Enhancement of Local Air Pollution by Urban CO_2 Domes

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Data suggest that domes of high CO_2 levels form over cities. Despite our knowledge of these domes for over a decade, no study has contemplated their effects on air pollution or health. In fact, all air pollution regulations worldwide assume arbitrarily that such domes have no local health impact, and carbon policy proposals, such as "cap and trade", implicitly assume that CO_2 impacts are the same regardless of where emissions occur. Here, it is found through data-evaluated numerical modeling with telescoping domains from the globe to the U.S., California, and Los Angeles, that local CO_2 emissions in isolation may increase local ozone and particulate matter. Although health impacts of such changes are uncertain, they are of concern, and it is estimated that that local CO_2 emissions may increase premature mortality by 50–100 and 300–1000/yr in California and the U.S., respectively. As such, reducing locally emitted CO_2 may reduce local air pollution mortality even if CO_2 in adjacent regions is not controlled. If correct, this result contradicts the basis for air pollution regulations worldwide, none of which considers controlling local CO_2 based on its local health impacts. It also suggests that a "cap and trade" policy should consider the location of CO_2 emissions, as the underlying assumption of the policy is incorrect.

Introduction

Although CO_2 is generally well-mixed in the atmosphere, data indicate that its mixing ratios are higher in urban than in background air, resulting in *urban CO_2 domes* (*1–6*). Measurements in Phoenix, for example, indicate that peak and mean CO_2 in the city center were 75% and 38–43% higher, respectively, than in surrounding rural areas (*2*). Recent studies have examined the impact of global greenhouse gases on air pollution (*7–13*). Whereas one study used a 1-D model to estimate the temperature profile impact of a CO_2 dome (*3*), no study has isolated the impact of locally emitted CO_2 on air pollution or health. One reason is that model simulations of such an effect require treatment of meteorological feedbacks to gas, aerosol, and cloud changes, and few models include such feedbacks in detail. Second, local CO_2 emissions are close to the ground, where the temperature contrast between the Earth's surface and the lowest CO_2 layers is small. However, studies have not considered that CO_2 domes result in CO_2 gradients high above the surface. If locally emitted CO_2 increases local air pollution, then cities, counties, states, and small countries can reduce air pollution health problems by reducing their own CO_2 emissions, regardless of whether other air pollutants are reduced locally or whether other locations reduce CO_2.

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Methodology and Evaluation

For this study, the nested global-through-urban 3-D model, GATOR-GCMOM (*13–17*) was used to examine the effects of locally emitted CO_2 on local climate and air pollution. A nested model is one that telescopes from a large scale to more finely resolved domains. The model and its feedbacks are described in the Supporting Information. Example CO_2 feedbacks treated include those to heating rates thus temperatures, which affect (a) local temperature and pressure gradients, stability, wind speeds, cloudiness, and gas/particle transport, (b) water evaporation rates, (c) the relative humidity and particle swelling, and (d) temperature-dependent natural emissions, air chemistry, and particle microphysics. Changes in CO_2 also affect (e) photosynthesis and respiration rates, (f) dissolution and evaporation rates of CO_2 into the ocean, (g) weathering rates, (h) ocean pH and chemical composition, (i) sea spray pH and composition, and (j) rainwater pH and composition. Changes in sea spray composition, in turn, affect sea spray radiative properties, thus heating rates.

The model was nested from the globe (resolution $4°SN \times 5°WE$) to the U.S. ($0.5° \times 0.75°$), California ($0.20° \times 0.15°$), and Los Angeles ($0.045° \times 0.05°$). The global domain included 47 sigma-pressure layers up to 0.22 hPa (~60 km), with high resolution (15 layers) in the bottom 1 km. The nested regional domains included 35 layers exactly matching the global layers up to 65 hPa (~18 km). The model was initialized with 1-degree global reanalysis data (*18*) but run without data assimilation or model spinup.

Three original pairs of baseline and sensitivity simulations were run: one pair nested from the globe to California for one year (2006), one pair nested from the globe to California to Los Angeles for two sets of three months (Feb-Apr, Aug-Oct, 2006), and one pair nested from the globe to the U.S. for two sets of three months (Jan-Mar, Jul-Sep, 2006). The seasonal periods were selected to obtain roughly winter/summer results that could be averaged to estimate annual values. A second 1-year (2007) simulation pair was run for California to test interannual variability. In each sensitivity simulation, only anthropogenic CO_2 emissions (emCO$_2$) were removed from the finest domain. Initial ambient CO_2 was the same in all domains of both simulations, and emCO$_2$ was the same in the parent domains of both. As such, all resulting differences were due solely to initial changes in locally emitted (in the finest domain) CO_2.

The model and comparisons with data have been described in over 50 papers, including recently (*13–17*). Figure 1 further compares modeled O_3, PM_{10}, and CH_3CHO from August 1–7 of the baseline (with emCO$_2$) and sensitivity (no emCO$_2$) simulations from the Los Angeles domain with data. The comparisons indicate good agreement for ozone in particular. Since emCO$_2$ was the only variable that differed initially between simulations, it was the initiating causal factor in the increases in O_3, PM_{10}, and CH_3CHO seen in Figure 1. Although ozone was predicted slightly better in the no-emCO$_2$ case than in the emCO$_2$ case during some hours, modeled ozone in the emCO$_2$ case matched peaks better by about 0.5% averaged over comparisons with all data shown and not shown.

