Endangerment and Cause or Contribute Findings for Greenhouse Gases Under Section 202(a) of the Clean Air Act:

EPA's Response to Public Comments

Volume 5: Impacts and Risks to Public Health and Welfare: Human Health and Air Quality

Impacts and Risks to Public Health and Welfare: Human Health and Air Quality

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FOREWORD

This document provides responses to public comments on the U.S. Environmental Protection Agency's (EPA's) Proposed Endangerment and Cause or Contribute Findings for Greenhouse Gases Under Section 202(a) of the Clean Air Act, published at 74 FR 18886 (April 24, 2009). EPA received comments on these Proposed Findings via mail, e-mail, and facsimile, and at two public hearings held in Arlington, Virginia, and Seattle, Washington, in May 2009. Copies of all comment letters submitted and transcripts of the public hearings are available at the EPA Docket Center Public Reading Room, or electronically through http://www.regulations.gov by searching Docket ID *EPA-HQ-OAR-2009-0171*.

This document accompanies the Administrator's final Endangerment and Cause or Contribute Findings for Greenhouse Gases Under Section 202(a) of the Clean Air Act (Findings) and the Technical Support Document (TSD), which contains the underlying science and greenhouse gas emissions data.

EPA prepared this document in multiple volumes, with each volume focusing on a different broad category of comments on the Proposed Findings. This volume of the document provides responses to public comments regarding human health and air quality.

In light of the very large number of comments received and the significant overlap between many comments, this document does not respond to each comment individually. Rather, EPA summarized and provided a single response to each significant argument, assertion, and question contained within the totality of comments. Within each comment summary, EPA provides in parentheses one or more lists of Docket ID numbers for commenters who raised particular issues; however, these lists are not meant to be exhaustive and EPA does not individually identify each and every commenter who made a certain point in all instances, particularly in cases where multiple commenters expressed essentially identical arguments.

Several commenters provided additional scientific literature to support their arguments. EPA's general approach for taking such literature into consideration is described in Volume 1, Section 1.1, of this Response to Comments document. As with the comments, there was overlap in the literature received. EPA identified the relevant literature related to the significant comments, and responded to the significant issues raised in the literature. EPA does not individually identify each and every piece of literature (submitted or incorporated by reference) that made a certain point in all instances.

Throughout this document, we provide a list of references at the end of each volume for additional literature cited by EPA in our responses; however, we do not repeat the full citations of literature cited in the TSD.

EPA's responses to comments are generally provided immediately following each comment summary. In some cases, EPA has discussed responses to specific comments or groups of similar comments in the Findings. In such cases, EPA references the Findings rather than repeating those responses in this document.

Comments were assigned to specific volumes of this Response to Comments document based on an assessment of the principal subject of the comment; however, some comments inevitably overlap multiple subject areas. For this reason, EPA encourages the public to read the other volumes of this document relevant to their interests.

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Acronyms and Abbreviations

ANPR	Advance Notice of Proposed Rulemaking
°C	degrees Celsius
CAIR	Clean Air Interstate Rule
CCSP	U.S. Climate Change Science Program
CDC	Centers for Disease Control and Prevention
CIRAQ	Climate Impact on Regional Air Quality
CH_4	methane
CO_2	carbon dioxide
D8H	daily 8-hour average
ENSO	El Niño Southern Oscillation
EPA	U.S. Environmental Protection Agency
GCM	general circulation model
GHG	greenhouse gas
GIT	Georgia Institute of Technology
HFC	hydrofluorocarbon
IA	integrated assessment
ICAP	Intercontinental transport and Climatic effects of Air Pollutants
IPCC	Intergovernmental Panel on Climate Change
MDA8	maximum daily 8-hour
N_2O	nitrous oxide
NAAQS	National Ambient Air Quality Standards
NERL	National Exposure Research Laboratory
NIPCC	Nongovernmental International Panel on Climate Change
NO_x	nitrogen oxides
O_3	ozone
OAQPS	Office of Air Quality Planning and Standards
ORD	Office of Research and Development
PAN	peroxyacetylnitrate
PFC	perfluorocarbon
PM	particulate matter
$PM_{2.5}$	fine particulate matter (particles less than 2.5 micrometers in diameter)
ppb	parts per billion
ppm	parts per million
RIA	regulatory impact analysis
SRES	IPCC Special Report on Emissions Scenarios
TAR	IPCC Third Assessment Report
TBE	tick-borne encephalitis
TSD	Technical Support Document
UK	United Kingdom
USGCRP	U.S. Global Change Research Program
VOC	volatile organic compound
WHO	World Health Organization

5.0 Impacts and Risks to Public Health and Welfare: Human Health and Air Quality

5.1 Human Health

General Issues

Comment (5-1):
Commenters (e.g., 0329, 2750, 3136.1, 3394.1, 3462.1, 3449.1:10, 3747.1, and 7020) state that specific aspects of the climate impacts evidence summarized in the Technical Support Document (TSD) with respect to human health do not support the Administrator's endangerment finding.

Response (5-1):
The specific issues that underlie these comments are addressed in the responses throughout this volume, and other volumes of the Response to Comments document. With regard to the commenters' conclusion that the current science does not support an endangerment finding with respect to human health and air quality, we disagree based on the scientific evidence before the Administrator. See the Findings, Section IV.B, "The Air Pollution is Reasonably Anticipated to Endanger Both Public Health and Welfare," for details on how the Administrator weighed the scientific evidence underlying her endangerment determination in general, and with regard to the human health and air quality in particular.

Comment (5-2):
A commenter (3394.1) acknowledges the paucity of studies addressing "the interaction effects of multi-sector climate impacts (they may be nonlinear) or of interactions between climate change health impacts and other kinds of local, regional, and global changes," and indicates that the U.S. Environmental Protection Agency's (EPA's) analysis demonstrates that the Agency assumes that such studies would demonstrate additional negative impacts as opposed to possible benefits or the potential for mitigation. The commenter notes EPA may not properly substitute speculation for adequate information.

Response (5-2):
The sentence the commenter is quoting was taken from the introduction of Section 7 of the TSD associated with the Advanced Notice of Proposed Rulemaking (ANPR) for this action. We have revised this statement in response to these comments and it now reads: "There are few studies that address the interactive effects of multiple climate change impacts or of interactions between climate change health impacts and other kinds of local, regional, and global socioeconomic changes (Field et al., 2007)." The commenter interprets this statement as evidence that the Agency is speculating in the absence of adequate information.

We disagree, and note that the next sentence following the one highlighted by the commenter summarizes another key finding from the Intergovernmental Panel on Climate Change (IPCC): "For example, climate change impacts on human health in urban areas will be compounded by aging infrastructure, maladapted urban form and building stock, urban heat islands, air pollution, population growth, and an aging population (Field et al., 2007)." In addition, the TSD summarizes the U.S. Climate Change Science Program (CCSP) findings that vulnerability is the summation of all the factors of risk and resilience that determine whether individuals experience adverse health impacts, and that climate change is very likely to accentuate the disparities already evident in the American health care systems, as many of the expected health effects are likely to fall disproportionately on the poor, the elderly, the disabled, and the uninsured (Ebi et al., 2008).

EPA has not speculated that there would be negative impacts. We have carefully reviewed the TSD, and conclude we have summarized the conclusions of the scientific assessment literature for both positive and

negative impacts. We note that the commenter has failed to cite any scientific literature to the contrary. Further, we note EPA has summarized both beneficial and adverse impacts of climate change that throughout the TSD. The assertion that the Agency is focusing on negative impacts and ignoring beneficial impacts is incorrect.

Finally, see the Findings, Section III.C, "Adaptation and Mitigation," for our response to comments on the treatment of adaptation and mitigation in the Findings.

Comment (5-3):

At least one commenter (3497.1) indicates that EPA in the endangerment finding has failed to consider health benefits from increases in carbon dioxide (CO_2). The commenter argues that any reductions in CO_2 increase the potential loss of agricultural product and threaten sufficient food supply and that we must maintain and increase our ability to produce food and fiber for a growing population. The commenter states that not having food, i.e. starvation, is a real human health hazard. Similarly, another commenter (2818) states that "if there is not enough food, if there is not enough water, then chronic diseases, heart disease, diabetes, as serious as those problems are, they really don't mean as much if there is not enough food and water to go around."

Response (5-3):

We examined the TSD and Findings in light of this comment, and conclude that the issues the commenter raises are addressed. First, the TSD addresses the beneficial and adverse effects of climate change on food production and agriculture in Section 9, and the scientific literature on CO_2 fertilization is discussed therein. See Volume 6 of the Response to Comments document for responses to comments on issues associated with food production and agriculture. Second, the impacts of climate change on water quantity and quality are discussed in Section 11 of the TSD, and responses to comments on these issues are provided in Volume 7 of the Response to Comments document.

Lastly, see the Findings, Section IV.B, "The Air Pollution Is Reasonably Anticipated to Endanger Both Public Health and Welfare," for our response to comments on how the Administrator weighed the scientific evidence underlying her endangerment determination. In addition, see Response to Comments Volume 9 for our responses to other significant comments on how evidence of specific climate impacts was considered in the Administrator's endangerment finding. Also see the Findings, Section III.C, "Adaptation and Mitigation," for our response to comments on the treatment of adaptation and mitigation in the Findings.

Comment (5-4):

A commenter (1468) states that if increased concentrations of the minor greenhouse gases (GHGs) including CO_2 have any significant warming effect (which is highly debatable), they could conceivably contribute to a warmer world that would be more propitious for human beings.

Response (5-4):

EPA disagrees with the commenter's assertion that the scientific literature does not suggest that increased concentrations of GHGs such as CO_2 affect temperatures. See Volume 3 of the Response to Comments document, "Attribution of Observed Climate Change," for responses to comments on the attribution of climate warming.

With respect to the commenter's general assertion that a warmer world would be better for human beings, we note that many commenters provided specific comments on this issue. See responses to other comments in this volume regarding the health impacts associated with a warmer world.

Comment (5-5):

A commenter (4003) indicates that perhaps the most significant indicator of all that the U.S. population does not seem to have been adversely affected by any vulnerabilities, risks, and impacts that may have arisen as the result of human-induced climate change is the increase in life expectancy, and provided the figure below. Similarly, several commenters cite the Nongovernmental International Panel on Climate Change (NIPCC) (Idso and Singer, 2009), which concludes that observations demonstrate that if the increases in temperature and CO_2 concentrations of the past two centuries were bad for our health, their combined negative influence was minuscule compared to whatever else was at work in promoting this vast increase in worldwide longevity.

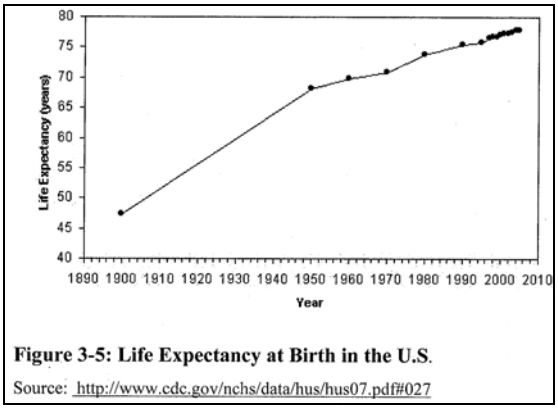

Figure 3-5: Life Expectancy at Birth in the U.S.

Source: http://www.cdc.gov/nchs/data/hus/hus07.pdf#027

Source: Commenter 4003, from Carlin, 2009

Response (5-5):

In light of this comment, we have reviewed the cited literature and examined the issue in the context of this action. We note that life expectancy over the last century in the United States (and globally) is determined by several factors, many of which are non-climatic (i.e., advances in health care, infrastructure, technology, and wealth). The chart provides no insight into the risk or trend of any specific factor on life expectancy. We also conclude that this measure is of limited meaning in the context of the evaluation of risks due to a changing climate, which can occur via various pathways. The single metric of life expectancy does not convey or provide information on the risks associated with changes in climate and whether or not they are increasing, either in the past or in the future. For example, were a particular epidemic to occur within this trend it may or may not be evident; however, this does not mean it is not of serious concern to public health.

While the commenter notes that this increase in life expectancy has occurred despite increasing temperatures and CO_2 levels, it does not represent future conditions in which climate is likely to become warmer and with likely more variable precipitation. The fact that public health measures are very effective (i.e., we do not have many cases of certain diseases) does not mean that the risk is not present or increasing. In addition, our prevention measures are not 100% effective and there remain concerns about potential risks (e.g., new diseases), growing resistance to antibiotics, reduction in effectiveness of vector-control measures over time, and an aging sanitation and drinking water infrastructure. See the Findings, Section IV.B, "The Air Pollution Is Reasonably Anticipated to Endanger Both Public Health and Welfare," for our response to comments on how the Administrator weighed the scientific evidence underlying her endangerment determination.

Comment (5-6):
A commenter (11348) indicates that we need more research on direct and indirect health effects of climate change, and more effort in adaptation, given the impossibility of preventing natural events.

Response (5-6):
Though EPA agrees more research would be valuable on the direct and indirect health effects of climate change, the current state of knowledge of the current and future risks of climate change and public health provides sufficient support basis for the Administrator's determination. Please refer to the Findings, Section III.A, "The Science on Which Decisions Are Based," and Section IV.B, "The Air Pollution Is Reasonably Anticipated to Endanger Both Public Health and Welfare," for our response to comments on how the Administrator weighed the scientific evidence underlying her endangerment determination. See the Findings, Section III.C, Adaptation and Mitigation, for our response to comments on the treatment of adaptation and mitigation in the Findings.

Comment (5-7):
A commenter (3136.1) asks why EPA includes the parenthetical "but may be modulated by public health measures" after the expanded range of vector-borne and tick-borne disease in the ANPR TSD. This applies equally to all of the other bullets in this section. The commenter states that there is conclusive evidence that public health measures have successfully reduced our sensitivity to heat waves (Davis et al., 2003a, 2003b).

Another commenter (3722) indicates that the fundamental factor which EPA repeatedly discounts or ignores in its Proposed Findings is human adaptability to temperature change. This adaptability, which underlies the increased life expectancy and improved health in U.S. cities and cities throughout the world over the last 100 years, despite increases in temperatures (of 4°C or more), is the single most important factor which has to be taken into account in predicting the health effects of future warming. In addition, studies in the United States indicate the importance of human adaptation in mitigating the mortality effects of climate. The commenter references Davis et al. (2003), which indicates that a 74% drop in heat-related deaths occurred despite an average increase in temperature of 1.0°C during the same period.

Response (5-7):
See the Findings, Section III.C, "Adaptation and Mitigation," for our response to comments on the treatment of adaptation and mitigation in the Findings.

With respect to the specific comment regarding the referenced parenthetical, we note that it is based on a specific IPCC (Field et al., 2007) finding and associated underlying scientific literature. Thus, we disagree with the commenter that it applies to all of the other bullets in this particular section. However, we note that Section 7(c) of the April 2009 version of the TSD, also notes an Intergovernmental Panel on Climate change (IPCC) conclusion that indicates more broadly that human health risks from climate change will be strongly modulated by changes in health care, infrastructure, technology, and accessibility to health care (Field et al., 2007). In response to these comments, and in acknowledgment that measures to modulate human health risks apply more broadly, we have revised the final TSD by moving the following language toward the beginning of Section 7:

> "The IPCC concludes that human health risks from climate change will be strongly modulated by changes in health care, infrastructure, technology, and accessibility to health care (Field et al., 2007). The aging of the population and patterns of immigration and/or emigration will also strongly influence risks (Field et al., 2007)."

While EPA finds that risks to human health can be reduced by investments in countermeasures, we conclude that this does not eliminate risk, especially to populations vulnerable to the array of direct and indirect health effects associated with climate change. EPA is familiar with the studies by Davis et al. (2003a, 2003b) but notes that net mortality is based on several factors beyond the influence of preventative and adaptive measures. Please refer to responses 5-24 and 5-48 for further discussion of Davis et al. (2003a; 2003b) in the context of temperature effects.

Comment (5-8):

A commenter (0329) asks the following questions: "Will the EPA recommend to U.S. lawmakers that population within the borders of the U.S. be limited by artificial means to further reduce emission of greenhouse gases? Can the EPA guarantee that if the U.S. curtails its production of greenhouse gases that citizens will see a notable and measurable improvement in the economic, social and medical standard of living within the U.S.? If GHG are "hazardous" to human health how many days/months/years will the average life expectancy of U.S. citizens be increased by implementation of controls?" Another (2818_GGG) wonders if EPA will require permits for having children given the contributions of human respiration and digestion to CO_2 and CH_4. One commenter (3712.1) claims that regulating CO_2 will regulate all activities, increasing prices for the poor and elderly, and restricting the amount of life that can be allowed to exist. Similarly, commenter (9717) worries that because people emit CO_2 in proportion to their weight that fat people will be taxed.

Response (5-8):

EPA notes that all of the commenters' questions focus on methods and implications of greenhouse gas (GHG) controls, which are not the focus of this action. This action concerns the Administrator's determination regarding whether GHG pollution is reasonably anticipated to endanger public health and welfare. In this action, EPA is not recommending any specific responses be taken to reduce greenhouse gases, and questions regarding the nature and effectiveness of measures to reduce greenhouse gas emissions are outside the scope of this action. When and if EPA proposes regulatory action to control greenhouse gas emissions, it would be accompanied by a regulatory impact analysis which would examine the costs and benefits of the specific proposed regulatory actions, and the public would have the opportunity to comment on any proposals.

We also want to state clearly that EPA has not, is not and does not plan to recommend any of the concepts raised by commenters (e.g., limiting population "by artificial means", requiring "permits for having children", "restricting the amount of life that can exist"). Further, it is unclear to us where the commenters got these ideas, as it is well known that CO_2 emissions from human respiration are not an emission source of concern, and are not contributing to the atmospheric buildup of CO_2. Human respiration of CO_2 is part of a closed system. There can be no net addition of CO_2 to the atmosphere because the amount of CO_2 people exhale cannot be greater than the carbon people put into their bodies by eating plants, or eating animals that eat plants.

Comment (5-9):

A commenter (11350) indicates that global warming could lead to human extinction, and the Administrator is justified in taking the most extreme measures to stop GHG pollution. The commenter references a book by Brian Fagan, "A Long Hot Summer" (Fagan, 2004) as evidence of "how Global Warming could lead to the extinction of Homo Sapiens by the year 2030."

Response (5-9):

EPA acknowledges that the risks and impacts of climate change are occurring now and will continue to increase over this century; however, EPA notes that the underlying scientific literature does not include predictions of human extinction.

Comment (5-10):
A commenter (4946) indicates that EPA's Proposed Findings make the case for action by demonstrating that climate change threatens "virtually every facet of the living world around us" and that its effects include "sickness and death." While coal plants are a major source of global warming pollution, it is clear that emissions from new motor vehicles and new motor vehicle engines contribute to global warming pollution too. The commenter applauds EPA for using its authority under the Clean Air Act to take action to protect us from the pollution that causes global warming and urge you to move rapidly by finalizing these determinations and issuing strong new rules to control global warming pollution and provide a sound foundation for our economy.

Response (5-10):
The commenters' view that this action demonstrates that climate change threatens human health is consistent with the Findings. The comments on GHG emission sources are generally consistent with the scientific literature summarized in the TSD on this issue.

Comment (5-11):
A commenter (8320) submits information and references on the potential health effects of global climate change. The commenter discusses the following topics: extreme heat events, wildfires, vector-borne diseases, water-borne diseases, air quality and human heath, aeroallergens and allergies, environmental justice, and the vulnerability of children to effects from air quality. The commenter stresses that "climate change will alter the global environment and present major challenges to the health and welfare of children."

Response (5-11):
EPA has reviewed the submitted comments and associated references, and finds that they include several very recent and relevant studies (e.g., St. Louis and Hess, 2008; Patz et al., 2008; Jerret et al., 2009) that confirm the scientific support for health related impacts summarized in the TSD.

In one example, Luber and McGeehin (2008) call extreme heat events "the most prominent cause" of weather-related human mortality in the United States, noting that they are responsible for more deaths annually than hurricanes, lightning, tornadoes, floods, and earthquakes combined. We note that the recently released U.S. Global Change Research Program (USGCRP) report (Karl et al., 2009) also concludes that mortality from heat is the number one weather-related cause of death and cites an analysis of nine U.S. cities showing that deaths rise with increases in temperature and humidity with no confounding or effect modification due to air pollution (Zanobetti and Schwartz, 2008).

Another recent study (Jerret et al., 2009) provided by the commenter indicates that high levels of ground-level ozone can increase the risk of asthma-related hospital visits and premature mortality and that the effect of long-term exposure to ozone on air pollution–related mortality was not known. The study indicates that ozone exposure is associated with the risk of death from respiratory causes, and that long-term, low-level exposure can be lethal. Researchers studied the outcomes of almost 500,000 adults in 96 metropolitan regions who enrolled in the American Cancer Society Cancer Prevention Study in 1982 and 1983 and were tracked for an average of 18 years. In addition, the study by Jerrett et al. (2009) looked at associations between ozone concentrations and the risk of death, in a single-pollutant model and in a two-

pollutant model with fine particulate matter ($PM_{2.5}$). In two-pollutant models, researchers demonstrated a significant increase in the risk of death from respiratory causes in association with an increase in ozone concentration. The study found that every increase in 10 parts per billion (ppb) in average ozone concentrations was associated with a roughly 4% increase in mortality from respiratory causes. This translated in Los Angeles to a 43% increase in the risk of dying from respiratory causes. Eastern cities like New York and Washington had an average increased risk of about 25% to 27%. EPA concludes this study fills important knowledge gaps in health impact literature from increases in ozone exposure (at low levels).

Another study provided by the commenter provides evidence of vector-borne disease shift attributed to climate. For example, in Europe, geographic shifts in the tick's distribution have been attributed to climate change. An expansion of the tick's range into higher elevations in the Czech Republic corresponded to rising temperatures. A shift toward higher latitudes in Sweden corresponded to a reduction in the number of very cold winter days (Gage et al., 2008).

Based on review of the cited literature from the commenter, we conclude that the information provided is generally consistent with, and in several cases even stronger than the assessment literature summarized in the TSD.

Comment (5-12):

A commenter (3402) indicates that the Earth is a dynamic system of balance, and all living things have a stake in the health of our shared atmosphere. While CO_2 as a gas is as critical as oxygen to life on Earth, like oxygen it becomes a toxin when the balance of concentration is disrupted. We must achieve atmospheric balance to protect not only human health, but also that of all living things. The commenter also indicates that her son is sensitive to air quality and notes there is a strong correlation between his sick days and poor air quality. The commenter indicates that, to protect public health and wellbeing, we must reduce health risks to those suffering from allergies. The commenter notes that warming climates have resulted in an earlier and longer allergic pollen season. She states: "Global warming pollution threatens the well being of today's youth as well as the future generations. Increased GHG emissions and climate change are putting more and more of our cities and communities in violation of air pollution standards. Children are most susceptible to these changes, particularly African American children who are more likely to be affected by asthma than their Caucasian counterparts."

Response (5-12):

We note the commenter's perspective and recognize that the risks and impacts mentioned by the commenter, while not supported by specific scientific literature, are in some cases the same impacts as those based on the scientific literature and summarized in the TSD. For example, the TSD notes that specific subpopulations may experience heightened vulnerability to climate-related health effects. As summarized in the TSD, climate change is very likely to accentuate the disparities already evident in the American health care system as many of the expected the health effects are likely to fall disproportionately on the poor, the elderly, the disabled, and the uninsured. The CCSP (Ebi et al., 2008) also notes that "climate can also affect the incidence of diseases associated with air pollutants and aeroallergens (Bernard et al., 2001)." The TSD summarizes the IPCC on the impact of climate change on ozone and particulate matter (PM), which states that "future climate change may cause significant air quality degradation by changing the dispersion rate of pollutants, the chemical environment for ozone and PM generation and the strength of emissions from the biosphere, fires and dust."

Comment (5-13):

Commenter(s) (2895) indicate that the World Health Organization (WHO) estimates that global warming is already responsible for 150,000 deaths and 5 million illnesses each year. The commenters note that these have come in the form of malnutrition, diarrhea, and vector-borne diseases like malaria and dengue fever. So far they have occurred predominantly in poor and developing countries; ironically, the countries that contribute the least to global warming are the most susceptible. The health impact of climate change is also evident here in the developed world, the commenters state, and will be increasingly felt if we do not take action.

The commenters state that there are four main categories of health effects:

> *Increased frequency, duration, and intensity of heat waves.* The associated health problems of heat cramps, heat exhaustion, and heat stroke will be become increasingly common. The very old and very young are especially vulnerable, as well as those who are poor, socially isolated or who have chronic illnesses.
> *Increased air pollution.* Increased temperatures cause increased production of ground level ozone, the main component of smog. This will increase rates of asthma and other respiratory diseases. It also makes breathing difficult for those who already have cardiac or respiratory ailments. Pollen production and allergies are also increasing as a result of increased CO_2 concentrations.
> *Infectious diseases.* Climate change is altering the range of disease-carrying organisms. West Nile virus carried by mosquitoes was not as prevalent in the United States until recently. More than 25,000 cases and more than 1,000 deaths have been recorded.
> *Extreme weather events.* This includes severe storms, increases in both drought and flooding, and associated features such as erosion and wild fires. The commenter indicates that we simply do not have the public health capacity to respond to increasing numbers of large-scale disasters that are difficult to predict.

Response (5-13):
EPA acknowledges the health effect categories as listed by the commenter and recognizes the rapid emergence of research and published literature on health effects associated with climate change. The TSD summarizes key conclusions of IPCC (2007), based on their review of over 500 articles on the effects of heat and cold; wind, storms, and floods; drought, nutrition, and food security; food safety; water and disease; air quality and aeroallergens and disease; vector-borne, rodent-borne, and other infectious diseases; occupational health; and ultraviolet radiation. The TSD also summarizes the conclusions of the recently released USGCRP report (Karl et al., 2009), which describes global climate change impacts for the United States. This report provides further evidence of the scope and magnitude of health effects, both direct (e.g., increased morbidity from heat waves or floods) and non-direct (e.g., increased or alteration in the spread and transmission rates of infectious diseases). As noted by the commenter, the literature shows poor countries and communities are likely to be disproportionately impacted by climate change health risks. For example, IPCC notes that adverse health impacts will be greatest in low-income countries and those at greatest risk include—in all countries—the urban poor, the elderly and children, traditional societies, subsistence farmers, and coastal populations (high confidence, Confalonieri et al. 2007).

Comment (5-14):
Commenters (3226, 3248, 4036.5) cite and submit a 2009 report published in *Lancet* (Costello et al., 2009). Similarly, a commenter (3226) submits an article from the Grist Web site entitled "New Lancet Report on Health and Social Effects of Climate Change" (Roberts, 2009). The commenters highlight the importance of the findings in the report with regard to public health effects from climate change.

Specifically, the commenters note from the report that "climate change is the biggest global health threat of the 21st century."

Response (5-14):
EPA has carefully reviewed the cited reference and notes that it relies heavily on IPCC (2007) reports and other sources of information summarized in the TSD. We note that the although the report (Costello et al., 2009) focuses on global health and the TSD has a primary focus on U.S. impacts, the overall conclusions on impacts and effects to human health and vulnerable populations are generally consistent with the TSD.

Comment (5-15):
A commenter (3574.1) indicates that ozone is detrimental to human health and references a book as evidence of impacts to lung function as a result of high ozone concentration (Casarett and Doull, 1991).

Response (5-15):
We have reviewed the reference and note the well-documented effects of ozone to lung function at concentrations of sufficient levels. The TSD notes climate change is expected to increase regional ozone pollution, with associated risks in respiratory illnesses and premature death.

Comment (5-16):
Several commenters (0296, 0489, 1470, 1529, 1548, 1582, 1586, 1672, 1807, 3280.1, 3501, 10298, 11249) express their support for the findings (and/or strong action on climate change) and describe various health impacts resulting from climate change, including increased heat-related illnesses and mortality, respiratory diseases, and both vector- and water-borne infectious diseases. Several commenters (2040, 3680.1, 4171) note that multiple and severe effects of climate change on health have been documented by numerous governmental and scientific bodies, including IPCC, CCSP, the World Health Organization (WHO), and/or the American Public Health Association. Several commenters (4171, 11249) provide WHO statistics regarding the number of annual deaths attributable to climate change and note that this number is expected to rise.

Response (5-16):
EPA has carefully considered the commenters' views, many of which are based on the same sources of information relied upon by to summarize science impacts and risk from climate change in the TSD. As stated in the findings, the Administrator has evaluated various health impacts, both direct (temperature effects) and indirect (e.g., changes in climate-sensitive diseases) that occur through human-induced climate change. For more specific information, see our responses to comments on topics throughout this volume of the Response to Comments document.

Comment (5-17):
A commenter (6749) states that environmental factors contribute to a multitude of cancers, and expressed concern that clean air, nutrient-rich foods, and clean drinking water are threatened by climate change. One commenter (6733) notes an increase in skin cancers resulting from ozone depletion.

