-related (temperature,

precipitation, and frost day frequency), each of which is “often considered to have an effect of malaria transmission,” they note. The three European researchers, however, found “indicators relating to socio-economic improvements such as wealth, life expectancy and urbanization were strongly correlated with the decline of malaria in Europe, whereas those describing climatic and land use changes showed weaker relationships.” More often than not, they found, changes in climate tended to run counter to observed trends in malaria; i.e., climate changes were thought to lead to an increased number of cases yet the actual numbers declined. It would appear that socioeconomic and land use factors are more than capable of compensating for any unfavorable changes in climate that may lead to malaria transmission and outbreaks. As long as countries continue to focus on improving these more important factors, malaria trends will continue to remain little influenced by future climate change, model projections notwithstanding.

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Figure 4.6.1.2
Effects of temperature and diurnal temperature range (DTR) on vectorial capacity



Source: Murdock *et al.*, 2016.

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4.6.2 Dengue Fever

Concerns over large increases in dengue fever as a result of rising temperatures are unfounded and unsupported by the scientific literature, as climatic indices are poor predictors for dengue fever.

According to Ooi and Gubler (2009a), “dengue/dengue hemorrhagic fever is the most important vector-borne viral disease globally,” with more than half the world’s population living in areas deemed to be at risk of infection. Kyle and Harris (2008) note “dengue is a spectrum of disease caused by four serotypes of the most prevalent arthropod-borne virus affecting humans today,” and “its incidence has increased dramatically in the past 50 years,” to where “tens of millions of cases of dengue fever are estimated to occur annually, including up to 500,000 cases of the life-threatening dengue hemorrhagic fever/dengue shock syndrome.”

Some of the research papers summarized in previous sections address dengue fever as well as malaria. With a few worthy exceptions, we do not repeat those summaries in this section. The most

important exceptions are papers written or coauthored by Paul Reiter (2001, 2003, 2010a, 2010b), one of the world's premier authorities on the subject. Reiter analyzed the history of malaria and dengue fever in an attempt to determine whether the incidence and range of influence of these diseases would indeed increase in response to higher global surface temperatures. His reviews established what is now widely accepted among experts in the field: that the natural history of these vector-borne diseases is highly complex, and the interplay of climate, ecology, vector biology, and a number of other factors defies definition by the simplistic analyses utilized in the computer models relied on by environmental activists and the IPCC.

That there has in fact been a resurgence of these diseases in parts of the world is true, but as Reiter (2001) notes, it is "facile to attribute this resurgence to climate change." This he shows via a number of independent analyses that clearly demonstrate factors associated with politics, economics, and human activity are the principal determinants of the spread of these diseases. He describes these factors as being "much more significant" than climate in promoting disease expansion. Reiter took up the subject again in 2003 with 19 other scientists as coauthors (Reiter *et al.*, 2003), and again in 2010.

Tuchman *et al.* (2003) conducted an empirical study of the impact of a doubling of atmospheric CO₂ concentrations (from 360 to 720 ppm) on development rates and survivorship of four species of detritivorous mosquito larvae eating leaf litter from *Populus tremuloides* (Michaux) trees. They report larval mortality was 2.2 times higher for *Aedes albopictus* (Skuse) mosquitos that were fed leaf litter that had been produced in the high-CO₂ chambers than it was for those fed litter that had been produced in the ambient-air chambers. In addition, they found larval development rates of *Aedes triseriatus* (Say), *Aedes aegypti* (L.), and *Armigeres subalbatus* (Coquillett) were slowed by 78%, 25%, and 27%, respectively. The researchers suggest "increases in lignin coupled with decreases in leaf nitrogen induced by elevated CO₂ and subsequent lower bacterial productivity [on the leaf litter in the water] were probably responsible for [the] decreases in survivorship and/or development rate of the four species of mosquitoes." Concerning the significance of these findings, Tuchman *et al.* write, "the indirect impacts of an elevated CO₂ atmosphere on mosquito larval survivorship and development time could potentially be great," because longer larval development times could result in fewer cohorts of

mosquitoes surviving to adulthood. With fewer mosquitoes, there should be lower levels of mosquito-borne diseases.

Kyle and Harris (2008) write "there has been a great deal of debate on the implications of global warming for human health," but "at the moment, there is no consensus." However, "in the case of dengue," they report, "it is important to note that even if global warming does not cause the mosquito vectors to expand their geographic range, there could still be a significant impact on transmission in endemic regions," because "a 2°C increase in temperature would simultaneously lengthen the lifespan of the mosquito and shorten the extrinsic incubation period of the dengue virus, resulting in more infected mosquitoes for a longer period of time." Nevertheless, they state there are "infrastructure and socioeconomic differences that exist today and already prevent the transmission of vector-borne diseases, including dengue, even in the continued presence of their vectors," citing Reiter (2001).

