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CLIMATE CHANGE AND PUBLIC HEALTH IN CALIFORNIA

A Report From:
California Climate Change Center

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Arnold Schwarzenegger, *Governor*

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Preface

The California Energy Commission's Public Interest Energy Research (PIER) Program supports public interest energy research and development that will help improve the quality of life in California by bringing environmentally safe, affordable, and reliable energy services and products to the marketplace.

The PIER Program conducts public interest research, development, and demonstration (RD&D) projects to benefit California's electricity and natural gas ratepayers. The PIER Program strives to conduct the most promising public interest energy research by partnering with RD&D entities, including individuals, businesses, utilities, and public or private research institutions.

PIER funding efforts focus on the following RD&D program areas:

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In 2003, the California Energy Commission's PIER Program established the **California Climate Change Center** to document climate change research relevant to the states. This center is a virtual organization with core research activities at Scripps Institution of Oceanography and the University of California, Berkeley, complemented by efforts at other research institutions. Priority research areas defined in PIER's five-year Climate Change Research Plan are: monitoring, analysis, and modeling of climate; analysis of options to reduce greenhouse gas emissions; assessment of physical impacts and of adaptation strategies; and analysis of the economic consequences of both climate change impacts and the efforts designed to reduce emissions.

The California Climate Change Center Report Series details ongoing center-sponsored research. As interim project results, the information contained in these reports may change; authors should be contacted for the most recent project results. By providing ready access to this timely research, the center seeks to inform the public and expand dissemination of climate change information, thereby leveraging collaborative efforts and increasing the benefits of this research to California's citizens, environment, and economy.

For more information on the PIER Program, please visit the Energy Commission's website www.energy.ca.gov/pier/ or contract the Energy Commission at (916) 654-5164.

Table of Contents

Preface	iii
Abstract	ix
1.0 Introduction	1
2.0 Approach.....	1
3.0 Results.....	2
3.1. Heat-Related Health Threats.....	2
3.1.1. Methodological Approaches and Challenges.....	2
3.1.1.1 Directly Heat-related Deaths.....	2
3.1.1.2 Observed Compared to Expected Deaths	2
3.1.1.3 Time-Series and Case-Crossover Analyses of the Relationship between Temperature and Mortality in the United States.....	3
3.1.1.4 Mortality Displacement.....	3
3.1.2. Insights on the Heat-Mortality Relationship	4
3.1.2.1 Shape of the Temperature — Mortality Relationship.....	4
3.1.2.2 Directly Heat-Related Deaths.....	4
3.1.2.3 Excess Mortality	5
3.1.2.4 Time-Series and Case-Crossover Studies	6
3.1.3. Additional Insights from Historical Heat Waves	7
3.1.3.1 The 2003 European Heat Wave	7
3.1.3.2 U. S. Heat Waves	9
3.1.3.3 The California Heat Wave of 2006	10
3.1.3.4 The Heat Island Effect	11
3.1.3.5 Additional Stresses from Air Pollution.....	11
3.1.3.6 Heat Wave Mortality and Mental Health.....	13
3.1.4. Insights on the Heat — Morbidity Relationship	13
3.1.4.1 Background	13
3.1.4.2 Preexisting Medical Conditions	13
3.1.4.3 Additional Stresses from Air Pollution.....	15
3.1.4.4 Conclusions on Heat Mortality and Morbidity	16
3.2. Cold-Related Health Threats.....	16
3.2.1. Background.....	16
3.2.2. Methodological Approaches and Challenges.....	17
3.2.3. Insights on the Cold-Mortality Relationship	17
3.2.3.1 Directly Cold-Related Mortality	17
3.2.3.2 Findings from Time-Series and Case-Crossover Analyses of the Relationship between Cold Temperatures and Mortality — U.S. Studies.....	17
3.2.3.3 Findings from Time-Series and Case-Crossover Analyses of the Relationship between Cold Temperatures and Mortality — European Studies.....	18

3.2.3.4	Conclusions:.....	20
3.3	Air Pollution-Related Health Effects.....	20
3.3.1.	Background.....	20
3.3.2.	Ozone.....	21
3.3.3.	Particulate Matter	22
3.3.4.	Conclusions:	23
3.4	Wildfires and Public Health.....	23
3.4.1.	Background.....	23
3.4.2.	Methodological Challenges.....	23
3.4.2.1	Wildfires and Mortality.....	24
3.4.2.2	Wildfires and Morbidity	25
3.4.3.	Conclusions:	26
3.5	Infectious Diseases.....	26
3.5.1.	Water-Borne Diseases	26
3.5.2.	Food-Borne Diseases	28
3.5.3.	Other Infectious Diseases	29
3.5.4.	Vector-Borne Diseases.....	30
3.5.5.	Rodent-Borne Diseases	32
3.5.6.	Conclusions	32
3.6	Climate Change and Public Health.....	32
3.6.1.	Future Estimates of Heat-Related Mortality and Morbidity	32
3.6.2.	Future Estimates of Cold-Related Mortality and Morbidity.....	34
3.6.3.	Implications of Increasing Ambient Temperature on Air Quality.....	34
3.6.4.	Implications of increasing Ambient Temperatures on Wildfires	34
3.6.5.	Implications of increasing Ambient Temperature on Infectious Diseases.....	35
3.6.6.	Water- and Food-Borne Diseases	35
3.6.7.	Other Infectious Diseases	37
3.6.8.	Vector-Borne Diseases.....	37
3.6.9.	Rodent-Borne Diseases	38
4.0	Conclusions and Recommendations.....	39
4.1.	Conclusions.....	39
4.1.1.	Temperature-Related Mortality and Morbidity	39
4.1.2.	Air Pollution	39
4.1.3.	Wildfires.....	40
4.1.4.	Infectious Diseases.....	40
4.2.	Recommendations	41
4.2.1.	Actions to Protect Public Health	41
4.2.2.	Research Needs.....	42
5.0	References.....	43

List of Tables

Table 1. Deaths attributed to the 2003 European heat wave	7
Table 2. Cold-related mortality in the Eurowinter study	18

Abstract

Population health is affected by a complex integration of many factors, including biological, ecological, social, and geographical inputs. Review of the literature on the public health consequences of climate change leads to the conclusion that the outcomes most likely to occur in California include death and illness related to temperature, air pollution, vector- and water-borne diseases, and wildfires. The population groups most at risk include the elderly, those with chronic heart or lung disease, children, people with mental illnesses or addictions, and the socially or economically disadvantaged. There are a number of actions and mitigations that can be adopted on the state-wide, local, and personal level that can reduce or prevent adverse health outcomes related to climate change. These actions include development of heat emergency action plans, continuing efforts to attain the health-based ambient air quality standards, strengthening surveillance for infectious diseases, expanding vector control programs, and review and modernization (as necessary) of water and sewage treatment facilities. It will also be important to expand public education on the health risks associated with climate change, including effective actions the individuals, caregivers, communities and health care providers can take to minimize personal risk.

Keywords: Climate change and public health; climate change and infectious diseases; heat mortality; air pollution and climate change; wildfires and public health

1.0 Introduction

The scientific community is in substantial agreement that the earth is warming, and that climatic patterns are changing, both worldwide and in California (IPCC 2007). Scenario analyses suggest that by the end of the twenty-first century temperatures in California could rise from 2°F to 9°F (1°C to 5°C), depending on the emissions scenario and general circulation model employed for the analysis (Cayan et al. 2009).

In June 2005 Governor Arnold Schwarzenegger issued Executive Order S-3-05, which set greenhouse gas emission reduction targets for California. This Executive Order also directed the Secretary of the California Environmental Protection Agency (CalEPA) to report to the governor and the state legislature by January 2006 (and biannually thereafter) on the impacts of global warming on California, including effects on public health.

Population health is affected by a complex integration of many factors, including biological, ecological, social, political, and geographical inputs. Because of this, the distribution of adverse public health impacts related to climate change is not expected to be uniform worldwide (Gamble et al. 2008), and not all potential public health impacts are expected to be significant concerns for the United States or California, compared to other regions of the world. The impacts identified as most likely to be concerns for public health in California are mortality and morbidity related to temperature, although adverse impacts related to air pollution, vector- and water-borne diseases, and wildfires are also of concern. This report, the second review of the public health impacts of global warming in California, builds on the literature review and conclusions reached in the 2006 report (Drechsler et al. 2006).

There have been a number of reviews on health-related issues related to climate change that take a fairly global perspective (e.g., IPCC, 2007; Ebi et al., 2008). The purpose of this paper is to summarize key findings on public health-related consequences of climate change that are relevant to California.

2.0 Approach

This paper is an update to the literature reviews in the 2006 scenarios analysis public health impacts report (Drechsler et al. 2006). It is based on a literature search performed with PubMed through December 2008. The review primarily focuses on studies performed in the United States, although papers from Europe or Australia that illustrate or explain key concepts are also included. Of the health endpoints that have been identified as being of concern with future climate change, heat-related mortality and morbidity have been most studied, generally with reference to specific heat waves. Other health endpoints identified in the 2006 report as of concern to California include air pollution, vector- and water-borne diseases, and wildfires, and the literature on these topics is also updated. The focus is on clinical health endpoints.

Key words used for the literature search included multiple permutations of: climate change, mortality, morbidity, hospitalization, heat, wild fires, vector-borne diseases, mosquito-borne diseases, tick-borne diseases, water-borne diseases, and food-borne diseases.

3.0 Results

This section summarizes the key conclusions on public health impacts that have been associated with ambient temperature.

3.1. Heat-Related Health Threats

3.1.1. *Methodological Approaches and Challenges*

There are several approaches to estimating the magnitude of temperature-related mortality and morbidity, along with a number of challenges. A key difficulty in estimating the number of cases of heat-related mortality stems from the lack of an “official” definition. Inconsistent definitions of heat-related mortality are applied between jurisdictions (Wolfe et al. 1999), and medical examiners frequently fail to consider heat as a cause of mortality when it is an uncommon diagnosis in their area. Heat-related mortality has been estimated in several ways, including counts based on death certificates, by comparison of observed deaths to expected deaths, and through application of time-series and case-crossover epidemiological models. Each of these methodologies addresses a different question, and it is difficult to compare studies that use different approaches. These methodologies are described below.

3.1.1.1 *Directly Heat-related Deaths*

The number of deaths due to heat is often estimated based on counts of death certificates where heat is listed either as the cause of, or as a directly contributing factor to death. These are often referred to as directly heat-related deaths because the direct cause of death is heat exposure.

3.1.1.2 *Observed Compared to Expected Deaths*

Estimates of excess deaths related to elevated ambient temperature are also made through comparison of the number of deaths during the time period of interest with that during a baseline period. This method defines heat-related deaths as this excess, regardless of the actual cause of death, and leads to estimates of heat-related mortality that are considerably larger than counts of directly heat-related deaths, as described above. However, in this method, most of the deaths are due to pre-existing chronic disease, and not necessarily due to heat-related causes (e.g., O’Neill et al. 2003).

A number of methodological choices influence the results of excess death analyses, making it often difficult to compare results between studies (Gosling et al., 2008). For example, calculation of expected (baseline) mortality is sensitive to the method used to estimate it. This in turn influences the calculated excess mortality, so that results from studies using different methodologies are not directly comparable. Additionally, the categories of deaths included in the analysis also influence the baseline mortality rate, which also makes it difficult to compare studies.

When long time periods are studied, failure to account for long-term trends, such as the changing age-structure of the population, changes and advances in health care, and other longer-term factors that influence vulnerability need to be accounted for to avoid bias in temporal comparisons. Other issues that influence comparison of studies include selection of the index of exposure, and consideration of factors that might modify or confound the relationship between ambient temperature and specific causes of death.

3.1.1.3 Time-Series and Case-Crossover Analyses of the Relationship between Temperature and Mortality in the United States

Retrospective analyses, typically covering several years, have investigated the relationship between high temperature and mortality in various regions of the United States. These analyses have generally used standard time-series epidemiological models, although some recent studies have used the case-crossover approach. The latter method is particularly useful for examining the association between a brief exposure and the acute onset of an adverse response. It is based on comparison of conditions on the day of death and those on referent days on which death did not occur, a method that allows cases to be their own controls (Basu et al. 2005).

These methods present challenges for analysis of long time-series, in that the methodologies are not able to account for long-term trends, such as the changing age-structure of the population, advances and changes in health care, and other longer-term changes that influence vulnerability and could bias temporal comparisons. As with the excess death approach, the index of exposure, and control for confounders, and potential effect modifiers can also influence the results of these types of studies.

These models allow investigation of the lag structure of the relationship between the time of heat stress and the advent of adverse effects. Lag refers to the time period between, for the present discussion, high temperature and an adverse health event. For example, a significant effect at lag 0 means that the temperature on the day of the adverse event is associated with the adverse outcome, while a significant association for lag 1 means that the effect is most associated with temperature on the day before the adverse event, etc.

3.1.1.4 Mortality Displacement

Several studies have investigated whether excess heat mortality represents mortality displacement (also called “harvesting”), which is defined as moving the time of death forward by a period of up to a few weeks in cases that would have occurred relatively soon in any event. The literature on whether or not exposure to elevated temperatures leads to mortality displacement reports somewhat inconsistent findings.

Braga et al. (2001) concluded from an analysis of 12 U.S. cities that excess heat-related mortality was primarily mortality displacement. Results suggesting some level of mortality displacement have also been reported in an 11 year study of 50 U.S. cities (Medina-Ramón & Schwartz, 2007), and in a study of the 1995 Chicago heat wave (Kaiser et al., 2007).

Rey et al. (2007) found evidence for mortality displacement in an analysis including all major heat waves that occurred in France between 1971 and 2003. Hajat et al. (2005, 2006) found significant short-term mortality displacement in London, but less in Delhi (Hajat et al. 2005); possibly because causes of death that are less heat-related, such as infectious diseases, still predominate in the latter city.

Several studies of the 2003 European heat wave also provide evidence that at least some deaths associated with heat waves represent mortality displacement, including in twelve French cities (Dear et al., 2005), and in 15 Mediterranean and north-continental European cities (Baccini et al. 2008). However, in contrast, analyses from Italy (Conti et al. 2007) and France (Fouillet et al. 2006; LeTertre et al. 2006) found no evidence for mortality displacement.

In summary, most studies of mortality displacement, both over several years and related to a specific heat wave, have found evidence for some level of mortality displacement, although the magnitude of the effect varies widely among studies. There are several possible explanations for these disparate findings, including the location of the study and the degree of heat adaptation in that population. It is also plausible that the duration, timing during the season and intensity of heat stress influence whether or not mortality displacement occurs. These factors have not been investigated to date. Another possible explanation of these disparate findings is provided by Gosling et al. (2007), who investigated mortality displacement in six international cities (two in the United States, three in Europe, and one in Australia) that have different climate patterns. The results suggest that a greater degree of mortality displacement may occur in cities that have heat mortality relationships with a higher threshold temperature and flatter slope, compared to cities with a lower threshold temperature and steeper slope. The results also suggest that mortality displacement was no longer evident 12 days after a heat wave.