Response (5-17):
Summarizing the assessment literature, Sections 7 and 8 of the TSD summarize health effects associated with climate change and attendant changes in air quality. The assessment literature does not highlight changes in cancer as a likely effect of climate change.

Comment (5-18):
A commenter (0293) indicates that reducing atmospheric CO_2 would have the co-benefit of reducing the disease burden of criteria pollutants, hazardous air pollutants, and other toxics, including cardiovascular disease, pulmonary disease, cancer, neurodegenerative disorders, immunosuppression, premature birth, and infant mortality.

Response (5-18):
See the Findings, Section III.C, "Adaptation and Mitigation," for our response to comments on the treatment of adaptation and mitigation in the Finding.

Comment (5-19):
A commenter (3921.1) submits the first page of a research paper by Joachim Bormann entitled "Memantine is a potent blocker of N-methyl-D-aspartate (NMDA) receptor channels" (1989). The commenter provides no comments in association with the reference.

Response (5-19):
EPA confirms review of the submitted reference; however, it does not appear to address any elements of climate change science discussed in the TSD or the Findings.

Comment (5-20):
A commenter (3501) indicates that dust storms and forest fires obviously have their own destructive impacts on the economy, the ecosystems, and the aesthetics of the region. But they also have an impact on public health.

The commenter indicates that air pollution from forest fires and dust storms has become commonplace in the West for several months of the year, even sometimes in the winter, and notes that recent studies have demonstrated that PM air pollution from forest forests may be much more toxic than that from typical fossil fuel combustion. The commenter also states it is well known that forest fires generate atmospheric mercury.

Response (5-20):
We note the commenter does not provide specific references for studies demonstrating that air pollution from forest fires may be much more toxic than that from typical fossil fuel combustion, or that forest fires generate atmospheric mercury. The climate change assessment literature does not discuss these issues and they are, therefore, not summarized in the TSD. However, we agree with the commenter that forest fires can have an impact on public health, and this is well-documented in the assessment literature.

Summarizing the assessment literature, Section 7(b) of the TSD notes that wildfires can increase eye and respiratory illnesses and injuries, including burns and smoke inhalation (Ebi et al., 2008). Further evidence comes from a study cited by IPCC (Confalonieri et al., 2007), which indicates that large fires are also accompanied by an increased number of patients seeking emergency services for inhalation of smoke and ash. Section 8(b) of the TSD provides additional evidence of the effects of PM from forest fires. For example, PM emissions from forest fires can contribute to acute and chronic illnesses of the respiratory system, particularly in children, including pneumonia, upper respiratory diseases, asthma, and chronic obstructive pulmonary diseases (Moore et al., 2006; Confalonieri et al., 2007).

Comment (5-21):
One commenter (3280.1), in supportive comments regarding the Findings, specifically discusses several expected challenges to the health and welfare of children due to climate change. The commenter provides a summary of recent studies and statements from organizations and leading officials that detail children's physical and social climate change health risks. The commenter lists several examples including respiratory diseases, heat stroke, increased rates of malnutrition, and allergies.

Response (5-21):
We reviewed the materials submitted and find them generally consistent with the information summarized in the TSD. We note that most of the studies and statements submitted (e.g., Frumkin, 2008; American Academy of Pediatrics, 2008; Bunyavanich et al., 2003) extensively cite the assessment literature to support their findings.

In Section 7's summary of the assessment literature (Field et al., 2007), the TSD notes that for heat waves populations most vulnerable to illness and death include the very young (children). Furthermore, in Section 8(b), the TSD (summarizing Confalonieri et al., 2007) states: "Particulate matter emissions from forest fires can contribute to acute and chronic illnesses of the respiratory system, particularly in children, including pneumonia, upper respiratory diseases, asthma, and chronic obstructive pulmonary diseases."

See other responses to comments in this volume for information on additional effects on human health from climate change.

Comment (5-22):
Many commenters (e.g., 1156.1, 1672, 3383.1,, 3455.1, 3570.1, 3893, 3995, 4171, 4184, 9786, 10809, 10838) state their support for the findings, noting observed and future changes in heat waves and heat stress as one of the environmental effects of climate change. Some commenters (e.g., 2263, 11249) state concern for an increase in heat-related illnesses due to global warming associated with heat exposure, including heat cramps, heat syncope (fainting), heat exhaustion, and heatstroke.

Response (5-22):
While the commenters do not provide specific supporting information, we have carefully considered their views and statements regarding health effects of increasing temperatures and future changes in heat waves and heat stress. We note in the TSD that the IPCC projects that severe heat waves are expected to intensify in magnitude and duration over the portions of the United States where these events already occur, with potential increases in mortality and morbidity, especially among vulnerable populations (e.g., elderly, young and frail). We note that these and other findings in the TSD support the commenters' views with respect to temperature effects and changes in climate.

Comment (5-23):
A commenter (3347.3) alleges that EPA's analysis of the temperature effects is flawed and violates the Information Quality Act. The commenter contends that EPA is presenting a biased scientific record suggesting that potential rising temperatures may cause adverse local or regional public health risks, while ignoring equally strong or stronger evidence that rising temperature would provide offsetting reductions in public health risk, equal or larger in magnitude according to the highest-quality scientific evidence.

Response (5-23):

Please see Volume 1, Section 1.5 of this Response to Comments document for EPA's general response to the information quality concerns submitted during the public comment process. The science upon which the Administrator relied, including a discussion of how the literature was identified, is discussed in Section III of the Findings, and our response to comments on this can be found in Volume 1 of the Response to Comments document. This section also describes our treatment of new and additional studies that are not incorporated into the assessment literature. EPA's approach is fully consistent with EPA's IQA guidelines in accordance with sound, transparent and objective scientific practices.

For specific discussion of the balance of warming-related temperature effects and related scientific evidence, please refer to responses in the rest of this section, and in particular, responses 5-24 and 5-27.

Comment (5-24):

Several commenters (e.g., 3347.3, 11453.1, 3187.3) note that the April 2009 TSD indicated that cold-related deaths presently exceed heat-related deaths in the United States and that this provides evidence that a warming climate will have beneficial effects on temperature-related mortality. Commenters note that on page 70 of the April 2009 TSD, EPA states that 5,983 heat-related deaths were reported in the United States between 1979 and 2002. In the same timeframe, 16,555 people died of extreme cold. A commenter (3187.3) provides a paper by Goklany (2007), which indicates that death from extreme cold exceed death from extreme heat.

Response (5-24):

We have revised the TSD's estimates of heat-related deaths based on the latest findings of the assessment literature (Karl et al., 2009). Based on these results, other supporting evidence presented in the TSD, and additional evidence cited below, we have determined that the available literature strongly supports the conclusion that extreme heat is, on an average annual base, the leading cause of weather-related death in the United States. We agree that the April 2009 TSD contained statistics that could be interpreted as suggesting that cold-related mortality has recently been higher in the United States than heat-related mortality. The cold-related mortality statistics in the TSD from Ebi et al. (2008) are similar to those cited by Goklany (2007). However, the methods and data for estimating heat-related mortality were recently updated and these revised values are presented in Karl et al. (2009).

The more recent heat-related mortality numbers from Karl et al. (2009) reflect results from the Centers for Disease Control and Prevention (CDC). CDC (2006) reports more than 3,400 deaths from 1999 to 2003 for which exposure to extreme heat was listed as either a contributing factor or the underlying cause of death. This result of roughly 680 heat-related deaths per year is almost identical to the 689 deaths per year from cold exposure reported by Ebi et al. (2008) and summarized in the TSD. CDC (2006) suggests that even the revised heat-related mortality numbers may underestimate total heat-related mortality, noting: "Because heat-related illnesses can exacerbate existing medical conditions and death from heat exposure can be preceded by various symptoms, heat-related deaths can be difficult to identify when illness onset or death is not witnessed by a clinician. In addition, the criteria used to determine heat-related causes of death vary among states. This can lead to underreporting heat-related deaths or to reporting heat as a factor contributing to death rather than the underlying cause." This issue has long been recognized in attempting to estimate the mortality impact of extreme heat using information from death certificates (American Medical Association Council on Scientific Affairs, 1997). As noted in a subsequent response (5-29), cold-related deaths are likely also underestimated. One complication with these death certificate–based estimates of extreme cold and heat is they are not limited to periods that would be considered heat waves or cold snaps in the location where the death occurs. Therefore, while these results are based in a consistent methodology and data source, they have an uncertain overlap with the occurrence of the weather events of primary interest to the TSD, cold snaps and heat waves. As a result, these data alone do not provide strong evidence the heat-related mortality is presently greater than cold-related mortality.

However, we note that alternative and much higher estimates of heat-related mortality come from analyses of daily urban summertime mortality patterns in Kalkstein and Greene (1997) and Davis et al. (2003a), which use a different methodology to compute heat-related deaths compared to CDC (2006). These studies first define extreme heat events by identifying threshold conditions for an event in a location and then calculate the number of extreme heat–attributable deaths based on differences in daily deaths on extreme heat days compared to longer-term averages. In these studies, heat's mortality impact is quantified in terms of the *excess deaths* that result during the extreme heat conditions. By evaluating changes in daily deaths attributable to all causes, this approach also effectively eliminates differences or restriction in using certain causes of death as potential sources of bias in estimating the extreme heat's mortality impact. This method is also more consistent with the view that heat waves are effectively identified through exceptional weather conditions that result in increases in daily mortality (e.g., Confalonieri et al., 2007; U.S. EPA, 2006a). Although differences in the time series, definitions of urban populations, and other analytical methods prevent an exact comparison of the results in these two studies, both studies (Kalkstein and Greene, 1997; Davis et al., 2003a) estimate that there are approximately 1,700–1,800 excess deaths per year during extreme heat events based on an evaluation of a subset of approximately 40 U.S. metropolitan areas (see U.S. EPA, 2006a). These estimates of extreme heat's mortality impact are much higher than the corresponding death certificate–based estimates for heat as well as the Ebi et al. (2008) estimate for cold-related mortality summarized in the TSD.

We also note that Davis et al. (2004) find that the net impact of the observed temperature increase from 1964 to 1998 (considering both reduced temperature mortality in winter and increased temperature mortality in summer) was an extra 2.9 deaths (per standard million) per city per year in 28 major U.S. cities. This indicates that extreme heat has been the larger cause of mortality in the recently observed record when temperatures have warmed.

Furthermore, we note that the USGCRP assessment (Karl et al., 2009) specifically refers to a recent study by Borden and Cutter (2008), which concludes heat is the most deadly natural hazard in the United States. It also cites Medina-Ramon and Schwartz (2007), which found that in 50 U.S. cities between 1989 and 2000, extreme heat increased death rates 5.7% while extreme cold increased death rates by only 1.6%. These results are summarized in the TSD.

Though we are aware of a recent study by Andersen and Bell (2009) that finds a similar mortality risk for extremely hot and cold days based on the synthesis of results from 107 U.S. communities (contrasting with Medina-Ramon and Schwartz), Andersen and Bell are clear that cold temperatures more indirectly affect mortality than heat. In addition to the longer lag times for exposure incorporated for the effects of extreme cold (up to 25 days, compared a one-day lag for heat), they note that infectious diseases, which are more common in industrialized countries during colder weather (when people spend more time indoors and in proximity) could account for a substantial portion of the cold-related effect.

Summarizing, both recent studies and the assessment literature provide strong evidence that heat-related mortality presently exceeds cold-related mortality in the United States.

Comment (5-25):

A commenter (3347.3) argues that key studies used by EPA to show higher mortality relating to increasing temperature rely on incorrect estimation methods. The comment refers to EPA's Excessive Heat Events Guidebook (U.S. EPA, 2006a), which discusses estimates of heat-related mortality in a set of U.S. cities from Kalkstein and Greene (1997) and Davis et al. (2003a). It notes that despite the similarity in the aggregate results of those studies (obtained by adding the mortality totals for the cities analyzed), there are large differences in the results of the individual cities, which suggests balance was achieved by

trading off mortality between cities. The commenter concludes that the mortality results are therefore sensitive to the methodology and unreliable.

Response (5-25):
We disagree with the commenter's conclusion about the reliability of these study results. The methods and results of these studies are legitimate. Both studies were peer-reviewed and cited in the assessment literature (Kalkstein and Greene, 1997, was cited in IPCC's Third Assessment Report, McMichael et al., 2001; Davis et al., 2003a, was cited in IPCC's Fourth Assessment, Confalonieri et al., 2007, and in CCSP, Ebi et al., 2008). We also note that in other comments (see 5-36 and 5-37) the commenter extensively relies on the Davis et al. (2004) study which, in fact, is dependent on the allegedly "unreliable" Davis et al. (2003a) study.

While noting that the studies share a similar methodological approach in terms of quantifying the mortality impact of extreme heat based on estimates of the excess mortality, we also have recognized the city-specific results appear sensitive to the detailed differences in methodologies employed as well as other factors (note that U.S. EPA, 2006a, states that there are "differences in the time series, definitions of urban populations, and other analytical methods that prevent an exact comparison between the studies").

Regardless of these specific differences, the bottom line is that, using an excess mortality–based approach, both studies (further discussed in response 5-24) produce heat-related mortality totals that are substantially larger than the death certificate-based results described in response 5-24.

Comment (5-26):
A commenter (3347.3) suggests that since deaths are expected following a heat wave, they are therefore more likely to be reported as heat-related. The comment contends that this results in an overestimation of mortality attributed to heat waves.

Response (5-26):
We strongly disagree with this comment. The comment directly conflicts with statements from CDC (2006) with respect to the death certificate–based estimates of the mortality impact of extreme heat.

Response 5-24 summarizes CDC's (2006) assessment of the difficulties in accounting for heat-related deaths that may lead to underestimates using death certificate information.

This comment also has no validity with respect to estimates of the mortality impact of extreme heat that rely on estimates of excess mortality based on all-cause mortality estimates. In these studies (e.g., Davis, 2003a, Kalkstein and Greene, 1997) differences in all-cause mortality during a period of extreme heat are compared with mortality levels during a reference period. As a result, the methodology effectively controls for possible bias in assigning the cause of death by considering all deaths on a day.

Comment (5-27):
Many commenters (e.g., 3347.3, 3449.1, 11453.1) indicate that EPA has not demonstrated that increased mortality from heat will exceed decreased mortality from cold in a warming climate and/or that EPA concedes that the balance is uncertain and additional research is needed to better understand this issue. Some commenters present literature (e.g., Keating et al., 1997; Campbell-Lendrum et al., 2003) that, they find, suggests that reduced deaths from cold will outnumber increased deaths from heat in a warming climate.

Response (5-27):

The TSD is very clear that the assessment literature (Confalonieri et al., 2007; Ebi et al., 2008; Karl et al., 2009) finds that the exact balance between increases in heat-related mortality and decreases in cold-related mortality is uncertain in a warming climate. However, we note that the latest USGCRP assessment (Karl et al., 2009) cites recent research by Medina-Ramon and Schwartz (2007) that concludes that increases in heat-related mortality due to global warming are unlikely to be compensated for by decreases in cold-related mortality, based on the finding that heat currently results in higher death rates than cold in the United States. Additional support for the conclusion that heat is, in fact, the leading cause of temperature-related mortality in the United States is given in response 5-24.

Our review of relevant recent literature and the studies submitted by commenters does not identify flaws in our characterization of this issue in either the TSD or the Findings. Recent (post-2001) studies that estimate the possible future effects of temperature-related mortality provide varying results:

> Many commenters submitted comments that discuss observed temperature-related mortality trends and projections in Europe (e.g. Keating et al., 1997). Those studies are generally not germane to the United States for reasons discussed in response 5-39 and 5-40.
> Campbell-Lendrum et al. (2003) project no or a slightly negative net change in the risk of temperature-related mortality by 2030 for developed countries under several emissions scenarios and varying assumptions about acclimatization. For additional details, refer to response 5-37.
> Davis et al. (2004), who found the net effect of observed increases in temperature between 1964 and 1988 had resulted in a net increase in mortality (see response 5-24), project mortality results in the future will vary depending on the seasonal distribution of warming. They project that a uniform 1°C warming (equal warming in summer and winter) would result in a net mortality decline of 2.65 deaths (per standard million) per metropolitan area analyzed, a summer-dominant warming would generate a net increase in mortality of 3.61 additional deaths (per standard million), and that a winter-dominant warming would produce a net decrease in mortality of 8.92 fewer deaths (per standard million).
> McMichael et al. (2006) suggest that additional heat-related deaths in summer would outweigh the extra winter deaths averted globally in 2050 (refer to Figure 2 in that study).
> Deschenes and Greenstone (2007) find under a "business as usual" scenario, climate change will lead to an increase in the overall U.S. annual mortality rate ranging from 0.5% to 1.7% by the end of the 21st century (depending on choice of climate model).
> Older studies which project future changes in net mortality are discussed in response 5-28.

Most of the studies above examine temperature changes across the year and then evaluate seasonal mortality relationships, which are incorporated to analyze the climate change impact or a summary of impacts across all temperatures. To quantitatively address the risk associated with the changes in extreme temperature events in a warming climate which tend to be most lethal, it would be necessary to project future changes in extremes while holding current conditions constant with respect to thresholds for extreme heat and cold and the associated mortality response, taking into account regional variation. The current literature lacks such an analysis.

Comment (5-28):

Commenter (3747.1) indicates that EPA removed from an earlier version of its TSD a statement describing how a majority of studies on the subject showed a larger decrease in cold-related deaths than increase in heat-related deaths.

Response (5-28):

The version of the TSD the commenter is referring to (from the June 28, 2008, ANPR) stated the following:

> In the TAR [Third Assessment Report], the IPCC cited several studies that indicate decreases in winter mortality may be greater than increases in summer mortality in some temperate countries under climate change (McMichael et al, 2001). However, it cites one U.S. study (Kalkstein and Greene, 1997) that estimates increases in heat-related deaths will be three times greater than decreases in cold-related deaths. Given the paucity of recent literature on the subject and the challenges in estimating and projecting weather-related mortality, IPCC concludes additional research is needed to understand how the balance of heat-and cold-related deaths might change globally under different climate scenarios (Confalonieri et al, 2007).

We decided to remove the first two sentences of this statement for two primary reasons:

1) Of the five studies assessing net temperature-related mortality changes in IPCC's Third Assessment Report, only two contained results applicable to the United States. One study showed a net decrease in mortality (Martens, 1998) and the other showed a net increase in mortality (Kalkstein and Greene, 1997, as described above) under climate change scenarios. So for the U.S., the studies in IPCC's Third Assessment Report did not provide a clear picture with respect to the balance of heat and cold-related deaths. The other studies indicating temperature-related mortality would decline pertained to Europe (Donaldson et al., 2001; Langford and Bentham, 1995) and Australia (Guest et al., 1999).
2) In the April 2009 TSD, we focused on the most recent assessment literature, and relied primarily on IPCC's Fourth Assessment Report (as opposed to the Third Assessment Report) as well as findings from the most recent assessment literature from CCSP/USGCRP (Ebi et al., 2008; Karl et al., 2009).

Note that the third sentence in the above statement remains in the TSD.

Comment (5-29):
A commenter (3347.3) notes the TSD fails to include the Ebi et al. (2008) statement finding that cold-related mortality is likely underestimated.

Response (5-29):
We have added to the TSD the statement that "Cold also contributes to deaths caused by respiratory and cardiovascular diseases, so the overall mortality burden is likely underestimated (Ebi et al., 2008)."

Comment (5-30):
Commenters (3411.1, 3347.3, 11348,) suggest that direct effects of possible climate warming on temperature-related mortality are likely to be beneficial due to increases in life expectancy.

Referencing Deschenes and Moretti (2009),[1] commenters (3347.3, 11348,) suggest the increased longevity that has arisen from migrations of large populations from the northern to the southern United

[1] Some commenters referred to Deschenes and Moretti (2007), whereas others referred to Deschenes and Moretti (2009). These are essentially the same studies. The 2007 paper was published as a working paper; the 2009 paper was published in the *Review of Economics and Statistics.* It is cited as Deschenes and Moretti (2009) in these responses to comments.

States illustrates the benefits of increasing temperatures. A commenter notes that there are similar effects beginning in Europe.

Response (5-30):
We find that the commenters are drawing inappropriate conclusions from future warming and migration trends and inappropriately applying the results from Deschenes and Moretti (2009).

The assessment literature does not explicitly address temperature effects on life expectancy or longevity. While we have reviewed Deschenes and Moretti (2009) and do not dispute migration to a warmer climate may have longevity benefits (whether in the United States or in Europe), we reject consideration of the estimated benefits from migration to a warmer climate as an appropriate analog to warming from GHG-induced climate change. The fundamental reason for this is that in estimating the health effects of climate change scenarios, individuals are explicitly assumed to stay in the same location (and the choice to migrate is a planned, adaptive response to temperature; see the Findings, Section III.C, "Adaptation and Mitigation," for our response to comments on the treatment of adaptation and mitigation in the Findings).

The assessment literature, as summarized in the TSD, is clear that local factors, such as climate, topography, heat-island magnitude, demographic and health characteristics of the population, and policies that affect the social and economic structures of communities, including urban design, energy policy, water use, and transportation planning, are important in determining the underlying temperature-mortality relationship in a population. In studies, mid-latitude metropolitan areas with greater summertime temperature variability have shown stronger adverse health responses to heat than desert and semi-tropical cities with less variable weather (e.g., Chestnut et al., 1998; Media-Ramon and Schwartz, 2007; Gosling et al., 2007, Davis et al., 2003b).

Importantly, the longevity increase is expected to be the result of many variables and societal factors associated with the more southern areas that are associated with increased average temperature, making it hard to isolate the impacts of average temperature. Also, the health effects at issue are not associated with the increase in average temperature itself, but with the increased likelihood of changes in temperature extremes.

Comment (5-31):
Commenters (3347.3, 3394.1, 3411.1, 11348) refer to Deschenes and Moretti (2009) and other studies (e.g., Kunst et al., 1993; Campbell-Lendrum et al., 2003) which indicate that heat deaths often represent a short temporal displacement but deaths due to cold usually result in months to years of life lost.

Response (5-31):
We disagree with the commenter's suggestion that there is a consensus with respect to the extent of mortality displacement that results from extreme heat or cold.

"Mortality displacement" refers to forward temporal shift in the rate of mortality in a given population resulting from an environmental stressor—often heat or cold. It typically describes a situation where weakened people die a few days or weeks sooner than they would have under ordinary circumstances. (Such displacement is sometimes also described as "harvesting.")

Deschenes and Moretti (2009) use fixed temperature thresholds to define cold and hot days in all locations across the United States. They then find that, while hot days appear to have significantly increased mortality rates in the days immediately following the event, there is no signal of a lasting mortality impact of these hot days after 30 days. Their results for cold days are the opposite. The authors interpret these results as indicating that the short-term increases in mortality on hot days are offset in the

longer run by mortality displacement while cold days have a lasting impact in terms of increasing mortality rates.

However, our review of the literature (including Kunst et al., 1993; Campbell-Lendrum et al., 2003) revealed considerable evidence that the concept of temperature-related mortality displacement is complex and observed impacts and estimates of the extent of mortality displacement may depend on the population/location affected and the characteristics of the heat or cold event. For example, Davis et al. (2004) caution that mortality displacement is not a universal feature in all cities, and the topic has not been examined systematically in the United States with respect to heat. Similarly, Kalkstein (1997) develops a range of estimates for discrete heat events in U.S. cities and finds varying signals with respect to the importance of mortality displacement.

In addition, Medina-Ramon and Schwartz (2007) find evidence of short-term displacement for less extreme heat events in the United States but little evidence for short-term mortality displacement for extreme heat events.

Studies following the European heat wave of 2003 found no evidence of short-term mortality displacement in France (Pirard et al., 2005) or Spain (Simon et al., 2005), while others have estimated that mortality displacement may have accounted for a very small fraction of the excess deaths in this event. Specifically, Le Tertre et al. (2006) estimated that less than 10% of the 3,000 excess deaths from nine cities in France could be attributed to mortality displacement, although the percentage varied widely among the individual cities.

These studies would suggest that the individuals who died still had substantial periods of life remaining.

The uncertainties surrounding the issue of mortality displacement and differing assumptions made about it contribute to the variability in projections of net mortality described in response 5-27. We note that Deschenes and Greenstone (2007), who explicitly apply methodologies to account for displacement, still project net increases in temperature-related mortality and decreases in longevity in the United States under future climate change scenarios. They indicate that, had they not accounted for the harvesting effects of heat and the fact that there is often a delay or lag between cold exposure and health impacts, the direct mortality impacts would have been overstated by roughly 47% to 202% (depending on the climate model).

Most importantly, the discussion of mortality displacement with extreme heat and cold recognizes that these events result in observable increases in the daily mortality levels of the populations experiencing the conditions over different time periods.

To summarize: given the uncertainty over the distribution and magnitude of mortality displacement, the importance we assign to mortality, the other evidence of the relative magnitude of heat versus cold mortality, and the precautionary nature of the Findings, we are not discounting the heat wave mortality risk in any fashion.

Comment (5-32):
A commenter (3136.1) notes: “Just because the IPCC doesn’t cite any studies looking at cold vs. heat related deaths in the U.S. in the future doesn’t mean that they don’t exist. For instance, the subject of Davis et al. (2004) is precisely this topic.”

Response (5-32):

The TSD does not cite results from individual studies unless the specific results have been addressed in the assessment literature. We discuss the mortality projections in Davis et al. (2004) in response 5-27.

Comment (5-33):
A commenter (3347.3) states that researchers have found that for heat waves, an increase above average mortality rates sometimes occurs, followed by a counterbalancing decrease in the average mortality rates in the following days to a few weeks, and that there is no statistical net effect in mortality incidence from exposure to heat waves (Campbell-Lendrum, 2003; Kalkstein, 1993; Huynen et al., 2001; Deschenes and Moretti, 2009).

Response (5-33):
We disagree that the evidence suggests that heat waves result in no statistical net effect in mortality incidence. The body of literature cited by the commenter does not support such an assertion.

Due to the mortality displacement described in response 5-31, it is true that the literature suggests there is *sometimes* a counterbalancing decrease in the average mortality rates in the days to few weeks following a heat wave. But "sometimes" is the key qualifier, as the literature contains examples in which there is no evidence of mortality displacement as described in response 5-31.

We reviewed all five studies submitted by the commenter and found that—with one exception—they do not conclude that heat waves result in no statistical net effect in mortality incidence.

> Campbell-Lendrum (2003) reports: "There also is evidence for a 'harvesting effect,' i.e. a period of unusually lower mortality following an extreme temperature period. This indicates that in *some cases extreme temperatures advance the deaths of vulnerable people by a relatively short period*, rather than killing people who would otherwise have lived to average life expectancy. However, this effect has not been quantified for temperature exposures and is not included in the model." (Italics indicate emphasis added.)
>
> Kalkstein (1993) reports results from two U.S. cities (New York and St. Louis) that indicate that "most extra mortality during heat episodes does reflect deaths that would not have happened shortly afterwards had there been no heat wave."
>
> Huynen et al. (2001) report: "Our results relating to the heat-induced forward displacement of deaths are inconclusive. Some heat waves show a decline in mortality in the longer lag periods after the extreme heat, which suggests that heat has a harvesting effect, whereas others do not show this decline in the number of deaths."
>
> Though Schwartz et al. (2004)[2] conclude that "much of the effect of hot temperatures is short-term displacement of events," their study is examining morbidity as opposed to mortality.

Deschenes and Moretti (2009) do conclude that the "increase in mortality that occurs in the days immediately following heat waves appears entirely driven by temporal displacement" and that as a result "there is virtually no lasting impact of heat waves on mortality." However, the authors caution: "Of course, every death is harvesting, because we all eventually die. The point here is that in a heat wave, some individuals die only a few days earlier than they would have, not a few months or years earlier." As discussed in response 5-27, Deschenes and Greenstone (2007) project net increases in temperature-related mortality and life years lost under a warming climate in the United States despite the short mortality displacement for heat deaths.

[2] The comment cites Schwartz et al. (2007); the commenter's bibliography, which we believe is the accurate citation, refers to Schwartz et al. (2004).

In addition, there are other studies (e.g., Medina-Ramon and Schwartz, 2007; Le Tertre et al., 2006) whose results suggest that mortality displacement may play a relatively insignificant role in the observed excess mortality following a period of exceptional heat (see response 5-31)

Considering all of these studies and those studies reviewed in response 5-31, we do not find the commenter's statement that there is no net effect in mortality incidence from exposure to heat waves is supported by the evidence.

Comment (5-34):
A commenter (3347.3) states that EPA is incorrect in suggesting that cold-related deaths are overestimated because it inappropriately considers influenza. The commenter refers to a study finding that influenza-like conditions are strongly correlated with low temperatures (Huynen et al., 2001), a study predicting that influenza will decrease with a warming climate (McMichael and Beers, 1994), and a study finding that controlling for influenza only diminished the aggregate mortality effect of cold weather by 34% (Kunst et al., 1993).

Response (5-34):
The commenter has misread the TSD: we do not state that cold-related deaths are overestimated and our review of the literature suggests that the strength of the association between temperature and influenza is uncertain.

