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Testimony for the Hearing,
"Healthy Planet, Healthy People: Global Warming and Public Health"
Select Committee on Energy Independence and Global Warming
United States House of Representatives
The Honorable Ed Markey, Chair
The Honorable F. James Sensenbrenner Jr., Ranking Member
April 9, 2008, 10:00 a.m.

By Mark Z. Jacobson

I would like to thank the Honorable Chairman and Ranking Member and the committee for inviting me to testify today. I will discuss scientific findings on the effects of carbon dioxide, emitted during fossil-fuel combustion in California, the U.S., and the world, on air pollution and health in California relative to the U.S.. I will then discuss how these scientific findings differ from the two main assumptions made by Environmental Protection Agency (EPA) Administrator Stephen L. Johnson that formed the basis of his decision to deny California's request for a waiver of Clean Air Act Preemption on March 6, 2008 (Johnson, 2008). These assumptions were (a) there is no difference in the impact of globally-emitted carbon dioxide on California versus U.S. health and (b) locally-emitted carbon dioxide does not affect California's air pollution any more than does carbon dioxide emitted anywhere else in the world.

Summary

On March 6, 2008, EPA Administrator Stephen L. Johnson published a summary of his decision to deny the California Air Resources Board request for "a waiver of the Clean Air Act's Prohibition on adopting and enforcing its greenhouse gas emission standards as they affect 2009 and later model year new motor vehicles" (Johnson, 2008). The decision was made following consideration of two issues:

> "The appropriate criteria to apply therefore is whether the emissions of California motor vehicles, as well as California's local climate and topography, are the fundamental causal factors for the air pollution problem of elevated concentrations of greenhouse gases, and in the alternative whether the effect in California of this global air pollution problem amounts to compelling and extraordinary conditions (Johnson, 2008, p. 12162)."

With regard to the first issue, Mr. Johnson decided that

> "GHG (greenhouse gas) emissions from California cars are not a causal factor for local ozone levels any more than GHG emissions from other sources of GHG emissions in the world (Johnson, 2008, p. 12163)."

In other words, Mr. Johnson believes that because GHGs emitted in California eventually mix globally, California's GHG emissions do not affect California ozone any more than another state

or country's GHG emissions affect California's ozone. With regard to the second issue, Mr. Johnson ruled,

> "While I find that the conditions related to global climate change in California are substantial, they are not sufficiently different from conditions in the nation as a whole to justify separate state standards. As the discussion above indicates, global climate change has affected and is expected to affect, the nation, indeed the whole world, in ways very similar to the conditions noted in California. While proponents of the waiver claim that no other state experiences the impacts in combination as does California, the more appropriate comparison in this case is California compared to the nation as a whole, focusing on averages and extremes, and not a comparison of California to the other states individually. These identified impacts are found to affect other parts of the United States and therefore these effects are not sufficiently different compared to the nation as a whole. (Johnson, 2008, p. 12168).

The two questions raised by Mr. Johnson are questions of scientific fact. Because no publicly-available scientific paper(s) on these specific issues (namely the effects of global carbon dioxide on California versus U.S. air pollution health and the effects of California versus global carbon dioxide emissions on California air pollution health), were available prior to 2008 and no such study was cited in Johnson (2008), it appears reasonable to conclude that Mr. Johnson made his decision based on his own assumption that what he stated was scientific fact. The appearance that the decision was made on his assumption rather than scientific information is relevant since Johnson (2008, p. 12159) states, "As the court in MEMA I stated, 'here, too, if the Administrator ignores evidence demonstrating that the waiver should not be granted, or if he seeks to overcome that evidence with unsupported assumptions of his own, he runs the risk of having his waiver decision set aside as 'arbitrary and capricious.'"

The purpose of this document is to address the questions Mr. Johnson raised from a scientific approach. In particular, I report results from a recent peer-reviewed scientific study submitted for publication on June 22, 2007 and published on February 12, 2008 (Jacobson, 2008) and funded in part by the EPA, additional analysis of data from that study, and results from a follow up study that have not yet been published. Research published in this paper commenced about two years ago, before the waiver question became an issue and before EPA funding commenced on the project. It was also the culmination of research on the effect of climate change on air pollution that I started eight years ago and of research on the causes and effects of air pollution that I started 18 years ago.

Results from the studies and analyses are as follows

(a) Global warming due specifically to carbon dioxide currently increases the air-pollution-related death rate of people in California more than it increases the death rate of people in the United States as a whole, relative to their respective populations. Specifically, for every 1 degree Celsius (1.8 degrees Fahrenheit) temperature rise due to carbon dioxide, the U.S. death rate due to ozone and particle pollution increases above the baseline air pollution death rate of about 50,000-100,000 per year by approximately 1000 (350-1800) per year. Of these additional deaths, more than 30% occur in California, Since California has only 12 percent of the U.S. population, California suffers disproportionately (2.5 times) more deaths per person than the U.S. as a whole due to carbon-dioxide-induced global warming. The reason is that higher temperatures and water vapor due to carbon dioxide increase pollution the most where it is already high (Jacobson, 2008), and California has six of the ten most-polluted cities in the United States. The deaths are currently occurring and will occur more as temperatures increase in the future.

(b) Any emissions of carbon dioxide, whether in California or elsewhere, increase air pollution health problems in California at a rate 2.5 times higher than in the United States as a whole, even if the carbon dioxide becomes well-mixed in the atmosphere immediately after emissions, which it does not. Conversely, controlling carbon dioxide from California will reduce the air-pollution-

related death and illness rate in California at a rate 2.5 times faster than it will reduce the death rate of the U.S. as a whole.