3.1.2. *Insights on the Heat-Mortality Relationship*

3.1.2.1 *Shape of the Temperature — Mortality Relationship*

It is well known that temperature influences risk of mortality, and that in temperate regions the relationship between temperature and excess mortality is usually “U” or “J” shaped (Curriero et al. 2002, 2003; Kunst et al. 1993; Davis et al. 2003a,b; Kysely 2004). The “trough” in the relationship represents a temperature threshold above and below which mortality increases. Although the general shape of this relationship has been reported for locations worldwide, threshold temperature, and the slopes of both the high and low temperature portions of the temperature-mortality relationship vary considerably among locations (Curriero et al. 2002; Keatinge et al. 2000b; Donaldson et al. 2003a; McGheehin and Mirabelli 2001; McMichael et al. 2008; Baccini et al. 2008). These studies demonstrate that high temperatures have a smaller effect on mortality in regions with typically hot summers compared to the same temperature in regions unaccustomed to high temperatures. This is demonstrated through the higher threshold temperature and the smaller slope of the high temperature arm of the temperature mortality relationship in hotter vs. cooler regions. In addition, these results lead to the conclusion that population responses to extreme temperature events, for example heat waves, and are most affected by the deviation from typical seasonal temperature. Moreover, this dependence on local climate conditions underscores the necessity of considering the temperature mortality relationship on a localized rather than large-scale basis.

Many questions remain as to the magnitude of temperature-related mortality. Several methodologies have been employed to estimate heat-mortality cases, although the available literature does not point to a standardized approach for investigating this topic. Each approach addresses different questions, and estimates derived from the different approaches vary. However, the consensus of the literature to date is that heat exposure does increase both mortality and morbidity. The next several sections summarize the insights gained from each analytical approach.

3.1.2.2 *Directly Heat-Related Deaths*

According to the Centers for Disease Control (CDC 2006), between 1999 and 2003 there were 3,442 deaths directly attributable to extreme heat exposure reported in the United States. This number includes 2,239 cases where excessive heat exposure was the direct cause of death,

although this is likely an underestimate due to under-reporting, and differences between localities in the criteria for ruling a death heat-related (Mirabelli and Richardson 2005; Basu et al. 2005). The remaining 1,203 cases had excessive heat exposure as a contributing cause of death. Of cases where age of the decedent was known, 7% were less than 15 years of age, 53% were between 15 and 64 years of age, and 40% were over 65 years of age. The states with the highest annual hyperthermia-related death rates during this time period were Arizona, Nevada, and Missouri.

When defined in this way, the majority of heat mortality cases are relatively young to middle-aged adults who were active in the heat prior to their demise, although a significant fraction of cases were older adults. The number of cases in children has been comparatively small, and often related to the child being trapped in a parked car. Among the 1,203 cases where hyperthermia was a contributing factor to death, cardiovascular disease was the underlying cause of death in 57%, while infection and psychiatric disorders accounted for 11%.

One of the largest subgroups with increased vulnerability to directly heat-related death is crop workers. The Centers for Disease Control (CDC 2008) reported that between 1992 and 2006, 432 crop workers died from exposure to environmental heat. Nearly all of these decedents were male, and 78% were between the ages of 20 and 54 years. Almost 60% of these deaths occurred in July, and most occurred in the afternoon. Twenty-one states have reported heat-related deaths among crop workers, but California, Florida and North Carolina accounted for 57% of all reported deaths. The highest rate of deaths in crop workers occurred in North Carolina. It is likely that this is an under-estimate of heat-related deaths in this subpopulation for the reasons noted above.

3.1.2.3 Excess Mortality

Excess mortality related to high ambient temperature has declined in recent years in both the United States and other regions. Davis et al. (2003b) found a decline in mortality rates related to high temperature in the 1980s and 1990s, compared to the 1960s and 1970s in 28 U.S. cities, although the reduction in temperature-related mortality was not uniform throughout the country. High temperature remained significantly related to mortality in the northeastern and north central parts of the country, while most southern cities no longer exhibited an increase in mortality rates on high temperature days by the 1990s. Further analysis of this data set (Davis et al. 2003a) showed that for the 28 cities as a group, annual excess heat-related death rates declined from about 41 per million people in the 1960s and 1970s, to 17 per million people in the 1980s, and to 11 per million people in the 1990s. The authors suggested that this decrease was due to the increasing use of air conditioning in the southern United States, improved health care, and heightened public awareness of the risks posed by elevated temperature.

Barnett (2007) investigated the influence of temperature on cardiovascular deaths in elderly Americans living in 107 U.S. cities by using daily cardiovascular mortality counts from the National Morbidity and Mortality Air Pollution Study (NMMAPS). The results showed that the average increase in cardiovascular deaths associated with a 5.5°C (10°F) increase in temperature was 4.7% in the summer of 1987, while by summer 2000 risk was -0.4%, suggesting either no effect of heat on mortality, or that heat provided a small benefit to survival.

Donaldson et al. (2003a) found that between 1971 and 1997 excess mortality related to temperature decreased in both North Carolina and South Finland, and remained unchanged in

Southeast England. This is in agreement with Carson et al. (2006), who reported that the percentage of all cause mortality attributable to heat was 0.4% in 1900–1910, and -0.9% in 1986–1996 in London, England.

3.1.2.4 Time-Series and Case-Crossover Studies

Basu et al (2005) compared the standard time-series approach with two case-crossover methods to estimate the influence of temperature on mortality in 20 U.S. cities (six in California) that were distributed throughout the country. The results were similar for the three different modeling strategies, and the odds ratio for heat-related mortality was greatest in the southwestern cities, which included Los Angeles, San Bernardino, Santa Ana, and San Diego. This study has several limitations that may explain why the results are not in agreement with those of others. The study included only one year of data, which was informative for addressing the main objective of the paper, which was to comparing several time-series and case-crossover analyses. In addition, data on some possibly relevant confounders or effect modifiers, such as air conditioner use, and socioeconomic status. Another factor could be that temperature exposure was based on a county-wide average because individual temperature exposure data were not available.

A four year retrospective analysis of the average relative risk of heat-related mortality for nine California counties (Basu et al., 2008) found a 2.3% (95% CI: 1.0–3.6) increase in mortality with a 5.5°C (10°F) increase in same-day mean apparent temperature (combination of temperature and humidity) using the case-crossover method, with a nearly identical relative risk using a time-series methodology. Relative risk of heat-related mortality was generally higher in the counties that had cooler climates. Further analysis of these data (Basu and Ostro 2008) examined factors that influenced vulnerability. The results showed increased risk for death for cardiovascular causes, especially ischemic heart disease. There was also increased risk for people over 65 years of age, infants less than one year of age, and for the African-American racial/ethnic group. Gender and educational attainment did not influence risk size. Medina-Ramón et al. (2006) has also identified African-American race as a risk factor for heat mortality.

A study using similar methods for nine U.S. cities (Zanobetti and Schwartz 2008) found a 1.8% (95% CI: 1.09–2.5) in mortality per 5.5°C (10°F) increase in apparent temperature using a case-crossover methodology, and a 2.7% (95% CI: 2.01–3.5) using a time-series methodology. The cities were in the eastern half of the United States, except for Phoenix, and had average summer apparent temperatures ranging from 20°C to 32°C (68°F to 90°F). Warmer cities had smaller effect estimates and/or wider confidence intervals than cooler cities. The results suggested that physiological and sociological acclimatization plays a role in reducing the heat effect in cities with a warmer climate compared to those with cooler climates.

Braga et al. (2001) carried out a six year time-series analysis for 12 U.S. cities to investigate the relationship between weather variables and acute mortality, along with the lag structure of the relationship. In the cities that had cooler weather patterns, both high and low temperatures were associated with increased deaths on the same or the preceding day. There was little effect of high or low temperature on deaths in the warmer cities, where the smaller effect of high temperature was attributed to the greater penetration of air conditioning in the warmer cities.

A case-cross-over study of the temperature-mortality relationship in 50 U.S. cities (Medina-Ramón and Schwartz, 2007) confirmed that heat increases risk of mortality, although the effect was quite heterogeneous among the cities, with the largest effects observed in cities with milder climates, less penetration of air conditioning, and higher population density. A case-only analysis for the same 50 U.S. cities (Medina-Ramón et al., 2006) found that the elderly, diabetics, African-Americans, less educated subjects, and those dying outside hospitals were more susceptible to death on extreme heat days.

These studies, drawing on different methodological approaches, support several conclusions: (1) there is a relationship between temperature and mortality even without extreme heat conditions; (2) the magnitude of risk relates to the typical local climate and is highly heterogeneous across the United States; and (3) some population sub-groups are more at risk than others. Below, additional insights are gathered from individual heat events.

3.1.3. Additional Insights from Historical Heat Waves

3.1.3.1 The 2003 European Heat Wave

The most extensively analyzed historical heat wave struck Europe for about 15 days in August 2003. This was the hottest period in Europe since 1500 (Poumadère et al. 2005). France, Italy, Spain, the United Kingdom, the Netherlands, and Switzerland all reported an increase in deaths (Table 1) during and shortly after this heat wave, although the death toll was highest in France.

Table 1. Deaths attributed to the 2003 European heat wave

Country	Region of Country	Excess Deaths	Change in Rate
France ^a	Whole country	14,802	+60%
Italy ^b	Whole country	7,659	+19%
England and Wales ^c	Whole country	2,139	+16%
The Netherlands ^d	Whole country	500	—
Switzerland ^e	Whole country	975	+7%
Portugal ^f	Mainland Portugal	1,316	+38%
Spain ^g	Provincial capitals	2,175	+17%

^a Pirard et al. 2005; ^bConti et al. 2007; ^c Johnson et al. 2005a,b; ^d Garssen et al. 2005; ^e Grize et al. 2005 (whole summer 2003); ^f Nogueira et al. 2005; ^g Simón et al. 2005

Multiple papers have examined the impact of the 2003 heat wave in France, which at the time had no public policy in place to address a heat emergency. The average Paris daytime high temperature during August is about 23°C (75°F) (weather.com), while during the 15 days from August 4 to 18, 2003, average daily high temperatures exceeded 35°C (95°F) (Canouï-Poitrine et al. 2006; LeTertre et al. 2006), and nighttime temperatures averaged about 3.5°C (6.3°F) higher than usual (Canouï-Poitrine et al. 2006). The unprecedented temperatures during this heat wave suggest what might result from an extreme heat wave with a lack of preparedness, including inadequate information for the public on effective protective practices and behaviors, absence of cooling centers, and minimal penetration of air conditioning into local building stock.

The increase in excess deaths was not uniform throughout the country. While almost 15,000 excess deaths are estimated to have occurred in France as a whole during this period, deaths were heterogeneously distributed across the country. The excess death rate for France as a

whole was about 60% higher than normal, although rates varied from a 36% increase in rural communities to a 140% increase in Paris (Canoui-Poitrine et al. 2006; LeTertre et al. 2006; Vandentorren et al. 2006). Even within the city of Paris, excess deaths were not evenly distributed, with the greatest number of excess deaths in the southern part of the city (Canoui-Poitrine et al. 2006). The increase in deaths lagged the start of the heat wave by one to three days (Vandentorren et al. 2006).

Although not mentioned in the literature reviewed here, several news media accounts point out that the heat wave occurred during the typical Parisian August vacation period. Many government officials were also away from Paris at the time of the heat wave. These factors led to slower government response to the emergency, and to disrupted social bonds and a reduced alternate safety net for the elderly who remained in Paris while family members were on vacation. (http://en.wikipedia.org/wiki/2003_European_heat_wave; <http://www.cnn.com/2003/WORLD/europe/08/29/france.heatdeaths/>).

The principal causes of death varied by age group, with directly heat-related deaths being much more common in younger, healthier individuals, while excess deaths were predominantly in the elderly. People 75 years of age and over accounted for 11,891 of the increased deaths in France, while there was little impact in children less than five years of age. Of the deaths in the over 75 years of age group, 2,852 were directly related to heat. The next largest fraction of deaths was from cardiovascular disease. Other frequent causes of death included undefined causes, respiratory diseases and diseases of the nervous system (Pirard et al. 2005; Fouillet et al. 2006; Poumadère et al. 2005). Among 2,565 deaths in people between 45 and 74 years of age, 434 were directly related to heat, while other deaths were due to undefined causes, cardiovascular diseases, cancer, mental illness, nervous system disorders, infectious diseases, and pulmonary diseases. There was a 19% increase in deaths of people under 45 years of age, which occurred principally in men, with the majority caused by undefined conditions, heatstroke, dehydration, and hyperthermia.

People less able to care for themselves were at increased risk of adverse outcomes, whether they lived at home or in retirement or nursing homes. A significant proportion of excess deaths occurred in Parisian nursing home patients, where the mortality rate was about four times higher than average during the heat wave (Holstein et al. 2005). These results suggested that level of dependency was a risk factor for death in these patients. Another study, Fouillet et al. (2006), found that excess mortality at home and in retirement institutions was greater than in hospitals. Belmin et al. (2007) also reported that level of dependency was a risk factor for heat-related death in community dwelling elderly people. Residents of institutions such as retirement and nursing homes are by definition less able to care for their own needs, and are often less able to recognize when they are at risk due to elevated ambient temperatures. The rarity of air conditioning in France, coupled with the frequently less robust health status of the elderly, and lack of planning for heat emergencies are all likely contributing factors to the increase in deaths in these sub-populations.

Several clinical factors on presentation at an emergency room were associated with increased risk of a poor outcome. Patients admitted to the emergency room who used antihypertensive medication or who had anuria, coma, or cardiovascular failure had poorer outcomes (Argaud et al. 2007). Follow-up with patients who survived heatstroke showed that they still had significant functional impairment one and two years later, and a substantial fraction had

become bedridden. In another study of patients evaluated in the emergency room of a Parisian hospital Davido et al. (2006) found that patients who had a core temperature $\geq 38^{\circ}\text{C}$ (100.5°F) and/or with clinical signs of dehydration were at increased risk of death within one month. Other factors associated with a poor outcome were impaired ability to care for oneself, more serious clinical condition on presentation, alterations in blood chemistry indicative of disrupted physiological homeostasis, pre-existing ischemic cardiomyopathy, pneumonia, or previous treatment with psychotropic medications.

Collectively, studies of the 2003 heat wave point to a number of factors that increase risk of heat-related mortality. These factors include age over 75 years, being female, and being single, particularly for men (Canoui-Poitrine et al. 2006; Poumadère et al. 2005; Pirard et al. 2005; Fouillet et al. 2006), poverty, isolation, living in an urban area (Poumadère et al. 2005), lack of mobility, pre-existing medical conditions, lack of insulation in the home, sleeping on the top floor of the building (directly under the roof), and an independent contribution from the heat island effect (Vandentorren et al. 2006). Poumadère et al. (2005) found that use of some medications commonly prescribed to treat cardiovascular disease and mental health conditions increased risk of adverse outcomes with heat exposure, a finding also reported in the U.S. (CDC, 2002; Kaiser et al., 2001). These medications, as a side effect, can reduce physiological thermoregulatory responses to heat through alteration of neurological control of the vasculature and sweat glands.

Similar findings have been reported for other European countries affected by the 2003 heat wave, including Spain (Simón et al. 2005), mainland Portugal (Nogueira et al. 2005), Italy (Michelozzi et al. 2005a; 2005b), England and Wales (Johnson et al. 2005a,b; Kovats et al. 2006), the Netherlands (Garssen et al. 2005), and Switzerland (Grize et al. 2005).

3.1.3.2 U. S. Heat Waves

Heat wave analyses in the United States have focused primarily on heat waves that struck the Midwest during 1995 and 1999, particularly Chicago. A record-setting heat wave struck Chicago in July 1995, during which both daily maximum and minimum temperatures were unprecedented, and relative humidity was high (Semenza et al. 1996).

Medical examiner's records suggested that there were 437 directly heat-related deaths (Naughton et al. 2002), and at least 700 excess deaths (Semenza et al. 1996) attributable to the heat wave. Mortality peaked two days after the hottest day, 40°C (104°F), suggesting that there was a lag time between peak heat exposure and death (Kaiser et al. 2007). The most frequent causes of death were cardiovascular and heat-related. The number of deaths from respiratory disease did not change during the heat wave (Kaiser et al. 2007). Kaiser et al. (2007) reexamined the effects of the 1995 Chicago heat wave on all-cause and cause-specific mortality, using advanced time-series analysis methods that allowed for control of the meteorological and air pollution variables, and found results suggesting that about 26% of the observed deaths were due to mortality displacement.