Summarizing the assessment literature (Ebi et al., 2008; Confalonieri et al., 2007), the TSD states that "Projections of cold-related deaths, and the potential for decreasing their numbers due to warmer winters, can be overestimated unless they take into account the effects of season and influenza, which is not strongly associated with monthly winter temperature." This statement reflects the fact that there are important uncertainties in projecting cold-related mortality relating to the association between cold and influenza.

The CCSP assessment on health (Ebi et al., 2008) states: "…many factors contribute to winter mortality, making the question of how climate change could affect mortality highly uncertain. No projections have been published that incorporate critical factors such as the influence of influenza outbreaks." This information has been incorporated into the TSD.

Regarding the literature submitted by the commenter, it does not provide evidence that there is stronger association between cold and influenza than implied by the TSD. Huynen et al. (2001) note that cold influences influenza "indirectly" and that "the fact that we were not able to correct for the effects of influenza is one of the flaws in our models." We note that McGeehin and Mirabelli (2001) state: "influenza contributes to, but does not fully explain, the association between winter temperature and mortality."

We also reviewed McMichael and Beers (1994) and do not dispute their qualified statement that winter deaths from influenza "could be expected to decrease" with global warming. However, we note the study offers no details, original analysis, or assessment to accompany this statement.

The Kunst et al. (1993) reference which finds that controlling for influenza incidence diminishes the aggregate mortality effect of cold weather by 34% clearly supports the finding of the assessment literature that not taking into account influenza in projections of cold-related mortality will result in an overestimate of its effects.

In summary, the assessment literature as summarized in the TSD appropriately characterizes the uncertain relationship between influenza and cold and the importance of accounting for this in projections of cold-related mortality associated with climate change.

Comment (5-35):
A commenter (3347.3) states that EPA treats sensitive and susceptible subpopulations differently with respect to impacts from heat and cold. The commenter notes that EPA lists the vulnerable populations for heat but not for cold. They refer to Deschenes and Moretti (2009), which find that susceptible populations for excess cold include older adults, the mentally ill (e.g., homeless), those working outdoors, and the socially isolated.

Response (5-35):
We disagree that we do not identify populations vulnerable to excessive cold. Citing the assessment literature (Confalonieri et al., 2007), the TSD states: "Accidental cold exposure occurs mainly outdoors, among socially deprived people (e.g., alcoholics, the homeless), workers, and the elderly in temperate and cold climates, but cold waves also affect health in warmer climates." These are essentially the same susceptible groups identified by the commenter and Deschenes and Moretti (2009).

Comment (5-36):
Many commenters (e.g., 3136.1, 3187.3, 3283.1, 3347.3, 3449.1:8, 3596.1, and 4632) indicate that technological changes and adaptation have already reduced heat-related health effects and will sufficiently reduce an increase in heat-related health effects in the future. Many refer to literature (e.g., Kalkstein and Davis, 1989; Lerchl, 1998; Davis et al., 2004) that discusses adaptation to heat and/or its benefits. Commenters also indicate that humans physiologically adapt or acclimatize[3] to hotter climates over time, citing literature (e.g., Harrison, 1998; Kovats and Jendritzky, 2006) as evidence.

Response (5-36):
The assessment literature indicates that physical acclimatization to heat, adaptive measures, and technology reduce the human health impact of heat and will continue to do so in the future, but it is nonetheless indisputable that heat presently causes substantial mortality in the United States. The assessment literature clearly concludes the risk of adverse health effects from heat will increase as the climate warms. Therefore, we strongly disagree that technological changes, physical acclimatization, and adaptation will "sufficiently" reduce heat-related health risks in the future.

We have reviewed the literature demonstrating the observed (e.g., Palecki et al., 2001; Fouillet et al., 2008; Davis et al., 2003a, 2003b) and/or projected (e.g., Guest et al., 1999) benefits of adaptive measures in terms of reduced adverse health effects from heat. Many of the studies submitted by commenters are cited in the assessment literature and a following key conclusion that is drawn is stated in Section 7 of the TSD: "…human health risks from climate change will be strongly modulated by changes in health care, infrastructure, technology, and accessibility to health care (Field et al., 2007)."

We also do not dispute that there is evidence (e.g., Kalkstein and Davis, 1989; Harrison, 1998; Kovats and Jendritzky, 2006) that humans physiologically adapt (or acclimatize) to hotter climates over time. In fact, the TSD, citing the assessment literature, states: "Estimates of heat-related mortality attributable to

[3] "Heat acclimatization" refers to biological adaptations that reduce physiologic strain (e.g., heart rate and body temperature), improve physical work capabilities, improve comfort, and protect vital organs (brain, liver, kidneys, muscles) from heat injury.

climate change are reduced but not eliminated when assumptions about acclimatization and adaptation are included in models."

Furthermore, we agree technology (e.g., less climate-sensitive architecture and air conditioning) can reduce the health impacts of heat but it is not clear that it can eliminate them. Refer to additional discussion of air conditioning in response 5-52.

However, despite the presence of physical acclimatization, technology, and adaptive measures, it is indisputable from the health statistics cited in the TSD (as summarized from the assessment literature) that heat kills. In fact, heat is the leading cause of temperature-related mortality in the United States, as stated in the assessment literature (Karl et al., 2009), summarized in the TSD, and discussed in response 5-24.

Under future climate change scenarios in which the United States warms, the argument that increased risk from hotter temperatures will be compensated by technology (e.g., air conditioning) and/or adaptation (which are interrelated) is not relevant in the context of these Findings. See the Findings, Section III.C, "Adaptation and Mitigation," for our response to comments on the treatment of adaptation and mitigation in the Findings. Commenters do not present evidence that acclimatization alone will compensate for heat-related health effects that have been projected into the future. Physical acclimatization, which is a form of autonomous adaptation, is considered relevant. However, a recent study (Kinney et al., 2008) indicates the timescales over which individuals or communities acclimate to local weather is not well understood.

Comment (5-37):
A commenter (3347.3) argues that U.S. society has changed; therefore, past relationships between climate and mortality are not a reliable predictive basis for the future. The commenter refers to a Davis et al. (2004) study, which finds that on average for the 28 cities analyzed in that study, the number of excess deaths on hot and humid days declined from 41.0 in the 1960s and 1970s to 17.3 in the 1980s to only 10.5 in the 1990s. The comment highlights the Davis et al. (2004) conclusions that temperature currently does not have a major influence on monthly mortality rates in the United States and that the lives saved in conjunction with warm winters tend to offset the deaths associated with warmer conditions in July and August. The comment also refers to a study by Campbell-Lendrum et al. (2003), which investigates the direct physiological effects of heat and cold on cardiovascular mortality. The study finds that when physiological adaptation and unmitigated emissions are assumed, there is no increase in mortality risk from cardiovascular disease attributable to climate change in any region of the world and a decrease in the developed world (including the United States).

Response (5-37):
We do not dispute the general notion that past relationships between climate and mortality are not necessarily a reliable indicator for the future. We recognize, as stated in the TSD, that non-climatic factors such as changing demographics will influence past as well as future trends along with adaptive measures that might be adopted—though these are not germane to the Findings (e.g., see responses 5-36 and 5-47). However, we disagree the references provided by the commenter provide strong evidence that a warming climate will not pose a risk of adverse health effects from heat in the future.

Regarding the results presented from the Davis et al. (2004) study, we agree that heat-related mortality declined from the late 1960s into the 1990s in the major U.S. cities studied and that adaptation contributed to the decline. But this decline seems to have leveled off since the mid-1990s according to Sheridan et al. (2009). Refer to response 5-48 where this is discussed further. Regarding the finding that temperature does not have a major influence on monthly mortality rates in the United States, we note that the latest health statistics indicate that there are conservatively over 680 heat-related deaths per year; refer

to response 5-24 for further discussion. Regarding the Davis et al. (2004) projection that lives saved in conjunction with warm winters tend to offset the deaths associated with warmer conditions in July and August under certain scenarios (see response 5-27), their own results from the observed record suggest that heat deaths exceeded cold deaths from 1964 to 1998 as the climate warmed (see response 5-24). In summary, we do not find that Davis et al. (2004) provide evidence that would lead us to conclude that a warming climate will not increase the risk of adverse heat-related health effects.

Reviewing Campbell-Lendrum et al. (2003), we agree that they find little net change in the risk of mortality in the scenarios presented for the year 2030. Though they note a very slight decrease in the risk of mortality for developed countries, which includes the United States, it is only after they make assumptions about physical acclimatization which they concede have "not been formally tested." Furthermore, they note important latitudinal differences that are not resolved on a country-by-country basis in their study: "Climate change is expected to affect the distribution of deaths from the direct physiological effects of exposure to high or low temperatures (i.e. reduced mortality in winter, especially in high latitude countries, but increases in summer mortality, especially in low latitudes). However, the overall global effect on mortality is likely to be more or less neutral." Though this study does not support an increased risk in temperature-related mortality in developed countries, its results are equivocal and consistent with the conclusions of the assessment literature that the net changes in temperature-related mortality are difficult to estimate. Refer to response 5-27 for additional discussion.

Comment (5-38):
A commenter (11348) asserts that the benefits in life expectancy from warming in cold periods may be much more than nine times greater than lifespan lost in warm periods.

Response (5-38):
The commenter (11348) provides no reference to support the assertion that life expectancy from warming in cold periods may be more than nine times greater than lifespan lost in warm periods. See our responses throughout this section for specific discussion of the broader issues.

Comment (5-39):
Commenters refer to studies (Kunst et al., 1993; Langford and Bentham, 1995; Keatinge et al., 1997; Martens, 1998; Keatinge et al., 2000; Keatinge and Donaldson, 2004; Deschenes and Moretti, 2009; Laaidi et al., 2006) that provide evidence of, document, or project mortality benefits in winter from warming temperatures.

Response (5-39):
We reviewed these studies and do not disagree with their findings that warmer winter temperatures reduce mortality during that season. In fact, the TSD summarizes the assessment literature conclusion that cold-related mortality is projected to decrease under future climate scenarios.

However, we also note that studies such as Medina-Ramon and Schwartz (2007) only consistently find a mortality impact for cold days across U.S. cities when cold days are defined based on the location's annual temperature distribution (i.e. for unusually cold days in that city) instead of by absolute temperature levels. This suggests that there may be limited reduction in the mortality impact of cold snaps in most U.S. locations as long as these events are defined relative to a location's weather experience.

Comment (5-40):

One commenter (11348), referencing a European study (Keatinge et al., 1997), suggests that there will be 25,000–50,000 fewer deaths in the United States per year for a 1°C temperature rise. The commenter compares this to 30,000 deaths per year from breast cancer, 30,000 for prostate cancer, or about 40,000 from motor vehicle accidents.

Response (5-40):
We do not find this comment supportable. The temperature-mortality results from specific regions in Europe—such as those from Keatinge et al. (1997)—cannot credibly be projected on the entire United States. The assessment literature, as summarized in the TSD, clearly reflects that local factors, such as climate, topography, heat-island magnitude, demographic and health characteristics of the population, and policies that affect the social and economic structures of communities, including urban design, energy policy, water use and transportation planning are important in determining the underlying temperature-mortality relationship in a population (Confalonieri et al., 2007; Ebi et al., 2008).

Climate conditions in the United States differ in important ways from Europe in that they are more variable with greater heat extremes, not to mention that there are important socioeconomic and demographic differences. In addition, Medina-Ramon and Schwartz (2007) report: "Despite a wide range of temperatures, the U.S .population seems fully acclimatised to cold temperatures, but not to heat. This may reflect the near universality of central heating in the United States, as opposed to central air conditioning, which explained a substantial fraction of the heterogeneity between cities in the heat effects."

Furthermore, the Keatinge et al. (1997) study reported estimated deaths associated with falling winter temperatures; it did not assess risks associated with rising summer temperatures, which the commenter fails to take into account. Finally, the commenter did not provide attribution for the statistics provided on death counts for breast cancer, prostate cancer, and motor vehicle accidents.

Comment (5-41):
One commenter (11348) cites a report by the Department of Health of the United Kingdom (UK) that there was no increase in heat-related deaths from 1971 to 2002 despite warming summers, suggesting that the UK population is capable of adapting to warmer conditions. The report also notes that winter deaths will continue to decline.

Response (5-41):
As discussed in previous responses (5-40), we find temperature-mortality studies from Europe should not be projected on the United States.

Furthermore, EPA reviewed the referenced report by the Department of Health of the United Kingdom; we note that—directly after the quote provided by the commenter, which indicates that "there was no increase in heat-related deaths from 1971-2002"—the report states, "But heat waves still present a serious risk" and "While, periods of very cold weather will become less common, periods of very hot weather (heat waves) will become more common." EPA notes the study projects temperature-related mortality out to 2012, yet predicts a 1 in 40 chance every year (a 1 in 4 risk in the decade centered on 2012) of a nine-day heat wave at 27°C in southeast England. The report indicates that "Without preventive action, this could cause more than 3,000 immediate deaths with more than 6,350 heat-related deaths throughout that summer (Department of Health UK, 2008)." The authors also note that "in terms of conventional thinking about risk to health a risk of 1 in 40 is high."

More directly, the 2003 European heat wave was estimated to result in over 2,000 excess deaths in England and Wales from August 4 through 13, clarifying the risk of extreme heat in the United Kingdom

(Koppe et al., 2004). These impacts also resulted in the development of a national heat strategy to address heat waves (Department of Health UK, 2009).

Regarding the report's finding of a decline in winter deaths in the future, EPA notes that this is consistent with the underlying science of the IPCC and USGCRP assessments which EPA relies upon. For instance, the USGCRP notes that the number of deaths caused by extremely low temperatures would be expected to drop (Karl et al., 2009).

Comment (5-42):
A commenter (11348) suggests that the consequences of widespread cooling are likely to be much more threatening than warming.

Response (5-42):
Climate change projections in the assessment literature do not project cooling and, therefore, the health effects of cooling are not relevant to these Findings.

Comment (5-43):
At least one commenter (11348) refers to studies by Moore (1995, 1998). The commenter claims that the 1998 study estimated that a temperature increase of 2.5°C in the United States would cause a drop of 40,000 deaths per year from respiratory and circulatory disease, based on U.S. mortality statistics as a function of monthly climate change.

Response (5-43):
We reviewed the submitted publications and note that their results specific to temperature-related mortality are not current, not supported by other peer-reviewed studies, and not cited in the assessment literature. We note that IPCC and CCSP/USGCRP assessments had several opportunities to incorporate the results of these studies since their publication and declined. Further, though Moore (1998) was published in the peer-reviewed literature, Moore (1995) was not. Refer to the Findings, Section III.A., for the science on which the decisions are based.

With respect to specific results from the Moore (1998) study, we note that the study models the relationship between monthly and annual mortality rates and monthly and annual temperatures. As the impacts of extreme heat events often manifest themselves over a series of days, this is an unsupportable assumption. This study, therefore, does not explicitly isolate extreme heat events, which are known to be associated with elevated mortality and projected to increase as a result of climate change.

Therefore, we do not agree the publications of Moore from 1995 and 1998 are relevant, nor that their findings are supportable.

Comment 5-44):
A number of commenters (e.g., 11348, 3411.1) contend that because all-cause mortality is greater in winter than summer in the United States, a warming climate will result in a net decrease in temperature-related mortality.

At least one commenter (11348) disagrees with the McMichael et al. (2006) finding that heat-related deaths will increase more than the lives saved by warming of the cold periods. The commenter refers to "previous data" showing that U.S. mortality in winter due to cardiac, vascular, and respiratory disease in

winter is seven times greater than in summer, and also that this ratio is about nine to 10 in Europe, from the data of Keatinge et al. (1997). The commenter (11348) further asserts that the most comprehensive data on U.S. daily mortality, from all causes, as a function of the day of the year, is found in Deschenes and Moretti (2009). The commenter states that these data show a clear relationship, with maximum mortality in January and minimum mortality in the warmest months of July and August. The commenter contends that these data strongly indicate that warming of average daily temperatures would cause a decrease in mortality in winter far greater than the slight increase of mortality from summer heat.

Other commenters (3187.3, 3411.1, and 4632) also reference the Laaidi et al. (2006) and/or others (i.e., Braga et al., 2002) as evidence that winter-related mortality is greater than summer-related mortality.

Response (5-44):
Though we do not dispute that all-cause mortality is greater in winter than in summer in the United States, this information alone does not and cannot substantiate that a warming climate will result in net reductions in mortality. Studies (e.g., Davis et al., 2004; Andersen and Bell, 2009) show that the direct relationship between winter temperatures and mortality is much weaker than for summer; this is especially true for infectious and respiratory illnesses. In fact, McGeehin and Mirabelli (2001) note: "Overall mortality is generally higher in winter than in summer, but there is little convincing evidence that weather patterns are solely responsible." (Regarding the comment stating that U.S. mortality is much greater in winter than in summer citing "previous data" we note this comment does not make clear which previous data it is referring to.)

Furthermore, EPA notes that the seasonality highlighted by the commenter and depicted in Figure 1 of Deschenes and Moretti (2009) is not representative of regional differences in temperature exposure (for example, see Figure 2 in the same study). These data do not convey that people in hotter cities are more affected by cold temperatures; on the other hand, it is well documented in the scientific assessment literature (Media-Ramon and Schwartz, 2007; Gosling et al., 2007; McMichael et al., 2006; Ebi et al., 2006; Curriero et al., 2002; Davis et al., 2002) that people in colder cities are more affected by warmer temperatures. This finding is also confirmed in the study referenced by the commenter (Laaidi et al., 2006), which finds that excessive heat is more likely to increase heat-related mortality in colder areas since the thermal optimum (critical temperature threshold) is lower for the population in these areas.

Accordingly, these studies do not warrant rejecting the conclusion that temperature-related mortality may increase as stated in McMichael et al. (2006) and Deschenes and Greenstone (2007) (refer to response 5-27).

Comment (5-45):
Commenters (3136.1, 3596.1) provide two side-by-side graphs of U.S. mortality trends and U.S. temperature trends from 1980 to 2006 (CDC, http://www.cdc.gov/nchs/data/nvsr/nvsr56/nvsr56_16.pdf; National Climatic Data Center, http://climvis.ncdc.noaa.gov/cgi-bin/cag3/hr-display3.pl). These graphs, state the commenters, show that we are greatly triumphing over climate change, or perhaps that a warmer climate is a healthier climate. The commenters indicate that perhaps the most direct evidence of health and welfare is mortality rates, which have improved significantly over the last 100 years, and submit a graph of all-cause mortality and U.S. mean surface temperature for the 1980–2006 period in support of this point. The commenter notes that it would take a true pessimist to think this strong trend toward decreasing mortality rates will reverse itself in the future, no matter what the climate holds in store—primarily because the impact of climate on human health in the United States is minuscule, according to findings by Davis et al. (2004).

Another commenter (2898.1) indicates that public health and welfare will continue to improve, even in a warming world. In support of this view the commenter submits a reference by Indur Goklany (2007) entitled, "The Improving State of the World: Why We're Living Together, Healthier, More Comfortable Lives on a Cleaner Planet."

Response (5-45):
EPA reviewed the graphs provided by the commenter, as well as the associated referenced literature, and finds that they do not support the commenters' conclusions with respect to heat-related mortality due to climate change.

These two graphs depict "all cause mortality" and "U.S. mean surface temperature" for the 1980–2006 period. The metrics of crude and age-adjusted mortality represented in these graphs are influenced by all known factors that lead to mortality and do not represent or isolate the role of temperature or any other individual factor. Over the period represented in the graphs, mortality has decreased in United States as a result of several factors, such as technological and medical advances, improved health care, regulatory statutes, increases in life-expectancy, and improved emergency preparedness. In addition, the general trend in average annual temperature indicates warming but does not represent the specific weather events that are of concern for heat-related mortality. As a result, the trends depicted in the graphs provide little information regarding the direct health effects associated with extreme heat exposure or temperature in general. Two graphs showing that "all cause" mortality declined do not provide evidence to support a claim that mortality from every cause declined. All they would show is that the overall morality declined, reflecting changes that reduced mortality outweighing any changes that increased mortality. It does not show what changes increased and what changes decreased mortality. Moreover, these graphs do not represent conditions in the future in which extreme heat events are expected to increase in frequency, duration, and intensity, nor the future drivers affecting vulnerability to heat-related mortality (i.e., urban heat island effects, demographic trends of older age brackets).

We do not dispute that Davis et al. (2004) find that the observed temperature-related mortality rates are very low relative to the baseline total (all-cause) death rate. However, as noted in a previous response, Davis et al. (2004) find that the net impact of the observed temperature change from 1964 to 1998 was to cause an extra 2.9 deaths (per standard million) per city per year in 28 cities, which represents thousands of total deaths.

Regarding the comment that measures to protect public health and welfare will continue to improve, even in a warming world based on evidence from Goklany (2007), we conclude for reasons stated above that this evidence does not support changing our Findings of the presence of risk or the potential for increasing risk in the future from climate change.

Comment (5-46):
A commenter (3347.3) states that Longstreth (1991) suggests that if, with global warming, populations in the northern U.S. cities became like those in the South, then full acclimatization will have occurred and little or no impact from global warming is predicted.

Response (5-46):
We find this comment to be a mischaracterization of the results of Longstreth (1991). In fact, the author states:

> Studies based on two general circulation model scenarios in urban areas that include several from North America predict that the number of heat-related deaths will double by the year 2020 and increase severalfold by 2050. These numbers may be reduced if

> changes in climate occur over an extended time so populations can become acclimatized to the new conditions (*1*,*9*). Although climate change could also bring milder winters and a drop in winter death rates, the predicted increase in death rates from hotter summers may be such that there is a net increase in deaths associated with changing climate (*21*).

This excerpt from Longstreth (1991) clearly demonstrates that the study does not state acclimatization will result in little or no impact from global warming. To the contrary, the study only suggests that there may be a decrease in the overall impact from heat stress as a result of acclimatization, and that the net effect of climate change may be to increase deaths.

Comment (5-47):

Commenters indicate the population's rapid adaptation to heat has been demonstrated after the 1995 Chicago heat wave (Palecki et al., 2001), after heat waves in Philadelphia in 1993 and 1994, and across Europe after the 2003 heat wave event (Fouillet et al., 2008). The commenter notes that it is one thing for EPA to state that summer extreme temperatures may be related to global warming; it is another to relate them to individual events without elaborating more fully. Furthermore, the commenter that notes a major adaptation to heat waves occurred in France as a result of the 2003 disaster. The commenter indicates that when a heat wave of similar magnitude struck in 2006, far fewer deaths occurred than were predicted (Fouillet et al., 2008). The commenter suggests that the words "depending upon progress in health care" in the TSD are an attempt to influence the reader into thinking that people are unlikely to adapt, when in fact there is incontrovertible evidence they already have.

Response (5-47):

Regarding recent heat wave events and the assertion (from commenter 3136.1) that EPA does not provide sufficient information about the human response: citing the assessment literature (Confalonieri et al., 2007; Karl et al., 2009), the TSD has added specific information about the European heat wave in 2003, its effects, and implications for the future:

> The excess mortality during the extreme heat wave in Europe in 2003 demonstrates the lethality of such events, which led to approximately 15,000 deaths in France alone (Confalonieri et al., 2007). Karl et al. (2009) report that an analysis of the European summer heat wave of 2003 found that the risk of such a heat wave is now roughly four times greater than it would have been in the absence of human-induced climate change.

The specific adaptive responses to the 2003 European heat wave or other individual heat waves such as those that occurred in Philadelphia and Chicago are not relevant to these Findings. See the Findings, Section III.C, "Adaptation and Mitigation," for our response to comments on the treatment of adaptation and mitigation in the Findings.

Comment (5-48):

One commenter (3136.1) indicates that he has shown that sensitivity to extreme heat events has been declining in major cities across the United States and notes this result is only apparent when you correctly standardize the population for changing size and age distribution. Furthermore, the commenter indicates that without properly standardizing, it is impossible to compare the climate impacts from one period to another (or one place to another) and that once you do standardize you find that extreme heat is having less of an impact as we better adapt to its occurrence. The commenter refers to Davis et al. (2003a, 2003b), who find that heat-related mortality declined in 28 major U.S. cities even as temperatures rose.

Response (5-48):
We do not dispute the findings of Davis et al. (2003a, 2003b) presented here. In fact, the assessment literature, as summarized in the TSD, refers to the Davis et al. (2003a, 2003b) papers; it states that heat-related mortality in major U.S. cities declined from 1964 to 1998 and that adaptive measure contributed to the decline. However, it is not clear that the results of Davis et al. remain relevant. We note that the assessment literature, as summarized in the TSD, finds that this decreased sensitivity or decline in heat-related mortality may not be as pronounced in the future since access to air conditioning is already high in most regions (Ebi et al., 2008). We note that recent work by Sheridan et al. (2009) indicates that the general decline in heat-related deaths that had been observed since the 1970s leveled off in the mid-1990s, which is summarized the latest assessment literature (Karl et al., 2009) and the TSD. Additional discussion of air conditioning can be found in response 5-52.

Comment (5-49):
A commenter (3136.1) indicates that the TSD's statement that "possible implications for human health of these projected changes in temperature extremes are discussed in Section 7(b)" is misleading, as it contains no reference to the highly documented phenomenon of adaptation to increasing heat-wave frequency. They suggest that EPA might substitute "some implications," which they state is more honest and also weakens the TSD as support for endangerment.

Response (5-49):
EPA notes that these comments pertain to the TSD associated with the ANPR released in July 2008. Reflecting this comment, we updated the version of the TSD released with the April 2009 proposal to include information about heat sensitivity to increasing temperatures in the context where the underlying references already take into account certain assumptions about adaptation in the context of climate change.

See the Findings, Section III.C, "Adaptation and Mitigation," for our response to comments on the treatment of adaptation and mitigation in the Findings.

Comment (5-50):
A commenter (3136.1) indicates that just because there are more elderly people living now and in the future does not mean they are at greater risk. If they were, the commenter indicates, life expectancy could not be increasing. The commenter notes that older adults are clearly living longer despite increased exposure to heat. The commenter posits that it is a hard argument to make that future heat wave mortality will increase despite the fact that historically the trend has been exactly the opposite, even with increasing heat and humidity. According to the commenter, the fact that more of the populace tends to live in cities but that age-adjusted death rates have not increased over time in these areas actually weakens EPA's argument for CO_2 impacts. Another commenter (3347.3) states that EPA incorrectly assumes that heat-related mortality risks will increase as the U.S. population ages because it ignores the likelihood of further development of climate-mitigating technology over this period.

Response (5-50):
EPA has reviewed the commenter's argument and finds that the reasoning is flawed and the conclusions are not supported by the scientific assessment literature. EPA notes that life expectancy is increasing in the United States and other developed countries for a variety of reasons (e.g., economic growth, public health measures, and technology). The fact that life expectancy is increasing does not mean that the mortality rate associated with every cause of death is declining, however. To the contrary, we know that death rates from some causes can and are increasing while others are decreasing, and that the overall trend

in recent years has been lower mortality from all causes combined. Thus, EPA concludes that life expectancy and heat-related mortality can both increase as long as some other factor(s) exceed(s) the rate of change in heat-related mortality. The issue before EPA concerns the impact of temperature-related mortality, and how this impact might incrementally affect the otherwise expected trends in life expectancy mortality.

In addition, the assessment literature unambiguously indicates that advanced age represents one of the most significant risk factors for heat-related death in the United States, and this is summarized in the TSD. Increased life expectancy and projected growth in older age brackets (e.g., the aging baby boomer generation, as noted in assessment literature) will increase the overall number of vulnerable individuals who may be exposed to more extreme heat conditions in the future (under documented climate change scenarios).

Finally, commenters are assuming that the risks posed to the elderly by an increase in heat waves will be modulated by adaptive actions. See the Findings, Section III.C, "Adaptation and Mitigation," for our response to comments on the treatment of adaptation and mitigation in the Findings.

Comment (5-51):

Commenters (3136.1, 3316.1, and 3596.1) contend that EPA misinterprets the series of the Davis et al. (2002, 2003, and 2004) studies summarized in the TSD (as cited in the assessment literature) due to the juxtaposition of the April 2009 statement:

> Estimates of heat-related mortality attributable to climate change are reduced but not eliminated when assumptions about acclimatization and adaptation are included in models. Confalionieri et al. (2007) cite a series of studies that suggests populations in the U.S. became less sensitive to high temperatures over the period 1964 to 1988, in part, due to these factors.

with the statement it precedes:

> On the other hand, growing numbers of older adults will increase the size of the population at risk because of a decreased ability to thermo-regulate is a normal part of the aging process (Confalonieri et al., 2007). In addition, according to a study in Confalonieri et al. (2007), almost all the growth in population in the next 50 years is expected to occur in cities where temperatures tend to be higher due to the urban heat island[32] effect increasing the total number of people at risk of adverse health outcomes from heat.

