

# On the causal link between carbon dioxide and air pollution mortality

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[1] Greenhouse gases and particle soot have been linked to enhanced sea-level, snowmelt, disease, heat stress, severe weather, and ocean acidification, but the effect of carbon dioxide (CO<sub>2</sub>) on air pollution mortality has not been examined or quantified. Here, it is shown that increased water vapor and temperatures from higher CO<sub>2</sub> separately increase ozone more with higher ozone; thus, global warming may exacerbate ozone the most in already-polluted areas. A high-resolution global-regional model then found that CO<sub>2</sub> may increase U.S. annual air pollution deaths by about 1000 (350–1800) and cancers by 20–30 per 1 K rise in CO<sub>2</sub>-induced temperature. About 40% of the additional deaths may be due to ozone and the rest, to particles, which increase due to CO<sub>2</sub>-enhanced stability, humidity, and biogenic particle mass. An extrapolation by population could render 21,600 (7400–39,000) excess CO<sub>2</sub>-caused annual pollution deaths worldwide, more than those from CO<sub>2</sub>-enhanced storminess. **Citation:** Jacobson, M. Z. (2008), On the causal link between carbon dioxide and air pollution mortality, *Geophys. Res. Lett.*, 35, L03809, doi:10.1029/2007GL031101.

## 1. Introduction

[2] Because carbon dioxide's (CO<sub>2</sub>'s) ambient mixing ratios are too low to affect human respiration directly, CO<sub>2</sub> has not been considered a classic air pollutant. Its effects on temperatures, though, affect meteorology, and both feed back to air pollution. Several studies have modeled the sensitivity of ozone to temperature [Sillman and Samson, 1995; Zhang *et al.*, 1998] and the regional or global effects of climate change from all greenhouse gases on ozone [Thompson *et al.*, 1989; Evans *et al.*, 1998; Dvortsov and Solomon, 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] and aerosol particles [Aw and Kleeman, 2003; Liao *et al.*, 2006; Unger *et al.*, 2006]. Some studies have highlighted the effect of water vapor on chemistry [Evans *et al.*, 1998; Dvortsov and Solomon, 2001; Stevenson *et al.*, 2005; Steiner *et al.*, 2006; Racherla and Adams, 2006; Aw and Kleeman, 2003]. However, none has isolated the effect of CO<sub>2</sub> alone on ozone, particles, or carcinogens, applied population and health data to the pollution changes, or examined the problem with a global-regional climate/air pollution model.

[3] Here, a box photochemistry calculation is first used to show how increases in water vapor and temperature inde-

pendently increase ozone more with high than low ozone. This analysis helps to explain the causal link between CO<sub>2</sub> and health in areas where most people live, as subsequently found in 3-D global-regional simulations.

## 2. Chemical Effects of CO<sub>2</sub> on Ozone

[4] The SMVGEAR II chemical solver was used first in box mode, without dilution or entrainment, to solve chemistry for 12 hours among 128 gases and 395 inorganic, organic, sulfur, chlorine, and bromine reactions (including 57 photoprocesses) (mostly given by Jacobson *et al.* [2007], also see the supplementary material of Jacobson [2007]). Cases with different initial NO<sub>x</sub> and organic gas were run.

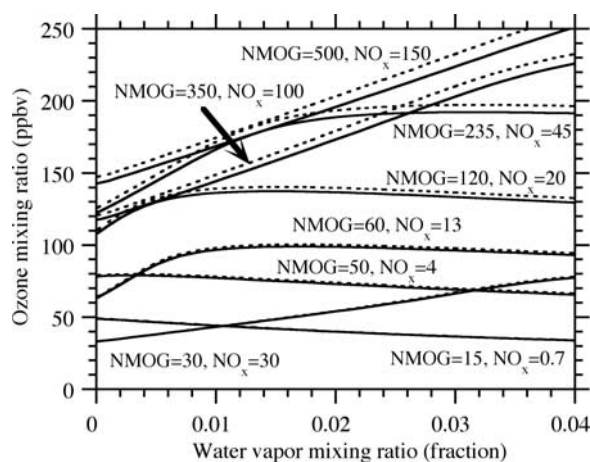
[5] Figure 1 shows the water-vapor (H<sub>2</sub>O) and temperature-dependence of ozone under several ozone precursor combinations. For initial NO<sub>x</sub> < 8 ppbv, ozone decreased with increasing H<sub>2</sub>O. For initial NO<sub>x</sub> > 80 ppbv and moderate initial NO<sub>x</sub> with low organics, though, ozone increased with increasing H<sub>2</sub>O, by up to 2.8 ppbv-O<sub>3</sub> per 1 ppbv-H<sub>2</sub>O. Between these extremes, ozone increased with increasing H<sub>2</sub>O at low H<sub>2</sub>O and stayed constant or slightly decreased at high H<sub>2</sub>O (see the auxiliary material).<sup>1</sup> Figure 1 also shows that, generally (but not always), increasing water vapor increased ozone more with higher ozone.