(c) Emissions of carbon dioxide do not mix immediately to the global atmosphere. Instead, carbon dioxide mixing ratios in polluted cites are higher than are those in surrounding areas. Although carbon dioxide in cities disperses to the global atmosphere, their continuous emissions from vehicles and power plants keep their levels high over cities. It is shown here that such elevated levels of carbon dioxide increase air pollution, particularly ozone. As such, locally-emitted carbon dioxide is a causal factor in increasing local air pollution.

The three conclusions here – that (a) carbon-dioxide-induced global warming increases air pollution health problems more in California per capita than it does in the U.S. as a whole, (b) controlling California carbon dioxide emissions will decrease the California death rate at more than 2.5 more per capita than it will decrease the death rate of the U.S. as a whole, and (c) local carbon dioxide emissions from vehicles in California causally increase local air pollution and health problems in California contradict both assumptions made by Mr. Johnson in his stated decision, namely (a) there is no difference in the impact of globally-emitted carbon dioxide on California versus U.S. health and (b) locally-emitted carbon dioxide does not affect California's air pollution any more than does carbon dioxide emitted anywhere else in the world.

Discussion

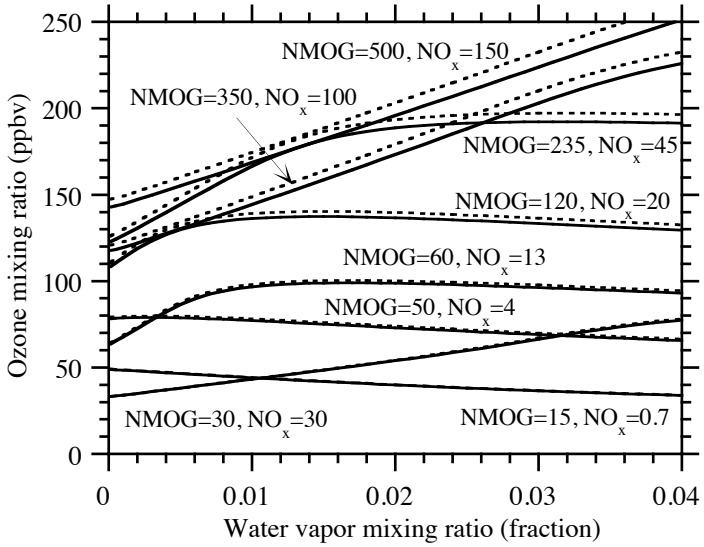
The effects of carbon dioxide on air pollution and the resulting effects on health can be determined only from large-scale computer model simulations, where the model treats the physics, chemistry, and meteorology of the atmosphere and has been evaluated thoroughly. Data measured in the atmosphere (e.g., from surface measurements, radiosonde, aircraft, satellite) can be used to show correlation only, not cause and effect. As such, it is not possible to use data alone to answer the question of the effects of carbon dioxide on air pollution. A computer model can show cause and effect when one input parameter at a time is changed. In the present case, the input parameter is carbon dioxide, and the goal is to determine the effect of carbon dioxide emissions on air pollution-related health problems in California and the United States.

Prior to 2008, many computer modeling studies had examined the sensitivity of near-surface ozone to temperature (Sillman and Samson, 1995; Zhang et al., 1998), the regional or global effects of climate change from all greenhouse gases on near-surface ozone (Thompson et al., 1989; Evans et al., 1998; Dvortsov et al., 2001; Mickley et al., 2004; Stevenson et al., 2005; Brasseur et al., 2006 Murazaki and Hess, 2006; Steiner et al., 2006; Racherla and Adams, 2006) or near-surface aerosol particles (Aw and Kleeman, 2003; Liao et al., 2006; Unger et al., 2006), and the effects of future global warming on regional ozone-related health problems (Knowlton et al., 2004; Bell et al., 2007). These studies generally found that higher temperatures increased ozone. However, no study had isolated the effect of carbon dioxide alone, emitted to date, on ozone, particles, or carcinogens, applied population and health data to the pollution changes over the U.S. as a whole, or examined the problem with a global-through-regional climate/air pollution model that treated feedback of gases and particles to clouds and meteorology. Jacobson (2008) performed a study accounting for these factors. The study used the computer model GATOR-GCMOM, which is a model developed over the last 18 years. It is described by Zhang (2008) as the first and still only unified, consistent global-to-urban scale air-quality-climate model worldwide and the "first fully-coupled online model to account for all major feedbacks among major atmospheric processes based on first principles (p. 1844)." As such, it was the most appropriate model for the type of study described here. The model had been evaluated against data in several published papers (e.g., Jacobson, 2001, 2004, 2007).

The model was first used to examine the effects of temperature alone and, separately, water vapor alone on ozone due to chemical reactions in the atmosphere. For this calculation, an exact numerical solver of chemical equations was used. No other process aside from photochemistry was solved. Figure 1 shows the resulting ozone predictions for a variety of initial levels of oxides of nitrogen (NO_x) and nonmethane organic gases (NMOGs). **A comparison of the solid lines (base temperature) with the dashed lines (higher temperature) in the figure**

shows that a 1 degree Kelvin or Celsius (= 1.8 degrees Fahrenheit) increase in temperature increases ozone when ozone is already high but has little or no effect on ozone when ozone is low. The figure also shows that water vapor (horizontal axis) independently increases ozone when ozone is high but generally has little effect or slightly decreases ozone when ozone is low.