Results

Figure 2a,b shows the modeled contribution of California's CO_2 emissions to surface and column CO_2, respectively, averaged over a year. The CO_2 domes over Los Angeles, the San Francisco Bay Area, Sacramento (38.58 N, 121.49 W),
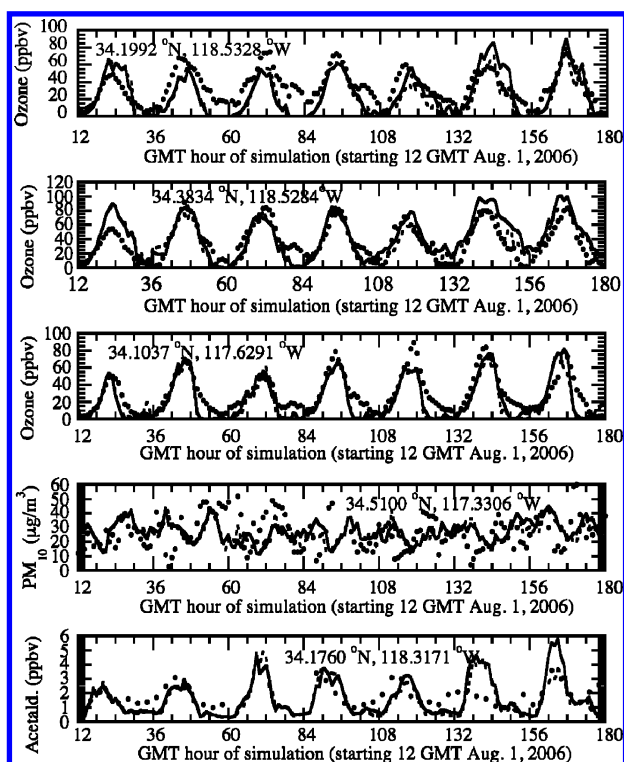
FIGURE 1. Paired-in-time-and-space comparisons of modeled baseline (solid lines), modeled no-emCO$_2$ (dashed lines), and data (22) (dots) for ozone, sub-10-μm particle mass, and acetaldehyde from the Los Angeles domain for August 1−7, 2006 of the Aug-Oct 2006 simulation. Local standard time is GMT minus 8 h.

and the Southern Central Valley are evident. The largest surface CO$_2$ increase (5%, or 17.5 ppmv) was lower than observed increases in cities (2) since the resolution of the California domain was coarser than the resolution of measurements. As shown below for Los Angeles, an increase in model resolution increases the magnitude of the surface and column CO$_2$ dome.

Population-weighted (PW) and domain-averaged (DA) changes in several parameters can help to elucidate the effects of the CO$_2$ domes. A PW value is the product of a parameter value and population in a grid cell, summed over all grid cells, all divided by the summed population among all cells. Thus, a PW value indicates changes primarily in populated areas, whereas a DA value indicates changes everywhere, independent of population. The PW and DA increases in surface CO$_2$ due to emCO$_2$ were 7.4 ppmv and 1.3 ppmv, respectively, but the corresponding increases in column CO$_2$ were 6.0 g/m^2 and 1.53 g/m^2, respectively, indicating, along with Figure 2a,b, that changes in column CO$_2$ were spread horizontally more than were changes in surface CO$_2$. This is because surface winds are usually slower than winds aloft, so only when surface CO$_2$ mixes vertically is it transported much horizontally, and when that occurs, surface CO$_2$ is quickly replenished with new emissions.

The CO$_2$ increases in California increased the PW air temperature by about 0.0063 K, more than it changed the domain-averaged air temperature (+0.00046) (Figure 2c). Thus, CO$_2$ domes had greater temperature impacts where the CO$_2$ was emitted and where people lived than in the domain average. This result held for the effects of emCO$_2$ on column water vapor (Figure 2d - PW: +4.3 g/m^2; DA: +0.88 g/m^2), ozone (Figure 2e - PW: +0.06 ppbv; DA: +0.0043 ppbv), PM$_{2.5}$ (Figure 2g - PW: +0.08 μg/m^3; DA: −0.0052 μg/m^3), and PAN (Figure 2i - PW: +0.002 ppbv;

DA: −0.000005 ppbv). The peak surface air temperature increases in Figure 2c (and in the Los Angeles simulations) were ∼0.1 K, similar to those found from 1-D radiative only calculations for Phoenix (3). Peak ozone and its health effects occurred over Los Angeles and Sacramento (Figure 2e,f), where increases in CO$_2$ (Figure 2a), temperature (although small for Sacramento, Figure 2c), and column H$_2$O (Figure 2d) occurred.