Factors identified as being associated with increased risk of heat-related death included having chronic medical problems, being unable to care for oneself, not leaving home daily, living alone, living on the top floor of the building, having few social contacts, being bed-ridden, and lack of access to air conditioning, low educational attainment (i.e., less than high school), age above 75

years, African-American race, and chronic cardiovascular disease (O'Neill et al. 2003; Semenza et al. 1996; Medina-Ramón et al. 2006; Hajat et al. 2001; Kaiser et al. 2007).

Several factors reduced risk of heat-related mortality, including having regular social contacts, access to transportation, and having a working air conditioner (Semenza et al. 1996; Medina-Ramón et al. 2006; Hajat et al. 2001).

One response to the 1995 heat wave was development of an Extreme Weather Operations Plan for the Chicago area to disseminate information on how to avoid heat illness and death and to provide interventions focused primarily on the elderly to reduce risk of heat-related health conditions. Another heat wave struck the Midwest from July 29 through August 1, 1999, during which the new Extreme Weather Operations Plan was activated. About 80 excess heat-related deaths occurred during the three-day 1999 heat wave (Naughton et al. 2002), considerably fewer than occurred in 1995 (about 700 excess deaths). Fifty-three percent of the 1999 cases were less than 65 years of age.

Environmental conditions during the 1995 and 1999 Chicago heat waves were similar, and the large reduction in mortality in 1999 compared to 1995 has been attributed to the effectiveness of the Extreme Weather Operations Plan, and judged not likely due to meteorological differences between the two heat waves (Palecki et al. 2001). Both the 1995 and 1999 heat waves affected much of the Midwest, in addition to Chicago, a substantial reduction in morbidity and mortality was also reported for Milwaukee, which has been attributed to increased public health preparedness and response actions put into place after the 1995 heat wave (Weisskopf et al. 2002).

3.1.3.3 The California Heat Wave of 2006

A two-week heat wave occurred in California in July 2006. Historical analysis of major heat waves in California shows that they are typically characterized as either daytime or nighttime events, based on whether the daytime highs or nighttime lows are unusually elevated. The July 2006 heat wave had daytime temperatures that were high, although not record-breaking. Nighttime temperatures, however, during this heat wave were unprecedented (Gershunov 2007).

Examination of coroner's records found that 140 deaths were directly attributable to heat exposure during the heat wave, with cases concentrated in Imperial, Stanislaus, San Joaquin, Fresno, Kern, and Sacramento Counties (Kim and Trent 2007). This is likely an underestimate, for reasons discussed previously.

Evaluation of the characteristics of the decedents showed that about two-thirds were white, non-Hispanics, and about one-fourth were Hispanics. Two-thirds of cases were male. About 80% of cases were over 50 years of age, with only a few less than 20 years of age. However, among Hispanics almost half were less than 50 years of age. Of the 140 cases, ninety-six died indoors, and among these, 45% had no air conditioning. Thirty-five decedents had air conditioning units in their homes, but the unit was not functional in 16 cases, and not used in 18 cases. Almost half of cases lived alone, and among these, almost half had no known recent social contacts. These results and characteristics of decedents are similar to those described above for the Chicago heat wave.

Several causes of death predominated. Forty-seven percent had chronic cardiovascular disease, 23% psychiatric illnesses, 17% alcoholism, 7% chronic pulmonary disease, and 2% were confined to bed.

Knowlton et al. (2008) analyzed county-level hospitalizations and emergency department visits for all causes, and for grouped causes, and reported 16,166 emergency department visits and 1182 excess hospitalizations statewide during the heat wave. Children up to four years of age, and adults over 65 years of age were at greatest risk. Risk of hospitalization was highest in the Central Coast region of the state (including San Francisco), which has fairly cool summers. This underscores previous findings that risk of adverse effects with heat exposure is related more strongly to deviations from the usual temperature range than to the actual recorded temperature.

3.1.3.4 The Heat Island Effect

The heat island effect refers to the observation that ambient temperature is typically higher in urban areas than rural areas. This effect results from the tendency for greater heat absorption and retention in urban areas due to the high concentration of buildings, pavement, and roadways. There was evidence for the influence of the heat island effect on mortality in analyses of the 1995 Chicago heat wave (Naughton et al. 2002; Semenza et al. 1996), and the 1999 heat wave in the Midwestern U.S. (Palecki et al. 2001). Canouï-Poitrine et al. (2006) and Vandentorren et al. (2006) observed a greater effect of heat on mortality in urban compared to rural areas of France during the 2003 heat wave, as did Hajat et al. (2007) in England.

3.1.3.5 Additional Stresses from Air Pollution

It is well known that air pollution, especially ozone, is often highest on hot, sunny days, raising the question of the effect of co-exposure to high heat and air pollution on heat-related mortality. Most studies that have investigated the health effects of air pollutants such as ozone and particulate matter have reported that when the epidemiological models control for temperature there is little evidence that temperature confounds or modifies the effects of air pollution on health endpoints (see section 3.1.5.3). In contrast, when model control for air pollutants has been included in studies investigating heat-related effects, results have been inconsistent as to whether air pollutants modify or confound the relationship between temperature and health endpoints. While these results may appear contradictory, they demonstrate that while air pollution has an independent effect, the temperature effect is stronger.

Several older studies suggest that air pollution has a small confounding effect on the temperature mortality relationship (Rainham et al., 2003; Smoyer-Tomic and Rainham, 2001), although until fairly recently, little attention has been devoted to investigation of the potential for air pollution to confound or modify the relationship between temperature and mortality.

Fischer et al. (2004) and Stedman (2004) analyzed data from the 2003 European heat wave, and suggested that although heat-related mortality dominated the increased mortality observed, the proportion of deaths related to air pollution was greater than had been appreciated. These studies estimated deaths from concentration-response functions developed for analyses of air pollution effects, and are unlikely to adequately account for the influence of the heat wave on mortality. Levels of peak ozone and particulate matter 10 microns or smaller (PM₁₀) were not above California ambient air quality standards in the Netherlands (Fischer et al. 2004), and only slightly above California standards in the United Kingdom (Stedman 2004). The number of

deaths attributed to air pollution by these papers for the two-week period of the 2003 European heat wave is very high given the population size, the duration studied, and the pollutant concentrations.

Studies published since 2004 have produced inconsistent results as to whether air pollution confounds or modifies the relationship between ambient temperature and mortality. The joint effects of temperature and ozone on cardiovascular mortality were substantially heterogeneous across 95 U.S. cities in a study based on the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) data set (Ren et al. 2008). In northern cities, ozone generally modified the association between temperature and cardiovascular mortality, but the effect was variable in southern communities. Effect modification or confounding was evident for Southern California communities, although when all 95 cities were combined in a Bayesian meta-analysis, ozone positively and significantly modified the temperature-cardiovascular mortality association. Baccini et al. (2008) and Filleul et al. (2006) also found significant heterogeneity in the influence of ozone on the temperature-mortality relationship in studies of 15 and nine European cities, respectively. Collectively, these papers suggest the influence of ozone on the temperature-cardiovascular mortality relationship tends to be smaller in populations regularly exposed to elevated ozone levels.

Several studies suggest that concomitant exposure to heat and ozone leads to higher risk of mortality than heat exposure alone. Dear et al. (2005) found that ozone significantly contributed to mortality during the August 2003 heat wave in France. A study from Sydney, Australia also found that ozone confounded the association between maximum temperature and mortality (Vaneckova et al. 2008a) during the six warm months of the year.

Several studies have found no evidence that ozone confounds or modifies the relationship between heat and mortality, including an analysis of the 1995 Chicago heat wave (Kaiser et al. 2007), and an analysis of nine French cities during the 2003 European heat wave (LeTertre et al. 2006). Keatinge and Donaldson (2006) investigated possible confounding of the relationship between mortality attributed to air pollution in the Greater London area using graphic and generalized additive modeling to include weather factors that have not typically been considered in these sorts of models. When temperatures were above 18°C (64°F), high levels of ozone were associated with sunshine, which increases heat stress. When confounding by sunshine was controlled for, there was no significant contribution of ozone to heat mortality.

Vaneckova et al. (2008b) used a synoptic (air mass) approach to investigate the relationship between weather and heat-related mortality over the warm seasons of 1993 to 2001 in Sydney, Australia. Days were classified into one of eleven different synoptic categories, and the correlation between these categories and heat-related mortality was estimated. Two synoptic categories were significantly associated with increased heat-related mortality. A relatively rare hot, dry category was most strongly associated with higher mortality rates, followed by a more frequent warm, humid category. Ozone was highest when the warm, humid category occurred, while PM₁₀ occurred in high concentrations with the hot, dry category. Overall, however, the influence of the air pollutants on the relationship between synoptic pattern and mortality was unclear. O'Neill et al. (2003) also found inconsistent confounding by PM₁₀, in that the direction of the influence of PM₁₀ (positive or negative) varied by location.

Two studies from Brisbane, Australia have investigated the effect of particulate matter exposure on the relationship between temperature and mortality. Ren et al. (2006) found that PM₁₀ increased the effects of temperature on all non-external-cause mortality, and cardiovascular mortality at high concentrations. Ren and Tong (2006) found a statistically significant interaction between PM₁₀ concentration and temperature on mortality, with a larger effect on warm days. However, the choice of the number of degrees of freedom used in adjusting for confounders, and the selection of arbitrary cut-offs for temperature affected the estimates of the size of the interaction, although the general conclusion held with all models evaluated.

A study from Sydney, Australia also found that PM₁₀ confounded the association between maximum temperature and mortality (Vaneckova et al. 2008a) during the six warm months of the year.

Keatinge and Donaldson (2006) also evaluated the influence of PM₁₀ using the same approach noted above for ozone. The results showed that at temperatures above 18°C (64°F), high levels of PM₁₀ were associated with sunshine, and low wind, both of which increase heat stress. When confounding by sunshine and wind speed was accounted for, PM₁₀ did not influence the relationship between temperature and mortality.

The inconsistent findings of these studies preclude a conclusion as to whether ozone and/or PM₁₀ confound or modify the relationship between elevated temperature and mortality, and thus are prime areas for further research.

3.1.3.6 Heat Extremes and Mental Health

Few studies have investigated the impact of heat waves and high temperature on mental health. A series of studies performed in Adelaide, Australia found that mortality in people with mental and behavioral disorders increased during heat waves among people between 65 and 74 years of age, and also in persons with schizophrenia, schizotypal and delusional disorders (Hansen et al. 2008; Nitschke et al. 2007). While it may appear odd that people with mental health conditions are at increased risk of heat mortality, several factors contribute to increased vulnerability in this population. Many mental illnesses include some level of judgment impairment, the extent related to the nature and severity of the illness. In addition, some medications commonly used to treat mental health conditions affect functioning of the autonomic nervous system in ways that impair physiological temperature regulatory mechanisms, including redistribution of blood flow and sweating, so that the body is less able to thermoregulate.

3.1.4. Insights on the Heat — Morbidity Relationship

3.1.4.1 Background

In comparison to investigation of the relationship between high temperatures and mortality, few papers have been published on the impact of hot weather and heat waves on emergency room visits and hospitalizations, and other morbidities.

3.1.4.2 Preexisting Medical Conditions

Several studies suggest that the relationship between temperature and hospitalizations and emergency room visits for exacerbation of chronic disease is not parallel to that between temperature and mortality, and that it appears to have a shallower slope. Kovats et al. (2004a) examined the relationship between daily emergency hospital admissions and hot weather and

several heat waves between April 1994 and March 2000 in Greater London, England. There was no clear relationship between total emergency hospital admissions and high ambient temperatures, except for an increase in emergency admissions for respiratory and renal disease in children under 5 years of age, and for respiratory disease in people over 75 years of age. During a heat wave that occurred between July 29 and August 3, 1995 there was a 10.8% increase in daily mortality (95% CI: 2.8–19.3%), while there was only a small non-significant increase in hospitalizations (2.6%: 95% CI -2.2–7.6) during the same time period. These results suggest different relationships between morbidity and hot weather than between temperature and mortality, at least in the United Kingdom. The authors suggested that a possible explanation for these findings may be that many heat-related deaths may occur before the cases come to medical attention.

The influence of the intensity, duration, and timing of heat waves on hospital admissions over the summers of 2002 and 2003 in the Veneto Region of Italy in persons over 75 years of age was investigated by Mastrangelo et al. (2007). Admissions for heat-related disorders (including heat stroke, disorders of fluid and electrolyte balance and acute renal failure) and respiratory diseases increased with heat wave duration, but not intensity. The results suggest that at least four consecutive hot, humid days were required before there was a significant increase in hospital admissions. Interestingly, similar increases in hospitalizations occurred with the first heat wave of the season, in early June as the last, in August.

Several Australian studies of heat-related morbidity have investigated the relationship between indices of morbidity and heat. Although Nitschke et al. (2007) found no excess mortality during heat waves in metropolitan Adelaide, there were modest increases in calls for ambulance transport, and for hospitalization for mental health issues, causes related to renal function, and ischemic heart disease. A follow-up analysis (Hansen et al. 2008) found a positive association between ambient temperature and hospital admissions for mental and behavioral disorders above a threshold of 26.7°C. A third paper from this research group (Hansen et al. 2008) reported an increase in hospital admissions for renal disease and acute renal failure during heat wave compared to non-heat wave periods, although there was no relationship between heat and hospitalizations for dialysis, or for patients with co-morbid diabetes.

Several studies have investigated the relationship between cardiovascular morbidity and elevated ambient temperatures. Hospital admissions for heart disease in people over 65 years of age in 12 U.S. cities increased monotonically with same and previous day average temperature, with no evidence for an effect related to humidity (Schwartz et al., 2004). Kiu et al. (2004) compared coronary care unit admissions for atrial fibrillation on the 30 hottest and 30 coldest days of 2001 in South Australia. The results showed a smaller effect on hot days than cold. Michelozzi et al. (2008), in contrast, found no increase in cardiovascular hospitalizations, but instead, an increase for respiratory causes.

Morabito et al. (2005) used a biometeorological approach to investigate the relationship between weather and myocardial infarction in Florence, Italy for the period 1998 to 2002. Myocardial infarction was associated with number of discomfort days, and with days where apparent temperature was over the 90th percentile of the historical distribution for at least nine hours. Unusually high nighttime temperatures were also a contributing factor. The association between heat and hospitalization for myocardial infarction was strongest with a lag time of three days. Hot weather conditions increased the rate of hospital admissions in general, and

especially in young people. These results are in contrast to other studies suggesting that the greatest increase in hospitalizations is in the elderly.

One study has reported that in contrast to temperature-related mortality data, hospitalizations increased for respiratory, but not cardiovascular, causes as temperature increased. The study compared hospital admissions for cardiovascular, cerebrovascular, and respiratory causes during the warm season in people over 65 years of age in 12 European cities (Michelozzi et al. 2008). The strength of the association varied among the 12 cities, although it was evident in all the cities. The authors suggested that the variability in the strength of the association, which was evident among the hotter cities, may be influenced by between-country differences in delivery of health care, such as admission policies, and availability of hospital beds, along with socioeconomic status, and local customs of clinical management.

One paper has investigated the influence of elevated temperature on a neurological condition. Tataru et al. (2006) found no statistically significant relationship between mean ambient temperature and the number of hospital admissions or relapses in patients with multiple sclerosis in France during the 2003 European heat wave.