Figure 1. Mixing ratio of ozone and several other gases as a function of water vapor mixing ratio after 12 hours of a box-model chemistry-only simulation initialized at 0430 under several NO_x and nonmethane organic gas (NMOG) mixing ratio combinations (ppbv) at 298.15 K (solid lines) and 299.15 K (dashed lines). The simulations assumed sinusoidally varying photolysis between 0600 and 1800.



The next step was to apply the numerical solution to chemical equations with solutions to equations for meteorological, aerosol microphysical, cloud, radiative, ocean, and surface processes within GATOR-GCMOM to examine the effect of carbon dioxide on ozone, particulate matter, and carcinogens. For this calculation, the model was set up in 'nested' mode whereby a high-resolution regional grid over the United States was fit within a coarser-resolved global grid. Both grids were three-dimensional and consisted of vertically-stacked layers of horizontally-adjacent boxes. Predicted meteorological, gas and aerosol variables from the global grid fed into the regional grid at the latter's boundaries. As such, it was possible to simulate the current global climate and the global climate with preindustrial levels of carbon dioxide emissions in both grids simultaneously and have the global-scale climate and air pollution variables from the global grid feed into the regional grid. Emissions for the simulations were spatially distributed. Thus, separate emissions occurred in each surface grid box in both grids.

Figures 2 shows results over the U.S. after taking the difference between the two simulations (e.g., one simulating present-day climate/air pollution and another simulating climate/air pollution at preindustrial carbon dioxide emission levels). It shows that human-emitted carbon dioxide caused an increase in near-surface temperatures and water vapor (Figures 2a,b). Increases in both thereby increased near-surface ozone (Figure 2c), as expected from Figure 1.

More specifically, Figure 2c indicates that carbon dioxide increased ozone by 0.12 ppbv over the U.S., with increases of 1-5 ppbv in the southeast and up to 2 ppbv along the northeast coast. In Los Angeles, the average temperature increase of 0.75 K (Figure 2a) and water vapor increase of 1.3 ppthv increased ozone by up to 5 ppbv.

Figure 2. Four-month (mid-July to mid-November) grid-averaged near-surface differences in (a) temperature, (b) water vapor, and (c) ozone between the present-day and preindustrial-carbon dioxide simulations. The grid-averaged (over land and water) change for each surface plot is given in parentheses.

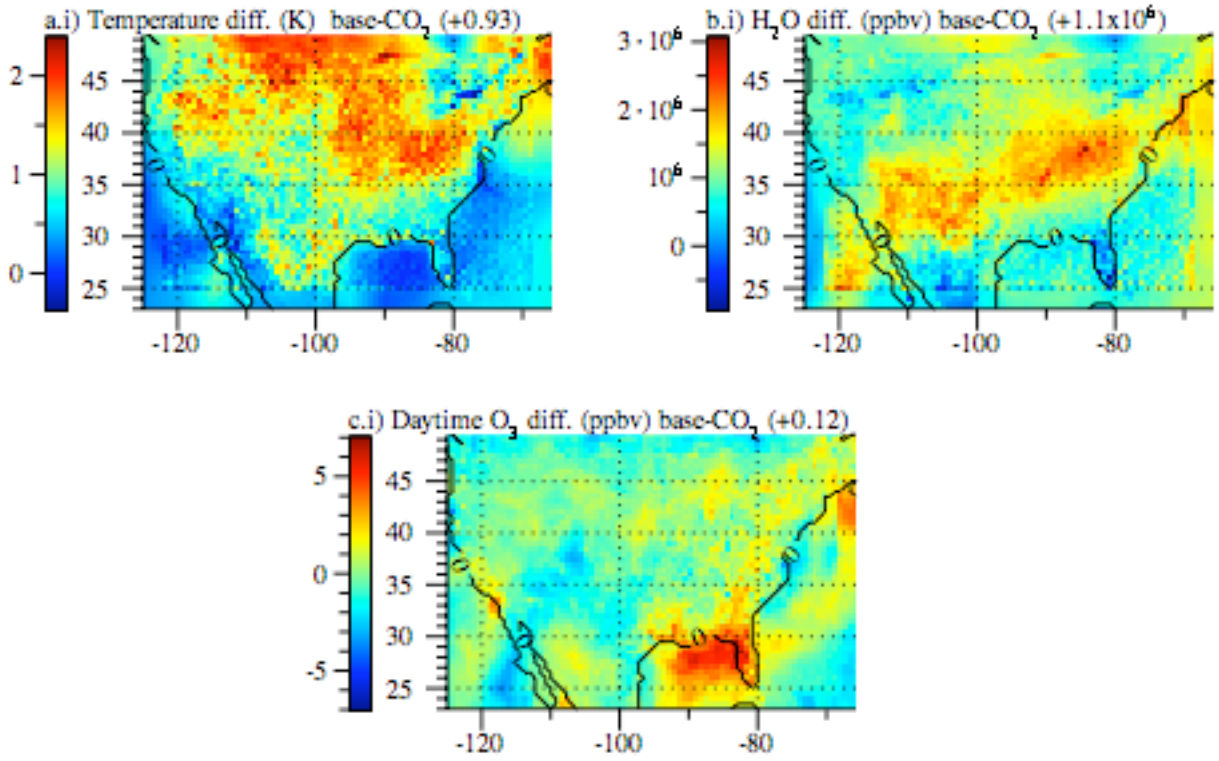


Table 1 indicates that the population-weighted ozone increase due to carbon dioxide was +0.72 ppbv, which compares with the land-averaged increase of 0.12 ppbv (Figure 2c), indicating a greater ozone increase over populated areas than less-populated areas. This result supports the hypothesis from the chemistry-only calculation that higher temperatures and water vapor due to carbon dioxide increase ozone the most where ozone is already high.