[6] Further, the more ozone present, the more temperature-dependent chemistry increases ozone (Figure 1), consistent with Sillman and Samson [1995] and Zhang *et al.* [1998]. The ozone increase (Δχ, ppbv) per 1 K change in temperature (ΔT) from all points in Figure 1 were fit to

$$\Delta\chi/\Delta T = -0.13034 - 0.0045585\chi + 0.00028643\chi^2 - 4.6893 \times 10^{-7}\chi^3 \quad (1)$$

where χ is ozone (ppbv) at 298.15 K (32–250 ppbv). A 1 K rise increased ozone by about 0.1 ppbv at 40 ppbv but 6.7 ppbv at 200 ppbv. Olszyna *et al.* [1997] reported an observed correlation in the rural southeast U.S. of 2.4 ppbv ozone per 1 K. If temperature-dependent chemistry alone were causing this increase, ozone would need to be about 115 ppbv (equation 1) in that study, but it was 30–90 ppbv. Thus, other factors not accounted for in Equation 1, such as H<sub>2</sub>O increases (described above) and biogenic gas emission increases [e.g., Guenther *et al.*, 1995], due to higher temperatures, may have caused the larger observed temperature-ozone correlation. Also, both temperature and ozone increase with sunlight, so all observed temperature-ozone correlations overestimate the magnitude of cause and effect.

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**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 initial  $\text{NO}_x$  and nonmethane organic gas (NMOG) mixing ratio combinations (ppbv) (given in the figure) at 298.15 K (solid lines) and 299.15 K (dashed lines). The simulations assumed sinusoidally varying photolysis between 0600 and 1800.

### 3. Health Effects of $\text{CO}_2$ From Global-U.S. Simulations

[7] The chemistry used for Figure 1 was applied with emission, aerosol, cloud, meteorological, radiative, transport, and surface processes in the nested global-urban 3-D model, GATOR-GCMOM. The model (see auxiliary material) has been evaluated against U.S. gas, aerosol, meteorological, and radiative data extensively [e.g., Jacobson, 2001; Jacobson *et al.*, 2004, 2007; Colella *et al.*, 2005].

[8] Two global simulations ( $4^\circ\text{-SN} \times 5^\circ\text{-WE}$ ) were run under present-day conditions. In the second, fossil-fuel  $\text{CO}_2$  ( $\text{fCO}_2$ ) ambient mixing ratios and emissions were set to preindustrial values. When U.S. temperatures were about 1 K higher in the present minus preindustrial- $\text{CO}_2$  global simulations, the U.S. regional domain ( $0.5^\circ\text{S-N} \times 0.75^\circ\text{W-E}$ ) in each global simulation was turned on and initialized with global-domain data (including ambient  $\text{CO}_2$ ). Global and regional domains were run another four months. Emissions of  $\text{fCO}_2$  were included in the present-day but not preindustrial- $\text{CO}_2$  global- and U.S.-domain simulations.

[9] Figures 2 and S3 show differences between the present-day and preindustrial- $\text{CO}_2$  simulations. Figure 2a compares modeled with radiosonde (1958–2006) vertical temperature differences. The population-weighted near-surface temperature increase over land was 1.07 K (Table S4), which increased population-weighted  $\text{H}_2\text{O}$  by 1.28 ppbv (Table S4) and U.S.-averaged  $\text{H}_2\text{O}$  by 1.1 ppbv (Figure 2b). The observed 1961–1995 U.S. water vapor increase and positive correlation between temperature and  $\text{H}_2\text{O}$  [Gaffen and Ross, 1999] support the modeled  $\text{H}_2\text{O}$  increase with increasing temperatures.