Figure 3 elucidates spatial correlations between annually averaged changes in local ambient CO$_2$ caused by emCO$_2$ and changes in other parameters. Increases in temperature, water vapor, and ozone correlated positively and with statistical significance ($p << 0.05$) with increases in CO$_2$. Ozone increases also correlated positively and with strong significance with increases in water vapor and temperature. A previous study found that increases in temperature and water vapor both increase ozone at high ozone but cause little change in ozone at low ozone (13), consistent with this result.

PM$_{2.5}$ correlated slightly negatively ($r = 0.017$) but without statistical significance, with higher temperature and much more positively ($r = 0.23$) and with strong significance ($p < 0.0001$) with higher water vapor in California. Higher temperature decreased PM$_{2.5}$ by increasing vapor pressures thus PM evaporation and by enhancing precipitation in some locations. Some PM$_{2.5}$ decreases with higher temperature were offset by biogenic organic emission increases with higher temperatures followed by biogenic oxidation to organic PM. But, in populated areas of California, biogenic emissions are relatively low. Some PM$_{2.5}$ decreases were also offset by surface PM$_{2.5}$ increases caused by slower surface winds due to enhanced boundary-layer stability from CO$_2$, which reduced the downward transport of fast winds aloft to the surface (13). While higher temperature slightly decreased PM$_{2.5}$, higher water vapor due to emCO$_2$ increased PM$_{2.5}$ by increasing aerosol water content, increasing nitric acid and ammonia gas dissolution, forming more particle nitrate and ammonium. Higher ozone from higher water vapor also increased oxidation of organic gases to organic PM. Overall, PM$_{2.5}$ increased with increasing CO$_2$, but because of the opposing effects of temperature and water vapor on PM$_{2.5}$, the net positive correlation was weak ($r = 0.022$) and not statistically significant ($p = 0.17$). However, when all CO$_2$ increases below 1 ppmv were removed, the correlation improved substantially ($r = 0.047$, $p = 0.07$). Further, the correlation was strongly statistically significant for Los Angeles and U.S. domains, as discussed shortly.

Health effect rates (y) due to pollutants in each model domain for each simulation were determined from

$$y = y_0 \sum_i \left\{ P_i \sum_t (1 - \exp[-\beta \times \max(x_{i,t} - x_{th}, 0)]) \right\}$$