Carbon dioxide similarly increased particles in populated areas (Table 1) by warming the air more than the ground, decreasing vertical and horizontal pollution dispersion, increasing particle buildup near sources. The water vapor increase due to carbon dioxide also increased the relative humidity, swelling aerosol particles, increasing absorption of these particles by other gases, increasing the size of these particles. Carbon dioxide warming also increased land precipitation increasing aerosol removal, offsetting some of the increases in particle mass due to other processes, but not nearly enough to cause a decrease in particle levels.

The spatially-resolved changes in ozone, particles, and carcinogens (benzene, butadiene, formaldehyde, acetaldehyde) from Figure 2 and similar results were combined with population and health-effects data to produce estimates of the U.S. health effect changes due to enhanced air pollution from anthropogenic carbon dioxide. Table 1 provides resulting statistics. Mortality increases due to carbon dioxide were +415 (+207 to +620)/yr for ozone and +640 (+160 to +1280)/yr for particles per 1.07 K (Table 1) or a total of near +1000 (+350 to +1800) per 1.00 K (a 1.1% increase relative to the baseline death rate - Table 1), with about 40% due to ozone.

A simple extrapolation from U.S. to world population (301.5 to 6600 million) gives 21,600 (7400-39,000) deaths/yr worldwide per 1 K due to carbon dioxide above the baseline air pollution death rate (2.2 million/yr). The ozone portion of this (8,500 deaths/yr) is conservative compared with 15,500 deaths/yr, calculated from *West et al*. (2006), who examined the global

health effects of ozone changes, but with a lower threshold for ozone health effects (25 ppbv versus 35 ppbv here).

Carbon dioxide increased carcinogens, but the increase was small. Isoprene increases due to higher temperatures increased formaldehyde and acetaldehyde. Reduced dispersion increased exposure to these carcinogens as well as benzene and 1,3-butadiene.

Table 1. Summary of CO_2's effects on cancer, ozone mortality, ozone hospitalization, ozone emergency-room (ER) visits, and particulate-matter mortality. Results are shown for the present-day ("Base") and present-day minus preindustrial ("no-fCO_2") 3-D simulations. All mixing ratios and concentrations are near-surface values averaged over four months (mid-July to mid-November) and weighted by population (!). Divide the last column by 1.07 K (the population-weighted CO_2-induced temperature change from Table S4) to obtain the health effect per 1 K.

	Base	Base minus no fCO_2
Carcinogens		
Formaldehyde (ppbv)	3.61	+0.22
Acetaldehyde (ppbv)	2.28	+0.203
1,3-Butadiene (ppbv)	0.254	+0.00823
Benzene (ppbv)	0.479	+0.0207
USEPA cancers/yr(+)	389	+23
OEHHA cancers/yr(+)	789	+33
Ozone		
8-hr ozone (ppbv) in areas ≥35 ppbv%	42.3	+0.724
Pop (mil.) exposed in areas ≥35 ppbv#	184.8	184.8
High ozone deaths/yr*	6230	+620
Med. ozone deaths/yr*	4160	+415
Low ozone deaths/yr*	2080	+207
Ozone hospitalizations/yr*	24,100	+2400
Ozone ER visits/yr*	21,500	+2160
Particulate matter		
PM2.5 (μg/m^3) in areas > 0 μg/m^3$	16.1	+0.065
Pop (mil.) exposed in areas ≥ 0 μg/m^3	301.5	301.5
High PM2.5 deaths/yr^	191,000	+1280
Medium PM2.5 deaths/yr^	97,000	+640
Low PM2.5 deaths/yr^	24,500	+160

(!) A population-weighted value is defined in the footnote to Table S4.

(+) USEPA and OEHHA cancers/yr were found by summing the product of individual CUREs (cancer unit risk estimates=increased 70-year cancer risk per μg/m^3 sustained concentration change) by the population-weighted mixing ratio or mixing ratio difference of a carcinogen, by the population, and air density, over all carcinogens, then dividing by 70 yr. USEPA CURES are 1.3×10^{-5} (formaldehyde), 2.2×10^{-6} (acetaldehyde), 3.0×10^{-5} (butadiene), 5.0×10^{-6} (=average of 2.2×10^{-6} and 7.8×10^{-6}) (benzene) (www.epa.gov/IRIS/). OEHHA CUREs are 6.0×10^{-6} (formaldehyde), 2.7×10^{-6} (acetaldehyde), 1.7×10^{-4} (butadiene), 2.9×10^{-5} (benzene) (www.oehha.ca.gov/risk/ChemicalDB/index.asp).

(%) 8-hr ozone ≥35 ppbv is the highest 8-hour-averaged ozone during each day, averaged over all days of the four-month simulation in areas where this value ≥35 ppbv in the base case. When base O_3>35 ppbv and no-fCO_2 O_3<35 ppbv, the mixing ratio difference was base O_3 minus 35 ppbv.

(#) The 2007 population exposed to ≥35 ppbv O_3 is the population exposed to a four-month-averaged 8-hour averaged ozone mixing ratio above 35 ppbv and was determined from the base case.