The commenters state the Davis et al. papers show that heat-related mortality is lowest in U.S. cities that have the oldest population distributions (Tampa and Phoenix). The commenters indicate that it is true that as the population ages, the number of individuals susceptible to extreme heat effects increases, but this has nothing to do with climate change. Further, the commenters indicate that the effects of population age structure are accounted for in the Davis et al. (2003) papers and the results indicate that the overall population's sensitivity to extreme heat has been declining across the United States, including for the elderly, from the 1960s through the 1990s. The commenters note the TSD indicates 1988 as the ending of the time period for this study, whereas it should be 1998.

Response (5-51):

We have carefully reviewed the TSD and find we have not misinterpreted the findings from Davis et al. The fact that some of the cities with the oldest population distributions have the lowest heat-related mortality as demonstrated in Davis et al. does not alter the fact that the population of this most vulnerable

age-group is projected to grow. It is well-established cities like Tampa and Phoenix that have lower heat-related mortality, as measured in terms of excess deaths, because climate variability is less there and the population is generally acclimated to warm temperatures (Curriero et al., 2002). Cities with highest climate variability, such as those in the Midwest and Northeast, are likely to experience the greatest number of illnesses and deaths in response to changes in summer temperatures (e.g., see McGeehin and Mirabelli, 2001; Curriero et al., 2002).

We agree with the commenters that projections for an increasing elderly population are independent of climate change and note the April 2009 and final TSD state: "…non-climatic factors related to demographics will have a significant influence on future heat-related mortality."

Please refer to response 5-48 for a discussion of the Davis et al. finding that the population's sensitivity to extreme heat has been declining. We also note that recent work by Sheridan et al. (2009) indicates that the general decline in heat-related deaths that had been observed since the 1970s leveled off in the mid-1990s, which is summarized the latest assessment literature (Karl et al., 2009) and the TSD.

EPA has updated the final TSD to reflect the study period (1968 to 1998) in reference to the series of studies (Davis et al., 2002, 2003, and 2004) cited by IPCC.

Comment (5-52):

Commenters (3347.1, 3347.3) assert that EPA incorrectly assumes that routine social and economic adaptation to temperature changes will not occur despite the increasing availability of air conditioning with increases in income. One of these commenters (3347.3) contends that clear historical evidence shows that air conditioning use reduces mortality in extreme heat events and refers to literature demonstrating the benefits of air conditioning in terms of reduced health effects from heat (e.g., Kilbourne et al., 1982; Kalkstein, 1993; Semenza et al., 1996, Davis et al., 2004; Seretakis et al., 1997; Basu and Samet, 2002). The commenter states although the use of air conditioning has grown continuously as Americans' incomes have risen and technology prices have fallen, EPA assumes that growth will now stop. The commenter refers to census data showing that the percentage of housing in the United States without air conditioning decreased from 46.5% in 1978 to 19.2% in 2005. The commenter cites a study (Stern et al. cited in Kalkstein et al., 1997) that projects that air conditioning will be "nearly universal" in 2050 and refers to a mathematical relationship between the availability of air conditioning and income (from Issaac and van Vuuren, 2009).

Response (5-52):

We do not assume that adaptation to temperature change will not occur and we find that the assessment literature suggests access to air conditioning is already high, if not reaching near saturation. Nonetheless, heat continues to harm or kill people in the United States.

As discussed in response 5-7, the TSD now states: "human health risks from climate change will be strongly modulated by changes in health care, infrastructure, technology, and accessibility to health care (Field et al., 2007)." Regarding air conditioning, we agree with the commenter that air conditioning reduces mortality in extreme heat events. We have reviewed the literature submitted on this issue and concur with findings presented. And we also agree with the statistics cited by the commenter showing that the percentage of housing in the United States without air conditioning has decreased. In fact, the TSD now states that the assessment literature has found access to air condition is high in most regions of the United States (Ebi et al., 2008).

Despite the increase in air conditioning, the assessment literature refers to Sheridan et al. (2009), which finds that the general decline in heat-related deaths observed from the 1970s to the mid-1990s seems to

have leveled off. This information is now summarized in the TSD. This evidence suggests that people remain subject to adverse health effects from heat in spite of increasing access to air conditioning. Furthermore, studies and reports from past heat events (e.g., National Weather Service, 2004; Sheridan, 2007; Luber and McGeehin, 2008) have demonstrated that not everyone with access to air conditioning uses it during heat events due to their financial situation and/or cognitive impairments. In other words, even if air conditioning prevalence were to increase independent of climate change, the evidence suggests that there would still be heat-related mortality risk.

Accordingly, despite projections in the references submitted for increasing access to air conditioning for the future—which we do not dispute—it is not clear that adverse heat-related health outcomes will decline. Finally, the argument that increased risk from hotter temperatures will be compensated for by technology (e.g., air conditioning) and/or adaptation (which are interrelated) is not relevant in the context of these Findings. See the Findings, Section III.C, "Adaptation and Mitigation," for our response to comments on the treatment of adaptation and mitigation in the Findings.

Comment (5-53):

Commenters (3502.1, Commonwealth of Massachusetts and the States of Arizona, California, Connecticut, Illinois, Iowa, Maine, Maryland, New Jersey, New Mexico, New York, Oregon, Rhode Island, Washington, and New York City) submit a 33-page document, including a manuscript of a paper accepted for publication in the July 2009 issue of *Journal of Epidemiology* entitled "Extreme High Temperatures and Hospital Admissions for Respiratory and Cardiovascular Diseases" (Lin et al., 2009). The commenters indicate that the study found the following: For each degree increase in temperature above a threshold (28.9°C), there was a significant increase in hospitalizations due to respiratory diseases on the same day (immediate effects). Similarly, for each degree increase above a threshold (29.4°C), there was a significant increase several days after exposure in hospitalizations due to cardiovascular diseases (likely due to the fact that it can take several days for cardiovascular effects to manifest). The diseases most associated with rises in temperature were asthma, chronic pulmonary obstructive disease, ischemic heart disease (heart attacks), and abnormal heart rhythms. For respiratory diseases, the risks for Hispanics were higher than those for non-Hispanics, and people living in low-income neighborhoods were at higher risk than those in high-income neighborhoods. Elderly people also had higher increased hospitalization risks for both disease types.

The commenters indicate that based on the results of the Lin et al. (2009) study, it is reasonable to conclude that the number of hospitalizations can be expected to significantly increase in the future due to a warming climate. In addition, the commenters indicate the results of this study further confirm EPA's conclusion that GHG emissions endanger public health. The commenters state: "We concur with EPA's approach to its proposed finding as to public health and strongly agree that even though the projected risks and impacts to public health are indirect, as opposed to resulting from direct exposure to greenhouse gas emissions, they constitute reasonably anticipated endangerment within the meaning of the statute."

Response (5-53):

EPA has reviewed the submitted reference and finds that it is consistent with the literature summarized in the TSD. The study (manuscript) by Lin et al. (2009) provides information on the association of high temperatures and morbidity, which is useful since such data are limited. It supports the TSD summary of the assessment literature, which finds: "Hot temperatures have also been associated with increased morbidity. A study cited in Field et al. (2007) indicates increased hospital admissions for cardiovascular disease and emergency room visits have been documented in parts of North America during heat events."

Comment (5-54):
A commenter (3722) indicates that the health impacts and mortality associated with increased temperature are considerably less than those associated with cold; and that anticipated warming over the next 50 to 100 years will actually decrease mortality. The commenter suggests that this is not a new issue and that older literature supports their assertion. The commenter (3722RI0A, B, C, D) submits four references in support of their comments (Bull, 1973; Bull and Morton, 1975a, 1975b, 1978). These references are also cited by the NIPCC report entitled, "Climate Change Reconsidered" (Idso and Singer 2009) (as referenced by commenter 7031, for example), which also contends that these studies suggest mortality decreases as temperatures rise.

Response (5-54):
EPA has reviewed the submitted references and we do not find they support the commenter's assertion. These studies mainly apply to England and Wales; in the one study that analyzes New York City, the results do not imply that warm weather reduces mortality rates.

These studies were published in the 1970s, and discuss the relationship of temperature changes and changes in death rates of specific health conditions. They are primarily focused on studying the relationships between temperature change and the health response of myocardial and cerebral infarction, particularly in the elderly.

We first note that the first three of the studies listed above find that changes in temperature typically are inversely associated with death rates except for stroke, and the second study (Bull and Morton, 1975a) does find that high temperature on the day of onset is associated with an increase in deaths on that day. These studies, however, were conducted in England and Wales, which do not experience the same climate variability as many parts of the United States; therefore, the results may not be applicable in the United States (see response 5-40 for further discussion).

We note that the most recent of the four studies (Bull and Morton, 1978), which includes New York City in its analysis, obtains results that differ importantly from the first three studies. It finds in all three locations, but particularly New York, "[a]bove 20°C deaths rise steeply as the temperature rises…," demonstrating the lethal effect of high temperatures, particularly for a U.S. location with a strongly variable climate. The study concludes: "there is a close association between temperature and death rates from *most* diseases and at *all* temperatures."

Importantly, these studies do not directly address climate change or long-term trends in health effects related to climate variables. Since their publication, many studies that are specifically focused on the impacts of climate change on health in the United States have been published, and the findings of this literature are summarized in the TSD.

Comment (5-55):
A commenter (3187.3) indicates that the claim that warming increases morbidity rates is a myth. The commenter states that Dr. Robert Mendelsohn, an environmental economist from Yale University, argues that heat-stress deaths are caused by temperature variability, not warming, and that those deaths grow in number not as climates warm but as the variability in climate increases. The commenter notes that excess deaths are greatest in metropolitan areas among the elderly and when the nighttime readings stay high (80^{o}F or greater) and the heat lasts more than a few days.

Response (5-55):
We disagree with the commenter's contention that warming will not increase morbidity or mortality.

(We note the comment indicates that morbidity does not increase with climate warming; however, the substance of the comment is related to mortality. Although both morbidity and mortality are important in terms of human health impacts and risks from climate change, EPA does not view these terms as interchangeable.) The assessment literature, as cited in the TSD, is very clear that excessive heat contributes to increases in both morbidity and mortality.

The commenter indicates that Dr. Robert Mendelsohn argues that heat-stress deaths are caused by temperature variability and not a warming climate. We disagree with this argument and, furthermore, find that the commenter does not substantiate the claim that Mendelsohn makes this argument. Numerous studies (e.g., Kalkstein and Greene, 1997; Curriero et al., 2002; Davis et al., 2003a; Laaidi et al., 2006) find that temperature-related mortality increases when threshold conditions, typically defined exclusively or in large part by temperature measures, are crossed. If climate change increases the probability of a location exceeding that threshold, mortality will rise, all else being equal. The commenter refers to Mendelsohn's book (Mendelsohn and Neumann, 1999), but we find no statement in that book to indicate that heat-stress deaths are not caused by a warming climate. In the introductory chapter, authors Mendelsohn, Smith, and Neumann summarize a study by Kalkstein and Davis (1989) that finds that climate change could increase heat stress mortality. However, the authors then state: "These judgments about sizable health effects from climate change may be premature. Uncertainties about the role of climate variability and human adaptation to heat stress make it difficult to predict the magnitude of the effect." Though the authors express some skepticism about the results of Kalkstein and Davis (1989), this is not at all the same as stating that heat stress deaths are not caused by a warming climate. We further note the book was published in 1999, and we now have a decade of additional science, which the TSD summarizes.

We agree that the risks from heat waves are highest for older adults and that heat waves of long duration with high nighttime minimum temperatures are often deadly.

Comment (5-56):
A commenter (0300) claims that the greenhouse effect will have beneficial moderation of nighttime low temperatures, but no effect on daytime highs, because clouds from water vapor feedback warm at all hours but cool during the day. Another commenter (3411.1) refers to the study of Robeson (2002) which shows temperature variability has declined with warming in the United States.

Response (5-56):
We disagree that daytime highs will not increase and that the effects of warming nighttime minimum temperature will have beneficial effects on human health.

While the observational record in the United States has shown greater increases in minimum temperatures than maximum temperatures in recent decades (as noted in Robeson, 2002), daytime highs are still rising. The TSD, citing the assessment literature (Field et al., 2007; Kunkel et al., 2008) notes in Section 4(c) that "daily minimum (nighttime) temperatures have warmed more than daily maximum (daytime) temperatures" but also states in Section 4(k) that "the annual percent of days exceeding the 90th, 95th, and 97.5 percentile thresholds for both maximum (hottest daytime highs) … have increased." These statements are further supported by a recent study by Meehl et al. (2009) that finds that the current ratio of daily record high maximum temperatures to record low minimum temperatures averaged across the United States is about two to one. Refer to Response to Comments Volume 2 for further discussion of observed maximum and minimum temperatures in the United States.

Future projections of climate warming are not limited to nighttime temperatures because a positive water vapor feedback does not operate solely at night—and the commenter presents no evidence to the contrary.

For further discussion of the water vapor feedback, please refer to Response to Comments Volume 4. We note Robeson (2002) finds: "Although the variability of air temperature may decrease or remain unchanged at most locations in the contiguous USA [under climate change], the probability of extremely high air temperatures should still increase…" In fact, climate model projections indicate substantial daytime warming. Summarizing the assessment literature in Section 6(e), the TSD notes: "Karl et al. (2008) find for a mid-range scenario (A1B) of future GHG emissions, a day so hot that it is currently experienced only once every 20 years would occur every three years by the middle of the century over much of the continental United States; by the end of the century, it would occur every other year or more." Furthermore,

As noted in the previous comment and response (5-55), high minimum temperatures also contribute to heat stress. A number of studies (e.g., Kalkstein, 1991; Chestnut et al., 1998; Dixon, 1999; Basu and Samet, 2002) have linked high minimum (nighttime) temperatures with elevated mortality. For example, Basu and Samet (2002) noted several temperature-mortality studies identified a minimum temperature above which effects of temperature on mortality were observed and that these temperatures varied by location.

To summarize: both daytime and nighttime temperatures are projected to increase, and the evidence suggests that both will result in increasing risk of adverse health effects.

Comment (5-57):
A commenter (3187.3) states that the risk of heat-related deaths may be increased by policies that limit reliable traditional energy sources in favor of renewable energy such as wind power, which increase the likelihood of brownouts and blackouts. The comment notes that it is during the normal heat waves that energy demand is the greatest. The commenter asserts that during extremes of heat and cold, high pressure dominates, causing winds to be below the threshold for energy generation, and that this has already been shown in Texas, California, and the UK. The commenter states that this type of policy, not the weather, puts the population at enhanced risk.

Response (5-57):
Regarding the commenter's perspective that policies favoring renewable energy are responsible for impacts during extreme weather, EPA notes that this action does not propose any policies concerning mitigation and considers this issue beyond the scope of the Findings.

Comment (5-58):
A commenter (3347.3) contends that EPA cites U.S.-specific heat-related mortality projections from IPCC that suffer from exposure mischaracterization. The commenter indicates that the projections are based on a "place-time" model which assumes that outdoor temperature is a surrogate for personal exposure to heat, even though people in the United States generally spend most of their time indoors. They suggest these projections should use the "micro-environmental" model, which uses a time-weighted sum of heat exposure to project mortality, citing Basu and Samet (2002).

Response (5-58):
We disagree that the projections of heat-related mortality for the United States referred to in the TSD suffer from exposure mischaracterization. Contrary to the commenter's assertions, the methodology used to project heat-related mortality does not assume people are spending most of their time outdoors.

In the TSD, EPA summarizes mortality projections described in IPCC (Field et al., 2007) from one study (Hayhoe et al., 2004). In the study, heat-related mortality projections for Los Angeles are developed using algorithms that are based on the historical, empirically developed relationships between mortality and air mass characteristics (e.g., temperature and humidity) in that city. Because the projections are based on empirically based associations, it is not necessary to control for exposure using methods described in Basu and Samet (2002).

Comment (5-59):
Some commenters (e.g., 1543, 3353, 5379, 6510, 6941, 11249) express concern about the effects that extreme weather—such as heat waves, droughts, floods, wildfires, and hurricanes—has had or would have on the safety and health of humans. The commenters state that such events can result in the spread of infectious disease, malnutrition, and poor air and water quality because runoff from these events contains agricultural wastes, metals, pesticides, pathogens, dangerous chemicals, and fecal indicator bacteria.

Response (5-59):
We have carefully reviewed the commenters' concerns regarding the effects of extreme weather events (heat waves, droughts, floods, wildfires, and hurricanes). We note that the TSD contains information on the human health effects of extreme events as well as information on the vulnerabilities of populations, communities, and locations to such events. In addition, we note that the TSD summarizes the scientific literature with regard to observations and projections of extreme events in Sections 4 and 6, respectively.

Comment (5-60):
A commenter (3475.10) submits a reference by Delfino et al. (2009) entitled, "The Relationship of Respiratory and Cardiovascular Hospital Administrations to the Southern California Wildfires of 2003." The commenter summarizes the study's general conclusions with respect to wildfire-related $PM_{2.5}$ leading to increased respiratory hospital admissions, especially asthma, and suggesting that better preventive measures are required to reduce morbidity among vulnerable populations.

Response (5-60):
EPA reviewed this study and finds its conclusions on the health impacts of wildfires to be consistent with the assessment literature summarized in Section 7(b) of the TSD. For example, the TSD indicates a study cited by IPCC (Confalonieri et al., 2007) indicating that large fires are also accompanied by an increased number of patients seeking emergency services for inhalation of smoke and ash. See also the discussion of wildfire frequency in Sections 8(b) and 10(b) of the TSD, and response to comments on this issue in Volume 6 of the Response to Comments document.

See the Findings, Section III.C, "Adaptation and Mitigation," for our response to comments on the treatment of adaptation and mitigation in the Findings.

Comment (5-61):
A commenter (3722) indicates that it is true that flood, severe storms, fire, and disease can cause human injury and death; however, the fundamental issue with this portion of the TSD is that of causation. The commenter indicates that EPA has not established elsewhere in the TSD that climate change that is occurring is in fact having a material impact on these kinds of disastrous events, or that human contribution to climate change is the causative factor in such impacts. This fundamental issue, states the commenter, undercuts Sections 7 and 8 of the TSD. The commenter provides an example of the extreme

event impacts from floods and storms as compared to the evidence provided in the TSD. The commenter refers to the following information in the TSD (as paraphrased by the commenter):

> The IPCC projects a very likely increase in heavy precipitation event frequency over most areas as described in Sections 6. Increases in the frequency of heavy precipitation events are associated with increased risk of deaths and injuries as well as infectious, respiratory and skin diseases. Flood health impacts include deaths, injuries, infectious diseases, intoxications and mental health problems.

The commenter then, referring to Figure 2c in study, notes that despite projections of increased flooding in the United States, no increased trends in flood-related damages have been detected (Downton et al., 2005) while temperatures and CO_2 have been rising.

Response (5-61):
We have carefully reviewed the comments and referenced literature provided by the commenter. We note that the Downton et al. (2005) study also provides two additional graphics (Figures 2a and 2b of that study) related to flooding trends. They both appear to support an upward (increase) trend flooding over the past century. Further, we conclude that this information does not convey an evaluation of risks associated with flooding and storms in future conditions of a warming climate. We will continue to rely on the science summarized in the TSD on this issue. Please also refer to Volume 7 of these responses to comments for further discussion of the changes in flood damage over time versus the increased risk of flood.

The commenter also appears to be challenging the notion of attribution of observed climate change and extreme events as the basis for projected climate change impacts. See Volume 2, "Attribution of Observed and Measured Climate," and Volume 3, "Change and Validity of Future Projections," of this Response to Comments document for our responses to comments on these issues. See Volume 9 for EPA's responses on the Administrator's determination, in general and with respect to climate impacts.

Comment (5-62):
A commenter (3283.1) indicates that EPA cites floods, storms, droughts, and wildfires as human health risks. The risks listed are death, injury, disease, intoxications, increased exposure to smoke and ash, and mental health problems. The commenter notes that while disease is a public health issue, in the future, improved medical technology will all but eliminate elevated disease risks due to extreme events. Similarly, the commenter indicates that improved forest management practices can be used to minimize health risks due to wildfires.

Response (5-62):
As noted in the TSD and summarized from the scientific literature, human health risks from climate change will be strongly modulated by changes in health care, infrastructure, technology, and accessibility to heath care (Field et al., 2007). However, the commenter fails to provide any supporting evidence that improved medical technology will all but eliminate elevated disease risks due to extreme events. Similarly, the commenter fails to provide supporting information that improved forest management practices will effectively reduce health risks. In addition, see the Findings, Section III.C, "Adaptation and Mitigation," for our response to comments on the treatment of adaptation and mitigation in the Findings.

Comment (5-63):

A commenter (10050.1) submits brief comments and several references on the subject of the impacts of global warming on the mental health of those impacted. The commenter indicates that as GHG emissions continue to soar and fuel the problems associated with climate change, severe harm will come to the mental wellbeing of millions of Americans and citizens around the world. The commenter further states that while we often hear that the public's physical health will face increasing threats from droughts, floods, storms, food shortages, higher temperatures, and climate change's related repercussions, many more members of the public will experience increased rates of anxiety, depression, conflict, and other psychological problems that damage lives and spirits for years to come.

The commenter indicates that WHO, CDC, and even the U.S. military now recognize that climate change is on track to be one of the most significant contributors to behavioral problems on the planet. In support of the proposed endangerment finding, the commenter indicates that projected climate impacts including rising temperatures, water scarcity, devastating storms (i.e., hurricane Katrina), air pollution, and a rise in infectious diseases will adversely affect society and the psychological wellbeing of millions of people around the world.

Another commenter (3421) notes that an important health consequence not mentioned in the TSD is the risk to mental health. The commenter states that the toll on mental health from surviving an extreme weather event, deprivation of food and water, forced relocation, and worry about "what comes next" can be expected to far surpass the physical injuries and illnesses of climate change.

Response (5-63):
EPA has reviewed and considered the submitted references concerning mental health effects. We note the commenter provides a study indicating that severe weather events, such as coastal areas experienced with hurricane Katrina in 2005, engender severe anxiety, depression, grief, anger, family problems, work problems, conflict, and, potentially, post-traumatic stress disorder that can last for years (Galea et al., 2007).

EPA agrees with commenter that climate change effects, particularly those related to some extreme events, may pose mental health risks. We conclude the commenter's views are supported in the TSD. For example, the assessment literature, as summarized in the TSD, refers to mental health problems that arise from flood impacts (Confalonieri et al., 2007), which are projected to become more extreme (Karl et al., 2009). The TSD also notes the IPCC (2007b) finding that intense hurricanes—which are projected to increase—are linked to increases in post-traumatic stress disorder. These findings are generally consistent with the literature as referenced by the commenter (e.g., Bourque et al., 2006; Kessler et al., 2008; Weems et al., 2007; Weisler et al., 2006), which documents mental health problems that arise from certain extreme weather events.

Comment (5-64):
A commenter (2818) indicates that EPA's technical documents did not really mention the mental health effects of climate change. The commenter indicates that mental health problems, although much harder to see, are likely to impact far more people than just injury and illness. The commenter states that mental health impacts would manifest themselves in things like depression, anxiety, and post-traumatic stress disorder from living through traumatic events. The commenter notes that hurricane Katrina demonstrated that people who have lived through a traumatic event such as that have far greater problems with medical conditions as well as mental health conditions; their health indicators have gone down precipitously.

Response (5-64):
EPA agrees with the commenter that mental health effects associated with extreme events are an important consideration, and notes that this issue is addressed in Section 7 of the TSD, though briefly.

Section 7(b) summarizes scientific findings that flood health impacts include deaths, injuries, infectious diseases, intoxications, and mental health problems (Confalonieri et al., 2007). Further the TSD notes, in Section 7, that high-impact events can overwhelm and disrupt social organizations, which can leave people displaced and without necessary post-event assistance. In addition, the TSD notes increased deaths, injuries, infectious diseases, and stress-related disorders and other adverse effects associated with social disruption and migration from more frequent extreme weather (Field et al., 2007). Our review of the assessment literature has also found that there is limited information on the mental health impacts associated with climate change.

Comment (5-65):
Some commenters specifically mention the migration of tropical diseases (0311) such as West Nile virus (3893, 4184, 11249) and malaria and dengue fever (1507, 3680.1, 10838, 11357). One commenter (6510) mentions the spread of disease-carrying vectors into new habitats as a specific impact, which will increase health-related morbidity and mortality. One commenter (6733) discusses the rise in the number of disease-carrying mosquitoes.

Response (5-65):
While the commenters do not provide specific references for these observations and views, we note that they are generally consistent with the scientifically based findings summarized in the TSD, especially in Section 6. We note that several human and other diseases are sensitive to climate. As indicated in the TSD, there are several important associations between climate and water-, vector-, and food-borne diseases. Based on the science and research from major assessments (IPCC, USGCRP), we conclude that certain disease may occur more often and affect new populations, as a result of changes in temperature and precipitation that allow these pathogens to expand into new geographic regions.

Comment (5-66):
Commenters (e.g., 3347.3, 3921.1) indicate that dengue fever and other vector-borne diseases have become an endangerment to American public health because of climate change. Commenters state that vectors and infectious agents are cold-blooded, and because breeding sites have increased due to changing temperature and precipitation, vector and agent development has accelerated, their geographic range has shifted to higher latitudes and altitudes, and their seasonal period of disease risk has lengthened. The commenters state that these diseases are only one piece of the huge threat to the United States and urge EPA to take action.

Response (5-66):
We agree that climate increases the risk of certain diseases to spread or emerge in areas where they had previously been limited or not existed, and we find that these risks are appropriately summarized in the TSD as reflected in the assessment literature. We also agree with the commenter that in addition to risks associated with infectious diseases, there are various other risks due to climate change. Please see the appropriate volumes of this Response to Comments document for our responses to comments on other impacts of climate change that affect public health and welfare.

Comment (5-67):
A number of commenters (e.g., 0339, 0700.1, 3283.1, 3316.1, 3347.3, 3449.1:10, 3722, and 10158) argue that statements made in the TSD regarding the spread of infectious and vector-borne diseases as a result of temperature and precipitation changes are speculative, that they are not scientifically supportable, and/or that there is there is no demonstrated link between climate change and disease. Some commenters

suggest that the TSD relies on inferred health effects based on temperature sensitivity as a proxy for public health impacts in the United States.

Response (5-67):
We have reviewed the assessment literature in light of these comments, and we find that the TSD's summary of the current state of the science on this topic as reflected in the assessment literature is accurate and sound. The assessment literature is clear that certain vector-borne diseases and other infectious diseases are sensitive to climate and that climate change increases the risk of exposure in a number of ways. As noted in the TSD, the incidence of airborne infectious diseases (e.g., coccidioidomycosis) varies seasonally and annually, due partly to climate variations such as drought (Field et al., 2007; Karl et al., 2008). It also notes that waterborne disease outbreaks are distinctly seasonal, suggesting potential underlying environmental or weather control, clustered in particular watersheds, and associated with heavy precipitation. It further states that food-borne diseases show some relationship with temperature based on Confalonieri et al. (2007).

The CCSP assessment *Analyses of the Effects of Global Change on Human Health and Welfare and Human Systems* includes additional detail on this topic, stating that "[s]everal important pathogens that are commonly transmitted by food or water may be susceptible to changes in replication, survival, persistence, habitat range, and transmission under changing climatic and environmental conditions" (Ebi et al., 2008). Similarly, IPCC (Confalonieri et al., 2007; Field et al., 2007) references a number of studies documenting links between climate and disease.

Based upon our careful review of the literature, we have determined that the comment that the TSD relies only on inferred health effects is incorrect. Our review of the assessment literature indicates that there are documented links between climate change and disease, and that these are appropriately summarized in the TSD. As the TSD notes, studies discussed in Field et al. (2007) linked above-average temperatures in the United States during the summers of 2002–2004 to the greatest transmissions of West Vile virus. The TSD also reports that associations between temperature and precipitation and tick-borne Lyme disease are noted by IPCC (Field et al., 2007), as well as the finding from CCSP (2008b) that there will likely be an increase in the spread of several foodborne and waterborne pathogens among susceptible populations depending on the pathogens' survival, persistence, habitat range, and transmission under changing environmental conditions.

CCSP (Ebi et al., 2008) includes extensive discussion of this topic that is consistent with the TSD's summary. Table 2.2 in Ebi et al. lists a number of pathogens, the climate-related driver, the possible influence of climate change, the likelihood of changes, the basis for assessment, and the peer-reviewed references underlying the assessment. It reports, among other things, that increasing temperature is likely to be associated with increasing clinical cases of *Salmonella*. It also reports that increasing temperature is extremely likely to be associated with higher environmental prevalence of *Vibrio* species and associated disease, and very likely to be associated with range expansion.

Comment (5-68):
Several commenters (e.g., 7031) referred to the NIPCC report (Singer and Idso, 2009) which contends that the projected spread of tick-borne diseases is based on a speculative assumption of steadily increasing temperature and other assumptions.