[10] Figure 2c indicates that  $\text{fCO}_2$  increased ozone by 0.12 ppbv in the U.S., 5 ppbv in Los Angeles, 1–5 ppbv in the southeast, and up to 2 ppbv along the northeast coast. In Los Angeles, the 0.75 K temperature increase (Figure 2a) and 1.3 ppbv water vapor increase increased ozone through chemistry (Figure 1).

[11] In the southeast, 0.5–1 K temperature increases increased isoprene and monoterpenes (Figure S3a), reducing the relative humidity (Figure S3c) and cloud optical depth (Figure S3d), increasing ultraviolet radiation (Figure S3e), and enhancing ozone. The 0.5–2 ppbv/K ozone increase in Tennessee is just below the correlated estimate of 2.4 ppbv/K from Olszyna *et al.* [1997] as expected (section 2). Averaged over the U.S. domain, higher temperatures from  $\text{fCO}_2$  increased biogenic soil  $\text{NO}_x$ , isoprene, monoterpene, and other organic carbon emissions by 6% (0.01 Tg/yr), 9% (0.47), 9.8% (0.15), and 8.9% (0.14), respectively. In the northeast, higher ozone due to higher temperatures was offset partly by higher cloud optical depth (Figure S3d) and lower ultraviolet radiation (Figure S3e), modestly increasing ozone.

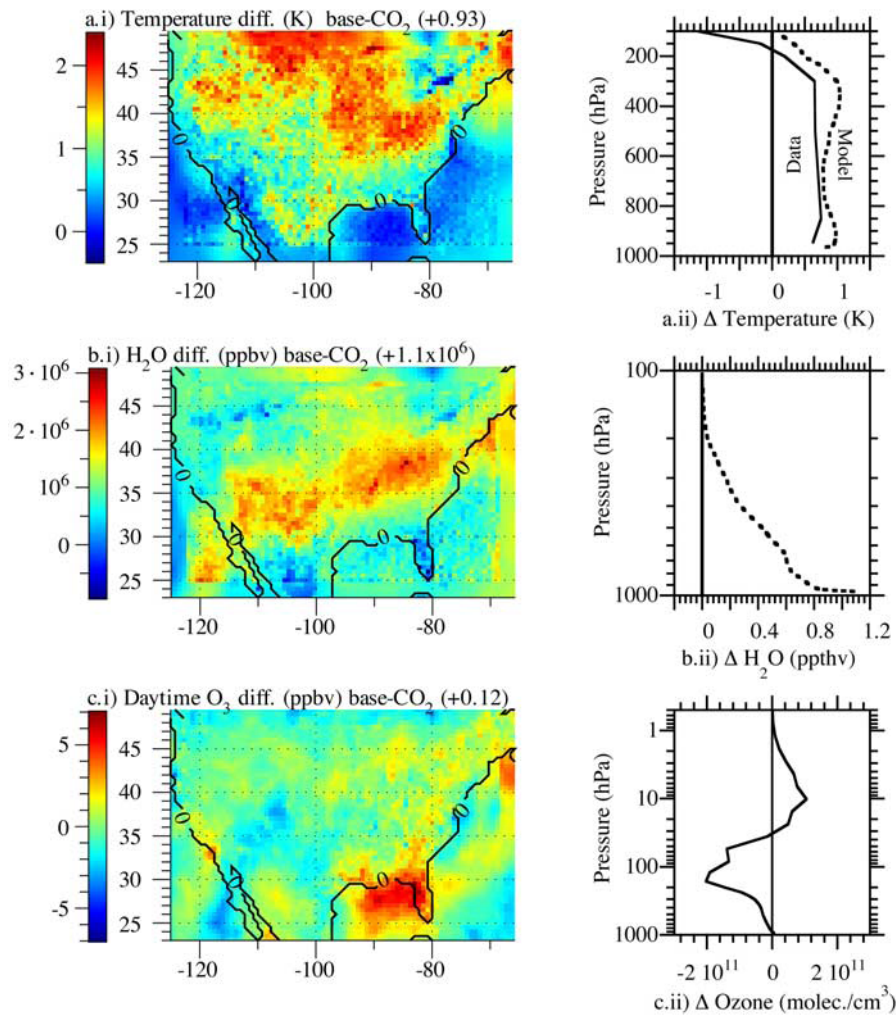
[12] The population-weighted 8-hr ozone increase due to  $\text{fCO}_2$  was +0.72 ppbv (Table 1), suggesting a greater increase over populated than less-populated areas.  $\text{FCO}_2$  increased particles in populated areas (Tables 1 and S4) by warming the air more than the ground, increasing stability (as with radiosonde data-Figure 2a, ii), decreasing turbulence, shearing stress, and surface wind speed (Table S4 and Figure S3), reducing dispersion. Reduced dispersion and wind speed are consistent with Mickley *et al.* [2004] who correlated warmer temperatures with reduced cyclone activity.  $\text{FCO}_2$  also increased isoprene and monoterpene emissions, thus secondary organic matter (SOM) (Table S4, Figures S3a and S3b); and increased relative humidity (Table S4) by increasing  $\text{H}_2\text{O}$ , swelling aerosol particles, increasing nitric acid and ammonia dissolution and the surface area for sulfuric acid and organic condensation.  $\text{FCO}_2$  increased land precipitation, consistent in direction with observed trends [Intergovernmental Panel on Climate Change, 2001], increasing aerosol removal, but less than other processes increased aerosol concentrations.