(*) High, medium, and low deaths/yr, hospitalizations/yr, and emergency-room (ER) visits/yr due to short-term O_3 exposure were obtained from Eq. 2 applied to each model cell, summed over all cells. The baseline 2003 U.S. death rate (y_0) was 833 deaths/yr per 100,000 [*Hoyert et al.*, 2006]. The baseline 2002 hospitalization rate due to respiratory problems was 1189 per 100,000 [*Merrill and Elixhauser*, 2005]. The baseline 1999 all-age emergency-room visit rate for asthma was 732 per 100,000 [*Mannino et al.*, 2002]. These rates were assumed to be the same in each U.S. county although they vary slightly by county. The fraction increases (β) in the number of deaths from all causes due to ozone were 0.006, 0.004, and 0.002 per 10 ppbv increase in daily 1-hr maximum ozone [*Ostro et al.*, 2006]. These were multiplied by 1.33 to convert the risk associated with 10 ppbv increase in 1-hr maximum O_3 to that associated with a 10 ppbv increase in 8-hour average O_3 [*Thurston and Ito*, 2001]. The central value of the increased risk of hospitalization due to respiratory disease was 1.65% per 10

ppbv increase in 1-hour maximum O_3 (2.19% per 10 ppbv increase in 8-hour average O_3), and that for all-age ER visits for asthma was 2.4% per 10 ppbv increase in 1-hour O_3 [*Ostro et al*., 2006] (3.2% per 10 ppbv increase in 8-hour O_3). All values were reduced by 45% to account for the mid-July to mid-November and year-around O_3 >35 ppbv ratio, obtained from detailed observations [*H. Tran, pers. comm*.].

($) This is the simulated 24-hr $PM_{2.5}$, averaged over four months, in locations where $PM_{2.5}$ ≥0 μg/m^3.

(^) The death rate due to long-term $PM_{2.5}$ exposure was calculated from Eq. 2. *Pope et al*., [2002] provide increased dearth risks to those ≥30 years of 0.008 (high), 0.004 (medium), and 0.001 (low) per 1 μg/m^3 $PM_{2.5}$ >8 μg/m^3 based on 1979-1983 data. From 0-8 μg/m^3, the increased risks were conservatively but arbitrarily assumed =¼ those >8 μg/m^3 to account for reduced risk near zero $PM_{2.5}$. Assuming a higher risk would strengthen the conclusion found here. The all-cause 2003 U.S. death rate of those ≥30 years was 809.7 deaths/yr per 100,000 total population. No scaling of results from the 4-month model period to the annual average was performed to be conservative, since $PM_{2.5}$ concentrations from July-November are lower than in the annual average based on California data [*H. Tran, pers. comm*.].

Impacts of Carbon Dioxide on California Versus U.S. Air Pollution Health

In sum, Jacobson (2008) showed by cause and effect that carbon dioxide emitted regionally around the global increases ozone, particle, and carcinogen air pollution health problems in the United States. The study also found that pollution increases the most where air pollution is already high. Subsequently, data from the study have been extracted to calculate the portion of air pollution health problems that occurred in California. The result was that, of the additional 1000 (+350 to +1800) deaths per year in the United States due to carbon dioxide, more than 30% (>300) occurred in California, which has only 12% of the U.S. population. As such, the death rate per capita in California was over 2.5 times the national average death rate per capita due to carbon dioxide-induced air pollution. This result is not a surprise since 6 of the 10 most polluted cities in the United States, with respect to photochemical smog, are in California: Los Angeles, Visalia-Porterville, Bakersfield, Fresno, Merced, and Sacramento (e.g., www.citymayors.com/environment/polluted_uscities.html).

The disproportionate effect of carbon-dioxide-induced global warming on California compared with the rest of the United States found in this analysis contradicts a major assumption by Mr. Johnson in his decision to deny California a waiver, namely that there is no difference in the impact of globally-emitted carbon dioxide on health in California versus the U.S. as a whole. (Johnson, 2008, p. 12168).

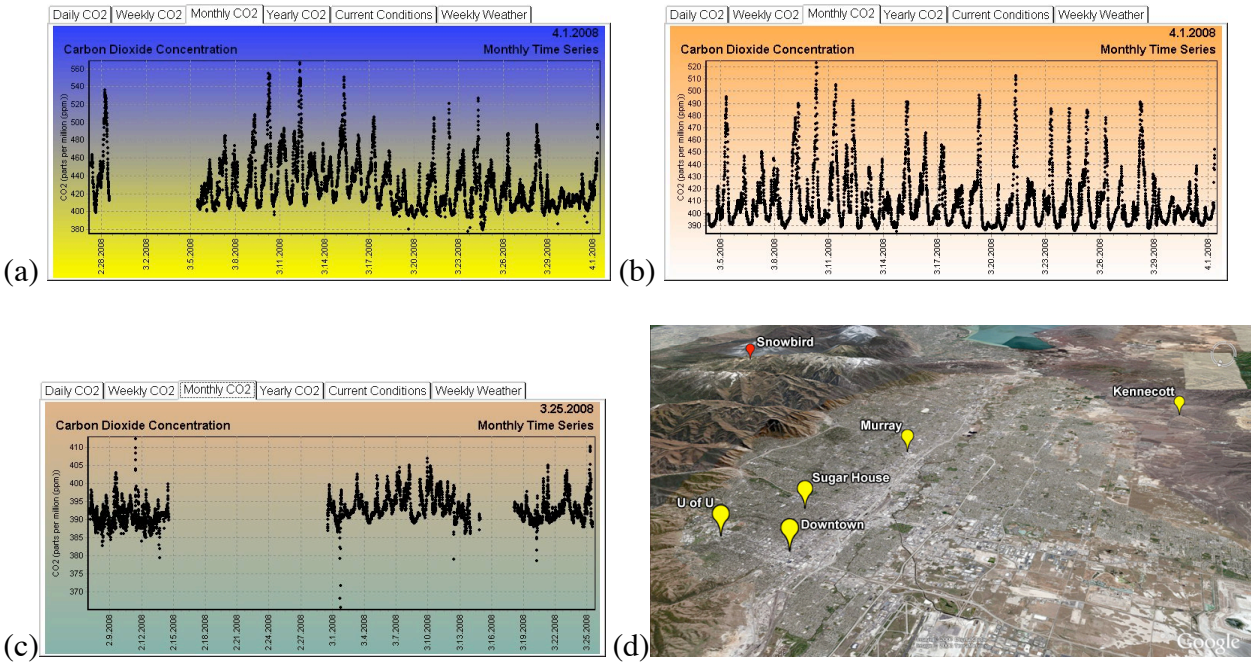
Impacts of California-Emitted Carbon Dioxide on California Health.