$$(1)$$

where $x_{i,t}$ is the concentration in grid cell i at time t, x_{th} is the threshold concentration below which no health effect occurs, β is the fractional increase in risk per unit x, y_0 is the baseline health effect rate, and P_i is the grid cell population. Table 1 provides sums or values of P, β, y_0, and x_{th}. Differences in health effects between two simulations were obtained by differencing the aggregated effects from each simulation determined from eq 1. The relationship between ozone exposure and premature mortality is uncertain; however, ref 19 suggests that it is "highly unlikely" to be zero. Similarly, ref 20 suggests that the exact relationship between PM$_{2.5}$ exposure and mortality is uncertain but "likely causal". Cardiovascular effects of PM$_{2.5}$ are more strongly "causal". Although health effects of PM$_{2.5}$ differ for different chemical components within PM$_{2.5}$, almost all epidemiological studies
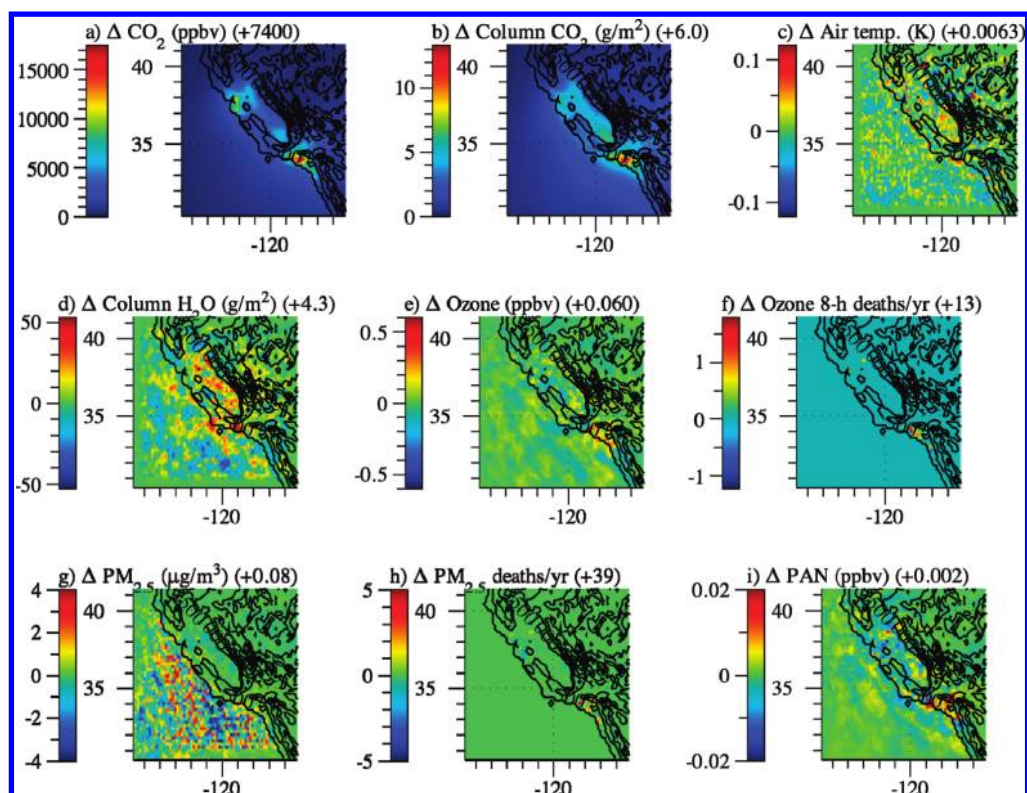
FIGURE 2. Modeled annually averaged difference for several surface or column (if indicated) parameters in California, parts of Nevada, and parts of New Mexico when two simulations (with and without emCO₂) were run. The numbers in parentheses are average population-weighted changes for the domain shown.
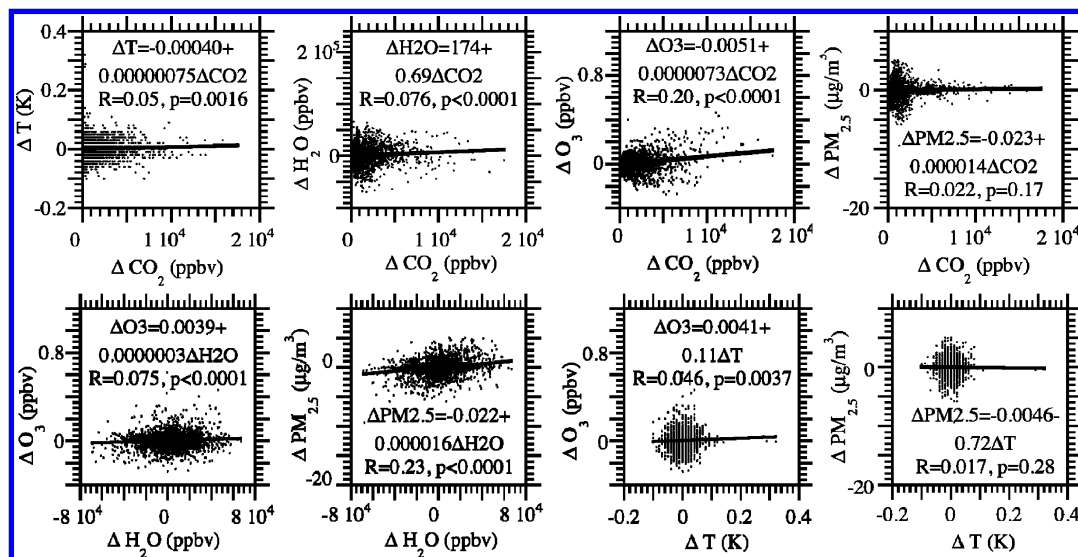

FIGURE 3. Scatter plots of paired-in-space one-year-averaged changes between several parameter pairs, obtained from all near-surface grid cells of the California domain. Also shown is an equation for the linear fit through the data points in each case and the *r* and *p* values for the fits. The equation describes correlation only, not cause and effect, between each parameter pair.

correlating particle changes with health use ambient $PM_{2.5}$ measurements to derive such correlations. For consistency, it is therefore necessary to apply β values from such studies to modeled $PM_{2.5}$ (*22*).

California's local CO_2 resulted here in ~13 (with a range of 6−19 due to uncertainty in epidemiological data) additional ozone-related premature mortalities/year (Figure 2f) or 0.3% above the baseline 4600 (2300−6900)/year (Table 1). Higher $PM_{2.5}$ due to emCO₂ contributed another ~39 (13−60) premature mortalities/year (Figure 2h), 0.2%

above the baseline rate of 22,500 (5900−42,000)/year. Changes in cancer due to emCO₂ were relatively small (Table 1). Additional uncertainty arises due to the model itself and interannual variations in concentration. Some of the model uncertainties are elucidated in comparisons with data, such as in Figure 1; however, it is difficult to translate such uncertainty into mortality uncertainty. Interannual variations in concentrations were examined by running a second pair of simulations for California, starting one year after the first. The results of this simulation

TABLE 1. Summary of Locally-Emitted CO_2's ($emCO_2$) Effects on Cancer, Ozone Mortality, Ozone Hospitalization, Ozone Emergency-Room (ER) Visits, and Particulate-Matter Mortality in California (CA), Los Angeles (LA), and the United States (U.S.)[d]