Larrieu et al. (2008) investigated morbidity in subsets of the participants in two established cohorts of elderly French people during the 2003 heat wave. The cohorts differed in the age of the subjects, and in the proportion of subjects that lived in urban compared to more rural areas. Subjects were questioned about their perceptions about their health during the heat wave, and for objective morbidity events, such as dizziness, fainting, falls, hospitalization or death, as well as behavioral changes they made to cope with the heat wave. During the heat wave, 8.8% of the subjects reported deterioration in their health, while 7.8% reported a clinically relevant outcome; however the subjects who reported deterioration in their health were not necessarily the same ones who experienced a clinically relevant event. Low educational level, being disabled, and being a woman increased risk of objective morbidity, while being able to ventilate the house and having a bathroom decreased risk of objective morbidity.

3.1.4.3 Additional Stresses from Air Pollution

It is well known that air pollution, especially ozone, is often highest on hot, sunny days, raising the question of the influence of co-exposure to high heat and air pollutants on heat-related morbidity. Only a few studies have considered how air pollution may influence heat-related morbidity.

Two studies from Brisbane, Australia have investigated the effect of particulate matter exposure on the relationship between temperature and adverse health impacts. Ren et al. (2006) found that PM10 increased the effect of temperature on respiratory and cardiovascular hospital admissions, although there was no clear evidence for an interactive effect on respiratory or cardiovascular emergency department visits. Ren and Tong (2006) found a statistically significant interaction between PM10 concentration and temperature on various indices of hospitalization, and emergency department visits, with a larger effect on warm days. However, the choice of the number of degrees of freedom used in adjusting for confounders, and the selection of arbitrary cut-offs for temperature affected the estimates of the size of the interaction, although the general conclusion held with all models employed.

An investigation of the influence of air pollution on the relationship between temperature and hospitalization for cardiovascular diseases among elderly people in Denver during July and August 1993 to 1997 was published by Koken et al. (2003). Once the relationship between temperature and hospitalization was modeled, the investigators introduced single air pollutants and evaluated whether air pollutants influenced the relationship between temperature and hospitalization. The results suggest that ozone is associated with an increase in risk of hospitalization for acute myocardial infarction, coronary atherosclerosis and pulmonary heart disease, in agreement with Linares and Díaz's (2007) results for Madrid, Spain. Koken et al. (2003) also found that particulate matter and nitrogen dioxide did not alter the relationship between temperature and any health outcomes. Sulfur dioxide increased the association between temperature and hospital stays for cardiac dysrhythmias, and carbon monoxide was significantly associated with congestive heart failure.

3.1.4.4 Conclusions on Heat Mortality and Morbidity

- The shape of the temperature-mortality relationship for extreme events differs from that for average seasonal temperature. Regions with hotter average temperatures have a higher threshold temperature for increased heat-related mortality, and the slope of the relationship between temperature and mortality is flatter, in contrast to areas with a cooler climate. Extreme heat events tend to have a smaller effect in areas with higher threshold temperatures.
- Heat mortality has a lag of 0-3 days.
- Different age groups are affected differently – causes of death vary by age. Directly heat-related deaths are more frequent in younger, healthier people than excess deaths, which occur predominantly among the elderly and people with chronic diseases.
- The wide variety of risk factors for heat-related mortality and morbidity identified to date includes personal characteristics and behavior choices, health status, socioeconomic, housing stock, and other factors. Since these factors touch on all areas of life, their variety presents challenges to development of public health interventions designed to promote population adaptation to future climate change.
- Hospitalizations related to heat predominately occur in the elderly and in people with chronic diseases.
- Incidence of heat-related morbidity does not parallel heat-related mortality.
- The influence of concomitant exposure to air pollution on heat-related mortality and morbidity remains unclear.

3.2. Cold-Related Health Threats

3.2.1. Background

Hypothermia, a preventable medical emergency, is a reduction in body temperature to less than 95°F (35.0°C). It is usually caused by prolonged exposure to cold temperatures without adequate clothing or other protection. The most common additional risk factors include advanced age, alcohol or drug use, altered mental status, and contact with substances that promote heat loss, such as water.

3.2.2. Methodological Approaches and Challenges

Similar approaches have been used to investigate cold-related mortality as were described for heat-related mortality. Fewer studies have investigated the contribution of low temperatures to mortality in the United States, than have investigated high temperatures.

3.2.3. Insights on the Cold-Mortality Relationship

3.2.3.1 Directly Cold-Related Mortality

Between 1979 and 2002, an average of 689 deaths per year was directly attributed to excess natural cold exposure in the United States. Using 2002 as an example (CDC 2005a), the majority of cold-related deaths occurred in males, and slightly over half in people over 65 years of age. The three key risk factors for directly cold-related mortality are advanced age, mental impairment (i.e., dementia), and substance abuse. The higher risk for older adults is believed to be related to reduced perception of the cold, and to a lower metabolic rate, which reduces body heat production. Persons with dementias and mental impairment due to substance abuse are particularly at risk due their lack of perception of the risks and dangers of cold exposure. Hypothermia-related death does not only occur under extreme conditions, and many cases are reported in areas with relatively mild cold weather. The U.S. annual death rate for hypothermia has declined since 1991 (CDC 2005a). While the subject of cold-related mortality may appear out of place in the context of global warming, it is essential to remember that research indicates that it will still play a role in total mortality even with increasing world temperatures.

3.2.3.2 Findings from Time-Series and Case-Crossover Analyses of the Relationship between Cold Temperatures and Mortality — U.S. Studies

Several studies have reported that cold-related mortality can occur not only during extremely cold days, but also at relatively mild temperatures. A study based on data from the National Morbidity and Mortality Air Pollution Study (Barnett 2007) that included data from 107 U.S. cities from 1987 to 2001 showed that although cardiovascular mortality was lower during warmer winters, the risk of cold-related mortality has persisted without significant change throughout the 14-year analysis period. In contrast, heat-related mortality declined during the same time period. In addition, the results showed that mortality risk not only increased on the coldest days of winter, but also was elevated on relatively cold days in spring and fall. The latter finding was confirmed by Cagle and Hubbard (2005), who reported a greater mortality rate for winter (0.64 per 100,000) than for summer (0.54 per 100,000) months in people over 55 years of age in King County, Washington, between 1980 and 2001, although the highest mortality rate was at temperatures below 5°C (41°F).

Braga et al. (2001) found that for 12 U.S. cities cold temperatures were most associated with deaths on the same day, although an increased number of deaths were observed over the following week or two, with no sign of a harvesting effect. The effect size varied among cities, and was larger in the warmer cities, in agreement with the results of Curriero et al. (2002, 2003).

Cold was most strongly associated with death from myocardial infarction and cardiac arrest, and was fairly homogeneous across 50 U.S. cities (Medina-Ramón and Schwartz, 2007) in a case-crossover study. The results showed a greater relative increase in risk of death for cardiovascular causes, and cardiac arrest on the coldest days. A case-only analysis for the same 50 U.S. cities (Medina-Ramón et al., 2006) also found that cardiovascular deaths showed the greatest relative increase on extremely cold days. They also found that susceptibility to death

related to temperature extremes varied for different primary causes of death. The results suggest that even though there is a wide range of winter temperatures in the U.S., the population seems well acclimatized to typical winter temperatures in their regions. The authors speculated that this may be due to the near universality of central heating in the U.S.

In contrast to the studies discussed above, Basu et al. (2005) found null or negative associations between temperature and mortality in the elderly during winter, spring, and autumn in the 20 largest U.S. cities. This difference in results may be related to several limitations of the study. The study included data for only one year, and information on several possibly important confounders and effect modifiers was not available.

3.2.3.3 Findings from Time-Series and Case-Crossover Analyses of the Relationship between Cold Temperatures and Mortality — European Studies

Perhaps the most comprehensive population-based study of cold-related mortality is the Eurowinter survey of cold-related mortality and protective measures employed in seven regions of Europe (North and South Finland, Athens, Baden-Württemberg, the Netherlands, London, and North Italy). These studies (The Eurowinter Group 1997; Keatinge et al. 2000a; Keatinge and Donaldson 2001; Keatinge 2002) found that cold-related mortality is more frequent in areas with milder winters than in areas with more severe winters, in agreement with U.S. studies (Barnett 2007; Curriero et al. 2002, 2003), and Analitis et al. (2008) in a study of 15 European cities. This somewhat counterintuitive finding is thought to be due to the greater understanding of the risk posed by cold, and preparedness to deal with it, among people who live in areas that frequently experience very cold temperatures. Table 3 shows the percentage increase in all-cause mortality per 1°C (1.8°F) when temperature falls below 18°C (64°F). Risk of cold-related mortality increased with age (The Eurowinter Group 1997; Keatinge et al. 2000a; Cordioli et al. 2000).

Table 2. Cold-related mortality in the Eurowinter study

City	Average Winter Temp. (°C)	% Increase in Mortality per 1°C Decrease in Temp.
N. Finland	-2.8	0.29
S. Finland	-1.0	0.27
Baden-Württemberg	5.1	0.60
Netherlands	6.2	0.59
London	7.6	1.37
North Italy	7.7	0.51
Athens	12.7	2.15
Palermo	15.4	1.54

Source: The Eurowinter Group 1997.

Several studies have investigated cold-related mortality in London (Keatinge and Donaldson 2001; Hajat et al. 2007; Donaldson and Keatinge 2003b). Keatinge and Donaldson (2001) found that atypical patterns of prolonged cold weather were associated with episodes of elevated air pollution, which could give false indications of mortality associated with sulfur dioxide, carbon

monoxide, or smoke when common epidemiological modeling techniques are used. The results underscore the importance of including all weather variables and lags that could impact the temperature-mortality relationship. In addition, a more extensive lag structure needs to be considered for cold-related mortality than heat-related mortality, because cardiovascular mortality related to cold occurs within about three days of minimum temperature, while cold-related respiratory mortality is associated with a lag of up to several weeks. The analyses showed no net increase in cold-related mortality related to current sulfur dioxide, carbon monoxide, or smoke concentrations in London. Monitoring data for particulate matter of any size range were too sparse to analyze.

There appears to be a 2–3 day lag time between cold exposure and cardiac events (Cagle and Hubbard 2005), while the lag is longer for respiratory deaths. Many excess winter deaths are due to influenza and other respiratory infections, possibly due to reduced building ventilation in cold weather and to the greater tendency for people to congregate in confined spaces, which increases the opportunity for cross-infection, and is consistent with the longer lag time in the association between cold exposure and respiratory mortality.

Hajat et al. (2007) found a relative risk for cold-related mortality in England and Wales of 1.06 per 1°C (1.8°F) decrease in temperature below the cold-related mortality threshold. Elderly people, particularly those in nursing or other care homes were at greatest risk of cold-related mortality, with risk increasing with advancing age. Respiratory and external causes of death accounted for the largest fractions of cold-related deaths. Among the population less than 65 years of age, cold-related mortality was primarily for causes related to chronic disease, that were not directly attributable to ambient temperature. The relationship between cold and mortality was not modified by deprivation, except for rural areas where the relative risk of cold-related mortality was slightly higher for more deprived people. An analysis of the influence of social class in working and retired age groups (Donaldson and Keatinge 2003b) found that men working in lower status, unskilled jobs, which generally involved greater physical activity, were at lower risk of cold-related mortality than men working in higher status positions, although the opposite was found for women. Risk of cold-related mortality in retired people tended to be higher in the lower social class compared to higher social class groups.

Pattenden et al. (2003) compared cold-related mortality in London, England, and Sofia, Bulgaria. Mean winter temperature in London was 8°C (12°F), and in Sofia, 3.9°C (7°F). For each 1°C (1.8°F) decrease in two-week average temperature below the local tenth percentile temperature, there was a 4.24% increase in mortality in London, and a 1.83% increase in Sofia, even though Sofia was colder than London.

Cold-related mortality is also influenced by home factors and personal behaviors. The Eurowinter study found that cold-related mortality varied inversely with the effectiveness of measures taken to avoid both indoor and outdoor cold, including such factors as home heating, the extent to which people regularly wore protective clothing and exercised while outdoors (The Eurowinter Group 1997). For example, people living in Finland, the coldest country studied, had better heating in their homes, dressed more protectively when outdoors, and were more physically active when outdoors than people living in the countries with warmer winters. These findings, supported by a study performed in Yakutsk, eastern Siberia (Donaldson et al. 1998) which found no increase in mortality related to cold even though the mean winter temperature for the period 1989 to 1995 was -26.6°C (-16°F), support the conclusion that many

cold-related deaths can be prevented with adequate home heating and insulation and by wearing proper clothing when outdoors.

Carson et al. (2006) reported that cold-related mortality in London has decreased steadily since 1900 for all causes, cardiovascular diseases, respiratory conditions, and non-cardiorespiratory causes. In the period 1900 to 1910, about 12.5% of all deaths in London were attributable to cold, while by 1986 to 1996 the attributable fraction was 5.42%. The authors posit that this reduction is related to improved housing, including home heating and improved insulation. The authors also suggest a role for increased car ownership, climate-controlled public transportation and shopping facilities, and improved clothing fabrics.

3.2.3.4 Conclusions

- Cold-related mortality is higher in areas with milder winters.
- Cold-related deaths are primarily due to cardiovascular or respiratory causes.
- Elderly people are at greatest risk.
- Cardiovascular deaths have a lag time of about three days, while respiratory deaths (primarily pneumonia and influenza) have a lag time of up to two weeks.

3.3. Air Pollution-Related Health Effects

3.3.1. Background

Exposure to various air pollutants, including ozone, particulate matter, nitrogen dioxide, sulfur dioxide, and carbon monoxide, has been associated with a variety of adverse health effects (CARB 2000). In this section, the focus is not on the confounding impacts of air pollution on heat or cold-related mortality and morbidity (as discussed above), but on the direct impacts of air pollution on human health. Health impacts of biogenic air pollutants (e.g., allergens) are not discussed, though they have been recognized as important climate-sensitive air pollutants that do and will increasingly affect human health (e.g., Kinney 2008; Ziska et al. 2008; Mohan et al. 2006). Because the available scientific literature suggests that ozone and PM are responsible for most of the health effects associated with criteria air pollutants, this review focuses on these two pollutants (Bernard et al. 2001; CARB 2000, 2002, 2005b).

Attainment of health-based ambient air quality standards is generally achieved through regulations that control emissions. Over the past 30 years, considerable progress has been made toward attainment of these health-based standards; however, many areas of California continue to be in non-attainment of the standards. State Implementation Plans (SIPs) have been developed to bring California into attainment with the federal ambient air quality standards. The current planning documents are available at:
<http://www.arb.ca.gov/planning/sip/sip.htm>.

Historically, climate change is not considered in the process of setting ambient air quality standards because the standards are based on health effects that are unrelated to ambient temperature. However, since many atmospheric chemical reactions contributing to the production of ozone and PM progress more rapidly at higher temperature, it is likely that all other factors remaining the same, the concentrations of these pollutants will increase, and that climate change could have an effect on the attainment process. Without additional emissions

control measures, climate change could slow progress toward attainment of the ambient air quality standards and increase control costs. The National Research Council (2004) recommended that "the air quality management system will need to ensure that pollution reduction strategies remain effective as the climate changes, because some forms of air pollution, such as ground-level ozone, might be exacerbated."