Response (5-68):
We have reviewed the assessment literature in light of this comment, and we disagree that the projected spread of tick-borne disease is "speculative." The assessment literature, as summarized in the TSD, clearly demonstrates relationships between tick-borne diseases and climate. IPCC (Field et al., 2007)

reports on studies indicating that Lyme disease, a prevalent tick-borne disease in North America, has an association with temperature (Ogden et al., 2004) and precipitation (McCabe and Bunnell, 2004). IPCC refers to a study (Ogden et al., 2006) that projects that the northern range limit for the tick responsible for transmitting Lyme disease could shift north by 200 kilometers by the 2020s, and 1,000 kilometers by the 2080s (based on model projections under IPCC Special Report on Emissions Scenarios (SRES) A2 emissions scenario) (Field et al., 2007). CCSP (2008b) reinforces these statements, reporting that studies suggest that temperature influences the distributions of *Ixodes* species ticks that transmit pathogens causing Lyme disease in the United States (Brownstein et al., 2003) and Canada (Ogden et al., 2006).

EPA concludes that the TSD's summary of the current state of the science on this topic as reflected in the assessment literature is accurate and sound.

Please refer to Volume 4 for EPA's responses to comments specific to the validity of future projections of climatic variables important for disease transmission such as temperature and precipitation.

Comment (5-69):

A commenter (3136.1) indicates that most tropical diseases occur in the tropics not because the weather is warm, but because public health measures are poor. Similarly, a commenter (3722) notes that EPA ignores the fact that public health advancements, availability of health care, and other socioeconomic factors unrelated to climate change play a far greater role in the spread of disease than warming temperatures. Commenters (e.g., 3136.1, 3283.1, and 3316.1) note that prior to public health intervention (such as in the late 1800s), malaria was common in non-tropical areas (including high latitudes). They indicate that the spread of malaria is easily preventable and that public health measures are far more effective at controlling disease outbreaks than attempting to control global climate. Commenters (3136.1, 3316.1) contend that malaria is weakly related to climate but strongly related to poverty, arguing that the resurgence of malaria in some developing countries is related to the ban of DDT, anti-malaria drug resistance, and the breakdown of public health systems, not any ascertainable changes in climate. Commenters also note that EPA acknowledges that locally acquired cases of malaria in the United States have been virtually eliminated, and thus the Agency relies on the effects to other countries under the guise of public health impacts in the United States. One commenter (4509) submitted an interview with Paul Reiter entitled "Global Warming Won't Spread Malaria (EIR, 2007). Another commenter indicates that the NIPCC book *Climate Change Reconsidered* states that the global spread of malaria related to CO_2-induced warming is not supported in the scientific literature.

Response (5-69):

Commenters are correct when they note that the TSD states that locally acquired cases of malaria have been virtually eliminated in the United States, in part due to effective public health interventions. We also agree that public health interventions have reduced malaria in other parts of the world. EPA never states that temperature is the only determinant in the transmission of malaria and the TSD is clear that a number of factors impact transmission. However, we disagree with commenters who dismiss the possibility that climate change may impact the spread of malaria in some areas (globally) based on our review of the assessment literature.

IPCC (Confalonieri et al., 2007), citing several studies (Hay et al., 2002a; Craig et al., 2004), reports the spatial distribution, intensity of transmission, and seasonality of malaria is influenced by climate in sub-Saharan Africa. It notes that studies have reported associations between interannual variability in temperature and malaria in the African highlands. IPCC (Confalonieri et al., 2007) also indicates that malaria risk is well associated with climatic factors such as rainfall patterns and the El Niño Southern Oscillation (ENSO) in Southern Asia and South America. IPCC (Confalonieri et al., 2007) concludes that there are known causal links between climate and malaria transmission dynamics, but that there is still

much uncertainty about the potential impacts of climate change on malaria at local and global scales because of the paucity of concurrent detailed historical observations of climate and malaria, the complexity of malaria diseases dynamics, and the importance of non-climatic factors, including socioeconomic development, immunity, and drug resistance, in determining infection and infection outcomes. Accordingly, we find the TSD's qualified discussion of possible linkages between climate change and malaria (in Sections 7 and 16b), appropriately summarizes the assessment literature.

See the Findings, Section III.C, Adaptation and Mitigation, for our response to comments on the treatment of adaptation and mitigation in the Findings.

Comment (5-70):
A commenter (3136.1) argues that climate change has nothing to do with the reason why West Nile virus now exists in the United States, and that the reason it was not found in the United States in the past was simply that it had not been introduced until recently.

Response (5-70):
The assertion that climate change has nothing to do with why West Nile virus currently exists in the United States is irrelevant when considering the effects climate change has on the transmission of this disease now and in the future. We have reviewed the assessment literature in light of this comment and we find that the TSD's summary is accurate. The TSD notes that a study cited by Field et al. (2007) linking above-average temperatures in the United States during the summers of 2002–2004 to the greatest transmissions of West Nile virus.

The assessment literature includes additional detail on this topic that is consistent with the TSD's overall discussion of climate-sensitive diseases. Karl et al. (2009) cite a study (Kilpatrick et al., 2008) which found that transmission of both strains of West Nile virus accelerated sharply with increasing temperature, such that small increases in temperature had relatively large effects on transmission. This laboratory study suggests that both viral evolution and temperature influence the distribution and intensity of transmission of West Nile virus, and provides a model for predicting the impact of temperature and global warming on virus transmission (Kilpatrick et al., 2008). Karl et al. (2009) report that the study suggests that greater risks from West Nile virus may result from increases in the frequency of heat waves, but note that the risk will also depend on the effectiveness of mosquito control programs.

Comment (5-71):
One commenter (3136.1) indicates that there is no evidence linking climate change to past or future Saint Louis encephalitis outbreaks in the United States. The comment contends the TSD makes unsubstantiated claims of increased risk of Saint Louis encephalitis during La Niña weather events.

Response (5-71):
The TSD does not state that there is explicit evidence linking Saint Louis encephalitis and climate change, but rather summarizes a study (Cayan et al., 2003) cited by IPCC (Field et al., 2007) finding a relationship between Saint Louis encephalitis and hot, dry La Niña years. We, therefore, disagree with the assertion that the TSD makes unsubstantiated claims on this topic. Rather, we find that it appropriately summarizes the conclusions of the assessment literature.

Comment (5-72):

A number of commenters (e.g., 3347.3, 3449.1:10, and 3747) state that many factors other than climate, such as the availability of an adequate health system, are more important to the incidence of disease than climate, or that the impacts of climate on food and water-borne pathogens are not the only factors determining the risk of human injuries, illness, and death. Some commenters state that the risk of disease following flooding in developed countries is generally low.

Response (5-72):

We agree that many non-climate factors are important for the incidence of disease. The TSD includes information from the assessment literature on this topic, noting, for example, the IPCC (Field et al., 2007) conclusion that human health risks from climate change will be strongly modulated by changes in health care, infrastructure, technology, and accessibility to health care.

The fact that many factors are important to the incidence of disease does not mean that climate change is not one of them. As discussed throughout this section of the Response to Comments document, the assessment literature clearly indicates that some diseases transmitted by food, water, and insects are likely to increase due to climate change (Karl et al., 2009).

In regard to the risk of disease due to flooding, the TSD notes that the United States has successful programs to protect water quality under the Safe Drinking Water Act and the Clean Water Act but adds that some contamination pathways and routes of exposure do not fall under regulatory programs (e.g., dermal absorption from floodwaters, swimming in lakes and ponds with elevated pathogen levels). The TSD additionally notes that the primary climate-related factors that affect these pathogens include temperature, precipitation, extreme weather events, and shifts in ecological regimes, adding that consistent with the latest understanding of climate change on human health, the impact of foodborne and waterborne pathogens will seldom be the only factor determining the burden of human injuries, illness, and death (CCSP, 2008b).

CCSP (Peterson et al., 2008) assesses this issue in more detail. It reports that "Floods directly promote transmission of water-borne diseases by causing mingling of untreated or partially treated sewage with freshwater sources, as well as indirectly from the breakdown of normal infrastructure causing post-flood loss of sanitation and fresh water supplies (Atherholt et al., 1998; Rose et al., 2000; Curriero et al., 2001; Patz et al., 2003; O'Connor, 2002)." The report goes on to report the following:

> Analyses of the U.S. indicate that the assumption that developed countries have low vulnerability may be premature, as independent studies have repeatedly concluded that water and food-borne pathogens (that cause diarrhea) will likely increase with projected increases in regional flooding events, primarily by contamination of main waterways (Rose et al., 2000; Ebi et al., 2006).
>
> A U.S. study documented that 51% of waterborne disease outbreaks were preceded by precipitation events in the top 10% of occurrences, with 68% of outbreaks preceded by precipitation in the top 20% (Curriero et al., 2001). These outbreaks comprised mainly intestinal disorders due to contaminated well water or water treatment facilities that allowed microbial pathogens, such as *E. coli*, to enter drinking water. In 1993, 54 people in Milwaukee, Wisconsin died in the largest reported flood-related disease outbreak (Curriero et al., 2001). The costs associated with this one outbreak were $31.7 million in medical costs and $64.6 million in productivity losses (Corso et al., 2003).

CCSP (2008b) also includes an analysis of the potential impacts of flooding, noting that "[f]ixed infrastructure itself has the potential to be adversely impacted by climate change, which can increase

vulnerability to climate change. For example, flooding can overwhelm sanitation infrastructure and lead to water related illnesses (Grambsch and Menne, 2003)."

To better explain the link between flooding and climate-sensitive diseases and incorporate the CCSP assessment of this topic, we have modified the summary of Section 7(c) of the TSD to read as follows:

> Waterborne disease outbreaks are distinctly seasonal (which suggests potential underlying environmental or weather control), clustered in particular watersheds, and associated with heavy precipitation. IPCC (Confalonieri et al., 2007) reports that the risk of infectious disease following flooding in high-income countries is generally low, although increases in respiratory and diarrheal diseases have been reported after floods. However, CCSP (Peterson et al., 2008) finds that analyses of the United States indicate that the assumption that developed countries have low vulnerability may be premature, citing to studies that "have repeatedly concluded that water and food-borne pathogens (that cause diarrhea) will likely increase with projected increases in regional flooding events, primarily by contamination of main waterways." In another report, CCSP (2008b) notes that flooding can overwhelm sanitation infrastructure and lead to water-related illnesses. A U.S. study documented that 51% of waterborne disease outbreaks were preceded by precipitation events in the top 10% of occurrences, with 68% of outbreaks preceded by precipitation in the top 20% (Curriero et al., 2001, cited in Peterson et al., 2008). After hurricanes Katrina and Rita in 2005, contamination of water supplies with fecal bacteria led to many cases of diarrheal illness and some deaths (Ebi et al., 2008; CDC, 2005; Confalonieri et al., 2007).

This new text replaces the previous text from the April 2009 version that read:

> Waterborne disease outbreaks are distinctly seasonal (which suggests potential underlying environmental or weather control), clustered in particular watersheds, and associated with heavy precipitation. The risk of infectious disease following flooding in high-income countries is generally low, although increases in respiratory and diarrheal diseases have been reported after floods (Confalonieri et al, 2007). For example, after Hurricanes Katrina and Rita in 2005, contamination of water supplies with fecal bacteria led to many cases of diarrheal illness and some deaths (Ebi et al., 2008; CDC, 2005; Confalonieri et al, 2007)

As the CCSP findings quoted above show, there is considerable evidence that high-precipitation events associated with climate change are likely to increase the likelihood of waterborne disease outbreaks in parts of the United States. Therefore, EPA concludes that the TSD's summary of the current state of the science on this topic as reflected in the assessment literature is accurate and sound.

Comment (5-73):

A commenter (3347.3) asserts that EPA ignores important contrary scientific conclusions on climate-sensitive disease. The commenter points to a statement in the CCSP assessment on health effects (Ebi et al., 2009) not in the April 2009 TSD that concludes: "For the U.S., it is not anticipated that climate change will lead to loss of life or years of life due to chronic illness or injury from waterborne or foodborne illnesses." The commenter requests that EPA add this statement to the TSD.

Response (5-73):

We have added this statement from Ebi et al. (2008) to Section 7c of the TSD. However, as stated in the TSD, Ebi et al. (2008) indicate that there will likely be an increase in the spread of several foodborne and

waterborne pathogens among susceptible populations depending on the pathogens' survival, persistence, habitat range, and transmission under changing climate and environmental conditions (CCSP 2008b). As noted in response 5-72, deaths were attributed to water contamination in the wake of Hurricanes Katrina and Rita.

Comment (5-74):
One commenter (1616.1) indicates that diseases naturally vary with climate and that as climate changes, diseases will undoubtedly change, but there is no reason to think there will be a net change for the worse in the future.

Response (5-74):
We disagree with this comment and note that it provides no references to substantiate its assertion that climate change will not make diseases worse. As discussed in response 5-67, the assessment literature (e.g., Ebi et al., 2008; Karl et al., 2009) finds that some diseases transmitted by food, water, and insects are likely to increase due to climate change.

Comment (5-75):
A commenter (3501) indicates that of unique concern in the western United States are rates of valley fever (coccidioidomycosis), which have quadrupled in the last 10 years, primarily due to weather pattern changes which will likely continue as the climate warms. The commenter also indicates that ocean temperature changes directly influence waterborne disease distribution; in particular, outbreaks of the waterborne disease cholera are directly linked to warmer ocean temperatures. The commenter indicates that studies have shown that ENSO events and associated warming of regional ocean surface waters induce algae blooms that harbor the pathogen *Vibrio cholerae,* ultimately leading to disease outbreaks in coastal areas. The commenter also indicate that rising temperatures directly impact urban air quality by accelerating ozone formation and heat-related illness in urban areas is also on the rise and states that approximately 70,000 people in Europe died during the 2003 heat wave. The commenter states that numerous studies show a clear relationship between temperature and such things as hospitalizations for respiratory diseases as well as sudden cardiac death, but does not reference any specific studies.

Response (5-75):
We agree that climate change will affect human health in a variety of ways and find that these comments are generally consistent with the underlying scientific literature summarized in the TSD; however, we note that the statements in the comment do not in all cases employ the careful qualifying language the TSD uses in summarizing the conclusions of the assessment literature. Please see other responses in this section for our responses to comments on specific diseases discussed in the TSD. Please see responses 5-11, 5-20, 5-86, and 5-92 for some example responses to comments on links between climate change and respiratory diseases.

Comment (5-76):
One commenter (3347.3) claims that "EPA often fails to adequately explain the context of research results that could significantly affect the applicability of those results to estimates from climate change," citing as an example the TSD's treatment of links between temperature and the incidence of human disease from food- and water-borne pathogens such as the *Salmonella* and *Vibrio* species. The commenter states that a study cited by Confalonieri et al. (2007) (Mclaughlin et al., 2005) includes important relevant information omitted by EPA, such as that the oysters involved were consumed raw.

Response (5-76):
We have reviewed our summary of the assessment literature in light of this comment. On the basis of our review, we have determined that the commenter's claim that we failed to adequately explain the context of research results that could significantly affect the applicability of those results to estimates from climate change is incorrect and not substantiated by the evidence cited.

We have reviewed the assessment literature's treatment of links between temperature and the incidence of human disease from food- and water-borne pathogens such as the *Salmonella* and *Vibrio* species and the source study referenced by the commenter. On the basis of this review, we have concluded that the TSD accurately summarizes this information. The fact that the outbreak at issue involved oysters consumed raw is in no way inconsistent with the TSD's summary of assessment literature conclusions on this issue, which is as follows:

> *Vibrio* spp. infections from shellfish consumption may also be influenced by temperature (Confalonieri et al., 2007). For example, Confalonieri et al. (2007) cited a study documenting a 2004 outbreak of *V. parahaemolyticus* linked to atypically high temperatures in costal water.

This statement is fully consistent with the conclusions of other assessment reports. Karl et al. (2009), for example, note that "[t]here is a close association between temperature, *Vibrio* sp. abundance, and clinical illness."

EPA concludes that the TSD's summary of the current state of the science on this topic as reflected in the assessment literature is reasonable and sound.

Comment (5-77):
With regard to EPA's summary of assessment literature conclusions on West Nile virus, one commenter (3347.3) states that "The incidence of infectious disease has a complex etiology, and cannot be ascribed to one factor alone." The commenter adds that "the incidence of a disease is not evidence of causation."

Response (5-77):
We agree that incidence of infectious disease has a complex etiology and cannot be ascribed to one factor alone. The TSD does not claim that West Nile Virus or any other infections disease will be affected by climate change acting in isolation. Rather, it notes that the study discussed in Field et al. (2007) linked above-average temperatures in the United States during the summers of 2002-2004 to the greatest transmissions of West Nile virus. Consistent with Field et al. (2007), the statement uses the world "linked" rather than "caused" but is still clearly in general agreement with the conclusion from Karl et al. (2009) that some diseases transmitted by food, water, and insects are likely to increase due to climate change. The term "likely" indicates the degree of certainty about the causal link between climate change and the increased transmission of some diseases.

Comment (5-78):
One commenter (3347.3) argues that statements in the TSD regarding Lyme disease are questionable and not well supported by existing research. The commenter states that "[r]ecent research shows that climate change is projected to decrease the geographic range of tick-borne encephalitis (TBE) in areas of lower latitude and elevation if transmission expands Northward (Randolph, 2004, Randolph and Rogers, 2000 in CCSP). These same researchers also found that changing patterns of TBE in Europe are not consistently related to changing climate." The commenter then references research in the Baltics (Sumilo

et al., 2007) that, according to the commenter, finds that a sudden jump in climate suitability for TBE in 1989 did not result in a landscape-wide increase in incidence. The commenter concludes that climate change alone cannot explain recent increases in the incidence of tick-borne diseases in North America, citing Confalonieri et al. (2007). The commenter asserts that the incidence of tick-borne diseases could decrease in areas where they are currently endemic, due to climate-related changes in habitat.

Response (5-78):
We have reviewed the relevant assessment reports in the light of this comment and disagree that statements in the TSD regarding Lyme disease are "questionable and not well supported by existing research." The relevant statements from the TSD on this topic are as follows: "Associations between temperature and precipitation and tick-borne disease are also noted by IPCC (Field et al., 2007). A study cited in Field et al. (2007) found that the northern range limit of *Ixodes scapularis*, the tick that carries Lyme disease, could shift north by 120 mi (200 km) by the 2020s and 620 mi by the 2080s."

These statements are consistent with both Field et al. (2007) and Ebi et al. (2008). The statements do not imply that recent changes in tick-borne diseases are due to climate change alone, nor do they imply that the incidence of tick-borne diseases will not decrease in some areas.

Ebi et al. (2008) assessed the Randolph (2004) and Randolph and Rogers (2000) studies referenced by the commenter. To further summarize the conclusions of the assessment literature on the regionally variable nature of potential changes in tick distribution, and to incorporated the findings of Randolph (2004) (which relates specifically to Europe) and Randolph and Rogers (2000), as assessed by the CCSP, we have added the following detail on this topic to the TSD:

> According to Ebi et al. (2008), studies suggest that higher minimum temperatures generally were favorable to the potential of expanding tick distributions and greater local abundance of these vectors. However, Ebi et al. (2008) add that: "changing patterns of tick-borne disease in Europe are not consistently related to changing climate (Randolph, 2004a). Climate change is projected to decrease the geographic range of TBE (tick-borne encephalitis) in areas of lower latitude and elevation as transmission expands northward (Randolph and Rogers, 2000)."

Comment (5-79):
One commenter (3347.3) argues that "the TSD includes statements regarding food-borne diseases that cannot be supported by the science." The commenter cites a recent CDC report (CDC, 2009) that suggests the incidence of food-borne illnesses over the past three years has hit a plateau. The commenter additionally states that "significant declines since 1996 were reported in the incidence rates involving numerous, other food-borne infections." The commenter adds that Kovats et al. (2005) find that campylobacteriosis rates peak before high annual temperatures and that according to CCSP (2008b), pathogenic species of *Campylobacter* cannot replicate in the environment and will not persist long under non-microaerophilic conditions, suggesting that high ambient temperatures would not contribute to increased replication in water or in food products. The commenter concludes that "[e]xisting scientific evidence does not support the notion that increasing global temperatures will increase rates of all food-borne diseases."

Response (5-79):
We have reviewed the assessment literature in light of this comment and the referenced studies, and we find that the TSD's summary of this issue appropriately reflects the judgment of the scientific community as reflected in the assessment literature. Therefore, we disagree with the comment that the TSD includes information regarding food-borne disease that "cannot be supported by the science."

The TSD does not state or imply that increasing global temperatures will increase rates of all food-borne diseases. Rather, it includes a much more qualified conclusion based on Confalonieri et al. (2007): "Foodborne diseases show some relationship with temperature (e.g., increased temperatures have been associated with increased cases of Salmonellosis)." This statement is fully consistent with the CCSP analysis (Ebi et al., 2008), which states that "[E]vidence supports the notion that increasing global temperatures will likely increase rates of salmonellosis," noting that studies in North America and several countries across Europe "report striking similarities in correlations between peak ambient temperatures (controlled for season) and peak in clinical cases of salmonellosis."

We find that the commenter's discussion of *Campylobacter* is largely consistent with the assessment report the commenter cites (CCSP, 2008b), which references the Kovats et al. (2005) study cited by the commenter. However, the commenter did not mention that *Campylobacter* transmission can be affected by factors other than replication in water or food products. CCSP (Ebi et al., 2008) concludes that it is more likely than not that increasing temperatures may expand typical peak season of clinical infection, or result in earlier peak. It also assesses that it is about as likely as not that increasing temperatures will result in shorter development times for flies, contributing to increased transmission by this proposed vector.

With regard to the CDC report referenced by the commenter, we note that a three-year period is not sufficient for analyzing long-term trends in food-borne illness and that the incidence of some food-borne infections could well change due to non-climate factors such as public awareness advances, food processing changes, or changes in eating habits. This does not mean that the incidence of food-borne diseases will not increase due to climate change—all else being equal—but rather, as the TSD notes, that other factors will also play a role. EPA concludes that the TSD's summary of the current state of the science on this topic as reflected in the assessment literature is accurate and sound.

Comment (5-80):

Several commenters (2633, 3077, 3961, 4791, 4818, 4820, 4923, and 9345) cite the increase in asthma cases as a health concern due to climate change. Many (3077, 4818, 4820, 4923, 9345) are specifically concerned about asthma in children. One commenter (3961) states that researchers have found that asthma incidence has quadrupled in the United States since 1980 and that the "drivers" include "rising CO_2 which increases the allergenic plant pollens and some soil fungi, and dust clouds containing particles and microbes coming from expanding deserts, compounding the effects of air pollutants and smog from the burning of fossil fuels." Another commenter (2633) notes that today nearly 30 million adults and children in the United States have been diagnosed with asthma.

One commenter (11249) notes that as temperatures increase historic seasonal patterns change, leading to an earlier and potentially longer allergy season. The commenter (11249) cites that allergic diseases are the sixth leading cause of chronic disease in the United States and impose a substantial burden on the U.S. population, noting that the global rise in asthma is an early health effect of climate change.

Response (5-80):

As indicated in the TSD, climate change, including change in CO_2 concentrations, could impact the production, distribution, dispersion, and allergenicity of aeroallergens and the growth and distribution of weeds, grasses, and trees that produce them (McMichael et al., 2001; Confalonieri et al., 2007). We also note the commenter's point that an earlier and potentially longer allergy season is consistent with information in the TSD that was reaffirmed in the recent USGCRP report (Karl et al., 2009). While the commenter does not provide a reference for chronic diseases in the United States, it is true that a

significant number of people suffer from allergic diseases. However, projections of the possible impacts of climate change on allergenic diseases are difficult to assess.

Comment (5-81):

Numerous commenters (e.g., 2750, 3347.1, 3347.3, 3394.1, 3449.1, 3509.1, 3722, 3747.1) contend that the proposed endangerment finding offers no support regarding the effect of GHG levels on aeroallergen-related health risks, and that the scientific literature does not provide definitive data or conclusions on how climate change might impact allergenicity and aeroallergens. This includes the incidence, prevalence, distribution, and severity of allergic disorders. Commenters note that there are numerous factors that affect aeroallergen levels and the prevalence of associated allergenic illnesses, such as changes in land use, air pollution, and adaptive responses, many of which are difficult to assess. One commenter (3722) additionally criticizes the indefinite language used (the word "could") and EPA's acknowledgment that the scientific literature lacks "definitive data or conclusions," as "reduc[ing] this issue to the level of speculation." Commenters suggest that the EPA has made a predetermination about the effect of GHG levels on aeroallergen-related health risks based completely on unfounded, scientifically unsupported, and hypothetical harms. Without evidence, the issue of aeroallegens should not figure into the calculation of harm at all. One commenter (3722) states that "it is surprising that EPA gives any weight whatsoever to aeroallergens in the Proposed Rule." Another (3349.1) states that EPA "reaches an arbitrary and capricious conclusion." Another (3347.3) asks EPA to revise the TSD to accurately portray scientific knowledge, and present that knowledge objectively. This commenter goes on to state that the TSD section on aeroallergens "does not meet the standard of objectivity because it includes only scattered information suggestive of a potential link, and it presents this information with a notable bias that exaggerates the likely effect of climate change."

Response (5-81):

EPA disagrees with the commenters that the TSD was developed without sensitivity to uncertainties in this area, that EPA did not accurately portray scientific knowledge, or that EPA presented "scattered information" with a "notable bias" that exaggerates the likely effect of climate change. A review of Section 7(d) of the TSD confirms that it accurately conveys the findings of the scientific literature. The TSD clearly states in Section 7(d) that "the scientific literature does not provide definitive data or conclusions on how climate might impact aeroallergens and subsequently the prevalence of allergenic illnesses in the U.S." The TSD notes that numerous other factors affect aeroallergen levels and the prevalence of associated allergenic illnesses. Consistent with the scientific literature, EPA describes the impacts of elevated temperature and CO_2 on aeroallergens, including IPCC's conclusion that "pollens are likely to increase with elevated temperature and CO_2," as well as discussion of impacts on season length and allergenic content of pollens. We agree with the commenters, and state in the TSD, that numerous factors affect aeroallergen levels and the prevalence of associated allergenic illnesses. The information provided in the section is not scattered, but rather presents the scientific assessment on a range of climate change impacts on aeroallergens. Additionally, the information is presented objectively and in an unbiased fashion. All of the references cited by EPA in the aeroallergen section of the TSD are from IPCC or CCSP, and thus are part of the body of peer-reviewed assessment literature upon which EPA relies for the TSD. See Section III.A., "The Science on Which the Decisions Are Based," for our response to comments on the use of the assessment literature and our treatment of new and additional scientific literature provided through the public comment process.

We disagree with the assertion by the commenter that EPA's use of "indefinite language (the word 'could')," and EPA's statement that the scientific literature lacks definitive data or conclusions, means that the TSD's conclusions on aeroallergens are speculative. We clearly characterize the existing level of scientific uncertainty regarding climate change impacts upon aeroallergens, based upon the peer-reviewed assessment literature. For a response to comments regarding EPA's characterization of scientific

certainty, please see Volume 1, Section 1.2, "Level of Scientific Certainty," in this Response to Comments document.

We also disagree with the commenters that EPA reached a predetermined or arbitrary and capricious conclusion. EPA conducted a careful review of the peer-reviewed assessment literature, which is reflected in the TSD, and upon which the Administrator based her judgment on aeroallergens. See the Findings, Section IV.B, "The Air Pollution Is Reasonably Anticipated to Endanger Both Public Health and Welfare," for our response to comments on how the Administrator weighed the scientific evidence underlying her endangerment determination.

Finally, we note that commenters did not submit any evidence that shows the TSD's discussion and conclusions to be inaccurate. Thus, EPA finds the discussion of aeroallergens in the TSD to be reasonable and sound.

Comment (5-82):
One commenter (7020) states that the TSD conclusion on aeroallergens shows that there are offsetting effects that cannot be resolved in one direction or the other that the EPA must improve this section to address offsetting effects, and that if this balance cannot be resolved, then aeroallergens cannot influence a potential endangerment finding. The commenter defines offsetting effects as when one mechanism produces an adverse effect and another mechanism produces a beneficial effect.

Response (5-82):
We disagree that the TSD conclusions presented on aeroallergens, which summarize the peer-reviewed assessment literature, are invalid because the TSD does not present a full accounting of "adverse" and "beneficial" effects. The TSD clearly describes the current state of scientific understanding on the issue of climate change impacts on aeroallergens. We also note that the commenter did not identify any potential beneficial effect from aeroallergens for us to consider. For EPA's discussion on the consideration of net effects in the TSD, please see Volume 1, Section 1.4: "Net (Adverse and Beneficial) Effects," of this Response to Comments document.

Comment (5-83):
One commenter (3283.1) states that EPA has not been able to cite any quantifiable risk with regard to aeroallergens, and that risks must be quantified before a massive regulatory structure is implemented to reduce this risk.

Response (5-83):
We disagree that climate change impacts must be quantified for inclusion in the TSD and consideration with regard to an endangerment finding. We note that the peer-reviewed assessment literature, on which we rely for the TSD, is limited with regard to projections of aeroallergen impacts, and thus we provide a qualitative, not quantitative, evaluation of this issue. We also note that the commenter did not cite any new literature for us to consider with regard to quantifying the risk to aeroallergens from climate change. For EPA's discussion on the quantification of climate change impacts in the TSD, please see Volume 1, Section 1.4, "Net (Adverse and Beneficial) Effects," of this Response to Comments document.