[13] Health effect changes ( $\Delta y$ ) due to ozone and  $\text{PM}_{2.5}$  changes in each model cell were determined from [e.g., Ostro *et al.*, 2006],

$$\Delta y = (1 - \exp[-\beta \Delta x]) y_0 P \quad (2)$$

where  $\Delta x$  is the simulation-averaged mixing ratio or concentration change in the cell,  $\beta$  is the fractional increase in risk per unit  $\Delta x$ ,  $y_0$  is the baseline health effect rate, and  $P$  is the cell population exposed to at least a minimum threshold. Table 1 and its footnote provide values of  $P$ ,  $\Delta x$ ,  $\beta$ ,  $y_0$ , and thresholds. Changes were summed over all cells and adjusted from a four-month to an annual average (Table 1, footnote).

[14] With this method, mortality increases due to modeled ozone and  $\text{PM}_{2.5}$  from  $\text{fCO}_2$  were 415 (207–620)/yr and 640 (160–1280)/yr, respectively, per 1.07 K (Table 1) or a total of near 1000 (350–1800) 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  $\text{fCO}_2$  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] (= 30,000 deaths/yr from 1 ppbv ozone multiplied by the 2006:2030 population ratio (66:92) and the ozone



**Figure 2.** . Four-month (mid-July to mid-November) domain-averaged near-surface and vertical-profile differences in (a) temperature, (b) water vapor, and (c) ozone between the present-day and preindustrial- $\text{CO}_2$  simulations. The domain-averaged (over land and water) change for each surface plot is given in parentheses. Also shown in Figure 2a (ii) is the 1958–2006 globally-averaged radiosonde temperature change [Thorne *et al.*, 2005], which is for reference only since the present simulations isolate the effects of  $\text{CO}_2$  and do not examine all forcing agents.

change ratio (0.72:1.0). Remaining differences may be due to different thresholds used (35 ppbv here vs. 25 ppbv).

[15] One estimate of severe weather-related fatalities worldwide in the 1990s was 33,000/yr (Worldwatch Institute, *Unnatural disaster: The lesson of Katrina*, available at [www.worldwatch.org/node/1822](http://www.worldwatch.org/node/1822), 2005). A 1 K rise will increase this number, but less than 23,000/yr given that hurricane and tornado deaths have declined due to better warning systems (e.g., the deadliest hurricane since 1910 was over 30 years ago – Honduras, 1974, 10,000 deaths). Global warming will increase heat stress- and disease-related deaths as well, but by uncertain rates [e.g., Medina-Ramon and Schwartz, 2007].

[16]  $\text{fCO}_2$  increased carcinogens, but the increase was small. Isoprene increases due to higher temperatures increased formaldehyde and acetaldehyde. Reduced dispersion increased exposure to these carcinogens and benzene and 1,3-butadiene.

[17] These simulations treated temperature effects on natural emissions but not power plant or vehicle emissions.