	annual base CA	base minus no emCO₂CA	annual base LA	base minus no emCO₂ LA	annual base U.S.	base minus no emCO₂ U.S.
ozone ≥ 35 ppbv (ppbv)	47.4	+0.060	44.7	+0.12	47.0	+0.044
$PM_{2.5}$ ($\mu g/m^3$) (pop-weight)	50.0	+0.08	36	+0.29	64.4	+0.041
$PM_{2.5}$ ($\mu g/m^3$) (all land)	21.5	−0.007	25.8	+0.06	32.8	+0.039
formaldehyde (ppbv)	4.43	+0.0030	4.1	+0.054	6.75	+0.066
acetaldehyde (ppbv)	1.35	+0.0017	1.3	+0.021	2.45	+0.016
1,3-butadiene (ppbv)	0.11	−0.00024	0.23	+0.0020	0.077	+0.0005
benzene (ppbv)	0.30	−0.00009	0.37	+0.0041	0.34	+0.020
Cancer						
USEPA cancers/yr[a]	44.1	0.016	22.0	+0.28	573	+6.9
OEHHA cancers/yr[a]	54.4	−0.038	37.8	+0.39	561	+11.8
Ozone Health Effects						
high O_3 mortalities/yr[b]	6860	+19	2140	+20	52,300	+245
med. O_3 mortalities/yr[b]	4600	+13	1430	+14	35,100	+166
low O_3 mortalities/yr[b]	2300	+6	718	+7	17,620	+85
O_3 hospitalizations/yr[b]	26,300	+65	8270	+75	200,000	+867
ozone ER visits/yr[b]	23,200	+56	7320	+66	175,000	+721
PM Health Effects						
high $PM_{2.5}$ mortalities/yr[c]	42,000	+60	16,220	+147	44,800	+810
med. $PM_{2.5}$ mortalities/yr[c]	22,500	+39	8500	+81	169,000	+607
low $PM_{2.5}$ mortalities/yr[c]	5900	+13	2200	+22	316,000	+201

[a] USEPA (U.S. Environmental Protection Agency) and OEHHA (Office of Environmental Health Hazard Assessment) cancers/yr were found by summing, over all model surface grid cells and the four carcinogens (formaldehyde, acetaldehyde, 1,3-butadiene, and benzene), the product of individual CUREs (cancer unit risk estimates=increased 70-year cancer risk per $\mu g/m^3$ sustained concentration change), the mass concentration ($\mu g/m^3$) (for baseline statistics) or mass concentration difference (for difference statistics) of the carcinogen, and the population in the cell and then dividing by the population of the model domain and by 70 yr. USEPA CURES were 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 were 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). [b] High, medium, and low mortalities/yr, hospitalizations/yr, and emergency-room (ER) visits/yr due to short-term O_3 exposure were obtained from eq 1, assuming a threshold (x_{th}) of 35 ppbv (23). The baseline 2003 U.S. mortality rate (y_0) was 833 mortalities/yr per 100,000 (24). The baseline 2002 hospitalization rate due to respiratory problems was 1189 per 100,000 (25). The baseline 1999 all-age emergency-room visit rate for asthma was 732 per 100,000 (26). The fractional increases (β) in the number of premature mortalities from all causes due to ozone were 0.006, 0.004, and 0.002 per 10 ppbv increase in daily 1-h maximum ozone (27). These were multiplied by 1.33 to convert the risk associated with a 10 ppbv increase in 1-h maximum O_3 to that associated with a 10 ppbv increase in 8-h average O_3 (23). The central value of the increased risk of hospitalization due to respiratory disease was 1.65% per 10 ppbv increase in 1-h maximum O_3 (2.19% per 10 ppbv increase in 8-h average O_3), and that for all-age ER visits for asthma was 2.4% per 10 ppbv increase in 1-h O_3 (3.2% per 10 ppbv increase in 8-h O_3) (25, 26). [c] The mortality rate due to long-term PM_{25} exposure was calculated from eq 1. Increased premature mortality risks to those ≥30 years were 0.008 (high), 0.004 (medium), and 0.001 (low) per 1 $\mu g/m^3$ $PM_{2.5} > 8$ $\mu g/m^3$ based on 1979−1983 data (28). From 0−8 $\mu g/m^3$, the increased risks were assumed to be a quarter of the risks for those >8 $\mu g/m^3$ to account for reduced risk near zero $PM_{2.5}$ (13). The all-cause 2003 U.S. mortality rate of those ≥30 years was 809.7 mortalities/yr per 100,000 total population. Reference 29 provides higher relative risks of $PM_{2.5}$ health effects data; however, the values from ref 28 were retained to be conservative. [d] Results are shown for the with-$emCO_2$ emissions simulation ("base") and the difference between the base and no $emCO_2$ emissions simulations ("base minus no-$emCO_2$") for each case. The domain summed populations (sum of P_i in eq 1) in the CA, LA, and U.S. domains were 35.35 million, 17.268 million, and 324.07 million, respectively. All concentrations except the second $PM_{2.5}$, which is an all-land average, were near-surface values weighted spatially by population. $PM_{2.5}$ concentrations in the table include liquid water, but $PM_{2.5}$ used for health calculations were dry. CA results were for an entire year, LA results were an average of Feb-Apr and Aug-Oct (Figure 4), and U.S. results were an average of Jan-Mar and Jul-Sep.

were similar to those for the first, with ∼51 (17−82) additional ozone- plus $PM_{2.5}$-related premature mortalities/year attributable to $emCO_2$.