3.3.2. Ozone

Ground-level ozone is a secondary air pollutant that primarily forms in the atmosphere through a complex series of photochemical reactions between nitrogen oxides and reactive hydrocarbons. In urban areas, the principal source of these precursors is motor vehicles, along with the fuel supply system that supports them, although vegetation can contribute significant amounts of reactive hydrocarbons. Because ozone formation is highly dependent on solar radiation to drive the photochemical reactions, significant concentrations of ozone typically appear only during May through October, and during daylight hours, although peak concentrations occur later in the day in downwind locations because of transport (USEPA 2006).

Collectively, the literature on controlled exposure of human subjects to ozone indicates that one- to three-hour exposures to ozone concentrations as low as 0.12 parts per million (ppm) with moderate to heavy exercise can induce decrements in pulmonary function and increases in respiratory and/or ventilatory symptoms for some subjects. Increased airways reactivity and inflammation have been reported with one- to three-hour exposures to 0.40 and 0.18 ppm ozone, respectively. Concerns about the impacts of longer averaging times led to studies using a protocol that simulates a day of active outdoor work or play. These studies demonstrate that statistically significant group mean decrements in lung function, increases in symptoms of respiratory and/or ventilatory irritation, and increased airways reactivity and airways inflammation can be induced in the most sensitive fraction of the population with 6.6 to 8-hour exposures to ozone concentrations as low as 0.08 ppm (CARB 2005b).

Animal toxicological studies have shown that chronic ozone exposure can induce morphological changes similar to those characteristic of chronic lung disease (Last et al. 1994; Reiser et al. 1987; Harkema et al. 1993). Studies in primates suggest that intermittent ozone challenges with periods of clean air alternated with ozone exposure can lead to greater lung injury than daily exposures to similar concentrations (Tyler et al. 1988), while similar intermittent exposures in juvenile monkeys has demonstrated alterations in lung development (Evans et al., 2003; Schelegle et al. 2003). These data provide a biologically plausible basis for considering that repeated inflammation associated with exposure to ozone over a lifetime may lead to chronic lung injury.

Epidemiologic studies have shown positive associations between short-term ozone levels and several health effects including hospitalization (e.g., Burnett et al. 1997; Anderson et al. 1997), emergency room visits for asthma (e.g., Tolbert et al. 2000), restrictions in activity (Ostro and Rothschild 1989), respiratory symptoms, particularly in asthmatics (e.g., Gent et al. 2003), and school absenteeism (Gilliland et al. 2001). Many of these findings are observed or studied only in the summer season, when ozone levels are usually highest. A growing body of data now suggests that ozone concentration may represent an independent risk factor for premature death (Dominici 2003; Bell et al. 2004, 2005; Levy et al. 2005; Gryparis et al. 2004; Ito et al. 2005).

Long-term ozone exposure has been associated with respiratory inflammation (Kinney et al. 1996), reduced lung function and respiratory symptoms (Kinney and Lippmann 2000), reduced growth of lung function in children (Galizia and Kinney 1999; Tager et al. 1998; Kunzli et al. 1997), and asthma prevalence (Abbey et al. 1999; McConnell et al. 2002). Individuals at greatest risk of experiencing adverse health effects from ozone exposure are those who spend prolonged periods of time outdoors while participating in activities that increase the breathing rate, since ozone concentrations indoors are typically considerably lower than those outdoors. Highly exposed groups include children, outdoor workers, and recreational and professional athletes.

3.3.3. Particulate Matter

Particulate matter (PM) is emitted by many sources, and the size and chemical composition of particles can vary considerably among particles from different sources. Particles smaller than 10 microns in diameter (PM_{10}) can be inhaled deeply into the lung, and there is particular concern about particles 2.5 microns or less in diameter ($PM_{2.5}$) (USEPA 2004). Some particles are directly emitted from combustion processes, such as through combustion of gasoline or diesel fuel by motor vehicles, or generation of electrical power through combustion of oil, natural gas, or coal. Particulate matter is also emitted through a variety of industrial processes. Natural sources of PM include soil and dust. Some particles form through chemical reactions in the atmosphere. Examples of these particles include sulfates, nitrates, and organic aerosols (Seinfeld and Pandis 1998; Finlayson-Pitts and Pitts 1999).

The literature on health effects of PM was most recently reviewed by the U.S. Environmental Protection Agency during review of the federal ambient air quality standards for PM (U.S. EPA 2004), and by ARB as part of the Goods Movement Reduction Plan (CARB, 2006). The majority of literature on the public health impacts of particulate matter comes from epidemiologic studies. This literature fairly consistently reports statistically significant associations between changes in PM_{10} and $PM_{2.5}$ concentrations and a range of adverse health outcomes, both on a daily and a long-term basis. Associations between daily and long-term average changes in both PM_{10} and $PM_{2.5}$ and mortality appear to be independent of the effect of weather factors, seasonality, time, and day of week (Dockery et al. 1993; Pope et al. 1995, 2002, 2004; Krewski et al. 2000; Burnett and Goldberg 2003; Fairley 2003; Ito 2003; Laden et al. 2006; Jerrett et al. 2005; Ostro et al., 2008, 2007, 2006). Mortality occurs primarily in elderly populations, but has also been reported for infants (Romieu et al. 2004; Diaz et al. 2004; Kaiser et al. 2004; Ha et al. 2003; Bobek and Leon 1999; Loomis et al. 1999; Woodruff et al. 1997). Studies over the past several years consistently report associations between PM_{10} and $PM_{2.5}$ and several different measures of hospitalization or urgent care for exacerbation of respiratory (i.e., chronic obstructive pulmonary disease, asthma) or cardiovascular diseases (i.e., congestive heart failure) (Atkinson et al. 2003; Zanobetti and Schwartz 2003; Sheppard 2003). These effects have been reported primarily among elderly individuals, but they have also been reported among all age groups, including children under age 18 (Ostro et al. 1999; Moolgavkar et al. 2000).

Data also suggest that long-term (i.e., months to years) PM exposures are associated with increased risk of mortality from cardiopulmonary causes (Dockery et al. 1993; Pope et al. 1995, 2002; Krewski et al., 2000; Laden et al. 2006; Jerrett et al. 2005). Associations with PM_{10} and $PM_{2.5}$ exposure have also been reported for chronic respiratory symptoms or disease, possibly decreased lung function in general (e.g., Ferris et al. 1973, Hodgkin et al. 1984; Mullahy and

Portney 1990), and with prenatal and lifetime exposure in asthmatic children (Mortimer et al. 2008).

3.3.4. Conclusions

- Both ozone and particulate matter have been associated with premature mortality and morbidity, including hospitalizations, emergency room visits, respiratory symptoms, school absences and work loss days.
- The most at-risk populations include the elderly, children, and people who are active outdoors, including outdoor workers and athletes.
- Federal and State ambient air quality standards are health-based, and are promulgated to protect public health. Because climate change will make attainment of ambient air quality standards more difficult, both federal and State actions to achieve attainment will need to continue. Consequently, the extent of future air pollution-related adverse health effects will be related to the extent to which attainment efforts succeed or fall short.

3.4. Wildfires and Public Health

3.4.1. Background

Wildfires can be a significant contributor to air pollution in both urban and rural areas, and they have the potential to significantly affect public health primarily through their smoke. Fires also affect the economy and public safety. Various climate change scenarios project that through the twenty-first century there will likely be an increase in the frequency, size, and intensity of wildfires (Westerling and Bryant 2006; Westerling et al., 2009). However, quantitative estimation of the public health impacts of future wildfire events is extremely difficult. The public health impacts of any fire are unique to that fire, and are influenced not only by the magnitude, intensity, and duration of the fire, but also the proximity of the smoke plume to a population.

Smoke from burning vegetation includes a large quantity of $PM_{2.5}$ and PM_{10} (Lee et al. 2008; Viswanathan et al. 2006). Schöllnberger et al. (2002) have shown that the particulate matter in wildfire smoke deposits throughout the respiratory tract, with deposition fractions of 54%–58% for particles less than 0.02 μ m in diameter, and 76%–78% for particles less than 10 μ m in diameter. Adult men and women had total deposition efficiencies at the lower end, and newborn infants had deposition efficiencies at the upper end of these ranges. Wildfire smoke impacts on public health can reasonably be inferred from the extensive literature on the health impacts of $PM_{2.5}$ and PM_{10} , which was discussed above in the section on air pollution-related health effects.

3.4.2. Methodological Challenges

Because wildfires are relatively rare, and most frequently occur in sparsely inhabited areas, there are few studies evaluating the public health impacts of wildfires. Assessment of the public health impacts of wildfires is also complicated by the fact that there are usually few, if any, air quality data available for fire-impacted regions. Since this precludes development of exposure assessments and concentration-response functions, analyses of public health impacts of wildfires are generally limited to retrospective assessments of the change in incidence of health endpoints during the fire compared to that observed during a baseline time period.

The largest group of studies has examined changes in emergency room visits or self-reported symptoms. However, because the decision to visit an emergency room is influenced by individual perceptions of risk and a decision to seek medical care is not necessarily based on objective assessment of medical need, these results are somewhat subjective. In addition, psychosomatic stress associated with wildfires and resulting air quality emergencies also can influence the decision to visit an emergency room (Mott et al. 2005; Lipsett et al. 1994). There are few studies of more objective and severe health endpoints, such as hospitalization and mortality, primarily because population densities in exposed communities are rarely large enough to yield sufficient sample sizes for assessment of epidemiologic relationships between poor air quality due to fires and health endpoints (Vedal and Dutton 2006), and because adequate air quality data are generally not available (Mott et al. 2005).

3.4.2.1 Wildfires and Mortality

There have been few investigations into the impact of wildfire smoke on mortality (non-burn-related). While not directly similar to the sort of fires that occur in California, two reports on a series of fires in Southeast Asia in 1997 suggest a combination of factors that created a particularly severe and long lasting smoke exposure scenario. The few air quality data available suggest that PM₁₀ levels varied widely during the fire period. Many days had typical PM₁₀ concentrations, but there were days on which PM₁₀ concentrations reached alarming levels. Between August and early November 1997, Singapore air quality reached the unhealthy range, based on the U.S. Environmental Protection Agency Pollutant Standards Index (PSI; values over 100 are considered unhealthy) on 12 days, with the highest PSI recorded being 138. Monthly average PM₁₀ values, typically between 30 and 50 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$), increased significantly to 60 to 100 $\mu\text{g}/\text{m}^3$ during September and October 1997 (Emmanuel 2000), although there was no significant change in mortality with an increase in PM₁₀ from 50 to 150 $\mu\text{g}/\text{m}^3$. Sastry (2002) found a 19% increase in mortality ($P < 0.05$) in Kuala Lumpur, Malaysia on the day following days with 24-hour average PM₁₀ measurements above 210 $\mu\text{g}/\text{m}^3$ in people 65 to 74 years of age, but not in other age groups at any PM₁₀ concentration. The ninetieth percentile PM₁₀ value was 99.4 $\mu\text{g}/\text{m}^3$. The study estimated a relative risk of mortality with a 100 $\mu\text{g}/\text{m}^3$ increase in PM₁₀ of 1.07. The results also suggested that displacement of deaths from the exposure to the smoke haze was short lived, although for those aged 65 to 74 there was an upward shift in death rate that lasted for a few weeks after the fires ended. Overall, the study found that mortality burden, in terms of days of life lost, was small.

Vedal and Dutton (2006) evaluated the relationship between smoke, measured as PM_{2.5}, and mortality consequent to a wildfire near Denver, Colorado (Denver metropolitan area population was about two million at the time of the study). On two days in June 2002 peak one-hour PM₁₀ and PM_{2.5} concentrations reached 372 $\mu\text{g}/\text{m}^3$ and 200 $\mu\text{g}/\text{m}^3$, respectively on June 9th, and 316 $\mu\text{g}/\text{m}^3$ and 200 $\mu\text{g}/\text{m}^3$, respectively, on June 18th, but there was no evidence for an increase in daily mortality attributable to these fire-related increases in PM₁₀ or PM_{2.5} level. Although not a wildfire, Lipsett et al.'s (1994) found no observable increase in respiratory mortality related to a supermarket warehouse fire that occurred in Richmond, California, in July 1988. Although not the usual sort of wildfire, these results are relevant, in that wildfires in California often spread into urban areas, adding combustion emissions to the smoke plume from building materials and household and consumer goods to those of vegetation. Analysis of coroner's records following an urban wildfire that burned parts of Alameda County, California, in late October

1991 (Shusterman et al.1993), found 25 fire-related deaths, all principally due to extensive burns, although many patients also had smoke inhalation injury.

3.4.2.2 Wildfires and Morbidity

Several studies of the impacts of a series of fires in 1997 in Southeast Asia have reported on morbidity endpoints, including increased respiratory symptoms, upper respiratory tract illnesses, and hospitalizations. As noted above, few air quality monitoring data were recorded during the fires PM₁₀ levels varied widely during the fire period, and reached very high levels on some days. Emmanuel (2000) found that an increase in PM₁₀ from 50 to 150 g/m³ was associated with a 12% increase in upper respiratory tract illness, a 19% increase in asthma exacerbation, and a 26% increase in rhinitis. However, these increases were not coupled with a significant increase in hospital admissions. In contrast, Mott et al. (2005) found an 8% increase in hospitalization rate for all causes compared to baseline, with the greatest increase for respiratory hospitalizations between August 1 and October 31, 1997, in the Kuching region of the Malaysian state of Sarawak. The number of persons over 65 years of age readmitted to the hospital during the fire period was significantly higher than expected, and re-hospitalization rates returned to the pre-fire level once the fires ended.

Several studies have investigated the public health impacts of wildfires in the western United States. Vedal (2003) reported an increase in the mean number of daily emergency room visits for respiratory causes on two days in June of 2002 when peak one-hour PM_{2.5} concentrations reached 352 and 390 g/m³, consequent to a wildfire near Denver, Colorado. Morris et al. (2003) found that those who were not evacuated from the area affected by smoke from the June 2002 Rodeo-Chedeski fire in Arizona reported significantly more respiratory symptoms than people who were evacuated. The most commonly reported symptoms were eye irritation, itchy sore throat, and cough. Prevalence of self-reported asthma exacerbation increased 86% among people living in the non-evacuated area, compared to 39% among evacuees. Sutherland (2005) followed a small panel of chronic obstructive pulmonary disease (COPD) patients living in Denver during the 2002 fire. There was a small increase in symptom scores on the days with the highest PM_{2.5} concentrations, followed by return to baseline on the following, lower PM_{2.5} day. Unfortunately, there were no objective measures of lung function, and since lung function changes and symptoms are not well correlated in COPD, the clinical and functional significance of the small increase in symptoms is unknown.

Several studies have investigated morbidity related to fires in California. Duclos et al. (1990) assessed the impact of a series of fires in late summer of 1987 that burned over 600,000 acres of California forests. There was a 40% increase above expected in the number of emergency room visits for asthma (120 vs. 86) and a 30% increase in visits for chronic obstructive pulmonary disease (74 vs. 57) during the period with the highest smoke levels. Analysis of emergency room visits to nine local hospitals during the two days of an urban wildfire that burned parts of Alameda County, California, in late October 1991 (Shusterman et al.1993) showed that smoke inhalation was the principal cause of about one-half of the emergency room visits during the fire. Lipsett et al. (1994) also reported an increase in emergency room visits for asthma and other lower respiratory conditions and for respiratory-related hospitalizations during a supermarket warehouse fire that occurred in Richmond, California, in July 1988.

In October of 2003 a series of wild fires burned a large portion of Southern California, including several Children's Health Study sites, providing a serendipitous opportunity to investigate the impact of wildfire smoke on the respiratory health of children (Künzli et al. 2006). Exposure to wildfire smoke was associated with increased eye and respiratory symptoms, medication use by asthmatics, and unscheduled physician visits. Symptoms were more severe with both higher levels of smoke and more smoky days. Surprisingly, the associations were strongest for children without asthma. Children with asthma were more likely to take preventive action, likely explaining the lower level of adverse effects in the asthmatic compared to the non-asthmatic children. This is the first study to report benefits of preventive actions, including remaining indoors, reducing physical activity, using air conditioning, and wearing masks when outdoors.