Comment (5-84):
One commenter (3283.1) states that "medical technology has developed techniques to minimize many allergic reactions" and that "new medicines will be available to nullify allergic reactions."

Response (5-84):
The commenter's implication seems to be that new medical techniques and medicines would serve as adaptation measures that would offset any adverse health effects from climate change impacts upon aeroallergens. See Section III.C, "Adaptation and Mitigation," of the Findings for our response to comments on the treatment of adaptation and mitigation in the Findings.

Comment (5-85):
One commenter (3347.3) states that the following statement from the TSD is unsupported: "Climate change, including changes in CO_2 concentrations, could impact the production, distribution, dispersion and allergenicity of aeroallergens and the growth and distribution of weeds, grasses, and trees that produce them (McMichael et al., 2001; Confalonieri et al., 2007). These changes in aeroallergens and subsequent human exposures could affect the prevalence and severity of allergy symptoms." The commenter states that relatively small increases in pollen exposure may have a protective effect against acquiring allergic rhinoconjunctivitis or asthma. The commenter cites one study in support of this (Burr et al., 2003), and states that the study reports a weak but consistent tendency for the prevalence of allergic symptoms to be inversely associated with pollen exposure. The commenter states that based upon this it seems that high pollen exposure either has no effect on the risk of acquiring allergic rhinoconjunctivitis or may even confer some protection against it. The commenter notes that this conclusion is backed up by the study, which cites evidence from several countries suggesting that the prevalence of hay fever and asthma is lower in rural than urban areas.

Response (5-85):
We disagree that the IPCC statement quoted by the commenter lacks adequate support and note that the commenter provides no evidence that counter the IPCC statement. See Section III.A., "The Science on Which the Decisions Are Based," for our response to comments on the use of the assessment literature and our treatment of new and additional scientific literature provided through the public comment process.

The commenter cites only one study (Burr et al., 2003) to support the assertion that the TSD statement was unsupported. However, this study does not contradict and in fact supports the information presented in the TSD. The study was cited in the peer-reviewed assessment literature (Ebi et al., 2008). As the Burr et al. (2003) study states in its concluding paragraph:

> Overall, it seems very unlikely that high exposure to pollen increases the risk of acquiring allergic rhinoconjunctivitis or asthma, despite its action in provoking attacks in susceptible persons. The associations in this study were very weak, and it may be that pollen exposure has no influence on the prevalence of atopic disease. Nevertheless, the degree of consistency in the negative direction of these associations suggests the possibility of a protective effect.

EPA concludes that IPCC's statement on aeroallergen impacts from climate change is reasonable and sound. To the extent there is a protective effect, this would mean that climate change would not be expected to increase the risk of acquiring these illnesses, and EPA does not rely on any such increased risk in making the findings. However, the literature does support the concern that climate change could increase the incidence of allergy-related symptoms or illness for those susceptible persons who do have allergic rhinoconjunctivitis or asthma.

Comment (5-86):
One commenter (3447.3) states that the following statement from the TSD lacks adequate support: "The IPCC concluded that pollens are likely to increase with elevated temperature and CO_2 (Field et al., 2007)." A number of other factors influence aeroallergen levels, including "changes in the spatial distribution of natural vegetation, introduction of invasive species, changes in land use, air pollution, adaptive responses, water availability, disturbance, nutrients, and meteorological factors (e.g. rainfall, humidity, wind)." The commenter notes that most studies focus only on the effects of increasing CO_2 concentrations and temperature increase without considering any other factors, and are thus misleading. More studies are needed on the combined effects of CO_2 and temperature that also consider other interactions with other variables, and three studies are presented to highlight the importance of other factors:

1. Schenk et al. (1995) is cited as looking at the effects of CO_2 enrichment and intraspecies competition on a range of plant attributes, with results showing that some plant attributes changed in response to CO_2 concentration, but other plant attributes were dependent on plant density.
2. Ziska et al. (2007) is cited as "indicat[ing] that CO_2/temperature differences associated with urbanization may increase initial ragweed productivity and pollen production, but suggest that long-term, multi-year persistence of ragweed may not occur in the urban macro-environment due to the influence of other factors."
3. Curtis and Wang (1998) is cited as showing that stress effects significantly reduce overall woody plant biomass accumulation from heightened CO_2.

Response (5-86):
We disagree that the IPCC statement quoted by the commenter lacks adequate support and note that the evidence provided by the commenter does not demonstrate that there is inadequate support for the IPCC statement. See Section III.A., "The Science on Which the Decisions Are Based," for our response to comments on the use of the assessment literature and our treatment of new and additional scientific literature provided through the public comment process.

We have reviewed the three studies cited by the commenter and find that two of them (Curtis and Wang, 1998; Schenk et al., 1995) do not directly assess pollen production and thus offer no direct support for the substance of the comment. We find that the third study (Ziska et al., 2007) indeed suggests that multi-year persistence of ragweed in the urban macro-environment may be dependent on factors other than CO_2 concentrations; however, we note that the study took place in one specific location at the urban-rural interface and furthermore found that urbanization may increase initial ragweed productivity and pollen production.

We note that the Van de Water (2008) study cited elsewhere by the commenter is highly consistent with the assessment literature conclusion on this topic. We further note that the most recent assessment report to address this issue, Karl et al. (2009), finds that "Rising temperatures and carbon dioxide concentrations increase pollen production and prolong the pollen season in a number of plants with highly allergenic pollen, presenting a health risk."

Additionally, Field et al. (2007) base their assessment on several peer-reviewed studies and state that "Warming and climate extremes are likely to increase respiratory illness, including exposure to pollen and ozone." EPA concludes that the IPCC (Field et al., 2007) conclusion that pollens are likely[4] to increase with elevated temperature and CO_2, as summarized in the TSD, is accurate and sound.

[4] According to IPCC terminology, "likely" conveys a 66% to 90% probability of occurrence. See Box 1.2 of the final TSD for a full description of IPCC's uncertainty terms.

Comment (5-87):
One commenter (3447.3) criticizes the following statement from the TSD as lacking support: "The IPCC (Confalonieri et al., 2007) noted that climate change has caused an earlier onset of the spring pollen season in North America and that there is limited evidence that the length of the pollen season has increased for some species. However, it is unclear whether the allergenic content of these pollens has changed." The commenter also states that "there is little evidence to support an increase of the length of the allergy season and changes in the pollen content that may increase its health impact." The commenter makes numerous points:

> Data are lacking to support an overall increase in the length of the pollen season or a change in intensity. The commenter cites WHO (2003) in noting that the observed trends in the end of the season phases as a result of climate change are unclear; therefore, it is uncertain how the pollen season length will be affected.
> Changes in the pollen season may differ for different plant species, and the length of the pollen season may shorten in certain geographical regions. The commenter cites an example from WHO (2003) in stating that "For example, increased snowfall (due to increased precipitation) takes longer to melt and may cause a later onset of flowering in Northern latitudes."
> "[T]he burden of immune-mediated (IgE) allergic diseases is related to the length of pollen season as well as the total pollen counts and the number/height of the pollen peaks and bioallergen availability." The commenter cites WHO (2003) as presenting several possibilities with regard to aeroallergens from climate change. Additionally, the commenter states that depending on the actual effects, it remains uncertain how they will impact the severity of symptoms:
>
> - An earlier start of the season, but with several smaller peaks.
> - Seasons are prolonged, but also divided into multiple sub-seasons with smaller peaks.
> - Winter temperature influences the waking of trees leading to effects on the size of the peak of the pollen season.
>
> It is unclear if the allergenic content of the pollen type has changed. The commenter cites Levetin and Van de Water (2008) as noting that data from U.S. sampling stations are limited.

Response (5-87):
We disagree that the IPCC statement from the TSD lacks support. The IPCC references three separate studies in support of its conclusion. The commenter, by contrast, did not provide any literature that contradicts the IPCC conclusion. We have reviewed the submitted references and on the basis of our review find that they are consistent with the relevant IPCC conclusions.

The TSD does not state that length of the pollen season has increased. Rather, it notes that climate change has caused an earlier spring pollen season in the Northern Hemisphere and that there is "limited evidence that the length of the pollen season has increased for some species." Neither of these statements is inconsistent with any of the WHO (2003) conclusions cited by the commenter. In fact, while the WHO report in question does discuss a number of uncertainties, its overall conclusions are clearly directionally consistent with the assessment literature as summarized in the TSD. The report states that "Evidence is growing that climate change might facilitate the geographical spread of particular plant species to new areas as they become climatically suitable. Warming is likely to further cause an earlier onset and may extend the duration of flowering and pollen season, for some species (such as grasses and weeds)." Finally, the commenter's note with regard to increased snowfall potentially causing a later onset of flowering in northern latitudes is the only example given for shorter pollen seasons in certain geographic regions, and it is worth noting that this report was written specific to Europe. Thus, we find that the report does not provide support for the commenter's claim that "there is little evidence to support an increase of

the length of the allergy season and changes in the pollen content that may increase its health impact, either as a general conclusion or one relevant to the U.S."

We have reviewed the Levetin and Van de Water (2008) study and find that it is highly consistent with the assessment literature as reflected in the TSD. The study does not state that data from U.S. sampling stations are "limited," though it does note that "Plant phenology data for North America are not as widely available as European datasets." The authors conclude that "Elevated levels of atmospheric carbon dioxide and other greenhouse gases have resulted in global warming. The effect of these changes has impacted plant production, with increases in plant biomass, number of flowers, pollen per plant, and potential changes in pollen per anther. These effects have been shown in select plants through studies at elevated carbon dioxide levels."

EPA concludes that the TSD's summary of the current state of scientific knowledge on this topic as reflected in the assessment literature is accurate and sound. See Section III.A., "The Science on Which the Decisions Are Based," for our response to comments on the use of the assessment literature and our treatment of new and additional scientific literature provided through the public comment process.

Comment (5-88):
One commenter (3447.3) criticizes the following statement from the TSD as poorly supported: "The IPCC concluded that introductions of new invasive plant species with high allergenic pollen present important health risks, noting that ragweed (*Ambrosia artemisiifolia*) is spreading in several parts of the world (Confalonieri et al., 2007)." The commenter notes that, in addition to climate change, there are other influences on vegetation patterns, which include land use and land cover change as well as "socioeconomic changes such as the impact of agricultural policy on plant species composition." The commenter states that EPA failed to separate out these influences.

Response (5-88):
We agree that a variety of factors can influence vegetation patterns, including that of ragweed. However, regardless of the underlying cause(s), the increased spread of ragweed that has been observed could have important implications for aeroallergen impacts as the climate changes. As noted by IPCC (Confalonieri et al., 2007): "Several laboratory studies show that increasing CO_2 concentrations and temperatures increase ragweed pollen production and prolong the ragweed pollen season."

EPA concludes that IPCC's statement that the spread of invasive plant species with high allergenic pollen presents important health risks is reasonable and sound. See Section III.A., "The Science on Which the Decisions Are Based," for our response to comments on the use of the assessment literature and our treatment of new and additional scientific literature provided through the public comment process.

Comment(5-89):
One commenter (3447.3) states that EPA must recognize that factors unrelated to aeroallergen levels have been implicated in allergic disease onset and susceptibility. The commenter cites WHO (2003) in making the following statement: "infections in early life, microbial exposure, exposure to endotoxins, indoor and outdoor air pollution as environmental tobacco smoke or traffic-related air pollution, poor indoor/outdoor climates, allergen exposure and nutrition seem to coincide with the onset of allergic disease. When the disease is established, some of these factors may also act as triggers of symptoms."

Response (5-89):

We have reviewed the WHO report referenced by the commenter and we agree that factors unrelated to aeroallergen levels can affect disease onset and susceptibility. The TSD addresses this point, stating that "there are numerous other factors that affect aeroallergen levels and the prevalence of associated allergenic illnesses, such as changes in land use, air pollution, and adaptive responses, many of which are difficult to assess (Ebi et al., 2008)." EPA concludes that the TSD's summary of the assessment literature conclusions on this topic is reasonable and sound.

Comment (5-90):

Commenters (0553, 3394.1, 3722, 7031, and 10158) indicate that it is well known that CO_2 is not a health hazard for humans or animals at atmospheric concentrations reasonably expected to ever be reached, even if all known fossil fuel reserves are combusted and CO_2 atmospheric retention times are several centuries. Commenters note that this does not support endangerment. One commenter notes that greenhouse operations use elevated CO_2 concentrations to promote plant growth, yet greenhouse workers are completely unaffected. One commenter notes that Occupational Safety and Health Administration limits for air contaminants indicate that human exposure to CO_2 should not exceed 5,000 parts per million (ppm) during an 8-hour work day, which is well above ambient concentrations of CO_2. Another commenter notes the maximum allowed CO_2 concentrations for U.S. submarines is 8,000 ppm and many office buildings register greater than 1,000 ppm CO_2 levels with no harmful effects.

Response (5-90):

EPA agrees with the commenter that exposure to current and projected elevated CO_2 concentrations in the atmosphere would not have direct adverse impacts on human health, and notes that the science on this issue is summarized in Section 3, "Direct Effects of Elevated Greenhouse Gas Concentrations," as well as in Appendix C: "Direct Effects of Ambient GHG Concentrations on Human Health." The TSD also summarizes health effects associated with climate impacts caused by elevated GHGs. See the Findings, Section IV.B.1(f), for our response to comments on the consideration of health impacts stemming from climate change impacts as relevant to a determination on endangerment of public health impacts.

Comment (5-91):

Commenter (3607.1) indicates that the TSD's Appendix C, "Direct Effects of Ambient GHG Concentrations on Human Health," which discusses direct effects of the six ambient GHG concentrations on human health, indicates that "both current and projected atmospheric concentrations of these GHGs do not pose exposure risks on human respiratory systems (i.e., breathing/inhalation)." Further, the commenter indicates that EPA notes that the projected concentrations in 2100 are "well below" the thresholds for adverse effects; however, this conclusion is rather obscure and is in the last sentence of the discussions for CO_2, methane (CH_4), and nitrous oxide (N_2O). The commenter indicates that EPA should move this conclusion to the first sentence of each of these sections as it has done for the fluorinated gases section.

Response (5-91):

EPA notes that the Executive Summary of the TSD states, "Current ambient air concentrations of CO_2 and other GHGs remain well below published exposure thresholds for any direct adverse health effects, such as respiratory or toxic effects." In addition, the first sentence of Appendix C in the TSD states: "Greenhouse gases, at both current and projected atmospheric concentrations, are not expected to pose exposure risks on human systems (i.e., breathing/inhalation)." Thus, we find that the TSD clearly states the known relationship between atmospheric GHG concentrations and human exposure risks. In addition, this point was made explicit in the proposed endangerment finding under the discussion of public health impacts. Therefore, we do not find that a change to the TSD is necessary.

5.2 Air Quality

Comment (5-92):
Two commenters (2123, 2633) state that breathing polluted air can cause burning eyes and throat irritation, and that tiny airborne particles and ground-level ozone can trigger respiratory problems, especially for people with asthma. These commenters (2123, 2633) note that some toxic chemicals released in the air such as benzene or vinyl chloride are highly toxic and can cause cancer, birth defects, long-term injury to the lungs, as well as brain and nerve damage.

Response (5-92):
We note the comments regarding respiratory illness and other health effects are associated with PM and ground-level ozone. We concur with the commenter regarding the health effects associated with these pollutants and note that Section 8(c) of the TSD summarizes the health effects due to CO_2-induced increases in tropospheric ozone and PM. Regarding toxic chemicals such as benzene or vinyl chloride we note these chemicals and their emissions are outside the scope of this action. This action pertains to the well-mixed GHGs identified in the Findings, which include CO_2, CH_4, N_2O, hydrofluorocarbons (HFCs), perfluorocarbons (PFCs) and sulfur hexafluoride.

Comment (5-93):
Several commenters (2898.1, 3136.1, 3283.1, 3347.1, 3347.3, 3347.4, 3394.1, 3449.1, 3603.1, 3722, 3747.1) indicated that the TSD analyses of the impact of GHG emissions on air quality should include scenarios that assume future emission control programs. The commenters indicate that EPA should conduct a robust national-scale risk assessment that considers the impact of climate change on ozone and PM under current conditions versus future conditions, including the impact of ongoing regulatory emission control programs. Until such an analysis is available, they state, it is not possible to determine if climate change will negatively affect health due to changes in air quality. According to these commenters, these future emission reduction programs (e.g., the Clean Air Act's National Ambient Air Quality Standards [NAAQS] implementation measures) will ensure no adverse impact on public health due to climate change. By excluding consideration of highly relevant modeling showing a much different future outcome for ozone and PM, these commenters argue, EPA undermines the link between credible science and the endangerment proposal.

Similarly, several commenters (2898.1, 3136.1, 3477, 3596.2, 4003, 9863, and 10499) ask how EPA can support an IPCC claim that declining air quality is a "virtual certainty" when, partly because of EPA efforts, air quality has been improving in the United States in a warming climate. Commenters note that ozone pollution in the United States as measured by the EPA continues to decline, and the assertion that ozone pollution will increase is not supported by these trends. One commenter (4003) submits a figure from EPA's Air Quality Trends Web site which indicates a 21% decrease in ozone based on annual fourth-highest 8-hour average[5] in support of their point that ozone concentrations have been decreasing since 1970. Commenters state that EPA's current description fails to capture the nature of observed trends in ground-level ozone concentrations during a period of increasing temperatures across the United States and describes projections that seemingly are oblivious to the existence of such trends. These comments question the utility of these projections in addressing the potential future of low-level ozone concentrations across the United States, and contend that EPA is making an endangerment finding that is not based upon the most relevant data. The commenters note that lacking any strong guidance that climate

[5] http://www.epa.gov/airtrends/ozone.html

change will somehow act to more than offset the gains through regulatory actions into the future, the best indication of what is to come should be gained from what we have observed—that is, declines in PM concentrations despite increases in human GHG emissions, increasing temperatures, and increasing trends in wildfire activity.

Commenters suggest that the TSD and EPA's integrated assessment (IA) of the impacts of global change on regional U.S. air quality ignored the National Exposure Research Laboratory (NERL) modeling of the companion FUT2 simulation; this scenario entails ozone precursor emissions reductions across the United States consistent with the 2015 implementation of the Clean Air Interstate Rule (CAIR). One comment (3347) suggested that a variety of future baseline ozone and PM reduction scenarios should be fully examined.

Response (5-93):

Contrary to the assertion of the commenters, EPA has conducted a national-scale assessment of the impact of climate change on air quality as represented in the IA and reflected in the TSD, and this assessment included available results on the combined impacts of climate change and changes in emissions of criteria pollutants.

The IA notes the challenges associated with developing spatially detailed, plausible scenarios of future U.S. and worldwide emissions of criteria pollutants. For example, the IA states that:

> Future emissions scenarios that are consistent across pollutants and geographic scales and that incorporate important processes such as fire, land use, biogenic emissions, and technological change are lacking, limiting the kinds of studies that can be accomplished at this time…Plausible scenarios for future emissions need to be developed that account for changes in urbanization, population growth, migration, industrialization, fuel, technology, etc.

Therefore, the current uncertainty in future ozone precursor emission scenarios is large, limiting their application for the purpose of determining the sensitivity of ozone air quality to GHG emissions compared to scenarios that hold ozone precursor emissions constant (and therefore allow the research community to isolate the signal of the sensitivity of ozone to climate change).

Despite these caveats, the IA fully considered available results from all of the modeling groups whose simulations included both climate and criteria pollutant emissions changes. For example, EPA considered all the results from the NERL CIRAQ effort that was singled out for comment. These results are reported on and extensively discussed in the IA (e.g., in the key synthesis chapter and conclusions), and all of the published results from the NERL CIRAQ, including the FUT2 scenario, are considered by EPA in the IA and are included in the literature summarized in the TSD. Additional details of the CIRAQ effort including the FUT2 scenario are provided in response 5-95 below. Beyond the NERL CIRAQ results upon which the commenters focus, the IA also considered all available results on combined climate and criteria pollutant emissions changes from the other regional modeling groups, including the Columbia, Georgia Tech, University of Illinois, and Washington State teams. Synthesizing across all these results, the IA concludes: "Preliminary work suggests that the impacts of climate change on future U.S. regional O_3 [ozone] concentrations remain significant when also considering possible future anthropogenic O_3 precursor emissions changes." Therefore, these results, in aggregate, support the overall finding that climate change will negatively affect health due to changes in air quality. Furthermore, the IA goes on to state (about the results from the studies just mentioned):

> A number of the modeling teams whose results are discussed in this report also carried out simulations with modified future air pollutant emissions generally derived from the

> assumptions used to formulate the various IPCC GHG emissions scenarios. These preliminary tests found that the combined effects of climate and anthropogenic precursor emissions changes are highly sensitive to the assumptions about future emissions trajectories.

To summarize, the commenters' assertion that alleged EPA selectivity about which studies to consider "undermines the link between credible science and the endangerment proposal" is demonstrably false. As described above, the IA, considered available simulation results that included scenarios of changes in criteria pollutant emissions along with climate change, when drawing their conclusions, and the findings of the IA are accurately summarized in the TSD.

At the same time, EPA properly recognized that such modeling of future climate change with future emission control programs does not isolate the impacts of GHGs on air quality. Because of this EPA also considered modeling of future climate change without future emissions control programs, to isolate the impacts of GHGs on air quality. Many of the projections of ozone increases and related mortality and morbidity are tied to modeling that focuses on isolating the impact of climate change, and this modeling shows significant adverse impacts from climate change, typically resulting from the combination of climate associated increases in biogenic emissions of volatile organic compounds (VOCs), climate change impacts on temperature and atmospheric chemistry, and climate change impacts on atmospheric circulation. Climate change can have an important impact on past trends in ozone reduction as well as future projections of ozone. For example, Leibensperger et al. (2008) use detailed analysis of long-term observations and conclude:

> …[T]he decrease in mid-latitude cyclones over the period 1980–2006 has offset half of the air quality gains in the Northeast U.S. that should have been achieved from reduction of anthropogenic emissions over that period. This suggests that climate change has already had a major effect on the accountability of emission control strategies over the past 2–3 decades, preventing achievement of the ozone air quality standard. It demonstrates the potential of climate change to dramatically affect air quality on decadal scales relevant to air quality policy.

The challenge that "EPA's current description fails to capture the nature of observed trends in ground level ozone concentrations during a period of increasing temperatures across the U.S. and describes projections that seemingly are oblivious to the existence of such trends" is not supported by the results presented in the IA and summarized in the TSD. First, the IA explicitly notes the observed trend of generally decreasing U.S. ground-level ozone concentrations and explicitly recognizes the contribution of EPA's regulatory efforts to this trend (in Section 1 of the IA). Second, while the process of coupling climate with atmospheric chemical processes in modeling systems presents considerable scientific and technical challenges, because of the large number of physical, chemical, and biological processes involved, the complex interactions between them, and the scientific uncertainties associated with each, the modeling systems whose results are synthesized in the IA have been shown to be able to capture the observed historical trends in U.S. ozone concentrations to which the commenters refer (as described in Section 3 of the IA). From the perspective of the IA's research questions, however, the major advantage of these modeling systems is that they permit the detailed exploration of the potential responses of air quality to climate change over the next few decades in a way that would be difficult or impossible with other approaches—most saliently the air quality implications of climate changes well outside the envelope of historical experience. It is precisely because 1) GHG-induced climate change over the next several decades is expected to be greater than over the last few decades and 2) there are large uncertainties associated with modeling future U.S. and worldwide criteria pollutant emissions changes (as described in the paragraphs above) that these model simulations are considered useful in providing

scientific support for the TSD. Under these circumstances they are able to provide useful information that is not obtained by just looking to observations or trends of the past.

Finally, substantial challenges remain with respect to achieving the air quality protection under the NAAQS for ozone, and these challenges will likely be exacerbated by climate change. See the Findings, Section IV.B.1.f and Volume 9 of this Response to Comments document for EPA's response to comments on the relevancy of NAAQS in the context of the Administrator's endangerment finding.

Comment (5-94):
One commenter (2898.1) states that the only way to isolate the "global warming effect" on ozone pollution would be to compare ozone levels in warming and non-warming scenarios based on realistic projections of precursor emissions in the 2020s, 2050s, and 2080s.

Response (5-94):
See response 5-93 in this section regarding the rationale for the most appropriate method for isolating the effect of climate in the selection of modeling scenarios.

Comment (5-95):
A number of commenters (3347.1, 3347.3, and 3347.4) contend that the TSD fails to mention the results of three important EPA-sponsored projects that considered the impact of climate change in conjunction with existing emission control programs: the Georgia Tech Phase I STAR Grant Project, the EPA Climate Impact on Regional Air Quality (CIRAQ) Project, and the EPA Intercontinental transport and Climatic effects of Air Pollutants (ICAP) project. The commenters state that these three research programs show that the impact of climate change alone on U.S. regional air quality is minor when compared to the effects of continued Clean Air Act regulatory strategies for emissions control. The CIRAQ future case FUT2 modeling clearly demonstrates that maximum daily 8-hour (MDA8) ozone levels in 2050 are likely to decrease as the result of continued regulatory programs, notwithstanding the countervailing effects of climate change. This EPA modeling shows reductions in 2050 8-hour ozone concentrations as large as 12 to 15 ppb over several sub-regions of the country.

The commenters state that, in sum, the Georgia Tech or Georgia Institute of Technology (GIT) ozone modeling shows that the combined effect of climate change and emission reductions lead to overall ozone reductions up to 15 ppb. The Georgia Institute of Technology projected 2050 emissions reductions of more than 60% in nitrogen oxides (NO_x) and sulfur dioxide in 2050 as the result of control programs and the A1B global growth scenario. In percentage terms, there is likely to be a 20% decrease in the mean summer MDA8 ozone mixing ratios. Regionally, the eastern United States appears to benefit more than the rest of the country from reductions in mean daily 8-hour average (D8H) ozone because of both spatial variations in the meteorological and emissions changes. The commenters indicate that reductions in the higher 8-hour maximum ozone concentrations were calculated for all U.S. sub-regions and there are likely to be fewer days with D8H ozone above the level of the former NAAQS (85 ppb) in a number of metropolitan areas. Further, the commenter indicates that independent modeling by EPA's Office of Research and Development (ORD) (Nolte et al., 2008, 2009[6]) and Office of Air Quality Planning and Standards (OAQPS) (Jang et al., 2008) corroborate the Georgia Tech results. On the other hand, the commenter also noted that the Georgia Tech results include finding that climate change by itself has mixed effects on the 8-hour ozone levels, but the maximum daily 8-hour ozone levels are anticipated to generally increase.

[6] Nolte et al. (2009) refers to a poster presentation which references the Nolte et al. (2008) study.

Response (5-95):
We disagree with the commenter that studies that consider emission controls in conjunction with climate change were not considered in the development of the IA and reflected in the TSD. Other studies (e.g., Georgia Tech STAR grant project and the EPA CIRAQ project) were undertaken as part of the IA. The results from both projects (along with STAR projects at the University of Illinois, Washington State University, Carnegie Mellon University, Harvard University, University of California–Berkeley, and Columbia University) are discussed in the key synthesis chapter of the IA and build the foundation of the conclusions of the report. Thus, we confirm these studies are considered by the IA and the literature underlying the TSD.

The IA clearly acknowledges that results from these two studies show that emission reductions can be very effective at reducing air pollution to healthful levels. We also note that the conclusions of the Georgia Tech study (Tagaris et al., 2009) are consistent with the summary in the TSD. In particular, Tagaris et al. (2009) conclude: "Results suggest that climate change driven air quality-related health effects will be adversely affected in more than two-thirds of the continental United States."

To provide further clarification, as described in the IA and in Nolte et al. (2008), EPA initiated the CIRAQ project in 2002 to develop a pilot modeling study to incorporate regional-scale climate effects into air quality modeling. Historical values for GHGs were used for 1950–2000, with future GHG forcing following the Intergovernmental Panel on Climate Change's A1B SRES scenario. Two future simulations were conducted, one with anthropogenic emissions held at 2001 levels (FUT1), and one with anthropogenic emissions reduced in accordance with the A1B scenario (in FUT2, emissions of NO_x were projected to decrease by 45%). Considering just climate change alone, with future anthropogenic emissions held at current levels (i.e., FUT1), ozone increases in much of the eastern United States. This is consistent with the findings of Hogrefe et al. (2004), who reported ozone increases in summertime for the 2050s as a result of climate change alone under the A2 GHG scenario. For the future reduced emissions case FUT2, however, ozone decreases nearly everywhere in the continental United States. These results imply that, at least for the magnitude of emissions control assumed for the United States under the IPCC A1B scenario, the decrease in surface ozone due to the change in emissions outweighs the increase due to climate change. This conclusion is contrary to that of Hogrefe et al. (2004), who found that climate change and future emissions changes under the A2 scenario would both lead to enhanced surface ozone levels in the eastern United States. Under the A2 scenario, NO_x and VOC emissions increase in the United States, which accounts for their dramatically differing results. The authors emphasize that although the difference in temperature change at 2050 predicted by a range of global climate models in response to the A1B and A2 scenarios is small at about 0.25°C (IPCC, 2001) and as a consequence the impact of climate change on regional air quality is similar between these two scenarios, the simulated ozone changes are substantially different, and indeed of opposite sign, if changes in ozone precursor emissions associated with these two scenarios are considered as well. Nolte et al. (2008) also caution that "the uncertainty in the future emission scenarios for ozone precursors is quite large."