A sensitivity test was run examining the impact of 1 K on power plant energy demand and emissions. The resulting ozone (Figure S4) may cause 80 more U.S. deaths/yr. However, warmer winter temperatures will also decrease natural gas and vehicle emissions, and warmer summers will increase vehicle emissions [Rubin *et al.*, 2006; N. Motallebi *et al.*, manuscript in review, 2007]. The feedbacks of temperature to anthropogenic emissions must be studied more but are expected to be smaller than the other feedbacks examined here. Further uncertainties arise from model resolution, current and future emissions, numerical treatments, health data, and extrapolation of four-month results to a year, as detailed in the auxiliary material.

#### 4. Effects of $\text{CO}_2$ on Stratospheric Ozone and UV Radiation

[18] Whereas,  $\text{fCO}_2$  warms the surface and troposphere, it cools the stratosphere (Figure 2a, ii). Measurements indicate a 1%/yr (0.45 ppmv/decade) stratospheric water vapor



**Table 1.** Summary of CO<sub>2</sub>'s Effects on Cancer, Ozone Mortality, Ozone Hospitalization, Ozone Emergency Room Visits, and Particulate-Matter Mortality<sup>a</sup>

	Base	Base Minus No fCO <sub>2</sub>
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 <sup>b</sup>	389	+23
OEHAHA cancers/yr <sup>b</sup>	789	+33
Ozone		
8-hr ozone (ppbv) in areas $\geq 35$ ppbv <sup>c</sup>	42.3	+0.724
Pop (mil.) exposed in areas $\geq 35$ ppbv <sup>d</sup>	184.8	184.8
High ozone deaths/yr <sup>e</sup>	6230	620
Med. ozone deaths/yr <sup>e</sup>	4160	+415
Low ozone deaths/yr <sup>e</sup>	2080	+207
Ozone hospitalizations/yr <sup>e</sup>	24,100	+2400
Ozone ER visits/yr <sup>e</sup>	21,500	+2160
Particulate matter		
PM2.5 ( $\mu\text{g}/\text{m}^3$ ) in areas $> 0$ $\mu\text{g}/\text{m}^3$ <sup>f</sup>	16.1	+0.065
Pop (mil.) exposed in areas $\geq 0$ $\mu\text{g}/\text{m}^3$	301.5	301.5
High PM2.5 deaths/yr <sup>g</sup>	191,000	+1280
Medium PM2.5 deaths/yr <sup>g</sup>	97,000	+640
Low PM2.5 deaths/yr <sup>g</sup>	24,500	+160

<sup>a</sup>Results are shown for the present-day ("Base") and present-day minus preindustrial ("no-fCO<sub>2</sub>") 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 (population-weighted value is defined in the footnote to Table S4). Divide the last column by 1.07 K (the population-weighted CO<sub>2</sub>-induced temperature change from Table S4) to obtain the health effect per 1 K.

<sup>b</sup>USEPA and OEHAHA cancers/yr were found by summing the product of individual CUREs (cancer unit risk estimates = increased 70-year cancer risk per  $\mu\text{g}/\text{m}^3$  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  $1.3 \times 10^{-5}$  (formaldehyde),  $2.2 \times 10^{-6}$  (acetaldehyde),  $3.0 \times 10^{-5}$  (butadiene),  $5.0 \times 10^{-6}$  (= average of  $2.2 \times 10^{-6}$  and  $7.8 \times 10^{-6}$ ) (benzene) (www.epa.gov/IRIS/). OEHAHA CUREs are  $6.0 \times 10^{-6}$  (formaldehyde),  $2.7 \times 10^{-6}$  (acetaldehyde),  $1.7 \times 10^{-4}$  (butadiene),  $2.9 \times 10^{-5}$  (benzene) (www.oehha.ca.gov/risk/ChemicalDB/index.asp).

<sup>c</sup>8-hr ozone  $\geq 35$  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  $\geq 35$  ppbv in the base case. When base O<sub>3</sub>  $\geq 35$  ppbv and no-fCO<sub>2</sub> O<sub>3</sub>  $< 35$  ppbv, the mixing ratio difference was base O<sub>3</sub> minus 35 ppbv.

<sup>d</sup>The 2007 population exposed to  $\geq 35$  ppbv O<sub>3</sub> 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.