In 1999 a large wildfire burned from August 23 to November 3 near the Hoopa Valley National Indian Reservation in Northern California. On 15 days, the PM₁₀ concentration exceeded the U.S. Environmental Protection Agency (U.S. EPA) 24-hour standard of 150 g/m³, and on October 21 and 22, PM₁₀ levels exceeded the U.S. EPA hazardous level of 500 g/m³. A survey of 26% of tribal households showed a 52% increase in medical visits for respiratory problems during the fire, compared to the same period of 1998. More than 60% of those surveyed reported an increase in respiratory symptoms during the smoke episode, and 20% continued to report increased respiratory symptoms two weeks after the smoke cleared (Mott et al. 2002).

3.4.3. Conclusions:

- The majority of studies of the health effects of wildfires focus on respiratory symptoms and emergency room visits.
- Since fire smoke produces particulate matter in the PM_{2.5} size range, health effects would be expected to be comparable to similar concentrations of PM_{2.5}, with similar exposure durations.
- Exposures to fire smoke for up to a few days induce respiratory and ocular symptoms, but the limited data available suggest that few serious effects would be expected.

3.5. Infectious Diseases

3.5.1. Water-Borne Diseases

Available data show that meteorological factors, such as ambient temperature and precipitation, can influence the occurrence of outbreaks of water-borne diseases. Water quality and safety concerns apply not only to the drinking water supply, but also to irrigation and recreational waters, since all can be sources of human illness due to water-borne pathogens (Lipp et al. 2002; Hunter 2003; Rose et al. 2000). Exposure to these pathogens can occur through the drinking water supply, with failure at various points throughout the delivery system, and through contact with contaminated ground or surface water (Liang et al. 2006). Water-borne diseases can be acquired through ingestion, or by physical contact with contaminated water.

Historical data link many outbreaks of water-borne disease to water contamination subsequent to runoff from heavy rainfall, flooding, and/or sewage overflow (Curriero et al. 2001, 2003; Charron et al. 2004; Colwell and Patz 1998; Louis et al. 2003; Auld et al. 2004; Lee et al. 2002; Rose et al. 2000). For example, there was a doubling in the rate of gastrointestinal illness following floods related to Hurricane Floyd among residents in the eastern part of North

Carolina (Setzer and Domino, 2004). Infections were linked to pathogens originating from the high concentration of hog farms in the area, and illustrate how widespread flooding can spread disease-causing pathogens to downstream areas. Another illustration of the extent to which flooding can lead to serious disease outbreak occurred in Walkerton, Ontario in May 2000 (Auld et al. 2004; Hrudehy et al. 2003). Rainfall, accompanied by record high temperatures, exceeded the 60 to 100 year event marks in the region surrounding this small town. Almost half of the local population became seriously ill, and seven died from ingestion of *E. Coli* and *Campylobacter* contaminated drinking water. Wade et al. (2004) found that contact with floodwater from the Mississippi River during a severe flooding event in 2001 was related to gastrointestinal symptoms and illness, although there was no relationship between tap water consumption and illness.

Flooding can also contaminate ground water, in addition to surface waters, leading to outbreaks of water-borne disease (Liang et al 2006). For example, groundwater can become tainted through contamination of the surface waters that feed the aquifers, as well as through leakage from septic systems, sewage overflows, and dumps. Contamination can also occur after water treatment if treatment is insufficient for the pathogen load, or if the water delivery system is breached (Rose et al. 2001). Irrigation water can become contaminated by similar sources and from flooding and storm runoff, particularly if the runoff contains livestock excrement (King and Monis 2006).

The most common water-borne illness is gastroenteritis, followed by dermatitis. These diseases are under-reported, and the majority of cases are self-limited and do not come to medical attention. However, a fraction of cases require medical attention or hospitalization, and there have been some deaths related to water-borne pathogens reported in the United States (Dzuiban et al. 2006; Lee et al. 2002; Liang et al. 2006).

The most commonly isolated causal agent in drinking water has been the bacterium *Cryptosporidium*, although a significant number of cases have also been associated with various *Vibrio* species, and giardia (Dzuiban et al. 2006; Furness et al. 2000). Liang et al. (2006) have reported a significant number of cases due to *Legionella* spp. associated with water that was contaminated in the drinking water distribution system.

Outbreaks of gastroenteritis associated with contaminated recreational waters have been commonly associated with *Cryptosporidium parvum* in treated recreational water such as swimming pools, and *E.Coli* in freshwater venues (Lee et al. 2002). Several bacteria, viruses and parasites, including *Campylobacter* (Dzuiban et al. 2006), *Cryptosporidium* (Lee et al. 2002), *Vibrio* species (e.g., Morris, 2003; Randa et al. 2004; Dzuiban et al. 2006; Furness et al. 2000), leptospirosis (Meites et al. 2004), and the amoeboflagellate *Naegleria fowleri* (Marciano-Cabral et al. 2003) have been linked to outbreaks of waterborne diseases associated with untreated recreational water.

Leptospirosis has recently been identified as a re-emerging zoonosis, after being eliminated from the list of reportable diseases in 1995 (Katz et al. 2002). Hawaii has consistently had the highest incidence rate, with cases predominantly in males. Cases related to occupational exposure have declined in recent years, while cases related to recreational water have increased.

Water-borne illnesses are more common during the warmer months of the year (Dzuiban et al. 2006; Furness et al. 2000; Janda et al. 1988), suggesting a link to ambient temperature. Several studies have evaluated the influence of water characteristics on *Vibrio* species. High concentrations of these species and outbreaks of water-borne disease related to *Vibrio* species are more frequently associated with warmer water (Janda et al. 1988; Pfeffer et al. 2003; Randa et al. 2004; Thompson et al. 2004) and lower water salinity (Lipp et al. 2002; Louis et al. 2003; Randa et al. 2004), turbidity (Pfeffer et al. 2003), and level of dissolved oxygen (Pfeffer et al. 2003). *Campylobacter* infections from non-food sources have also been found to correlate with temperature (Louis et al. 2005). Environmental factors have also been associated with survival of *Cryptosporidium* oocysts (King and Monis 2006).

At-risk populations identified include the immunocompromised exposed to contaminated surface water (King and Monis, 2006), children and adults between 31 and 40 exposed to recreational water contaminated with giardia (Furness et al. 2000), children exposed to flood waters (Wade et al. 2004). Although not specifically addressed by the literature cited above, children and the elderly are more likely to require hospitalization for gastroenteritis, regardless of the cause, than other sub-populations (Charles et al. 2006; Gangarosa et al. 1992; Louis et al. 2005).

3.5.2. Food-Borne Diseases

Food-borne illnesses are a significant public health concern worldwide. These illnesses can be caused by a variety of microorganisms, including the pathogens discussed above under water-borne diseases. Other pathogens related to food-borne diseases include *Listeria*, *Salmonella*, *E. Coli*, *Shigella*, and *Yersinia* (Vugia et al. 2006). These pathogens typically cause gastroenteritis (Lynch et al. 2006). Many cases of food-borne disease are mild and self-limited, but food-borne illnesses cause a significant number of hospitalizations and deaths annually (Mead et al. 1999).

Several studies have found that ambient temperature influences the number of food-borne disease reports. D'Souza et al. (2004) has reported that seasonal patterns in salmonellosis reports can be explained by changes in temperature. The results also suggest that elevated temperature during earlier phases of the food production pathway may have a greater influence on whether or not food becomes infected with salmonella than at the food preparation phase. A similar linear association between temperature above 6°C and reported cases of salmonellosis has been reported for a number of European countries, with the strongest association for adults 15 to 64 years of age (Kovats et al. 2004), and for temperature one week before the onset of illness. Kovats et al. (2005) have also investigated the influence of ambient temperature on campylobacter infection. The results of this analysis indicated a distinct seasonality in campylobacter transmission, along with geographical variation in the time of seasonal peak activity that suggests that climate may be a contributing factor in disease transmission. Zhuang et al. (1995) have reported that the population of *Salmonella* Montevideo on tomatoes, both whole and chopped, increased significantly as storage temperature increased.

Seafood caught in waters infected with various species of *Vibrio* is a fairly common source of food-borne illness, in fact, seafood ranked third on the list of causes of food-borne disease in the U.S. between 1983 and 1992 (Lipp and Rose 1997). Raw oysters are one of the more common sources of *Vibrio* infection, and water temperature has been related to oyster contamination and subsequent disease (Morris 2003). Since 1997 mean Alaskan sea water temperature has

increased, leading to a significant outbreak of *Vibrio* poisoning that was traced to Alaskan oysters that had been harvested when mean daily water temperatures exceeded 15°C (McLaughlin et al. 2005). Several viruses associated with fecal contamination, and toxins related to toxic algal blooms have also been responsible for outbreaks of seafood-related food-borne illness (Lipp and Rose 1997).

3.5.3. Other Infectious Diseases

Several other infectious diseases that are spread by oral-fecal or respiratory routes have patterns of seasonality.

Diarrheal illness is common in the United States, with estimates of approximately 200 million episodes each year (Herikstad et al. 2002). Gastroenteritis associated with rotaviruses is primarily a winter disease in temperate climates (Charles et al. 2006; Cook et al. 1990). Fleury et al. (2006) have reported a strong non-linear association between ambient temperature and occurrence of *Salmonella*, *E. Coli* and *Campylobacter* infections in Alberta, Canada, and *Campylobacter* in Newfoundland-Labrador. Enteroviruses, including the family of echo viruses and coxsackie viruses, also have a seasonal pattern of infection; the greatest number of confirmed cases occurring between June and October (Khetsuriani et al. 2006). There appears to be a two to fourteen day lag between peak ambient temperature and peak incidence of *Campylobacter* and *Salmonella* infection, while *Cryptosporidium*, *Shigella* and *Giardia* infections peaked about 40 days relative to peak temperature (Naumova et al. 2006). Risk of hospitalization and death related to diarrheal disease is widely recognized for young children. However, the risk to the elderly, among whom the case-fatality ratio is higher than for children, has not been widely appreciated (Gangarosa et al. 1992).

Several respiratory diseases also have distinct seasonality. Several studies have investigated the influence of season on influenza. Although the influenza season is typically during the colder part of the year, cold weather alone does not predict influenza deaths (Dushoff et al. 2005).

Several papers have investigated the influence of El Niño on respiratory diseases. Both the circulating subtype and magnitude of the El Niño Southern Oscillation (ENSO) are associated with the impact of influenza epidemics (Viboud et al. 2004). Choi et al. (2006), in an analysis focused in California, found that mean influenza-related mortality was lower during non-ENSO periods, compared to El Niño periods. An analysis of the influence of El Niño on hospitalization for viral pneumonia in females in six California counties (Ebi et al. 2001) found that temperature had an inconsistent relationship with hospitalization among the various counties. There was a significant increase in hospitalizations in San Francisco and Los Angeles with increasing temperature, with the association independent of season. The results also suggested that viral pneumonia could continue to be a public health issue as climate warms. El Niño was only associated with hospitalizations in Sacramento, with a significant decrease for girls, and an increase for women.

Two papers have investigated the influence of climate conditions on incidence of valley fever, which is endemic to arid regions of the Western Hemisphere, and is caused by a soil-dwelling fungus. Antecedent temperature and precipitation in are important predictors of incidence (Kolivras and Comrie 2003). The results suggested that temperature and precipitation in the winter season lagged by a year or more were most predictive of incidence. In contrast,

Talamantes et al. (2007) found that fluctuations in incidence of valley fever were related to biological and/or anthropogenic factors, and only to a small extent to weather anomalies.

3.5.4. Vector-Borne Diseases

Vector-borne diseases are caused by a variety of viruses, bacteria, and protozoa that spend part of their life cycle in a host species (mosquitoes, fleas, and ticks) and are spread to humans and animals during insect feeding. Although twelve mosquito-borne diseases are known to occur in California, only Western Equine Encephalitis (WEE), West Nile Virus (WNV), and St. Louis Encephalitis Virus (SLV) are significant causes of human disease. Currently, West Nile Virus is the most important in California. These diseases are maintained through a cycle that depends of wild birds and mosquitoes, with humans as an incidental host.

The primary mosquito vector in California appears to be *Culex tarsalis*, although other sub-species of mosquitoes can also carry these diseases (California Department of Health Services 2004). The *Culex* family of mosquitoes is unique in that it is urban dwelling and prefers to breed in foul water such as that found in storm drains and catch basins (Epstein 2001). Consequently, drought tends to increase the richness of rotting organic material in storm drains, improving the mosquito habitat, while heavy rains flush the drains, and reduce the quality of the habitat leading to reduction in vector survival. Reisen et al. (2006) has reported that warm temperatures are necessary for virus development, that the zero virus development level is approximately 14.3°C, and that during the epidemic summers of 2002 to 2004, WNV dispersal and epicenters were closely linked to above average summer temperatures. An investigation of the influence of weather parameters on mosquito abundance and WEE transmission in Kern County California showed that the water content of snow in the Sierra Nevada during winter was correlated with spring river runoff, mosquito abundance and WEE activity in the San Joaquin Valley (Wegbreit and Reisen 2000). The strongest predictor of host-seeking females collected per trap night per month during summer was river runoff one month earlier.

DeGaetano (2005) found that between June and August, climatological conditions accounted for between 40% and 50% of the variation in the number of trapped *Culex* mosquitoes in two metropolitan New Jersey counties. In central Illinois, the *Culex pipiens* mosquito is the principal carrier of West Nile virus, although *Culex restuans* Theobald is also endemic (Kunkel et al. 2006). The time at which the two species of *Culex* are in equal abundance (crossover) marks the beginning of an increase in infections. This crossover time varies from year to year, and is influenced by temperature, in that warmer temperatures are correlated with earlier crossover dates.

The most common tick-borne disease in the United States is Lyme disease, caused by the bacteria *Borrelia burgdorferi*, and transmitted in the western United States by the *Ixodes pacificus* tick. Seventy-six cases of Lyme disease were reported in California in 2007, covering 23 counties (CDPH 2007). The primary hosts for this disease are deer and mice, and risk of infection increases in proportion to the populations of each (Gubler et al. 2001). Lindgren et al. (2000) reported a northern shift in the distribution of ticks related to fewer very cold days during the winter season in Sweden. Although the ticks studied are not of the same species as that seen in California, the results of the analysis support the notion that a vector's geographical range can alter with changing ambient temperature and humidity, as well the availability of suitable host

species (i.e., deer and mice), which can also be affected by climate and weather conditions (Gubler et al. 2001).

A series of studies by Ogden et al. (2004; 2005; 2006a; 2006b) investigated the influence of temperature and other weather-related factors on development of *Ixodes scapularis* ticks, and the potential for them to spread into currently non-endemic regions of Canada. Although this species of tick is not found in California, where Lyme disease is spread by the *Ixodes pacificus* tick, the findings of this series of studies are illustrative of the complex interaction between the life cycle of ticks and environmental factors. Ogden et al. (2004) found that while temperature affected several developmental phases of the tick life cycle, other phases were more dependent on availability of a suitable host for obtaining a blood meal or on length of daylight. It was also clear that at temperatures above 30° C few female ticks produced viable eggs. Ogden et al. (2005) found that, based solely on temperature, it was possible to apply a dynamic population model and predict areas of Canada that were conducive to establishment of tick populations. It is important to note that not all areas that met the temperature criteria for supporting tick populations in fact had tick populations, and that factors other than temperature affect tick viability and whether or not tick populations can be sustained or become established.