Wilder-Smith and Gubler (2008) conducted a review of the scientific literature, noting "the past two decades saw an unprecedented geographic expansion of dengue" and "global climate change is commonly blamed for the resurgence of dengue," but they add, "there are no good scientific data to support this conclusion." The two researchers report, "climate has rarely been the principal determinant of [mosquitoes'] prevalence or range," and "human activities and their impact on local ecology have generally been much more significant." They cite as contributing factors "urbanization, deforestation, new dams and irrigation systems, poor housing, sewage and waste management systems, and lack of reliable water systems that make it necessary to collect and store water," further noting "disruption of vector control programs, be it for reasons of political and social unrest or scientific reservations about the safety of DDT, has contributed to the resurgence of dengue around the world."

In addition, Wilder-Smith and Gubler write "large populations in which viruses circulate may also allow more co-infection of mosquitoes and humans with more than one serotype of virus," which would appear to be borne out by the fact that "the number of dengue lineages has been increasing roughly in parallel with the size of the human population over the last two centuries." Most important, perhaps, is "the impact of international travel," of which they say "humans, whether troops, migrant workers, tourists, business travelers,

refugees, or others, carry the virus into new geographic areas,” and these movements “can lead to epidemic waves.” The two researchers conclude, “population dynamics and viral evolution offer the most parsimonious explanation for the observed epidemic cycles of the disease, far more than climatic factors.”

Ooi and Gubler (2009b) examined “the history of dengue emergence” in order to determine “the major drivers for the spread of both the viruses and mosquito vectors to new geographic regions.” The two researchers note “frequent and cyclical epidemics are reported throughout the tropical world, with regular importation of the virus via viremic travelers into both endemic and non-endemic countries.” They state, “there is no good evidence to suggest that the current geographic expansion of the dengue virus and its vectors has been or will be due to global warming.”

Russell *et al.* (2009) showed the dengue vector (the *Aedes aegypti* mosquito) “was previously common in parts of Queensland, the Northern Territory, Western Australia and New South Wales,” and it had “in the past, covered most of the climatic range theoretically available to it,” adding “the distribution of local dengue transmission has [historically] nearly matched the geographic limits of the vector.” This being the case, they conclude the vector’s current absence from much of Australia “is not because of a lack of a favorable climate.” Thus, they reason “a temperature rise of a few degrees is not alone likely to be responsible for substantial increases in the southern distribution of *A. aegypti* or dengue, as has been recently proposed.” Instead of futile attempts to limit dengue transmission by controlling the world’s climate, therefore, the medical researchers recommend “well resourced and functioning surveillance programs, and effective public health intervention capabilities, are essential to counter threats from dengue and other mosquito-borne diseases.”

Johansson *et al.* (2009) studied the association between the El Niño Southern Oscillation (ENSO) and dengue incidence in Puerto Rico (1986–2006), Mexico (1985–2006), and Thailand (1983–2006) using wavelet analysis as a tool to identify time- and frequency-specific associations. The three researchers report they “did not find evidence of a strong, consistent relationship in any of the study areas,” and Rohani (2009), who wrote a Perspective piece on their study, states the three researchers found “no systematic association between multi-annual dengue outbreaks and El Niño Southern Oscillation.” Thus,

as stated in the “Editors’ Summary” of the Johansson *et al.* paper, their findings “provide little evidence for any relationship between ENSO, climate, and dengue incidence.”

Shang *et al.* (2010) analyzed dengue cases in Taiwan at their onset dates of illness from 1998 to 2007, in order to “identify correlations between indigenous dengue and imported dengue cases (in the context of local meteorological factors) across different time lags.” The researchers write, “the occurrence of indigenous dengue was significantly correlated with temporally-lagged cases of imported dengue (2–14 weeks), higher temperatures (6–14 weeks), and lower relative humidity (6–20 weeks),” and “imported and indigenous dengue cases had a significant quantitative relationship in the onset of local epidemics.” The six Taiwanese researchers conclude, “imported dengue are able to serve as an initial facilitator, or spark, for domestic epidemics” while “meteorology alone does not initiate an epidemic.” Rather than point to global warming, they state unequivocally that “an increase in viremic international travelers has caused global dengue hemorrhagic fever case numbers to surge in the past several decades.”