Simulations for Los Angeles echo results for California but allowed for a more resolved picture of the effects of $emCO_2$. Figure 4a (Feb-Apr) indicates that the near-surface CO_2 dome that formed over Los Angeles peaked at about 34 ppmv, twice that over the coarser California domain. The column difference (Figure 4b) indicates a spreading of the dome over a larger area than the surface dome. In Feb-Apr and Aug-Oct, $emCO_2$ enhanced PW ozone and $PM_{2.5}$, increasing mortality (Figure 4, Table 1) and other health effects (Table 1). The causes of such increases, however, differed with season.

During Feb-Apr, infrared absorption by $emCO_2$ warmed air temperatures (Figure 4c) up to ∼3 km altitude, increasing

the land-ocean temperature gradient by about 0.2 K over 50 km, increasing surface sea-breeze wind speeds by ∼0.06 m/s, and increasing water vapor transport to and soil−water evaporation in Los Angeles (Figure 4d). Higher temperatures and water vapor slightly increased ozone and $PM_{2.5}$ for the reasons given in ref 13. The high wind speeds also increased resuspension of road and soil dust and moved PM more to the eastern basin.

During summer, Los Angeles boundary layer heights, temperature inversions, land-sea temperature gradients, sea breeze wind speeds, water evaporation rates, column water vapor, and stratus cloud formation are greater than in summer. Since boundary-layer heights were higher during the Aug-Oct simulations, CO_2 mixed faster up to higher altitudes during summer. Initially, the higher CO_2 warmed the air up to 4 km above topography, but the higher
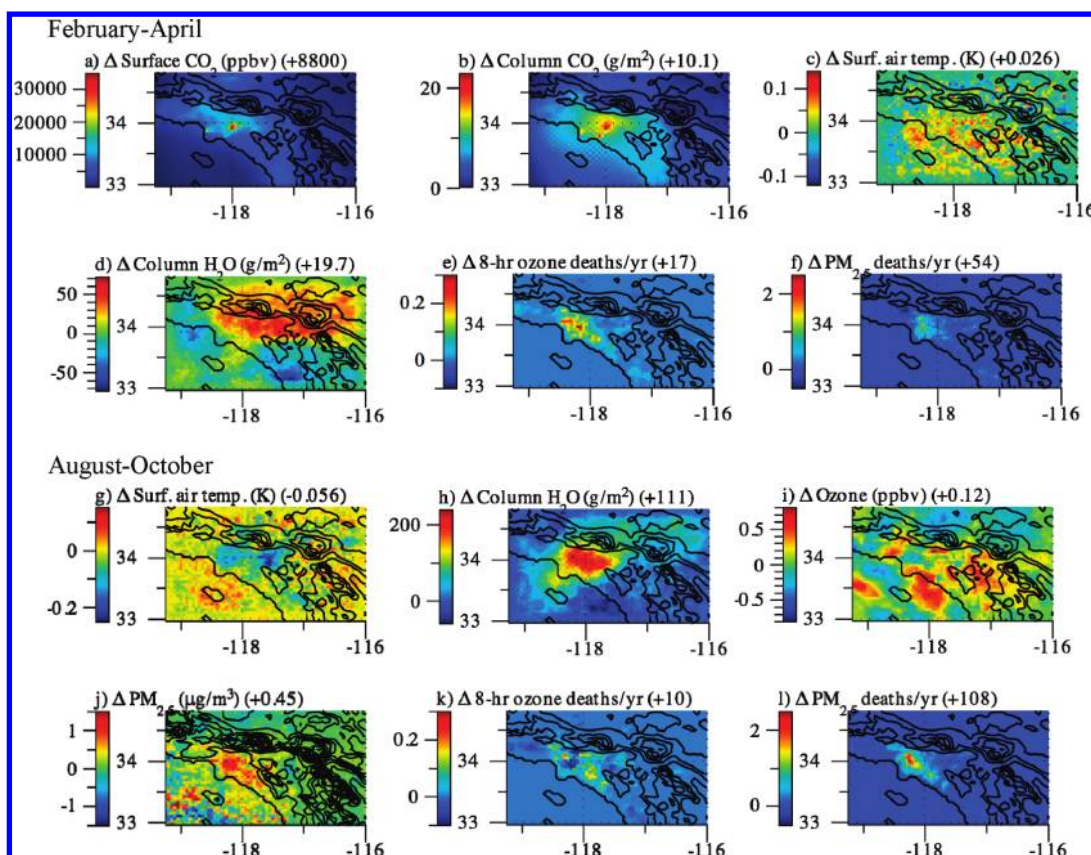
FIGURE 4. Same as Figure 2 but for the Los Angeles domain and for Feb-Apr and Aug-Oct.