As stated in the final TSD:

> [T]he results in the IA demonstrate that O_3 responds to climate change in a qualitatively consistent manner across the simulations from multiple research groups. The patterns of relative changes in regional climate vary across the same simulations. Figure 3-11 of the IA graphically illustrates the net change in daily average ozone values across the research results for summertime ozone. Ozone concentrations increase across most areas of the country with decreases limited to some parts of the Southwest. The net increases of ozone concentrations in the large population centers of the northeastern and middle Atlantic United States are the results with the highest confidence. The net increases in the

Southeast and the small net changes in the Northwest are the features with the lower confidence.

Response 5-102 below provides Figure 3-11. Thus, the net impact of climate change and emissions control programs may be a net decrease or a net increase in ozone levels, depending on the emissions control scenario that is modeled. However, in all cases the overall effect of climate change itself is to increase ozone levels compared to what would occur without the climate change. See response (5-93 for our response to comments regarding the selection of modeling scenarios in the context of the Findings.

Comment (5-96):
A commenter (3722) indicates that EPA already has all the authority needed and indeed the legal obligation, enforceable by citizen suit, to limit ozone to levels protective of human health and welfare.

Response (5-96):
See the Findings, Section IV.B.1.f, and Volume 9 of this Response to Comments document for EPA's response to comments on the relevancy of the NAAQS in the context of the Administrator's endangerment finding. We note that this action does not concern the scope or effectiveness of current or projected EPA authorities; its focus is on assessing the impacts of climate change on the United States and providing the foundation for the Administrator to determine whether GHG air pollution endangers public health or welfare.

Comment (5-97):
A commenter (3347.4) believes that inadequate attention is given to the time and space scales of available global and regional modeling results compared to the resolutions needed for reliable health risk assessments and ozone/$PM_{2.5}$ attainment demonstrations. Essentially all of the modeling considered by the TSD and IA had a horizontal grid spacing no finer than 36 kilometers. In contrast, EPA's 8-hour ozone attainment test requires that modeled concentrations possess substantially finer time and space resolutions and cover at least three contiguous years. The ozone attainment test requires use of the average of the fourth-highest 8-hour ozone concentration in the base year within a 3-by-3 array of 4-kilometer grid cells. $PM_{2.5}$ attainment modeling should be applied to grid meshes of 12 kilometers. Thus, the commenter concludes that the modeling relied upon in the TSD is too coarse for reliable estimates of NAAQS compliance in future years and insufficiently resolved to provide the gridded ground-level concentrations needed when performing realistic health risk assessments.

Response (5-97):
The TSD has been developed to inform the Administrator's endangerment analysis, not to inform area specific health risk assessments or attainment demonstrations (e.g., NAAQS). To support its intended purpose, the summary in the TSD properly focuses on the projected impact of global GHG emissions on large-scale patterns of air quality via induced climate changes as represented in general circulation models (GCMs). Confidence in these projections accordingly decreases the finer the spatial scale and further out into the future. Thus, larger grid spacing is utilized to minimize uncertainties associated with methods of downscaling and to focus on regional- and national-scale air quality changes, rather than the local-scale that is currently used for attainment modeling. We conclude the spatial and temporal scales used in the TSD and in the context of the Findings are appropriate and consistent with the scales used by the scientific assessment literature. See the Findings, Section IV.A.1.f, and Volume 9 of this Response to Comments document for response to comments on the relevancy of NAAQS in the context of the Administrator's endangerment finding.

Comment (5-98):
Two commenters (3136.1, 3722) state that if the Brewer-Dobson circulation is indeed enhanced via increasing CO_2 levels, then the related high-latitude ozone increases would 1) mitigate against polar ozone depletion issues and the concomitant health impacts from surface ultraviolet radiation and 2) not necessarily result in more stratosphere-troposphere exchange. Commenters further state that the latter occurs primarily in streamers linked to the coldest winter air masses near the polar jet—a warming of the coldest air masses, as is being observed over the Northern Hemisphere, and could just as easily result in a decrease in tropospheric ozone levels. Commenters request a correction to delete this issue from the TSD given that IPCC's Fourth Assessment Report is unsettled in this area.

Response (5-98):
In light of this comment, EPA has reviewed this discussion in Section 8(a) of the TSD, which references statements on this topic from IPCC (Denman et al., 2007). We conclude that a correction to the TSD is not necessary. We note that the TSD indicates that an influx of ozone from stratosphere to troposphere could occur with shifts in large-scale atmospheric circulation shifts in response to climate warming, which is based on multiple modeling studies in the scientific assessment literature. Since the commenters do not provide supporting literature indicating that high-latitude ozone increases will mitigate against polar ozone depletion issues, we conclude that this is currently not supported by available science. Additionally, we note that the Denman et al. (2007) statement indicates that ozone concentrations could increase via more stratosphere-troposphere exchange and further evidence of shifts in atmospheric circulation due to climate warming is provided by CCSP (2008b), which reports that stagnant air masses related to climate change are likely to degrade air quality in some densely populated areas. We acknowledge that stratosphere-troposphere exchange is associated with colder winter air masses; however, the current scientific literature does not support that a warming of polar jets will lead to decreases in tropospheric ozone levels. For these reasons we have determined that our discussion of this issue in the TSD is accurate and sound.

Comment (5-99):
A few commenters (2972.1, 3136.1, and 3722) suggest that despite the simulation provided by Mickley et al. (2004), observations show something different. Commenters cite the study by Strong and Davis (2007), which calculated a 0.6% increase (per decade) in the frequency of jet cores and increasing jet stream speed over much of central and eastern Canada over the 1958–2004 period in conjunction with a regional contraction of the 300-hectopascal circumpolar vortex. Commenters state that given the lack of upward trend in pollution episodes over the last 30 years, these observations call into question the likelihood of future air quality degradation from climate change. The commenters state that the TSD's inclusion of this issue is not based on the best available science and is inappropriate; therefore, they request a correction to the TSD.

Response (5-99):
This comment concerns the discussion in the TSD regarding statements of increases in the severity and persistence of regional pollution episodes in the eastern United States due to the reduced frequency of ventilation by storms. These results come from IPCC (Denman et al., 2007) based on the results of Mickley et al. (2004).

We disagree with the commenters' assertion that observations show something different. The commenters' view of Strong and Davis (2007) is incorrect on this issue. Strong and Davis (2007) report changes in the frequency of jet cores and increasing jet stream speed for wintertime (December–February). The Mickley et al. (2004) results are for summertime, which is the period of interest for high

ozone pollution episodes. Mickley et al. (2004) do cite multiple peer-reviewed journal articles with observed trends that are consistent with the model results of decreasing summertime cyclone frequency. More recent work by Mickley and co-investigators (Leibensperger et al., 2008) extends the model results through detailed analysis of long-term observations and concludes:

> …[T]he decrease in mid-latitude cyclones over the period 1980–2006 has offset half of the air quality gains in the Northeast United States that should have been achieved from reduction of anthropogenic emissions over that period. This suggests that climate change has already had a major effect on the accountability of emission control strategies over the past 2–3 decades, preventing achievement of the ozone air quality standard. It demonstrates the potential of climate change to dramatically affect air quality on decadal scales relevant to air quality policy.

Importantly, we note that the Leibensperger et al. (2008) work uses the same dataset as Strong and Davis (2007). For these reasons, we conclude that we have accurately reflected the science in the TSD regarding observations and the future likelihood of air quality from climate change. We will continue to rely on the synthesis reports, including this IPCC (Mickley et al. 2004) conclusion, and no changes to the TSD are warranted.

Comment (5-100):

Several commenters (3136.1, 3347.3, 3394.1, 3411.1, 3596.2, and 3722) argue that the statement in the TSD Executive Summary that the directional effect of climate change on ambient PM levels remains uncertain is misleading and is inconsistent with the underlying research reported in the rest of the report. These comments state that another factor to consider with respect to urban ozone levels is the possibility that precipitation will increase over urban areas because of the presence of the urban heat island coupled with a moister and more unstable atmosphere, resulting in more wet deposition (see, for example, Shepherd, 2005). The commenters claim that if EPA were to proceed to a final endangerment finding, it would have to first address the potential health benefits from possible climate change–related PM reductions.

Response (5-100):

The TSD discusses the issue of the directional effect of climate change on ambient PM levels as highlighted by commenters in Section 8(b): "The overall directional impact of climate change on PM levels in the United States remains uncertain (CENR, 2008), as too few data yet exist for PM to draw firm conclusions about the direction or magnitude of climate impacts (CCSP 2008b)." Further, recent analyses (e.g., EPA's IA) indicate a range of increases and decreases in PM concentrations related to changes in climate. The literature that we cite in the TSD has assessed several effects climate change could have on PM, including the one emphasized by the commenter, and found that the directional effect remains uncertain. After extensive review of the TSD and associated underlying scientific literature, we disagree with the commenters' assertion that the TSD is misleading or inconsistent with respect to the influence of climate change on PM and find the discussion in the TSD to be accurate and in agreement with the scientific literature.

As stated in the TSD, precipitation is a more important primary meteorological driver of PM than of ozone, due to its role in removing PM from the atmosphere (wet deposition). Precipitation, however, is particularly difficult to model and shows greater disagreement across simulations than other variables. Thus, it is reasonable to conclude that the impacts of climate change on PM will both be affected by wet deposition or removal from precipitation. However, model parameterizations of wet deposition are highly uncertain and not fully realistic in their coupling to the hydrological cycle (NRC, 2005). For models to simulate accurately the seasonally varying pattern of precipitation, they must correctly simulate a number

of processes (e.g., evaporation, condensation, transport) that are difficult to evaluate at a global scale (Randall et al., 2007). We determine these complexities and differences in directional effect for PM preclude the overall assessment of health benefits based on this modeling assessment which is consistent with the most recent scientific literature.

Comment (5-101):
A few commenters (3347.3, 3394.1, and 3603.1) cite EPA's IA report *Assessment of the Impacts of Global Change on Regional U.S. Air Quality: A Preliminary Synthesis of Climate Change Impacts on Ground-Level Ozone* (U.S. EPA, 2009d) as evidence that significant regions of the United States are likely to see ozone decreases associated with climate change and the science in this area is uncertain with respect to both ozone and PM. The commenters state that EPA pays little attention to the basic uncertainties underlying its modeling.

Response (5-101):
We reviewed the IA in light of this comment, and do not agree that EPA paid little attention to the basic uncertainties underlying its modeling. We note that uncertainties are fully discussed in the IA. For example, directly after the Executive Summary, modeling uncertainties are discussed in the section "Summary of Policy Relevant Findings." For instance, there are large differences across modeling groups, and/or across different model configurations used by the same group, in the specific spatial patterns of future simulated changes in meteorology that lead to differences in simulated future concentrations of ozone. Further, Sections 1.6 and 3 of the IA specifically discuss the uncertainty in the assessment effort. Here, the IA discusses the differences in modeling systems among the various groups. Section 3 of the IA provides a detailed discussion of the major uncertainties associated with the coupled climate and air quality modeling systems upon which rests the synthesis of the science of the report.

The IA report also states that individual simulations show "some regions of little change, or even decreases, in addition to the O_3 increases." We note, however, the report goes on to state, "For nearly every region of the country, at least one (usually multiple) of the modeling groups found that climate change caused increases in summertime O_3 concentrations." Further, the report clearly indicates these modeling studies "show increases in summertime O_3 concentrations over substantial regions of the country as a result of simulated 2050 climate change." The TSD clearly summarizes the modeling studies discussed in the IA that show simulated climate change causes increases in summer O_3 concentrations over substantial regions of the country, thought this was not uniform, and some areas showed little change or decreases. Therefore, we disagree with the commenters that we pay little attention to the uncertainties associated with the modeling analyses of the IA. See response to comment (5-103), which contains additional information regarding the various modeling groups.

Comment (5-102):
According to some commenters (3394.1, 3411.1, 3449.1, and 3747.1), the endangerment proposal admits that current models and studies do not reliably predict future ozone impacts. The TSD cites IPCC's 2007 conclusion that there are major discrepancies with observed long-term trends in ozone concentrations over the 20th century, and resolving these discrepancies is needed to establish confidence in the models. The commenters contend that conclusions drawn from models where discrepancies need to be resolved cannot support a health-based endangerment finding. EPA makes clear in its ozone IA, they state, that scientific analysis of these issues and the development of models that might project climate-related ozone changes are only now in the most preliminary of phases. The commenters suggest that the TSD notes various uncertainties without addressing their implications for the document's assertions, and they ask that EPA explain these implications and incorporate them into the Agency's conclusions.

Specifically, the commenters note the following uncertainties related to ozone impacts:

> "Relative to the other greenhouse gases, there is less confidence in reproducing the changes in ozone associated with large changes in emissions or climate, and in the simulation of observed long-term trends in ozone concentrations over the 20th century (Forster et al., 2007)."
> "More frequent occurrences of stagnant air events in urban or industrial areas could enhance the intensity of air pollution events, although the importance of these effects is not yet well quantified (Denman et al., 2007)."

The commenters suggest that these weakly stated acknowledgements that climate change will result in some ozone decreases and that specific posited effects are uncertain are still insufficiently reflective of the actual scientific record, which undermines EPA's reliance here on any supposed increase in ozone. They further state that EPA's own report, *Assessment of the Impacts of Global Change on Regional U.S. Air Quality: A Preliminary Synthesis of Climate Change Impacts on Ground-Level Ozone* (U.S. EPA, 2009d), indicates that there are significant regions of the United States that are indeed likely to see ozone decreases associated with climate change; that reductions in ozone precursor emissions likely will overwhelm any increases that may result from climate change; and that, ultimately, the science in this area is riddled with uncertainties with respect to ozone (and PM). The commenters indicate that the TSD and the proposed endangerment finding omit any reference to these facts, even though they were publicly reported by the Agency itself in the very month in which it issued the proposed endangerment finding. The commenters contend that EPA's approach here of ignoring or distorting scientific assessments, including even its own contemporaneous report, is arbitrary and indefensible.

Response (5-102):
We have examined the TSD in light of this and other comments, and have determined that the TSD accurately conveys current scientific understanding and the associated uncertainties.

The commenters contend that uncertainties in the scientific literature are "weakly stated" in the TSD. However, they do not provide adequate support or specific examples to support this assertion. In fact, the examples the commenters cite are actually taken from the TSD itself, which confirms that EPA provided a comprehensive discussion. Thus, EPA strongly disagrees with the contention that we omitted references to uncertainties or the need for additional research. To the contrary, the IA clearly identifies uncertainties and areas where further work is needed, and these findings are summarized in the TSD. We find that the commenters' assertions to be unsupported with specific scientific literature. Uncertainties in our modeling are transparently described and quantified; the models are widely accepted and scientifically sound to make conclusions on the things that we mention in the TSD and IA. Therefore, based on the discussions in the TSD and the IA, we conclude that our summary of this issue in the TSD is reasonable and supported by scientific literature.

We further note that the fact that particular issues are uncertain or could be better understood through additional research should not be taken to imply that such issues are not sufficiently well characterized to inform the Administrator's endangerment analysis. As noted in the quote above from the Executive Summary of the TSD, our goal has been to provide a complete and accurate summary of the science, including uncertainties, to ensure that the Administrator has a comprehensive foundation for her endangerment analysis.

For these reasons, we disagree with the comment that results of modeling studies should be excluded if it is also recognized that such results could be improved through additional research. The ozone simulations employ state-of-the-art models and up-to-date databases, and the assessment literature has found that the models are sufficiently accurate to indicate general increases in summertime ozone concentrations over

substantial regions of the country as a result of simulated 2050 climate change. The IA, as noted in the following excerpts, projects ozone increases over "substantial" regions of the country while "some" regions show little change or even decreases, though the decreases tend to be less pronounced than the increases.

> The new modeling studies discussed in this report show increases in summertime ozone concentrations over substantial regions of the country as a result of simulated 2050 climate change.
> For nearly every region of the country, at least one (usually more than one) of the modeling groups found that climate change caused increases in summertime ozone concentrations.
> Though in agreement on the results above, the different modeling systems did not necessarily simulate the same regional patterns of climate-induced ozone changes. The individual simulations showed some regions of little change, or even decreases, in addition to the ozone increases.

As stated in the final TSD:

> [T]he results in the IA demonstrate that O_3 responds to climate change in a qualitatively consistent manner across the simulations from multiple research groups. The patterns of relative changes in regional climate vary across these same simulations. Figure 3-11 of the IA graphically illustrates the net change in daily average ozone values across the research results for summertime ozone. Ozone concentrations increase across most areas of the country with decreases limited to some parts of the Southwest. The net increases of ozone concentrations in the large population centers of the northeast and middle Atlantic United States are the results with the highest confidence. The net increases in the Southeast and the small net changes in the Northwest are the features with the lower confidence.

Thus, the overall effect of climate change itself is to increase ozone levels compared to what would occur without future climate change, over broad areas of the country, especially on the highest ozone days and in the largest metropolitan areas with the worst ozone problems. Ozone decreases are projected to be less pronounced and to be limited to some parts of one area of the country, with generally less overall population. See Fig. 3-11 of the IA, which provides mean ozone changes across all seven modeling experiments

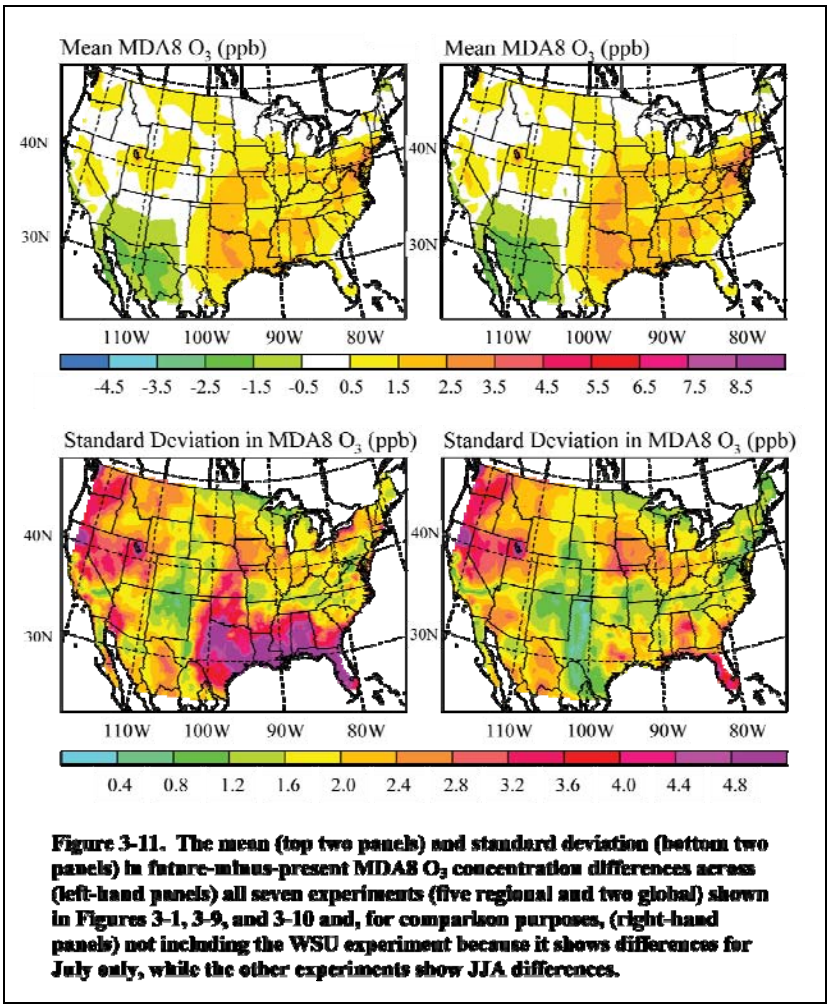

Figure 3-11. The mean (top two panels) and standard deviation (bottom two panels) in future-minus-present MDA8 O_3 concentration differences across (left-hand panels) all seven experiments (five regional and two global) shown in Figures 3-1, 3-9, and 3-10 and, for comparison purposes, (right-hand panels) not including the WSU experiment because it shows differences for July only, while the other experiments show JJA differences.

Source: Assessment of the Impacts of Global Change on Regional U.S. Air Quality: A Preliminary Synthesis of Climate Change Impacts on Ground-Level Ozone (U.S. EPA, 2009d)

We further disagree with the commenters' characterizations of the conclusions of the IA. It does not indicate that there are "significant" regions of the United States that are likely to see ozone decreases associated with climate change, nor does it indicate that reductions in ozone precursor emissions likely will "overwhelm" any increases that may result from climate change. The IA states: "Preliminary work suggests that the impacts of climate change on future U.S. regional O_3 concentrations remain significant when also considering possible future anthropogenic O_3 precursor emissions changes." The IA also states: "These preliminary tests found that the combined effects of climate and anthropogenic precursor emissions changes are highly sensitive to the assumptions about future emissions trajectories." As noted above, the IA projects ozone increases over "substantial" regions of the country while "some" regions show little change or even decreases, though the decreases tend to be less pronounced than the increases and to be limited to some parts of the Southwest.

We also note that the commenter failed to acknowledge an additional statement from IPCC: the TSD refers to the IPCC (Denman et al., 2007) conclusion that "the current generation of tropospheric ozone models is generally successful in describing the principal features of the present-day global ozone distribution."

It appears that the commenter is also concerned with how uncertainty in the scientific findings has been weighed and evaluated by the Administrator in her endangerment analysis. See the Findings, Section IV.B, "The Air Pollution Is Reasonably Anticipated to Endanger Both Public Health and Welfare," for our response to comments on how the Administrator weighed the scientific evidence underlying her endangerment determination.

Comment (5-103):
A number of commenters (3347.1, 3347.3, and 3347.4) state that the TSD selectively presents research findings, giving scant (if any) attention to results that contradict its main theme of large climate-related air pollution impacts. One commenter indicates that EPA selectively picked model results that support the endangerment proposal's policy objective. A commenter indicates that well over a hundred conference papers, final and "in review" journal papers, PowerPoint presentations, poster papers, STAR grant proposals, and project narratives are available that may be used to supplement, and in some cases challenge, the information in the TSD and IA. In light of the full array of relevant climate-air quality modeling data available by mid-April 2009, the commenters express concern that the TSD science foundation has been fashioned largely by information that agrees with the proposed finding of endangerment.

Specifically, a commenter (3347.3) states that the EPA's conclusions do not reflect the variability of the findings and EPA accepts as valid the conclusions of a modeling group when they demonstrate an increase in ozone in a certain region; however, when the same modeling group reports a decrease in ozone in another region, EPA then relies on another group's modeling results that show an increase in ozone in that particular region. The commenter notes, for example, that even though three of four modeling groups report that climate change will decrease ozone in the northwestern United States, EPA concludes climate change will increase ozone in this region based on the one positive study. The commenter suggests that when there is disagreement in the results across modeling groups in a region, EPA will use results from the group showing that ozone will increase due to climate change. The commenter concludes that this demonstrates a consistent lack of scientific objectivity, both in the substance and presentation of the findings.

Response (5-103):
EPA disagrees with the comments that analyses in the TSD do not reasonably and adequately reflect the current state of knowledge, including uncertainties, on this topic. See our response above (5-95) to critiques that EPA's analyses of the impacts of climate change on U.S. air quality were selective. See Section III.A., "The Science on Which the Decisions Are Based," of the Findings for our response to comments on the use of the assessment literature and our treatment of new and additional scientific literature provided through the public comment process. Similarly, see Volume 1 of the Response to Comments document for responses to comments on the use of major assessment reports as the primary scientific basis of the TSD.

EPA disagrees with the commenter's general assertion that we have selectively and non-scientifically presented the results of modeling studies. EPA's TSD relies not only on analysis provided by the IA but also relies on several peer-reviewed synthesis documents, including findings published by IPCC and USGCRP (formerly CCSP). The TSD summarizes findings from the IA, which assessed the results of numerous modeling simulations performed by a number of different modeling groups. A large proportion of Section 3 of the IA, the key scientific synthesis section upon which the conclusions of the IA are based, is devoted to detailed discussions of precisely the sorts of differences among the various modeling results that the commenter mentions. The full diversity of ozone results from the various modeling groups were explicitly presented in numerous maps and figures in the IA, clearly illustrating which simulations showed both climate-induced ozone increases and decreases, and in which parts of the country. Assertions

that the EPA drew conclusions in the IA such as "even though three of four modeling groups report that climate change will decrease ozone in the Northwest U.S. EPA concludes climate change will increase ozone in this region based on the one positive study" are false. For example, in Fig.3-11 of the IA (above), and as referenced in the TSD, the Northwest part of the country shows generally a neutral impact from climate change on ozone levels when the results across all seven different modeling experiments are combined. The analysis in the IA showed clearly that there are numerous, important model-based uncertainties in simulating the potential sensitivity of ozone to climate change, leading to differences among the results from the different models analyzed. These inter-model differences were embraced and fully examined in the IA to improve our understanding of the pathways by which meteorological changes in an altered climate lead to changes in ozone concentrations and the differences in the way these pathways are represented in the modeling systems used in the leading research groups. They were not highlighted in order to selectively discount the results from any particular model.

Nowhere in the IA or the TSD was any judgment made about any model being any "better" or "worse" than any other model, and nowhere did the IA give any primacy to any simulation result over any other for the purpose of drawing its conclusions. Completely contrary to the commenter's assertion, no model result was discarded, and all contributed equally to the stated conclusions of the IA. For regions for which there were larger differences among the models as to the simulated effect of climate change on ozone concentrations (i.e., portions of the western and southeastern parts of the United States), this was explicitly noted in the IA in the Executive Summary, the Summary for Policy Relevant Findings, and the main body of the report (in the conclusions of Section 3). To summarize, the conclusions in the IA that climate change has the potential to exacerbate ozone pollution in many parts of the country were arrived at from the improved scientific understanding provided by the complete range of available model results surveyed, and they flow logically and scientifically from this full diversity of findings. Regarding the general comment that there are well over a hundred conference papers, final and "in review" journal papers, PowerPoint presentations, poster papers, STAR grant proposals, and project narratives available for use, we would note that many of those peer-reviewed studies are already considered in the IA.

Comment (5-104):

A commenter (3347.4) states that the TSD relies on the "climate penalty" concept to describe air pollution increases under a changed future climate and encourages EPA to promote a more scientifically based, balanced means of describing the linkages between air quality and climate change impacts.

Response (5-104):
While the IA uses the term "climate penalty," the TSD does not. In any case, "climate penalty" has become an accepted term among researchers in this field since its introduction in the literature (introduced by Wu et al., 2008; see also Tagaris et al., 2009; Avise et al., 2009; and Bloomer et al., 2009). In general, "climate penalty" refers to the incremental impact of climate change on air quality under the assumption of constant criteria pollutant emissions, such that the effect of climate change on air quality can be isolated. It can be assessed from either simulations that hold air pollution emissions constant, from simulations with continuing air pollution emission controls, or from historical measures. The climate change penalty for ozone air pollution is the best available scientifically based means to describe linkages between air quality-climate change impacts across different emission scenarios. It was originally developed by researchers at Harvard and has been widely adopted by scientists working in the areas of climate change impacts on air quality. Not only is the climate change penalty for ozone air pollution a robust feature in modeling simulations, but a recent journal article (Bloomer et al., 2009) provides observational evidence for the climate change penalty consistent with modeling results. This helps to isolate the climate signal to understand and describe the linkages between air quality and climate change impacts. This serves as a scientifically accepted method for determining air pollution changes under future climate conditions, as is consistent with the larger body of scientific literature.

Comment (5-105):
A commenter (3347.1) states that inadequate consideration is given to projected future intercontinental transport of criteria air pollutants and the roles that sources outside the United States (e.g., China, India, and Mexico) will have on ozone and $PM_{2.5}$ impacts in coming decades. The commenter indicates that for the regional air quality models relied upon in the TSD, the impact of intercontinental transport of emissions is treated by boundary conditions and the TSD does not consider this subject. The commenter continues to note the IA states "...the strong influence of the GCM-simulated climate on the downscaled results is inescapable, regardless of the methodological details." The commenter notes that EPA affirms in the IA that interactions between the global and regional models are important for air quality modeling and emphasizes the need to quantify the biases and characteristics of the specific global model simulations being relied upon for boundary conditions to the air quality models. The commenter states that this need is not mentioned at all in the TSD.

Response (5-105):
The modeling systems from the IA indeed consider the effects of international emissions of criteria pollutants or their precursors and the transport of those criteria pollutants or their precursors to the United States (for example, via the chemical boundary conditions imposed at the borders of the regional air quality modeling domain). The results of the IA are reflected in the TSD along with other findings from major scientific assessments thus, we conclude this subject is fully considered by the TSD. For the climate-only sensitivity simulations, these international emissions were held constant at present-day levels (just like the U.S. emissions). Explicitly teasing apart the combined impacts of changes in future U.S. emissions, changes in future intercontinental transport, and changes in future climate was not the primary focus of the IA, though the intercontinental transport issue is acknowledged as important, called out as a research need, and also investigated somewhat by some of the participating modeling groups (and cited in their publications referenced in the IA).