The results from Jacobson (2008) and the subsequent analysis of the disproportionate death rate in California versus the U.S. as a whole due to carbon dioxide provide further insight into the effect of locally-emitted carbon dioxide on local California air pollution health.

First, let's examine the effect of carbon dioxide as if local emissions were instantaneously mixed globally, which is not the case in reality. In such a case, the carbon dioxide emitted from California or the United States has the effect of increasing the death rate more in California than the rest the United States because increases in global-scale carbon dioxide increase air pollution health problems more per capita in California than in the United States as a whole (analysis above). As such, controlling local carbon dioxide in California alone would reduce the air-pollution-related death and illness rate in California at a rate 2.5 times greater per capita than it would reduce such rates in the U.S. as a whole.

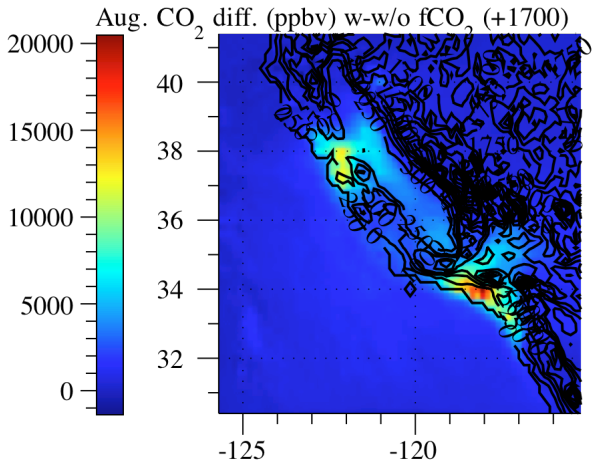
The above discussion assumed that carbon dioxide emissions mix quickly to the global atmosphere, as Mr. Johnson assumed in his waiver denial (Johnson, 2008, p. 12160). However, emissions of carbon dioxide do not mix immediately to the global atmosphere. Instead, carbon dioxide mixing ratios in polluted cites are much higher than are those in surrounding areas, as shown with data in Figure 3. Although the global background mixing ratio of carbon dioxide is currently about 385 ppmv (http://www.esrl.noaa.gov/gmd/ccgg/trends/), the data in Figure 3 indicate that the average mixing ratios in a medium-sized city's downtown area (Fig. 3a) or nearby (Fig. 3b) can be 420-440 ppmv and can peak at over 500 ppmv. Even just outside of a city, mixing ratios can average about 395 ppmv (Fig. 3c).

Figure 3. Measured mixing ratios (ppmv) of carbon dioxide in (a) downtown Salt Lake City, (b) the Sugar House monitoring site in Salt Lake City, and the Kennecott monitoring site in Salt Lake City over a month or more preceding April 1, 2008. (d) Map of the locations. Data and maps from the Ehleringer Lab at the University of Utah (http://co2.utah.edu).

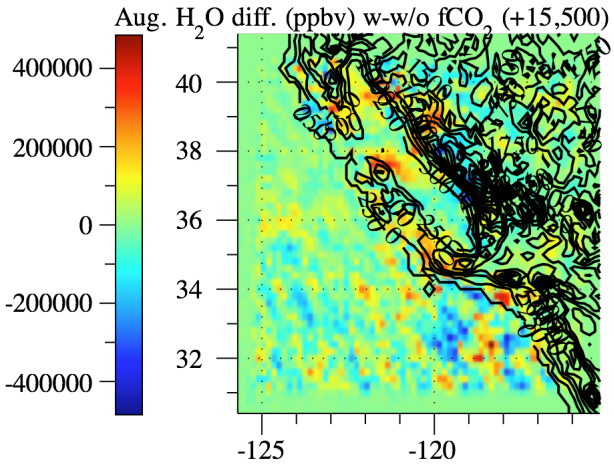


Although carbon dioxide in cities disperses to the global atmosphere, its continuous emissions from vehicles, power plants, and other sources keep its levels high over cities. It is shown here that such elevated levels of carbon dioxide can increase ozone. Figure 4a shows the computer-modeled changes in carbon dioxide in California for the month of August when simulations with and without carbon dioxide emissions were run. The elevated carbon dioxide over the urban areas (Los Angeles, San Francisco, Central Valley) is consistent with the expectations of elevated carbon dioxide in a city, as determined from data (e.g., Figure 3). It should be noted that the model grid cells for the simulations had resolution of around 15 km. A more highly-resolved domain results in higher peaks in carbon dioxide. For example, with a 5 km domain, the peak carbon dioxide above the background in Los Angeles is about 90 ppmv.