Ticks over-winter and rest while seeking hosts during warmer seasons in vegetation litter. Since ticks require sufficient humidity for survival, vegetation litter serves as a refuge from heat and dehydration. Ogden et al. (2006a) compared tick survival at several sites in south-eastern Canada that differed by vegetation type. The results suggested that this tick species will be able to establish itself in some new types of habitats, provided that there is a suitable density of hosts for the ticks (i.e., rodents, and deer). In addition, a theoretical investigation of the potential for ticks to expand their range in response to climate change (Ogden et al. 2006b) found that a northward range expansion was likely to be evident by 2020. One uncertainty in this modeling exercise is that rainfall was not included in the model, and this factor affects whether or not humidity and available moisture are sufficient to support the life cycle of ticks. In addition, some climate change scenarios suggest changes in rainfall pattern toward alternating periods of drought and heavy rainfall, which would likely reduce tick survival and range expansion.

Brownstein et al. (2003) developed a model to predict areas that could support established populations of *Ixodes scapularis* in the United States based on habitat suitability. The results showed that maximum, minimum and mean temperatures, along with vapor pressure all significantly contributed to tick population maintenance, although not all areas that were suitable based on habitat alone had resident tick populations. The resulting probability map was linked to a climate change model to estimate areas that will likely become suitable habitat areas for this species of tick in the future as climate warms. The model predicted that much of the United States will become potentially suitable habitat for this species of tick. It remains important to remember, however, that other factors, including availability of a suitable host species are required to support a tick population.

A study of the relationship between precipitation and occurrence of Lyme disease in the north eastern U.S. found that late spring/early summer precipitation was a significant climate-related factor affecting occurrence of Lyme disease (McCabe and Bunnell 2004). Ostfeld et al. (2006) found that the strongest predictors of current year risk of Lyme disease were the previous year's abundance of mice and chipmunks, and the abundance of acorns two years previously. Inclusion of meteorological factors or deer did not improve the predictions. These results

suggest that risk is related to prior abundance of hosts for the immature stages of the tick vector, and to availability of food for these hosts. Significant correlations between Lyme disease incidence and June moisture index two years previously and warmer winter weather a year and a half previous have been reported for the northeastern U.S. (Subak 2003). These factors may lead to higher survival of the important vector host, the white-footed mouse.

3.5.5. Rodent-Borne Diseases

Rodent-borne diseases are usually transmitted to humans through direct contact with rodent urine, feces or other body fluids. The most well-known rodent-borne human disease in the Southwest United States is hanta virus, although rodents are also hosts for fleas and ticks that can spread plague and several other infectious diseases (McMichael and Githeko 2001). The potential for human infection with hanta virus appears to be primarily related to the size of the rodent population. The impact of climate change on rodent-borne diseases is likely to be related to relatively short cycles, for example a year or two rather than longer-term, and based primarily on the availability of food, and consequently the rodent population (Gubler et al. 2001). There have been 49 cases of hanta virus cardiopulmonary syndrome in California since 1993 (CDPH 2008).

3.5.6. Conclusions

- Incidence of water-borne infectious diseases is influenced by the amount of precipitation, and storm intensity. These diseases are associated with contaminated runoff due to heavy precipitation and subsequent flooding and sewage overflow.
- The influence of climate and meteorology on vector-borne diseases is multifactorial. There are multiple points in the life cycles of the vector, the infectious agent, and host species that are potentially sensitive to local meteorology, and integration of the responses of each factor to climate change will determine the extent to which the ranges of these diseases alter.

3.6. Climate Change and Public Health

3.6.1. Future Estimates of Heat-Related Mortality and Morbidity

While numerous studies project that extreme heat conditions will increase with climate change in the future, and conjecture that this will increase the risk to human health and likely heat-related mortality (e.g., Karl et al., 2009), there are very few published studies that have attempted to make quantitative estimates of the likely future effects of increasing temperature on heat mortality. Two of these were done in California. The first, Hayhoe et al. (2004; Union of Concerned Scientists 2004) found that several different general circulation models employing several emissions scenarios pointed to the conclusion that there will likely be more hot days in the future, although not necessarily hotter temperatures on each day, under each scenario, along with an increase in the number and intensity of heat waves.

The second analysis, a part of the 2006 scenarios analysis project (Drechsler et al. 2006) focused on five California cities (Los Angeles, San Francisco, Fresno, San Bernardino, and Sacramento). The analysis concluded that climate change will likely result in a significant increase in the number of days with temperatures exceeding the ninetieth percentile of the historical temperature distribution (T90 day). All emissions scenarios (A1fi-high emissions, A2-medium-high emissions, and B1-lower emissions; IPCC 2007) and climate models (HadCM3, GFDL2.1,

and PCM)¹ point to longer, more intense, and more frequent heat waves, with the magnitude of the changes correlated with emissions; that is, scenarios based on higher greenhouse gas emissions predict greater increases than those with lower emissions, as illustrated in Figure 1 (Drechsler et al. 2006). By mid-century (2035–2064), temperatures currently seen on only 10% (or ~36 days) of the year will be exceeded on average 1.5 to 2.5 times more frequently each year. By the end of the century, extreme temperatures will be exceeded up to 4 times their current frequency under the high emissions scenario.

The frequency, length, and intensity of individual heat waves, as well as the duration of the entire heat wave season, are also projected to increase for all five cities studied. Inter-scenario differences are evident by mid-century, with significantly greater increases by century's end under the higher emissions (A1fi/A2) scenarios and for the more sensitive climate change models (GFDL and HadCM3). Results indicate large increases in the projected heat intensity, as compared to historical averages.

The analysis suggests that heat-related mortality with the high emissions scenarios (A1fi/A2) is likely to be almost double that for the lower emissions scenario (B1) by the end of the century. Unacclimatized mid-century projections show increases of 1.5 to 3 times the 1971–2000 values under B1 and 2 to 4.5 times under A1fi/A2, while end-of-century increases range from 2 to 4 times the historical values for each city for B1 and 3.5 to 9.5 times for A1fi/A2. The greatest overall increase in the heat-related mortality rate (per 100,000 people) is projected for Los Angeles. Since it is also the city with the largest population it is also projected to experience the greatest absolute impact as well. Overall, acclimatization is most successful at reducing projected mortality rates for inland cities that already experience extended periods of extreme heat and smallest for coastal cities where cool air masses off the ocean prevent consistent acclimatization to hotter conditions. For example, acclimatization reduced the mortality rates projected for Sacramento by 40% and for Fresno by 75%.

The only other U.S. study that has estimated heat-related mortality related to future climate change focuses on the New York metropolitan area. Knowlton et al. (2007) investigated the impact of projected regional increases in heat-related premature mortality to the year 2050. The results suggest an increase between 47% and 95% compared to the 1990s, without adaptations. The magnitude of the increase varied considerably across the region, and it was greater in urban counties than in less-urbanized counties. Inclusion of adaptations in the models reduced regional increases in summer heat-related premature mortality by about 25%.

It is important to note that demographic changes, societal decisions affecting implementation of adaptation measures, and changes in the health care sector will determine actual mortality rates. However, model uncertainties notwithstanding, extreme heat and associated human health risks under the lower-emissions scenario are significantly less than under higher-emissions scenarios. It is also important to note that increases in summer mean temperatures and the frequency, intensity, and duration of extreme heat events have significant implications for energy demand, particularly for additional electricity needed to support increased penetration of air conditioning in parts of the state where it is currently uncommon.

¹ The Hadley Centre Coupled Model, version 3; Geophysical Fluid Dynamics Laboratory model, version 2.1; and Parallel Climate Model.

There have been no projections of future morbidity related to increasing ambient temperatures related to climate change for any area of the United States.

3.6.2. Future Estimates of Cold-Related Mortality and Morbidity

Similar to the generic statements about heat-related health risks, general conjectures about declining cold-related diseases related to projections of increasing winter temperatures abound, but the knowledge base from research directly examining future cold-related mortality is slim indeed. There have been no projections of cold-related mortality or morbidity related to the projected shift in the range of ambient temperatures related to future climate change in the U.S.

3.6.3. Implications of Increasing Ambient Temperature on Air Quality

Rising temperature will increase the rate at which atmospheric chemical reactions proceed, increasing concentrations of ozone and possibly PM. However, relative humidity, wind speed, and mixing height also interact with temperature to affect the resulting pollutant concentrations (Drechsler et al. 2006; Steiner et al., 2006; Kleeman 2008; Mahmud et al. 2008; Jacobson, 2008; Millstein and Harley, 2009). These analyses also indicate that changes in the concentrations of ozone and PM are unlikely to be uniform across an air basin, making a health impacts analysis based on a simple incremental approach inappropriate. To date, air quality projections for California are limited to investigating the influence of perturbation of meteorological parameters on data from a few high air pollution episodes in the South Coast Air Basin and the San Joaquin Valley. These episodes lasted only a few days. While these analyses have provided important information as to the influence of meteorological parameters on air quality, the data are insufficient for projecting future health impacts.

Only one paper has attempted to quantitatively estimate the impact of future increases in ozone concentrations on mortality, and none have been published on PM. Knowlton et al. (2004) estimated a 4.5% increase in ozone-related mortality for the summer season in the New York metropolitan area based on modeled regional ozone concentrations using projected ozone precursor emissions with several climate change scenarios. The models, however, did not investigate whether ozone concentrations confounded or modified the temperature-mortality relationship. However, modeling studies investigating the influence of climate change on ozone concentrations suggest that without implementation of new ozone control measures mortality related to ozone could increase in the future, while the influence of climate change on future PM concentrations is less certain (Millstein & Harley, 2009; Drechsler et al. 2006).

California is taking steps toward attaining the existing ambient air quality standards for ozone and PM. Although limited, the information available on the influence of climate change on ozone and PM makes it clear that increasing temperature will make attainment of these standards more difficult in the future (Millstein & Harley, 2009; Drechsler et al. 2006). Since health impacts attributable to air pollution are related to ambient concentrations, the magnitude of health impacts attributable to ozone and PM in the future will be proportional to the degree to which ambient air quality standards are not attained.

3.6.4. Implications of increasing Ambient Temperatures on Wildfires

Wildfires can be a significant contributor to air pollution in both urban and rural areas, and they have the potential to significantly affect public health primarily through their smoke. Fires also affect the economy and public safety. Various climate change scenarios project that through the

twenty-first century there will likely be an increase in the frequency, size, and intensity of wildfires (Westerling et al. 2009). However, quantitative estimation of the public health impacts of future wildfire events is extremely difficult for several reasons. The public health impacts of any fire are unique to that fire, and are influenced not only by the magnitude, intensity, and duration of the fire, but also the proximity of the smoke plume to a population. Predictive assessments are further complicated by the fact that there are usually few, if any, air quality data available during fires other than those from the ambient air quality monitoring network, which are often not representative of air quality in the fire-impacted area. In addition, population densities in exposed communities are rarely large enough to yield sufficient sample sizes for assessment of epidemiologic relationships between poor air quality due to fires and health endpoints.

3.6.5. Implications of increasing Ambient Temperature on Infectious Diseases

Meteorological changes can influence human disease through both direct and indirect effects on pathogenic microorganisms, vectors, reservoirs, and hosts (Colwell and Patz 1998). Available evidence suggests that the incidence and spread of a number of infectious diseases can be affected by various weather-related factors on a time scale of a few years, and that climate change has the potential to affect their range, incidence and spread (Colwell and Patz 1998).

Most research to date on the impacts of climate change on infectious diseases has focused on short-term changes in weather patterns, primarily in rainfall, humidity, and ambient temperature, as opposed to long-term changes related to global climate change, largely because of the significant influence of relatively short-term weather patterns on the ecology and range of pathogens. The interactions between host and infectious organisms are complex, and the impact of climate on the ecology of infectious diseases increases that complexity, making it difficult to predict changes that may result from climate change. However, on a global scale, there is concern that rising temperatures may encourage the growth of infectious organisms, and thus increase global disease burden (Rose et al. 2001). Global travel may also increase the risk of introducing infectious diseases that are not currently endemic to California.

A National Research Council report (2001) that reviewed the relationships among climate, ecosystems and infectious diseases made several conclusions, including:

- Changes in weather, in both the short and longer terms can influence infectious diseases.
- Observational and modeling studies must be interpreted cautiously because they are unable to fully account for all of the factors that influence infectious diseases.
- Climate change may alter the evolution and ranges for infectious diseases.
- Since infectious diseases are highly dependent on local-scale and comparatively short-term weather parameters, extrapolation of climate and disease relationships from one spatial or temporal scale to another is not likely to yield valid estimates of future impacts.

3.6.6. Water- and Food-Borne Diseases

It is not possible to predict the likely incidence of diseases related to contaminated water or food, due to the complex interactions between the contamination potential of these pathogens, the effectiveness of public health programs designed to minimize exposure of the public to

contaminated water and food, and the public's compliance with these programs and recommendations.

Climate modeling suggests that extreme precipitation events, with significant runoff, are likely to become more commonplace as California's climate warms (Karl et al. 2008; Mastrandrea et al. 2009; Cayan et al. 2009; Dettinger et al. 2009). Coastal areas of California, where urban growth and development and land use decisions influence the quality of runoff water flowing through creeks and rivers to coastal beaches, are especially vulnerable to water contamination. This is particularly the case in the southern part of the state, where expanding urbanization, development and an increasing population generate pollution that ultimately flows into the Pacific Ocean (Dwight et al. 2002). Temporal and spatial analysis of bacterial levels near the mouths of several Los Angeles area rivers that empty into the Pacific Ocean showed that bacteria levels were highest near river mouths and adjacent beaches, and that precipitation events were significantly associated with increased bacterial loads in the same areas (Dwight et al. 2002). Louis et al. (2003) reported similar findings for the Chesapeake Bay on the East Coast of the United States. Severe precipitation events could increase opportunities for sewage treatment facilities to fail, leading to public exposure to contaminated water. Combined sewer systems, which carry both storm water and raw sewage to treatment plants, are a significant source of drinking and recreational water contamination, and although they are considered antiquated, some communities in California continue to be served by these systems. When overtaxed, these systems overflow directly into a surface water body, for example a river or lake, introducing untreated storm runoff water and sewage, along with any pathogens, pollutants, chemicals, and industrial wastes they contain, directly into the water body, thereby contaminating the water body, beaches, fish, and shellfish (Charron et al. 2004).

In the coastal zone, toxic algal blooms will likely be more frequent as water temperature rises, increasing the risk of illness originating from aquatic recreation, such as swimming and surfing, and from contamination of seafood (Rose et al. 2001). Food supplies could also become contaminated through contaminated runoff and through lack of field sanitation that results in contaminated irrigation water (Rose et al. 2001).

Food-borne diseases can originate with contamination of fruits, vegetables, and seafood by flooding, contaminated runoff following heavy rain, contaminated irrigation water, or for seafood by toxic substances released by algal blooms (Rose et al. 2001). The extent to which these diseases will increase is affected by multiple factors. It is likely that irrigation water contamination due to flooding, runoff, or seepage from adjacent livestock operations could increase due to more rapid organism replication under future warming conditions (Lipp et al. 2002; Hunter 2003). In addition, more frequent toxic algal blooms may occur under conditions of increased water temperature, leading to increased incidences of seafood contamination, in addition to increased contamination with *Vibrio* species.