Comment (5-106):
A commenter (3411.1) states that the Center for the Study of Carbon Dioxide and Global Change provided EPA with experimental evidence that the increased levels of CO_2 in the atmosphere may actually reduce the emission of isoprene per unit of biomass. Since isoprene is a highly reactive biogenic

hydrocarbon that is emitted in copious amounts by vegetation and is a major source of ozone in the atmosphere, the commenter requests that EPA permit testimony on how increased levels of CO_2 in the atmosphere would affect isoprene levels.

Response (5-106):
We reviewed the source of information provided by commenter and note it is derived from the organization's Web site. We note that this source of information does not specifically reference peer-reviewed scientific literature related to this issue. Thus, we conclude that the commenter's view of changes in isoprene emissions is not adequately supported by the scientific assessment literature. We will continue to rely on the synthesis reports, including the IPCC finding, and no changes to the TSD are warranted. See the Findings, Section III.A., "The Science on Which the Decisions Are Based," for our response to comments on the use of the assessment literature and our treatment of new and additional scientific literature provided through the public comment process.

As noted in the TSD, IPCC (Meehl et al., 2007) reports that biogenic emissions are projected to increase by between 27% and 59%, contributing to a 30 to 50% increase in ozone formation over northern continental regions (for the 2090–2100 timeframe, relative to 1990–2000). We addressed the issue of isoprene in the IA, stating, "Another important impact is that climate change leads to changes in the natural emissions of VOCs, e.g., isoprene from vegetation. All of the modeling groups found climate-induced increases in these biogenic VOC emissions over most of the United States, with especially pronounced increases in the Southeast."

See the Findings, Section I.C.1(e) for our response on the request to permit testimony under a formal rulemaking under the Administrative Procedures Act.

Comment (5-107):
Commenters (3411.1 and 3347.3) cite Tagaris et al. (2007) and state that this work reported that both ozone and fine PM were substantially reduced in the 2050 case, confirming that the nation's air quality will continue to improve in spite of projected climate change. The commenter also indicated that Tagaris et al. (2007) included a model simulation in which the projected climate change in 2050 was combined with the 2001 emissions and, while this is an unrealistic simulation of the future, it is a direct way to separate out the impact of climate change. The commenter indicates that the study found that summer ozone over the entire United States was essentially unchanged with climate change, although five individual regions had ozone increases or decreases ranging up to 3% and that summer fine PM was reduced by about 10% overall with reductions from 2% to 18% in the five regions.

Response (5-107):
We reviewed this comment and have determined that the Tagaris et al. (2007) study was addressed in the IA and appropriately reflected in the TSD. In particular, the findings of this study were taken into account in the IA, as part of its "Synthesis of Results Across Groups." (This journal article comes from the Georgia Tech/NESCAUM research group whose activities were funded through an EPA STAR grant and analyzed in detail in the IA.). The study confirms that substantial reductions in emissions (more than 50%) are effective for improving future air quality. We disagree with the commenters' assertion that the study found that the summer ozone over the entire United States was essentially unchanged with climate change. Importantly, the study indicates that GHG emissions will increase "the number of days with ozone concentrations above 85 ppb in most regions except the Midwest." A more recent journal article from the Georgia Tech/NESCAUM team estimates a net increase in ozone mortality of an additional 300 deaths per year due to climate change in 2050. This confirms that the results from the Georgia Tech/NESCAUM team indicate a net increase in ozone over the United States (Tagaris et al., 2009).

Thus, we disagree with the commenter that the results of Tagaris et al. (2007) as described the commenter were essentially unchanged.

For both the climate change only and the combined climate and emission changes cases, the magnitude of the "climate change penalty" for controlling ozone is found to be consistent with the work of Wu et al. (2008). The impact of climate change is to worsen air quality compared to both cases without climate change. The findings further suggest that compliance with air quality standards in areas at or near the NAAQS in the future would be sensitive to the amount of future climate change. For these reasons we find this work (Tagaris et al., 2007) to be consistent with climate-only analysis, thereby supporting the TSD's summary of the IA.

Comment (5-108):
A commenter (3347.4) states that "[t]here is no indication that OAQPS staff was involved in the science development of the proposed endangerment finding" and "[t]he TSD's perfunctory treatment of air quality-climate change modeling findings from the CIRAQ and ICAP programs suggests inadequate communication between the agency's modeling scientists and policy staff."

Response (5-108):
The commenter is in error. The EPA team of TSD authors and reviewers included staff from ORD and OAQPS. Further, we disagree that the TSD's treatment of air quality/climate change modeling findings from the CIRAQ and ICAP programs was "perfunctory." The TSD does not include the ICAP information contained in two PowerPoint presentations cited in comments (Jang, 2008, 2004) because, as explained in a previous response to comment, EPA is relying on peer-reviewed, synthesis reports such as IPCC (2007). The results from the NERL CIRAQ project, as published in Nolte et al. (2008), were a key part of the IA analysis and were fully considered in the TSD. We note in the IA report the critical role of the CIRAQ project and other offices within EPA had in providing modeling expertise for the development of the IA assessment. This assessment team, as identified in the IA report, provides the air quality modeling expertise to develop these simulations, to interpret the sensitivity of air quality to the future climate changes simulated, and to consider the regulatory implications of potential changes in air quality.

Comment (5-109):
A commenter (3347.4) asserts that the TSD relies almost exclusively on EPA's IA report to support assertions related to future ozone levels associated with climate change.

Response (5-109):
The EPA IA represents the current state of the science based on analysis of results from eight research groups that examined how climate change may affect U.S. air quality. However, we disagree with the commenter's assertion that the TSD relies almost exclusively on EPA's IA report for the section on air quality. The TSD relies on many studies relating climate change and ozone levels, and includes several references to findings of IPCC, USGCRP, and other sources in the assessment literature.

Comment (5-110):
One commenter (3347.3) states that EPA's conclusion on page ES-4 of the TSD fails to acknowledge that ozone levels may decrease in rural areas with low nitrogen oxide levels (U.S. EPA, 2009d). Such findings have a significant impact on the assessment of potential health impacts of changing air quality due to climate change. In regions of the country where ozone levels are expected to decline (e.g., rural and

certain coastal areas), the commenter asserts that EPA should account for the potential benefits of these lower ozone levels in these regions. The commenter further asks EPA to make its estimates of health effects from ozone methodologically consistent with the methods EPA uses in the recent ozone regulatory REA, or explain why the ozone regulatory impact analysis (RIA) is methodologically biased.

Response (5-110):

EPA disagrees with the commenter that we do not acknowledge regional changes in ozone levels such as in rural and urban locations. We note that the TSD summarizes the IA's assessment of the differing impacts of climate change in clean rural versus polluted rural and urban areas. In the IA and TSD we draw directly from the studies cited and do not alter the data or methodology conducted in those studies. However, because most of the population resides in urban areas, the overall impact of this aspect (clean rural versus urban areas) of climate change on ozone likely will be adverse; i.e., a large part of the population experiencing increased ozone levels. Overall, IPCC states that climate change is expected to decrease background tropospheric ozone due to higher water vapor and to increase regional and urban-scale ozone pollution due to higher temperatures and weaker air circulation (Denman et al., 2007; Confalonieri et al., 2007).

In addition, we note that climate change is projected to increase surface layer ozone concentrations in both urban and polluted rural environments due to decomposition of peroxyacetylnitrate (PAN) at higher temperatures (Sillman and Samson, 1995; Liao and Seinfeld, 2006). Further, warming temperatures enhances decomposition of PAN, releasing NO_x, an important ozone precursor (Stevenson et al., 2005). Model simulations (using the high-end A2 emissions scenario) with higher temperatures for the year 2100 showed that enhanced PAN thermal decomposition caused this species to decrease by up to 50% over source regions and ozone net production to increase (Hauglustaine et al., 2005). Thus, there is strong, credible scientific evidence that ozone in rural areas may end up higher in the future due to greater PAN decomposition.

We determined that the commenter meant RIA instead of REA and have responded accordingly. The RIA method for analyzing ozone attainment is appropriate for analyzing attainment with the NAAQS and EPA employs another method to analyze what the NAAQS should be and whether it should be revised. However, the present action related to endangerment is not a regulatory action; therefore, the RIA method does not apply. In addition, EPA is not trying to quantify the health benefits of a specific change in the NAAQS, and the resulting modeling of a certain ozone levels, which the RIA methodology then uses.

Modeling studies discussed in EPA's IA show that simulated climate change causes increases in summertime ozone concentrations over substantial regions of the country, though this was not uniform and some areas showed little change or decreases, though the decreases tend to be less pronounced than the increases. For those regions that showed climate-induced increases, the increase in maximum daily 8-hour average ozone concentration, a key metric for regulating U.S. air quality, was in the range of 2 to 8 ppb, averaged over the summer season. The increases were substantially greater than this during the peak pollution episodes that tend to occur over a number of days each summer. Thus, the overall effect of climate change itself is to increase ozone levels, compared to what would occur without future climate change, over broad areas of the country, especially on the highest ozone days and in the largest metropolitan areas with the worst ozone problems. Ozone decreases are projected to be less pronounced and to be limited to some parts of one area of the country, with generally less overall population.

Comment (5-111):

A commenter (3347.3) asserts that EPA has failed to cite data for locations other than the eastern United States where ozone is predicted to increase and adversely impact air quality and health (e.g., Bell et al., 2007; Field et al., 2007). On the other hand, the commenter states, EPA does not report findings where

climate change is expected to produce no change or a decrease in mortality due to no change or a decrease in the level of ozone; thus, the Agency only reports on areas for which a trend toward an increase in health effects is predicted. The commenter states that this is further evidence of a lack of substantive and presentational objectivity because EPA failed to perform a comprehensive nationwide analysis considering both the regional variability (increases and decreases) in ozone subsequent to climate change, and the regional variability in concentration function responses that are apparent in the health effects data (even though EPA possesses the data to do so). The commenter concludes that it is not possible at this time to make a definitive conclusion concerning whether, overall, U.S. health effects will increase or decrease due to the influence of climate change on ozone.

Response (5-111):

EPA strongly disagrees with the comment that the IA failed to cite data for locations other than the eastern United States with regard to ozone air quality and health effects. The IA extensively discusses the changes in ozone across the United States based on nationwide results from seven modeling groups and regional results from two additional modeling groups. Results across these modeling groups show greater increases than decreases, with the increases more predominant in high-population areas and for high-air-pollution episodes. Thus, we strongly disagree that we did not consider national-scale analyses in the TSD. The IA is a comprehensive nationwide analysis that considered the regional variability in ozone subsequent to climate change. The TSD has been developed to inform the Administrator's endangerment analysis, not to inform attainment demonstrations under the NAAQS. To support its intended purpose, the summary in the TSD properly focuses on the projected impact of global GHG emissions on large-scale patterns of air quality in the United States via induced climate changes as represented in GCMs.

The findings described in the IA as well as other peer-reviewed, synthesis documents, including findings published by IPCC and USGCRP (formerly CCSP), clearly indicate that ozone concentrations in urban and polluted rural areas are expected to increase during summer episodes due to climate changes including changes in temperature, wind patterns and chemical mechanisms involving PAN. The TSD also indicates that in some areas ozone concentrations are expected to decrease due to the higher absolute humidity which decreases ozone concentrations. Because the increases in ozone are projected to occur primarily in urban areas, we expect the overall impact to be adverse to public health. For example, the results in the IA demonstrate that ozone responds to climate change in a qualitatively consistent manner across the simulations from multiple research groups. The patterns of relative changes in regional climate vary across these same simulations. Figure 3-11 of the IA graphically illustrates the net change in daily average ozone values across the research results for summertime ozone. As mentioned in previous responses in the section, ozone concentrations increase across most areas of the country with decreases limited to some parts of Southwest. The net increases of ozone concentrations in the large population centers of the northeastern and middle Atlantic United States are the results with the highest confidence. The net increases in the Southeast and the small net changes in the Northwest are the features with the lower confidence. Recent results from the Georgia Tech group (Tagaris et al., 2009) estimate a net increase in ozone mortality of an additional 300 deaths per year due to climate change in 2050.

See the Findings, Section IV.B, "The Air Pollution Is Reasonably Anticipated to Endanger Both Public Health and Welfare," for our response to comments on how the Administrator weighed the scientific evidence underlying her endangerment determination.

Comment (5-112):

A commenter (3347.3) states that the EPA also relies heavily and selectively on the study by Jacob and Winner (2009). EPA cites this reference to support the conclusion that "there is now consistent evidence from models and observations that 21st-century climate change will worsen summertime surface ozone in polluted regions of North America compared to a future with no climate change." According to the

commenter, EPA fails to note that Jacob and Winner (2009) also state that while most of these models predict increases in the Northeast, the results in other regions show "large differences between models." Furthermore, this study did not account for existing regulatory control programs and reductions in anthropogenic emissions in making projections of future levels of ozone.

Response (5-112):
Jacob and Winner (2009) is a journal article that reviews and synthesizes results across all known peer-reviewed studies as of late 2008. As such, it provides a very recent representation of the current state of knowledge on the impacts of climate change on air quality. It assessed numerous individual studies to draw its conclusions that were reviewed not only through the journal's peer review process, but also by EPA, and we have determined the study by Jacob and Winner (2009) has been well-vetted and accurately reflects the consensus among knowledgeable peers in the field of climate science and air quality.

The statement in the TSD that "There is now consistent evidence from models and observations that 21st-century climate change will worsen summertime surface ozone in polluted regions of North America compared to a future with no climate change" accurately reflects the conclusions of Jacob and Winner (2009), and this statement does take into account the differences between models.

Jacob and Winner (2009) discuss several studies that account for existing regulatory control programs and reductions in criteria air pollutant emissions. We note that Jacob and Winner state: "Simulations that assume emission reductions far beyond the full implementation of current regulations indicate that climate change will partly offset the benefit of the emissions reductions (Tao et al., 2007; Tagaris et al., 2007; Nolte et al., 2008). Wu et al. (2008) refer to this 'climate penalty' as the need for stronger emission controls to achieve a given air quality standard." Thus, the EPA finds clear evidence from the scientific assessment literature that GHGs will impact air quality adversely in the future. For these reasons we conclude the study by Jacob and Winner (2009) is appropriate and reflects the current state of the scientific literature on this issue. See response (5-103) for our response to comments on the results and reasons for the relative differences between models.

Comment (5-113):
One commenter (3347.4) notes that notwithstanding the prodigious amount of modeling data generated by EPA and other research programs in the climate science field, certain basic data sets are simply unavailable to independent scientists or can only be obtained from participating researchers through time-consuming data requests.

Response (5-113):
We are not aware of any instance in which someone formally requested access to modeling data for the purpose of the proposed endangerment finding and was denied access. Additionally, most of the models used in these studies are publicly available for free download, such as CMAQ, CAMx, MM5, and the GISS GCM.

Comment (5-114):
A commenter (3347.4) notes that a few literature citations are missing in the reference section of the TSD. These include WHO (2002), Bowman and Johnston (2005), and Moore et al. (2006). The citation for Brasseur, et al. (2006) should include the page numbers 3932-3951.

Response (5-114):

We appreciate the commenter's point of clarification. The TSD was revised to reference the primary source of the finding (Confalonieri et al., 2007) and then note additional references therein, rather than adding the individual references to reflect the additional sources which contribute to the finding. We note this change is consistent with how other supporting conclusions from IPCC are referenced in the TSD.

Comment (5-115):
A commenter (3347.4) quotes the following from the TSD: "increased biogenic emissions alone add 1–3 parts per billion (ppb) to summertime average daily maximum 8-hour ozone concentrations in the Midwest and along the eastern seaboard (Hogrefe et al., 2004)." The commenter goes on to state that a closer inspection of the Hogrefe et al. (2004) paper reveals that over most of the Midwest and eastern United States, the biogenic impact ranges from -1 to 1 ppb. The ozone increases of 1–3 ppb mentioned in the TSD are largely confined to a narrow band of grid cells aligned along the Ohio River and along the East Coast margin from Philadelphia to Boston. The commenter concludes that the TSD has emphasized the high end of the modeled ozone enhancements (1–3 ppb) from biogenic increases, essentially neglecting broad regions across the country where ozone perturbations in the -1 to 1 ppb range were calculated.

Response (5-115):
We disagree with the commenter and maintain that the TSD accurately reflects the results reported by Hogrefe et al. (2004). The TSD summarizes this study in the context of evidence that the influence of climate induced changes of biogenic VOC emissions may be regionally substantial. Hogrefe et al. (2004) state: "Results from these simulations are shown in Figures 5a–5c and indicate that increased biogenic emissions alone add 1–3 ppb to summertime average daily maximum 8-hour O_3 concentrations in the Midwest and along the eastern seaboard." Other regions of the United States may not experience this increase, which is consistent with respect to expected overall variability in ozone levels associated with climate change. We note that Hogrefe et al. (2004) found the strongest increases in emissions in the Southeast, similar to the results from the NERL and Illinois 1 and 2 experiments modeling teams, but found that the largest ozone concentration changes that could be attributed to biogenic emissions changes occurred instead in parts of the Ohio Valley and coastal Mid-Atlantic. We conclude that the TSD accurately reflects these findings from Hoegefe et al. (2004) and we do not emphasize the high end of the ozone increases as the commenter implies.

Comment (5-116):
A commenter (3347.4) suggests that given differences among mechanism predictions and difficulty in proving which one is "correct," regulatory decisions based on model predictions should account for the uncertainty due to differences in chemical representation. The commenter further suggests that this is an area where the TSD should be expanded to include 1) the recent chemistry-related modeling studies within EPA's ORD as well as by outside researchers and 2) the range of predictions resulting from mechanism comparison studies in order to shed light on the expected range of uncertainty for ozone and $PM_{2.5}$.

Response (5-116):
The various chemical mechanisms used in several models are described in the IA, and the TSD summarizes findings from many references, including the IA. The TSD, for example, indicates that "the response of ozone to changes in biogenic emissions depends on how isoprene chemistry is represented in the models—models that recycle isoprene nitrates back to NO_x will tend to simulate significant O_3 concentration increases in regions with biogenic emissions increases, while models that do not recycle isoprene nitrates will tend to simulate small changes, or even O_3 decreases." We provide appropriate detail on the chemical mechanisms and reflect the associated uncertainties and references in the IA; we find it seems unnecessary to provide and repeat the same level of detailed information from the IA in the TSD, which is a summary of major conclusions from the underlying scientific assessments.

Comment (5-117):
A commenter (3347.4) notes that the TSD briefly mentions an area of increasing importance for reliable regional air quality modeling—reactive nitrogen chemistry feedbacks. Given the importance of the reactive nitrogen budget for future air quality simulations, the commenter requests that the TSD should be expanded to reflect recent research findings as well as a clear explanation of the values of dinitrogen pentoxide used in the various platforms employed in the Phase I, CIRAQ, and ICAP modeling (e.g., GEOS-Chem, MOZART, CMAQ, CAMx) and the assumptions regarding isoprene nitrate partitioning and yields.

Response (5-117):
In light of this comment and after further review of the TSD, we find these areas are adequately summarized in the TSD. Additional detailed discussions of both well understood and highly uncertain ozone photochemistry can be found in the U.S. EPA Air Quality Criteria Document for Ozone (U.S. EPA, 2006).

Comment (5-118):
A commenter (3347.4) states that the TSD paints a pessimistic view of the adequacy of state-of-science coupled chemistry-aerosol-climate models for providing guidance for decision-making and recommends the TSD incorporate this newer perspective of photochemical aerosol modeling capabilities. Although much work remains, the modeling tools are useful in their present stage of development to provide useful information to public decision-makers.

Response (5-118):
We agree that the modeling tools are useful in their present stage of development; however, no changes to the TSD are warranted. As described in the IA, improvements in our ability to model chemistry of air pollution are desirable in a number of areas to better understand the influence of climate change on air quality. Notable among these is the ability to introduce simulation of two-way interactions between climate and chemistry such as changes in the distribution of particulates resulting from changes in climate or emissions could have important impacts on the Earth's radiation budget, thereby further influencing climate.

Comment (5-119):
A commenter (5846) indicates that EPA has adopted the following false or misleading belief: that climate change will lead to degraded air quality specifically, that regional ozone pollution will increase. The commenter indicates that ozone is a natural chemical produced by natural processes in quantities much greater than any amounts produced by humans and ozone production is driven by natural, not

anthropogenic, processes. The commenter further indicates that any warming effect of increased ozone on health will be offset by a reduction in the severe cold events which are the true drivers of the increased rates of most respiratory complaints. The commenter argues that EPA presents the opposite of the most likely results of warming as fact and the true consequences are not presented.

Response (5-119):
EPA does not agree with the commenter that the potential for increases in regional ozone pollution is false or misleading. We rely on strong evidence from the scientific literature to support our statements regarding climate change and effects on air quality. As indicated in the TSD, tropospheric (including ground or surface-level) ozone is both naturally occurring and, as the primary constituent of urban smog, a secondary pollutant formed through photochemical reactions involving NO_x and VOCs in the presence of sunlight. IPCC (Denman et al., 2007) reports that climate change can affect ozone by modifying emissions precursors, atmospheric chemistry, and transport and removal. Further, there is now consistent evidence from models and observations that 21st century climate change will worsen summertime surface ozone in polluted regions of North America compared to a future with no climate change (Jacob and Winner, 2009). Thus, EPA considers there to be clear evidence that climate change will likely increase ozone pollution, with associated risks to human health from respiratory illness and premature death.

Comment (5-120):
A commenter (10116) indicates that this document is an important step for the federal government. However, on the issue of air quality and human health, it downplays an important body of work, funded by EPA, that represents the first comprehensive assessment of climate change impacts on ozone-related and heat-related health impacts in the United States: the New York Climate Health Project. The commenter recommends that EPA discuss Kinney et al. (2006, 2008) and Knowlton et al. (2004, 2008) and states that the EPA has overlooked the only health-based paper on climate change and health with a U.S. focus (Kinney et al., 2008).

Response (5-120):
In light of this comment, EPA has reviewed the reference literature in the TSD. We note that some of the studies referenced by the commenter (Knowlton et al., 2004) are cited by IPCC (Field et al., 2007) and summarized in Section 6 of the TSD section (6) which is referred to by the commenter. Similarly, the USGCRP assessment report (Karl et al., 2009) relies on the same findings and associated studies, which are also consistent with information summarized in the TSD. Further, we note several of the studies reviewed in Kinney et al. (2008) are cited in the assessment literature, TSD and this document. While EPA considers the studies referenced by the commenter to be consistent with the assessment literature and supportive of conclusions summarized in the TSD, we conclude that revising the TSD to incorporate these studies is unnecessary.

Comment (5-121):
Commenter (2750) notes that the endangerment proposal indicates climate change can exacerbate tropospheric ozone levels in some parts of the United States. However, with more frequent and intense storms which clean the atmosphere also projected, how will more ozone be produced?

Response (5-121):
EPA disagrees with the commenter's general assertion that the United States will have more frequent storms. Future changes in precipitation due to human-induced climate change are more difficult to project than temperature. Further, we note the overall frequency of summertime storms will likely decrease in many areas (e.g., Mickley et al., 2004).

Changes in the frequency and intensity of storms may decrease ozone, but this ignores other possible changes such as increasing biogenic emission of ozone precursors or increases in the reaction rates of chemical process that form ozone due to higher temperatures. Formation of tropospheric ozone is a complex system of chemical and physical processes. This is why the IA relies on comprehensive modeling systems to analyze this complex system. Based on this assessment, more ozone will be produced over much of the United States under future climate conditions.

Comment (5-122):
Commenter (3387.2) submits a study by Corbett et al. (2007), *Mortality From Ship Emissions: A Global Perspective*, on health effects and emissions from marine transport as part of an appendix to comments on EPA's Proposed Findings. The commenter indicates that epidemiological studies consistently link ambient concentrations of PM to negative health impacts, including asthma, heart attacks, hospital admissions, and premature mortality. Corbett et al. estimate global and regional mortalities by applying ambient PM increases due to ships to cardiopulmonary and lung cancer concentration risk functions and population models. The results indicate that shipping-related PM emissions are responsible for approximately 60,000 cardiopulmonary and lung cancer deaths annually, with most deaths occurring near coastlines in Europe, East Asia, and South Asia. The study notes that under current regulation and with the expected growth in shipping activity annual mortalities could increase by 40% by 2012.

Response (5-122):
EPA reviewed this reference and acknowledges the important health effects associated with emissions of PM and PM precursors. These findings are concerned with the causes of human-induced climate change. Please see Volume 9, Section 9.1.6, "Additional Substances" (black carbon, other substances, under the proposed definition of "air pollution") for our response to comments concerning the contribution to climate change of human emissions of black carbon and other short-lived pollutant species beyond the six GHGs associated with this action.

Comment (5-123):
One commenter (3893) voices support for the Findings, pointing out that global warming will undermine efforts to improve air quality, as higher temperatures will accelerate ozone formation during summer months.

Response (5-123):
We note that this is consistent with information summarized in the TSD. For example, increases in regional ozone pollution relative to ozone levels without climate change are expected due to higher temperatures and weaker circulation in the United States and other world cities relative to air quality levels without climate change. As further stated in the TSD, increases in regional ozone pollution relative to ozone levels without climate change are expected due to higher temperatures and weaker circulation in the United States and other world cities relative to air quality levels without climate change.

Comment (5-124):
Commenters (0660, 8320) indicate that reducing atmospheric CO_2 concentrations would result in significant co-benefits by reducing the disease burden caused by the criteria pollutants and other toxics. Knowledge regarding the extensive health consequences of air pollution has expanded exponentially in the last decade, with thousands of studies published in the mainstream medical literature.

Response (5-124):
See the Findings, Section III.C, "Adaptation and Mitigation," for our response to comments on the treatment of adaptation and mitigation in the Findings.

Comment (5-125):
Several commenters (2633, 6679, 8874, 11233) express concern over the impacts of climate change on the degradation of air quality, specifically, that climate change will lead to higher levels of ozone and other air pollution. One commenter (2633) notes that toxic air pollutants and the chemicals that form acid rain and ground-level ozone can damage trees, crops, wildlife, aquatic life, lakes and other bodies of water, including drinking water supplies. Some commenters (0293, 1507, 11249) express concern that climate change led to increased ozone formation and mercury contamination. One commenter (11249) notes that global warming will undermine efforts to improve air quality, and states that rising ozone concentrations would cause serious respiratory and cardiovascular health problems. The commenter (11249) notes that more than 100 million Americans live in areas where ozone levels exceed the 8-hour NAAQS.

Response (5-125):
EPA notes the concerns expressed by the commenters regarding the impacts of climate change on air quality, human health, and the environment. We also note the commenters' concerns regarding current levels of air pollutants and ecosystem health. The TSD and supporting information in the Findings cover the effects and impacts from climate change across of variety of environmental media (i.e., water, air, soil) and pathways.

Comment (5-126):
One commenter 72818) stated that just last month, EPA's USGCRP released its assessment of the impacts of global change on regional U.S. air quality, a synthesis of climate change impacts on ground-level ozone. The commenter indicates that the report finds that climate-induced ozone, averaged over the summer season, was in the range of approximately 2 to 8 ppb in the maximum daily 8-hour ozone concentration and this is a key metric for regulating U.S. air quality. According to the commenter, the study also found that the largest increases in ozone would be during the peak pollution events. The commenter believes this study tells us that on average these models agreed that these events will become more severe. The commenter further states that some of the modeling groups also found that climate-induced increases in ozone in some regions would extend into the spring and fall, which means that the ozone season could become longer.

Response (5-126):
We have reviewed these comments and note they are generally consistent with EPA's analyses in the Interim Assessment report and as reflected in the final TSD. The TSD now incorporates results from the latest USGCRP report.

Comment (5-127):
A commenter (2895) indicates that the effects of climate change will also be very significant for air quality, and in particular for ambient ground-level ozone levels. The commenter notes that this is not just a problem in notorious places like Los Angeles or Houston—it is also in Seattle. The commenter notes that as temperatures rise, as we can expect with climate change, it's going to put the area much more at risk for exceedances of the ozone standards. This is something that will affect human health; asthmatics and others at risk will be particularly severely impacted. Increased ozone will obscure the iconic vistas of

Mount Rainier. The commenter indicates that these are real effects that clearly show EPA needs to take action. And, states the commenter, EPA has a mandate to look at current as well as future risks in evaluating what action to take.

Response (5-127):

We note the concerns of the commenter regarding the risk of increasing ozone concentrations in Seattle, Washington. We concur with the commenter that breathing ozone at sufficient concentrations can reduce lung function, thereby aggravating asthma or other respiratory conditions. EPA's IA report, which is reflected in the TSD, provides a synthesis of the potential effects of climate change on air quality including ground-level ozone based on current and future conditions.

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