Figure 4. Modeled difference in the mixing ratios (all ppbv) of (a) carbon dioxide, (b) water vapor, and (c) daytime ozone in California during August when two simulations were run: one with fossil-fuel emissions of carbon dioxide (fCO_2) and one without such emissions. For both simulations, two nested grids were used: a global and California grid. Initial ambient levels of carbon dioxide were the same in both simulations on the California grid. Both emissions and ambient levels of carbon dioxide were the same in the global and grids in both simulations in order to ensure that local effects of carbon dioxide in California were isolated. This differs from Jacobson (2008), where both ambient and emission levels of carbon dioxide were set to preindustrial values in all grids to test whether global and local carbon dioxide would impact local pollution. The numbers in parentheses are average changes over all land points in the figure.



a)



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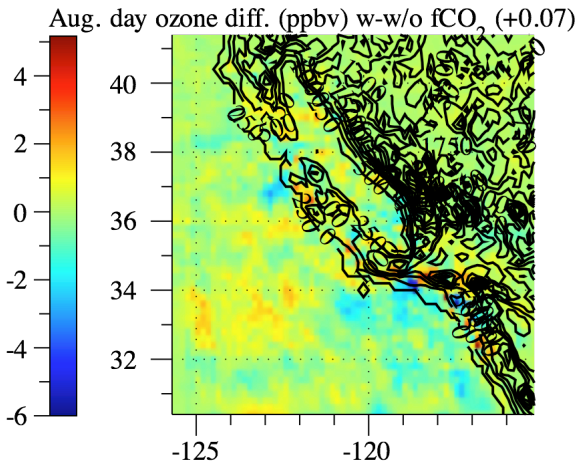

c)

Figure 4b shows that the increases in carbon dioxide in California led to an increase in water vapor, and this resulted in a net increase in ozone over all land in California, with increases in the Central Valley of up to 2 ppbv and in Los Angeles of up to 4-5 ppbv. These changes compare with polluted-air mixing ratio of above 100 ppbv and California-average daytime ozone mixing ratios in August of around 55 ppbv. Decreases also occurred in some location, but ozone increased on average over land (Figure 4c). The increases should be larger over a longer simulation period as the carbon dioxide changes from Figure 4a spread to a greater extent horizontally and vertically over California. Nevertheless, since carbon dioxide emissions outside

of the grids shown were not perturbed for the simulations, the simulations during the limited time simulated demonstrate that the effects on ozone found here were due solely to locally-emitted carbon dioxide. The figures thus demonstrate by cause and effect (since carbon dioxide emission in California was the only variable changed) that increases in locally-emitted carbon dioxide increase local ozone in California.

In sum, locally-emitted carbon dioxide is a fundamental causal factor of air pollution in California. This result contrasts with Mr. Johnson's assumption that "GHG emissions from California cars are not a causal factor for local ozone levels any more than GHG emissions from other sources of GHG emissions in the world (Johnson, 2008, p. 12163)."

Conclusions

This analysis finds the following:

1) Globally-emitted carbon dioxide increases air pollution-related mortality and other health problems in California at a rate at least 2.5 times that of the United States as a whole. The main reason is that higher temperatures and water vapor due to carbon dioxide increase pollution the most where pollution is already bad, and California has the highest levels of air pollution in the United States.

2) If emitted carbon dioxide were mixed instantaneously to the globe, which it doesn't, a decrease in California-emitted carbon dioxide would decrease the local air pollution death rate in California by at least a factor of 2.5 times more than it would decrease the death rate of the U.S. as a whole. Similarly, decreases in U.S.-emitted carbon dioxide would decrease the air pollution death rate in California at a rate at least 2.5 times higher than it would decrease the death rate of the U.S. as a whole.

3) Continuous local carbon dioxide emissions cause an increase in local outdoor carbon dioxide relative to the global average, particularly in cities. The higher carbon dioxide in cities, increasing ozone. As such, carbon dioxide is a fundamental causal factor of local air pollution.

4) Scientific findings 1-3 contradict the two assumptions that served as the basis for Mr. Johnson's decision to deny California a waiver – namely that (a) there is no difference in the impact of globally-emitted carbon dioxide on California versus U.S. health and (b) the effect of locally-emitted carbon dioxide emissions on California air pollution is no greater than the effect of U.S. or worldwide carbon dioxide emissions on California air pollution. I am unaware of any scientific publication or unpublished study that supports either assumption.

References

Aw, J., and M.J. Kleeman (2003), Evaluating the first-order effect of intraannual temperature variability on urban air pollution, *J. Geophys. Res., 108* (D12), 4365, doi:10.1029/2002JD002688.

Bell, M.L., R. Goldberg, C. Hogrefe, P.L. Kinney, K. Knowlton, B. Lynn, J. Rosenthal C. Rosenzweig, and J.A. Patz, Climate change, ambient ozone, and health in 50 U.S. cities, *Climatic Change, 82*, 61-76, 2007.

Brasseur, G.P., M.M. Schultz, C. Granier, M. Saunois, T. Diehl, M. Botzet, and E. Roeckner (2006), Impact of climate change on the future chemical composition of the global troposphere, *J. Clim., 19*, 3932-3951.

Johnson, S.L (2008) California State Motor Vehicle Pollution Control Standards; Notice of Decision Denying a Waiver of Clean Air Act Preemption for California's 2009 and Subsequent Model Year Greenhouse Gas Emission Standards for New Motor Vehicles, Federal Register, 73 (45), 12,156-12,169, March 6, 2008.