Reiter (2010a) observed “the introduction and rapidly expanding range of *Aedes albopictus* in Europe is an iconic example of the growing risk of the globalization of vectors and vector-borne diseases,” and “the history of yellow fever and dengue in temperate regions confirms that transmission of both diseases could recur, particularly if *Aedes aegypti*, a more effective vector, were to be re-introduced.” He states “conditions are already suitable for transmission.” Much more important than a rise or fall of a couple degrees of temperature, Reiter says, is “the quantum leap in the mobility of vectors and pathogens that has taken place in the past four decades, a direct result of the revolution of transport technologies and global travel.”

Carbajo *et al.* (2012) evaluated the relative contributions of geographic, demographic, and climatic variables to the recent spread of dengue in Argentina. They found dengue spatial occurrence “was positively associated with days of possible transmission, human population number, population fall and distance to water bodies.” When considered separately, the researchers write, “the classification performance of demographic variables was higher than that of climatic and geographic variables.” Thus, although useful in estimating annual transmission risk, Carbajo *et al.* conclude temperature “does not fully describe the distribution of dengue occurrence

at the country scale,” and “when taken separately, climatic variables performed worse than geographic or demographic variables.”

Williams *et al.* (2014) used a dynamic life table simulation model and statistically downscaled daily values for future climate to assess “climate change induced changes to mosquito bionomics,” focusing on “female mosquito abundance, wet weight, and the extrinsic incubation period for dengue virus in these mosquitoes.” They based their work on simulations of *Ae. aegypti* populations for current (1991–2011) and future (2046–2065) climate conditions for the city of Cairns, Queensland (which has historically experienced the most dengue virus transmission in all of Australia), as derived from the MPI ECHAM 5 climate model for the IPCC-proposed B1 and A2 emission scenarios.

Their work revealed “*Aedes aegypti* abundance is predicted to increase under the B1, but decrease under the A2, scenario,” and “mosquitoes are predicted to have a smaller body mass in a future climate.” Williams *et al.* say “it is therefore unclear whether dengue risk would increase or decrease in tropical Australia with climate change.” They conclude their findings “challenge the prevailing view that a future, warmer climate will lead to larger mosquito populations and a definite increase in dengue transmission.”

These several observations indicate concerns over large increases in dengue fever as a result of rising temperatures are unfounded and unsupported by the scientific literature, as climatic indices are poor predictors for dengue fever.

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4.6.3 Tick-borne Diseases

Climate change has not been the most significant factor driving the recent temporal

patterns in the epidemiology of tick-borne diseases.

Sarah Randolph of the University of Oxford's Department of Zoology is a leading scholar on tick-borne diseases. She and fellow Oxford faculty member David Rogers observed in 2000 that tick-borne encephalitis (TBE) "is the most significant vector-borne disease in Europe and Eurasia," having "a case morbidity rate of 10–30% and a case mortality rate of typically 1–2% but as high as 24% in the Far East" (Randolph and Rogers, 2000). The disease is caused by a flavivirus (TBEV) maintained in natural rodent-tick cycles; humans may be infected if bitten by an infected tick or by drinking untreated milk from infected sheep or goats.

Early discussions on the relationship of TBE to global warming predicted the disease would expand its range and become more of a threat to humans in a warmer world. However, Randolph and Rogers (2000) note, "like many vector-borne pathogen cycles that depend on the interaction of so many biotic agents with each other and with their abiotic environment, enzootic cycles of TBEV have an inherent fragility," so "their continuing survival or expansion cannot be predicted from simple univariate correlations." Confining their analysis to Europe, Randolph and Rogers first matched the present-day distribution of TBEV to the present-day distributions of five climatic variables: monthly mean, maximum, and minimum temperatures, plus rainfall and saturation vapor pressure, "to provide a multivariate description of present-day areas of disease risk." They applied this understanding to outputs of a general circulation model of the atmosphere that predicted how these five climatic variables may change in the future.

The results indicate the distribution of TBEV might expand both north and west of Stockholm, Sweden in a warming world. For most other parts of Europe, however, the two researchers say "fears for increased extent of risk from TBEV caused by global climate change appear to be unfounded." They report, "the precise conditions required for enzootic cycles of TBEV are predicted to be disrupted" in response to global warming, and the new climatic state "appears to be lethal for TBEV." This finding, they write, "gives the lie to the common perception that a warmer world will necessarily be a world under greater threat from vector-borne diseases." In the case of TBEV, they report the predicted change "appears to be to our advantage."