Although food can be contaminated in the fields, or during harvest or post-harvest processing, most outbreaks of food-borne diseases are related to improper food handling or storage, either at home, in restaurants, or at food stores. Food-borne illnesses could increase due to more rapid bacterial proliferation at warmer temperatures in food not handled correctly during harvesting or processing, or not maintained at proper storage temperatures (Colwell and Patz 1998). A study done in England by Kovats et al. (2004b,c) found that increasing ambient temperature contributed to transmission of salmonella and campylobacter through food.

Those most at risk of experiencing serious effects from water- and food-borne diseases are the very young, the elderly, the infirm, and people with compromised immune systems. The California Department of Public Health and California counties and cities conduct surveillance for these diseases, as well as having protocols that are activated when outbreaks of disease occur. However, surveillance for these diseases is relatively poor, since most cases do not require or seek medical attention, leading to underreporting.

3.6.7. Other Infectious Diseases

Few studies have investigated the influence of weather factors on infectious diseases spread through oral-fecal or respiratory routes, such as enteritis and influenza. However, several studies suggest that increasing temperature and the ENSO could influence incidence of these diseases, although there are insufficient data available to estimate future cases of disease.

3.6.8. Vector-Borne Diseases

Vector-borne diseases are caused by a variety of viruses, bacteria, and protozoa that spend part of their life cycle in a host species (mosquitoes, fleas, and ticks) and are spread to humans and animals during insect feeding. The literature is clear that disease transmission rates are not only related to the complex interactions between factors related to vector and infectious organism survival, but also to other factors that indirectly affect the vector and organism. Such factors include the availability of suitable habitat for vector growth, reproduction, and maturation; land use patterns; vector control programs; and personal behaviors—including use of window and door screens, insecticides and repellents, clothing selection, availability of preventative vaccines and drugs to treat active cases, and being outdoors when the vector is active (Kovats et al. 2001; Sutherst 2004).

Both ticks and mosquitoes and the viruses they can spread are temperature sensitive, although the transmission and maturation of these viruses is dependent on many factors in addition to ambient temperature, such as availability of a suitable habitat. Each of these viruses has a different pattern of susceptibility to climate factors, including temperature, humidity, and rainfall.

Natively acquired cases of other mosquito-borne diseases such as malaria and dengue fever, which require no animal host, are extremely rare in the United States (Gubler et al. 2001), although malaria was once endemic to most of the country. However, predictions of increased numbers of people globally living in areas where malaria is endemic, coupled with increased world travel, increases the possibility of additional cases of malaria and other currently non-endemic diseases in California (Pherez 2007).

It is likely that the ranges for these infectious diseases will shift (Gubler et al. 2001) in response to global warming. However, modeling to date does not generally take into account the large number of factors unrelated to climate that also influence vectors, infectious organisms, and disease transmission rates. These factors include land use patterns, availability of suitable habitats, availability of suitable hosts, and natural and artificial barriers to species dispersal (Kovats et al. 2001; Pherez 2007). Analysis of the history of three vector-borne diseases (malaria, yellow fever, and dengue fever) not currently endemic in the United States (Reiter 2001) suggests that climate, at least on the order of several years, has rarely been the principal determinant of the prevalence or range of these diseases. Rather, the most important determinants have been human activities and their impact on local ecology, suggesting that the

use of climate modeling to estimate future vector-borne disease rates from likely vector ranges may be inappropriate.

Several studies have emphasized the human behavior aspect of these diseases. Randolph et al. (2008) found that there were significant differences in tick-borne encephalitis rates among European countries that were more related to human behavior, including extent of outdoor activity in tick-infested regions, than to tick abundance and activity. Although focused on dengue virus infection, which is not endemic in California, results by Reiter et al. (2003) are illustrative of the influence of human behavior on transmission of vector-borne diseases. Laredo, Texas and Nuevo Laredo, Mexico are separated only by the Rio Grande River. Although there was greater abundance of the mosquito vector that carries dengue virus in Laredo, the disease rate was higher in Nuevo Laredo. Human behavior and environmental factors that influence contact with mosquitoes appear to account for this paradox. Houses in Laredo were more likely to have window screens and air conditioning, and people were less likely to sit outside in the evenings, compared to in Nuevo Laredo, suggesting that human exposure to the vector is an important factor in disease transmission rate.

The people most at risk for serious effects from these diseases are the very young, the elderly, the infirm, and people with compromised immune systems. Most healthy people are at little risk of serious disease from infection with these organisms (California Department of Health Services 2004). Exposure to these organisms leads to antibody formation that persists for at least several years; however, the degree and duration of protection provided by antibodies from an initial infection is unknown because these diseases are rare enough that there are no data available on reinfection rates (Personal communication, California Department of Public Health).

California has well-developed protocols involving the State, counties, and cities for surveillance for these diseases. The State also has a multi-level protocol that is activated based on risk of disease transmission (CDPH 2004; Barker et al. 2003). It is possible that the small number of cases of these diseases in California, compared to the large populations living in endemic areas, is related to the effectiveness of these and similar programs and recommendations such as use of window screens, insect repellants, and not going outdoors during times of high mosquito activity (Barker et al. 2003).

3.6.9. Rodent-Borne Diseases

Rodent-borne diseases are usually transmitted to humans through direct contact with rodent urine, feces or other body fluids. The most well-known rodent-borne human disease in the Southwest United States is hanta virus, although rodents are also hosts for fleas and ticks that can spread plague and several other infectious diseases (McMichael and Githeko 2001). The potential for human infection with hanta virus appears to be primarily related to the size of the rodent population. The impact of climate change on rodent-borne diseases is likely to be related to relatively short cycles, for example a year or two rather than longer-term, and based primarily on the availability of food, and consequently the rodent population (Gubler et al. 2001). There have been 49 cases of hanta virus cardiopulmonary syndrome in California since 1993 (CDPH 2008).

4.0 Conclusions and Recommendations

4.1. Conclusions

4.1.1. *Temperature-Related Mortality and Morbidity*

The temperatures at which heat and cold-related mortality occur vary among locations, and are primarily related to deviations from local average temperatures. Consequently, temperature deviations both above and below the typical levels are associated with an increased risk of temperature-related mortality for both excess deaths, and for deaths directly caused by heat or cold. This means that temperature alert programs must be tailored to specific regions, rather than application of a “one-size fits all” approach.

The majority of excess deaths related to heat appear to occur in cities, pointing to the influence of the heat island effect in urban areas, where temperatures are several degrees higher than in less densely populated areas outside of central city areas.

Most excess heat-related deaths occur in older people, particularly over age 75, and more frequently in women than men. There is also increased risk of mortality directly attributable to heat for younger people who are active outdoors in the heat, particularly outdoor workers. Identified risk factors for heat-related mortality include being bed-ridden or otherwise having an increased level of dependency, not leaving home daily, living alone, living of the top floor of a building, having few social contacts, lack of access to transportation, and lack of access to or not using air conditioning. A number of common clinical conditions have been associated with increased risk of heat mortality, including cardiovascular, respiratory, neurological, and psychiatric diseases. In addition, a number of common medications used to treat these diseases adversely affect the body’s ability to thermoregulate, and increase risk of heat-related mortality.

Analyses in Chicago, Milwaukee, Philadelphia, Boston, and other cities worldwide have demonstrated that heat health watch, warning, and emergency action plans are effective in reducing heat-related morbidity and mortality.

Populations accustomed to cold climates typically experience less cold-related mortality than areas with milder climates, and many cold-related deaths occur at relatively mild temperatures. This counterintuitive finding suggests that people living in areas with mild winters may lack appreciation of the dangers of cold, may have insufficient home heating and insulation, or fail to wear adequate clothing for the ambient conditions. As California warms, there could be a decrease in cold-mortality, but it would be a mistake to conclude that cold temperatures no longer will pose a risk. People tend to be unprepared for conditions outside the typical. Failure to remain cognizant of the public health risks associated with cold temperatures will continue to put the elderly, the poor, and the homeless at increased risk of cold-related mortality, even if the episode is relatively mild or lasts for only a few days. Awareness of the behavioral aspect of people’s response to heat and cold may also aid in modifying existing watch/warning systems and provide appropriate reminders to individuals from official sources.

4.1.2. *Air Pollution*

Air pollution-related health effects are fundamentally tied to the extent to which ambient air quality standards are not attained. The influence of increasing temperatures on both ambient and global background concentrations of air pollutants will likely increase the difficulty of

attaining current ambient air quality standards for ozone (and possibly for PM10 and PM2.5), and increase the cost and time period required to reach attainment. It is also likely that as temperatures increase, people will alter their behavior and activity patterns, resulting in increased time indoors, which would tend to reduce exposure, particularly as air conditioning becomes increasingly prevalent. Air conditioning, which is projected to become increasingly prevalent statewide, typically reduces the concentrations of ozone and PM indoors compared to outdoors. Indoor ozone levels typically range between 20% and 80% of outdoor levels, depending on such factors as season, building ventilation rate and microenvironmental factors (Weschler et al. 1989). Consequently, if global warming causes people to increase the amount of time they spend indoors in air conditioned environments, ozone- and PM-related health effects could, in theory, decrease, even if outdoor concentrations increase. However, it is not reasonable to assume that people will remain indoors at all times as California's climate warms. Also, the indoor environment can contain toxic air contaminants at concentrations that are several-fold higher than observed in outdoor ambient air. In addition, it should not be forgotten that there will continue to be a significant population of outdoor workers (e.g., gardeners, construction, and agricultural workers), as well as children and adults engaged in outdoor activities, who will continue to be at risk of adverse health outcomes related to ambient concentrations of air pollutants.

4.1.3. Wildfires

Review of the literature on wildfires indicates that fire smoke exposure can increase adverse respiratory health effects, including hospitalization, emergency room visits, and respiratory symptoms. The number of people who will be affected by future fires cannot be estimated due to the unique nature of each fire, and the necessity that the smoke plumes cover an inhabited area. However, the literature suggests that the people most at risk will be those with existing cardiopulmonary disease, and that risk increases with advancing age. Published analyses of morbidity and mortality related to wildfires suggest that the number and severity of additional cases related to future wildfires is likely to be modest.

The California Thoracic Society (1997), and the California Air Resources Board (CARB 2008) have published fire fact sheets that describe personal actions that can minimize lung injury when exposed to a fire. In addition, the document "Wildfire Smoke: A Guide for Public Health Officials (available at www.arb.ca.gov/smp/progdev/pubeduc/wfgv8.pdf) describes health risks related to fire smoke inhalation and actions that can be taken to protect oneself. It also provides guidance to public health officials on assessing the level of public health risk a particular fire poses, and recommended responses.

4.1.4. Infectious Diseases

A variety of infectious diseases could increase with climate change, although few data are available that address this question directly. These include diseases such as cryptosporidiosis or salmonellosis, which are contracted respectively through contaminated water or food, and those spread by various vectors, such as mosquitoes, ticks, and rodents, and include West Nile virus, Lyme disease, and Hanta virus. The local, State, and federal governments have active surveillance protocols in place to identify outbreaks of these diseases. Maintaining and strengthening California's public health infrastructure, along with active surveillance for water-, vector-, and food-borne diseases, are critical to preventing increased disease. Those most at risk

of experiencing serious effects from these infectious diseases are the very young, the elderly, the infirm, and people with compromised immune systems.

California also has an extensive protocol for combating vector-borne diseases. There are no human vaccines for these diseases, and consequently mitigations rely heavily on disease surveillance and vector control programs, as well as rapid and effective response once a disease has been contracted. A variety of personal behaviors, such as use of window screens and air conditioning, along with public health advisories to wear clothing that provides skin coverage, use insect repellent, and remain indoors during heightened vector activity times (for example, at dusk) are effective in reducing risk of contracting these diseases. Drainage of marshy areas, unmaintained swimming pools, and other sources of standing water, such as old tires, cans, and flower pots are also effective means of reducing habitat for mosquitoes, and thus the number of insects. Other possible habitats for mosquitoes include water storage, and distribution systems related to irrigation

4.2. Recommendations

4.2.1. Actions to Protect Public Health

The following are recommended actions that can help alleviate the impacts of climate change on public health.

- Strengthen surveillance for temperature-related mortality and adverse health effects of air pollution exposure and wildfires, as well as infectious diseases related to water-, vector-, and food-borne pathogens.
- Expand public education on the risks of extreme temperatures, high air pollution, wildfires, and infectious diseases related to water-, vector-, and food-borne pathogens to individuals and their caregivers. There should be an emphasis on effective steps that the individual can adopt to protect him- or herself. Educational materials should also cover personal risk factors for adverse outcomes, including age, health status, and commonly used prescription medications. Educational materials should be available in multiple languages to reach wider audiences.
- Improve coordination among alert systems and emergency personnel responding to public health emergencies related to temperature, air pollution, infectious diseases, and wildfires.
- Develop Health Heat Watch Warning Systems for regions of the State that have not yet adopted them. To date, only the San Jose area has a Health Heat Watch Warning System in place. These systems should be coupled with community-level programs to provide outreach and services to people in need of cooling centers and other assistance to prevent heat-related illness or death. These programs should focus primarily on the elderly and infirm but should not overlook the economically disadvantaged.
- Review, strengthen, and enforce occupational safety standards to protect outdoor workers from heat illness and mortality.
- Increase access to air conditioning, particularly in areas where it is currently not common and in population groups that lack access. Consider expanding existing programs that help low-income people pay for residential heating to include residential cooling as well.

- Evaluate the capacity of statewide water and sewage treatment facilities, and modernize and expand these facilities as necessary to meet predicted scenarios of extreme precipitation and runoff events. Replace remaining combined sewage systems in the State with modern systems.
- Review and expand existing vector control programs as necessary.
- Consider climate change as part of planning efforts directed at attaining the health-based ambient air quality standards.
- Encourage individuals and families to have an evacuation plan in the event of wildfire, keeping in mind that a wildfire may disrupt usual travel routes. These plans should include provisions for sheltering in place if not directed by authorities to evacuate, and should include planning for food and especially water in the event that utility service is disrupted by the fire.

4.2.2. Research Needs

This review suggests several avenues for future research.

- The health-related endpoints discussed above are influenced by many factors. Climate is one contributor, but these health endpoints are also influenced by many other factors unrelated to climate, including personal behaviors. Consequently, the ultimate health outcome is the result of the integration of all of these factors. Increasingly sophisticated statistical models will be required to make credible estimates of future climate change-related health impacts. These models will need to account for changes in population, healthcare, the physical environment (i.e., land use, building stock characteristics), life style factors, concurrent non-climatic stressors, and introduction of climate change adaptation strategies. The interactions among these various factors are unlikely to be simply additive, but synergistic, thus requiring sophisticated modeling.
- Research into the effects of climate change on air quality is still in its infancy, and results to date are primarily based on perturbations of single meteorological parameters within short, historical high air pollution episodes. These methods need to be expanded to better investigate the influence of projected complex meteorological changes on air pollution. These results will help to inform and guide development of new control technologies and regulations aimed at attaining the health-based ambient air quality standards.
- While there has been quite a bit of research into the short-term influence of meteorological factors on infectious diseases, for example as a means to predict incidence in the next season, little is known about the longer-term influence of climate-related factors on these diseases, particularly when vector control programs are factored into the analysis.
- Since most, if not all of the climate-related health effects discussed above are preventable with appropriate action on the part of health care providers, emergency responders, communities, individuals and their caregivers, a particularly fruitful avenue for future research would be development of more effective public health education and intervention strategies to equip communities, health care providers, and citizens to best protect themselves from weather and climate-related health impacts. This area of

research should also include investigations into improved methods for effecting behavior modifications that reduce health-related impacts of climate change.

- Significantly more research is needed into the potential, costs, and effectiveness of various adaptation strategies and their feasibility in different community contexts, including how to communicate these strategies most effectively.

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