Hoyert, D.L., M.P. Heron, S.L. Murphy, and H.-C. Kung (2006), National Vital Statistics Reports, Vol. 54, No. 13, http://www.cdc.gov/nchs/fastats/deaths.htm.

Intergovernmental Panel on Climate Change (IPCC), Third Assessment Report, Climate Change 2001: The Scientific Basis, J.T. Houghton *et al.*, eds., Cambridge University Press, New York (2001).

Jacobson, M.Z. (2001), GATOR-GCMM: 2. A study of day- and nighttime ozone layers aloft, ozone in national parks, and weather during the SARMAP Field Campaign, *J. Geophys. Res., 106,* 5403-5420.

Jacobson, M.Z., J.H. Seinfeld, G.R. Carmichael, and D.G. Streets (2004), The effect on photochemical smog of converting the U.S. fleet of gasoline vehicles to modern diesel vehicles, *Geophys. Res. Lett., 31*, L02116, doi:10.1029/2003GL018448.

Jacobson, M.Z., Y.J. Kaufmann, Y. Rudich (2007) Examining feedbacks of aerosols to urban climate with a model that treats 3-D clouds with aerosol inclusions, *J. Geophys. Res., 112*, doi:10.1029/2007JD008922.

Jacobson, M.Z. (2008) On the causal link between carbon dioxide and air pollution mortality, *Geophysical Research Letters, 35*, L03809, doi:10.1029/2007GL031101.

Knowlton, K., J.E. Rosenthal, C. Hogrefe, B. Lynn, S. Gaffin, R. Goldberg, C. Rosenzweig, K. Civerolo, J.-Y. Ku, and P.L. Kinney, Assessing ozone-related health impacts under a changing climate, *Environ. Health Perspectives, 112*, 1557-1563, 2004.

Liao, H., W.-T. Chen, and J.H. Seinfeld (2006), Role of climate change in global predictions of future tropospheric ozone and aerosols, *J. Geophys. Res., 111*, D12304, doi:10.1029/2005JD006852.

Mannino, D.M., D.M. Homa, L.J. Akinbami, J.E. Moorman, C. Gwynn, S.C. Redd (2002), Center for Disease Control Morbidity and Mortality Weekly Report, Surveillance Summaries 51 (SS01); 1-13.

Merrill, C.T., and A. Elixhauser (2005), HCUP Fact Book No. 6: Hospitalization in the United States, 2002. Appendix, www.ahrq.gov/data/hcup/factbk6/factbk6e.htm.

Mickley, L.J., D.J. Jacob, B.D. Field, and D. Rind (2004) Effects of future climate change on regional air pollution episodes in the United States, *Geophys. Res. Lett., 31*, L24103, doi:10.1029/2004GL021216.

Murazaki, K., and P. Hess (2006), How does climate change contribute to surface ozone change over the United States? *J. Geophys. Res., 111*, D05301, doi:10.1029/2005JD005873.

Ostro, B.D., H. Tran, and J.I. Levy (2006), The health benefits of reduced tropospheric ozone in California, *J. Air & Waste Manage. Assoc., 56*, 1007-1021.

Pope, C.A. III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston (2002), Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution, *JAMA, 287*, 1132-1141.

Racherla, P.N. and P.J. Adams (2006), Sensitivity of global tropospheric ozone and fine particulate matter concentrations to climate change, *J. Geophys. Res., 111*, D24103, doi:10.1029/2005JD006939.

Sillman, S. and P.J. Samson (1995), Impact of temperature on oxidant photochemistry in urban, polluted rural, and remote environments, *J. Geophys. Res., 100*, 11,497-11,508.

Steiner, A.L., S. Tonse, R.C. Cohen, A.H. Goldsten, and R.A. Harley (2006), Influence of future climate and emissions on regional air quality in California, *J. Geophys. Res., 111*, D18303, doi:10.1029/2005JD006935.

Stevenson, D., R. Doherty, M. Sanderson, C. Johnson, B. Collins, and D. Derwent (2005), Impacts of climate change and variability on tropospheric ozone and its precursors. *Faraday Disc., 130*, 1-17.

Thompson, A.M., R.W. Stewart, M.A. Owens, and J.A. Herwehe (1989), Sensitivity of tropospheric oxidants to global chemical and climate change, *Atmos. Environ., 23*, 519-532.

Thurston, G.D., and K. Ito (2001), Epidemiological studies of acute ozone exposures and mortality, *J. Exposure Analysis and Env. Epidemiology, 11*, 286-294.

Unger, N., D.T. Shindell, D.M. Koch, M. Ammann, J. Cofala, and D.G. Streets (2006), Influences of man-made emissions and climate changes on tropospheric ozone, methane, and sulfate at 2030 from a broad range of possible futures, *J. Geophys. Res., 111*, D12313, doi:10.1029/2005JD006518.

West, J.J., A.M. Fiore, L.W. Horowitz, and D.L. Mauzerall (2006) Global health benefits of mitigating ozone pollution with methane emission controls, *Proc. Nat. Acad. Sci., 103*, 3988-3993.

Zhang, Y., C.H. Bischof, R.C. Easter, and P.-T. Wu, Sensitivity analysis of a mixed phase chemical mechanism using automatic differentiation, *J. Geophys. Research, 103,* 18,953-18,797 (1998).

Zhang, Y., Online coupled meteorological and chemistry models: history, current status, and outlook, *Atmos. Chem. Phys. Discuss., 8*, 1833-1912, 2008.