temperatures from 1.5−4 km decreased the upper-level sea-breeze return flow (figures not shown) decreased pressure aloft, reducing the flow of moisture from land to ocean aloft (increasing it from ocean to land), increasing cloud optical depth over land by up to 0.4−0.6 optical depth units, decreasing summer surface solar radiation by at most 3−4 W/m^2 locally, decreasing local ground temperatures by up to 0.2 K (Figure 4g) while retaining the warmer air aloft. The excess water vapor aloft over land mixed to the surface (Figure 4h), increasing ozone (which increases chemically with water vapor at high ozone) and the relative humidity, which increased aerosol particle swelling, increasing gas growth onto aerosols, and reducing particle evaporation. In summary, $emCO_2$ increased ozone and $PM_{2.5}$ and their corresponding health effects in both seasons, increasing air pollution mortality in California and Los Angeles by about 50−100 per year (Figure 4e,f,i,j, Table 1). The spatial positive correlations between increases in near-surface CO_2 and near-surface O_3 and $PM_{2.5}$ were both visually apparent (Figure 4) and strongly statistically significant (e.g., Aug-Oct, $r = 0.14$, $p < 0.0001$ for ΔCO_2 vs ΔO_3; $r = 0.24$, $p < 0.0001$ for ΔCO_2 vs $\Delta PM_{2.5}$).

For the U.S. as a whole, the correlations between increases in CO_2 and increases in O_3 and $PM_{2.5}$ premature mortality were also both visually apparent (Figure 5) and statistically significant ($r = 0.31$, $p < 0.0001$ for ΔCO_2 vs ΔO_3 mortality; $r = 0.32$, $p < 0.0001$ for ΔCO_2 vs $\Delta PM_{2.5}$ mortality). The Jun-Aug correlation between ΔCO_2 and $\Delta PM_{2.5}$ concentration ($r = 0.1$, $p < 0.0001$) was weaker than that between ΔCO_2 and $\Delta PM_{2.5}$ mortality, since local CO_2 fed back to meteorology, which fed back to $PM_{2.5}$ outside of cities as well as in cities, but few people were exposed to such changes in $PM_{2.5}$ outside of cities. Nevertheless, both correlations were strongly statistically significant.

The annual premature mortality rates due to $emCO_2$ in the U.S. were ~770 (300−1000), with ~20% due to ozone. This rate represented an enhancement of ~0.4% of the baseline mortality rate due to air pollution. With a U.S. anthropogenic emission rate of 5.76 GT-CO_2/yr (Table S2), this corresponds to ~134 (52−174) additional premature mortalities/GT-CO_2/yr over the U.S. Modeled mortality rates in Los Angeles for the Los Angeles domain were higher than those for Los Angeles in the California or U.S. domains due to the higher resolution of the Los Angeles domain; thus, mortality estimates for California and the U.S. may be low.

Implications

Worldwide, emissions of NO_x, HCs, CO, and PM are regulated. The few CO_2 regulations proposed to date have been justified based on its large-scale feedback to temperatures, sea levels, water supply, and global air pollution. No proposed CO_2 regulation is based on the potential impact of locally emitted CO_2 on local pollution as such effects have been assumed not to exist (21). Here, it was found that local CO_2 emissions can increase local ozone and particulate matter due to feedbacks to temperatures, atmospheric stability, water vapor, humidity, winds, and precipitation. Although modeled pollution changes and their health impacts are uncertain, results here suggests that reducing local CO_2 may reduce 300−1000 premature air pollution mortalities/yr in the U.S. and 50−100/yr in California, even if CO_2 in adjacent regions is not controlled. Thus, CO_2 emission controls may be justified on the same grounds that NO_x, HC, CO, and PM emission regulations are justified. Results further imply that the assumption behind the "cap and trade" policy, namely that CO_2 emitted in one location has the same impact as CO_2 emitted in another, is incorrect, as CO_2 emissions in populated cities have larger health impacts than CO_2 emissions in unpopulated
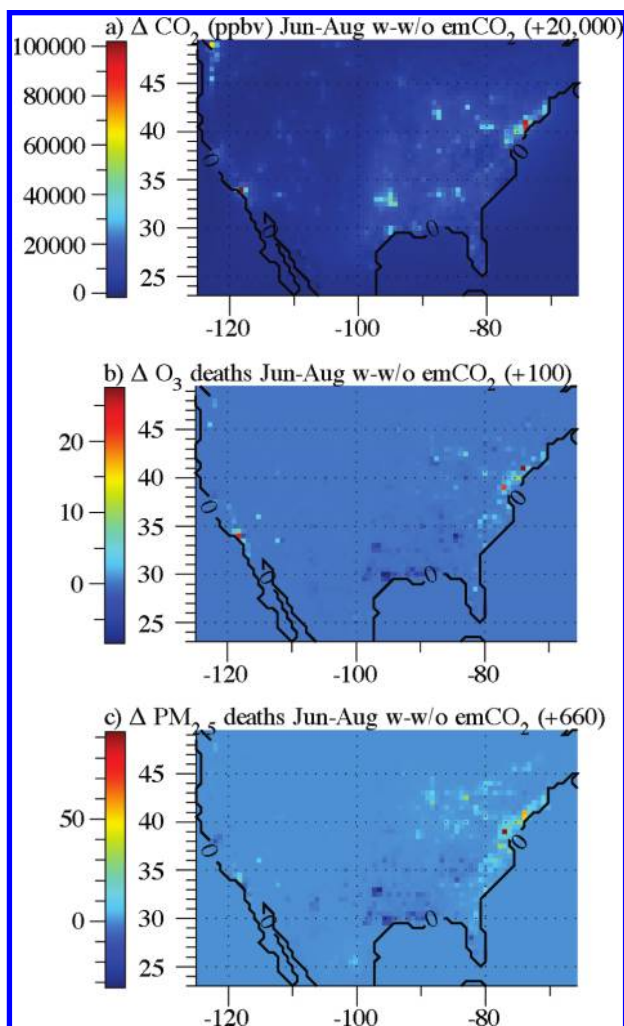
FIGURE 5. Same as Figure 2 but for the U.S. domain and for Jun-Aug. Numbers in parentheses Jun-Aug averaged changes (for CO_2) or total changes (for mortalities) over the domain.