Estrada-Peña (2003) evaluated the effects of various abiotic factors on the habitat suitability of four tick species that are major vectors of livestock pathogens in South Africa. They report "year-to-year variations in the forecasted habitat suitability over the period 1983–2000 show a clear decrease in habitat availability, which is attributed primarily to increasing temperature in the region over this period." In addition, when climate variables were projected to the year 2015, Estrada-Peña found "the simulations show a trend toward the destruction of the habitats of the four tick species," just the opposite of what is often predicted about this disease.

Randolph (2010) examined the roles played by various factors that may influence the spread of tick-borne diseases. After describing some of the outbreaks of tick-borne disease in Europe over the past couple of decades, Randolph states, "the inescapable conclusion is that the observed climate change alone cannot explain the full heterogeneity in the epidemiological change, either within the Baltic States or amongst Central and Eastern European countries," citing Sumilo *et al.* (2007). Instead, she writes, "a nexus of interrelated causal factors – abiotic, biotic and human – has been identified," and "each factor appears to operate synergistically, but with differential force in space and time, which would inevitably generate the observed epidemiological heterogeneity."

Many of these factors, she continues, "were the unintended consequences of the fall of Soviet rule and the subsequent socio-economic transition (Sumilo *et al.*, 2008b)," among which she cites "agricultural reforms resulting in changed land cover and land use, and an increased reliance on subsistence farming; reduction in the use of pesticides, and also in the emission of atmospheric pollution as industries collapsed; increased unemployment and poverty, but also wealth and leisure time in other sectors of the population as market forces took hold."

Randolph concludes "there is increasing evidence from detailed analyses that rapid changes in the incidence of tick-borne diseases are driven as much, if not more, by human behavior that determines exposure to infected ticks than by tick population biology that determines the abundance of infected ticks," as per Sumilo *et al.* (2008a) and Randolph *et al.* (2008). She ends her analysis by stating, "while nobody would deny the sensitivity of ticks and tick-borne disease systems to climatic factors that largely determine their geographical distributions, the evidence is that climate change has not been the most

significant factor driving the recent temporal patterns in the epidemiology of tick-borne diseases.”

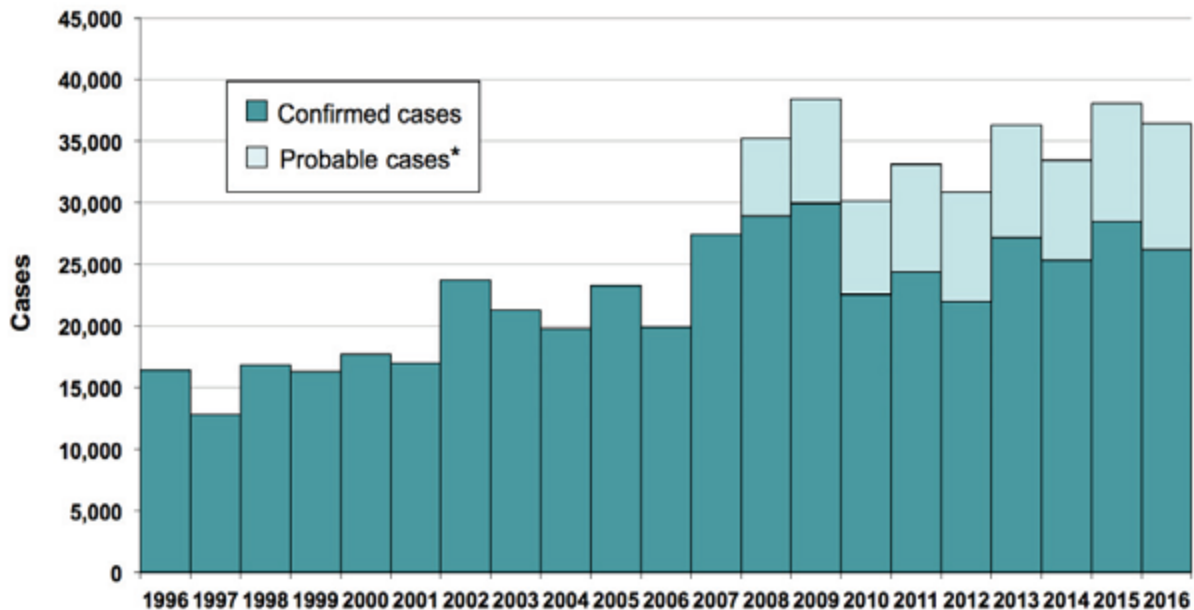
Lyme disease is the most common tick-borne human disease, with an estimated annual incidence of 300,000 in the United States (Shapiro, 2014) and at least 85,000 in Europe (Lindgren and Jaensen, 2006). It is caused by the spirochete bacteria, *Borrelia burgdorferi* and sometimes by *Borrelia mayonii* (Pritt *et al.*, 2016). It is transmitted in the eastern United States and parts of Canada by the tick, *Ixodes scapularis*, and on the Pacific Coast by *I. pacificus* (Clark, 2004).