<sup>e</sup>High, medium, and low deaths/yr, hospitalizations/yr, and emergency-room (ER) visits/yr due to short-term O<sub>3</sub> exposure were obtained from Equation 2 applied to each model cell, summed over all cells. The baseline 2003 U.S. death rate (y<sub>0</sub>) 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 ( $\beta$ ) 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<sub>3</sub> to that associated with a 10 ppbv increase in 8-hour average O<sub>3</sub> [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<sub>3</sub> (2.19% per 10 ppbv increase in 8-hour average O<sub>3</sub>), and that for all-age ER visits for asthma was 2.4% per 10 ppbv increase in 1-hour O<sub>3</sub> [Ostro *et al.*, 2006] (3.2% per 10 ppbv increase in 8-hour O<sub>3</sub>). All values were reduced by 45% to account for the mid-July to mid-November and year-around O<sub>3</sub>  $\geq 35$  ppbv ratio, obtained from detailed observations (H. Tran, personal communication, 2007).

<sup>f</sup>This is the simulated 24-hr PM<sub>2.5</sub>, averaged over four months, in locations where PM<sub>2.5</sub>  $\geq 0$   $\mu\text{g}/\text{m}^3$ .

<sup>g</sup>The death rate due to long-term PM<sub>2.5</sub> exposure was calculated from Equation 2. Pope *et al.* [2002] provide increased death risks to those  $\geq 30$  years of 0.008 (high), 0.004 (medium), and 0.001 (low) per 1  $\mu\text{g}/\text{m}^3$  PM<sub>2.5</sub>  $> 8$   $\mu\text{g}/\text{m}^3$  based on 1979–1983 data. From 0–8  $\mu\text{g}/\text{m}^3$ , the increased risks were conservatively but arbitrarily assumed =  $1/4$  those  $> 8$   $\mu\text{g}/\text{m}^3$  to account for reduced risk near zero PM<sub>2.5</sub>. Assuming a higher risk would strengthen the conclusion found here. The all-cause 2003 U.S. death rate of those  $\geq 30$  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<sub>2.5</sub> concentrations from July–November are lower than in the annual average based on California data (H. Tran, personal communication, 2007).

increase from 1954–2000 [Rosenlof *et al.*, 2001], but a slight lower-stratospheric decrease from 2001–2005 [Randel *et al.*, 2006]. The simulations here, which accounted for chlorine and bromine gas and heterogeneous chemistry, found that the temperature and H<sub>2</sub>O changes due to fCO<sub>2</sub> increased middle and upper-stratospheric ozone but decreased upper tropospheric and lower stratospheric (UTLS) ozone, where its column abundance is greater, causing a net U.S. column ozone loss of 2.7% (Figure 2c, ii, and Table S4). The UTLS ozone losses were due to increases in H<sub>2</sub>O there (Figure 2b, ii), as indicated by Figure S2b and Dvortsov and Solomon [2001]. The upper- and middle-stratospheric gains can be explained by Figure S1, which shows that, at 25 km, stratospheric ozone decreases by 1.5% as H<sub>2</sub>O increases by 1 ppmv. As temperature

decreases by 1.5 K, though, ozone increases by 3.6%, suggesting an overall ozone increase from H<sub>2</sub>O and cooling. The ozone increase upon stratospheric cooling is due to reduced loss from O+O<sub>3</sub> [Evans *et al.*, 1998]. Despite the column ozone loss due to fCO<sub>2</sub>, surface UV hardly changed (Table S4) because fCO<sub>2</sub> increased cloud optical depth, offsetting UV increases from ozone loss.

## 5. Summary

[19] A climate-air pollution model showed by cause and effect that fossil-fuel CO<sub>2</sub> increases increase U.S. surface ozone, carcinogens, and particulate matter, thereby increasing death, asthma, hospitalization, and cancer rates. Increased water vapor and temperatures due to higher CO<sub>2</sub>

each increase ozone increasingly with increasing ozone. At low ozone, more water vapor decreases ozone slightly but higher temperatures increase biogenic emission in many areas, offsetting ozone decreases in such areas. CO<sub>2</sub> increases stability, the relative humidity, and biogenic particle mass thus PM<sub>2.5</sub>. Finally, CO<sub>2</sub> decreases column ozone over the U.S. by increasing upper tropospheric/lower stratospheric water vapor.

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