areas. As such, CO_2 cap and trade, if done, should consider the location of emissions to avoid additional health damage.

Supporting Information Available
Model and emissions used for this study (Section 1), feedbacks in the model (Section 2), and a description of simulations (Section 3). This material is available free of charge via the Internet at http://pubs.acs.org.

Literature Cited

(1) Idso, C. D.; Idso, S. B.; Balling, R. C., Jr. The urban CO_2 dome of Phoenix, Arizona. *Phys. Geogr.* **1998**, *19*, 95–108.

(2) Idso, C. D.; Idso, S. B.; Balling, R. C., Jr. An intensive two-week study of an urban CO2 dome in Phoenix, Arizona, USA. *Atmos. Environ.* **2001**, *35*, 995–1000.

(3) Balling, R. C., Jr.; Cerveny, R. S.; Idso, C. D. Does the urban CO_2 dome of Phoenix, Arizona contribute to its heat island? *Geophys. Res. Lett.* **2001**, *28*, 4599–4601.

(4) Gratani, L.; Varone, L. Daily and seasonal variation of CO_2 in the city of Rome in relationship with the traffic volume. *Atmos. Environ.* **2005**, *39*, 2619–2624.

(5) Newman, S.; Xu, X.; Affek, H. P.; Stolper, E.; Epstein, S. Changes in mixing ratio and isotopic composition of CO_2 in urban air from the Los Angeles basin, California, between 1972 and 2003. *J. Geophys. Res.* **2008**, *113*, D23304, doi:10.1029/2008JD009999.

(6) Rigby, M.; Toumi, R.; Fisher, R.; Lowry, D.; Nisbet, E. G. First continuous measurements of CO_2 mixing ratio in central London using a compact diffusion probe. *Atmos. Environ.* **2008**, *42*, 8943–8953.

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

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

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

(10) Unger, N.; Shindell, D. T.; Koch, D. M.; Ammann, M.; Cofala, J.; Streets, D. G. 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.* **2006**, *111*, D12313, doi:10.1029/2005JD006518.

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

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

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

(14) Jacobson, M. Z.; Streets, D. G. The influence of future anthropogenic emissions on climate, natural emissions, and air quality. *J. Geophys. Res.* **2009**, *114*, D08118, doi:10.1029/2008JD011476.

(15) Jacobson, M. Z. 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.* **2001**, *106*, 5403–5420.

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

(17) Jacobson, M. Z. The short-term effects of agriculture on air pollution and climate in California. *J. Geophys. Res.* **2008**, *113*, D23101, doi:10.1029/2008JD010689.

(18) Global Forecast System. 1°x1° reanalysis fields; 2007; http://nomads.ncdc.noaa.gov/data/ (accession July 1, 2008).

(19) *Estimating mortality risk reduction and economic benefits from controlling ozone air pollution*; National Research Council, The National Academies Press: Washington, DC, 2008.

(20) *Integrated science assessment for particulate matter, Second External Review Draft*; U.S. Environmental Protection Agency: 2008; EPA/600/R-08/139B.

(21) Johnson, S. L. 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. *Fed. Register* **2008**, *73* (45), 12,156–12,169.

(22) *AIR Data*; United States Environmental Protection Agency: 2006; http://www.epa.gov/air/data/ (accession August 1, 2009).

(23) Thurston, G. D.; Ito, K. Epidemiological studies of acute ozone exposures and mortality. *J. Exposure Anal. Environ. Epidemiol.* **2001**, *11*, 286–294.

(24) Hoyert, D. L.; Heron, M. P.; Murphy, S. L.; Kung H.-C. National Vital Statistics Reports; Vol. 54, No. 13, 2006. http://www.cdc.gov/nchs/fastats/deaths.htm (accession August 1, 2009).

(25) Merrill, C. T.; Elixhauser, A. HCUP Fact Book No. 6: Hospitalization in the United States, 2002; Appendix, 2005. www.ahrq.gov/data/hcup/factbk6/factbk6e.htm (accession August 1, 2009).

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

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

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

(29) Pope III, C. A.; Burnett, R. T.; Thurston, G. D.; Thun, M. J.; Calle, E. E.; Drewski, D.; Godleski, J. J. Cardiovascular mortality and long-term exposure to particulate air pollution. *Circulation* **2004**, *109*, 71–77.

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