Modeling by Brownstein *et al.* (2005) “generated the current pattern of *I. scapularis* across North America with an accuracy of 89% ($P < 0.0001$). Extrapolation of the model revealed a significant expansion of *I. scapularis* north into Canada with an increase in suitable habitat and a retraction of the vector from Florida and Texas, so that the exposed population actually diminishes, by 28% in the 2020s, by 12.7% in the 2050s, and by 1.9% in the 2080s. The connection between *I. scapularis* and deciduous forest is so strong that the authors state: “recent emergence of Lyme disease throughout the

northeastern and mid-Atlantic states has been linked to reforestation.” The automobile may thus have contributed to the emergence of Lyme disease by converting numerous redundant horse-paddocks into woodlands and by fertilizing those woodlands with carbon dioxide. The reported incidence increased in the United States during the warming hiatus from 1998 to 2009 and then stabilized or even fell slightly despite warming in 2015–16, as shown in Figure 4.6.3.1.

In Europe and Asia, the vectors of Lyme borreliosis (LB) are *I. ricinus* (Europe) and *I. persulcatus* (Lindgren and Jaensen, 2006). Like its North American cousin, *I. ricinus* prefers forest to open land and deciduous to conifer (Zeman and Januska, 1999). Late twentieth century warming has been linked to ticks spreading into higher latitudes and altitudes (observed in the Czech Republic (Daniel *et al.*, 2004) and to higher incidences of LB (Lindgren and Gustafson, 2001), though distorted by better reporting over time in most regions. Moreover, the incidence of LB actually declined after 1995 in the Czech Republic and Lithuania (Lindgren and Jaensen, 2006).

Figure 4.6.3.1
Reported cases of Lyme disease in the United States, 1996–2016



Source: Centers for Disease Control and Prevention, n.d.

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4.7 Conclusion

Fossil fuels directly benefit human health and longevity by powering labor-saving and life-protecting technologies and perhaps indirectly by contributing to rising surface temperatures.

Fossil fuels have benefited human health by making possible the dramatic increase in human prosperity since the first Industrial Revolution, which in turn made possible the technologies that are essential to protecting human health and prolonging human life. If, as the United Nations' Intergovernmental Panel on Climate Change (IPCC) claims, the combustion of fossil fuels leads to some global warming, then the positive as well as negative health effects of that warming should be included in any cost-benefit analysis of fossil fuels. Medical science explains why warmer temperatures are associated with health benefits. Empirical research confirms that warmer temperatures lead to a net decrease in temperature-related mortality in virtually all parts of the world, even those with tropical climates.

Climate change is likely to reduce the incidence of fatal coronary events related to low temperatures and wintry weather by a much greater degree than it increases the incidence associated with high temperatures and summer heat waves. Non-fatal myocardial infarction is also less frequent during unseasonably warm periods than during unseasonably cold periods. Warm weather is correlated with a lower incidence of death due to respiratory disease in many parts of the world, including Canada, Shanghai,

Spain, and even on the subtropical island of Taiwan. Low minimum temperatures have been found to be a stronger risk factor than high temperatures for outpatient visits for respiratory diseases.

An extensive scientific literature contradicts the claim that malaria will expand across the globe or intensify in some regions as a result of rising global surface temperatures. Concerns over large increases in mosquito-transmitted and tick-borne diseases such as yellow fever, malaria, viral encephalitis, and dengue fever as a result of rising temperatures are unfounded and unsupported by the scientific literature. While climatic factors do influence the geographical distribution of ticks, temperature and climate change are not among the significant factors

determining the incidence of tick-borne diseases.

In the face of extensive evidence of the positive effects of fossil fuels on human health, the IPCC's claims of a rising "risk of severe ill-health" and "mortality and morbidity during periods of extreme heat" ring hollow. The computer models relied on by the IPCC can be programmed to produce whatever results their sponsors want, and the IPCC has sponsored many models to predict a world filled with disease and misery. But that is not what actual medical science and empirical data allow us to predict. Thanks to fossil fuels, humanity can look forward to living longer and healthier lives than ever before.