The enhancement of local air pollution by urban CO$_2$ domes

Mark Z. Jacobson
Department of Civil and Environmental Engineering, Stanford University, Stanford, California 94305-4020, USA; Email: jacobson@stanford.edu; Tel: (650) 723-6836

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Data suggest that domes of high CO$_2$ levels form over cities$^{1-5}$. Despite our knowledge of these domes for over a decade$^1$, no study has contemplated their effects on local temperature or water vapor or the resulting feedback to air pollution and health. In fact, all air pollution regulations worldwide assume arbitrarily that such domes have no local health impact$^6$ 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 by cause and effect that local CO$_2$ emissions indeed increase local ozone, particulate matter, and mortality. As such, reducing locally-emitted CO$_2$ will reduce local air pollution mortality even if CO$_2$ in adjacent regions is not controlled. This result contradicts the basis for all air pollution regulations worldwide, none of which considers controlling local CO$_2$ based on its local health impacts. It also suggests that implementation of a “cap and trade” policy should consider the location of CO$_2$ emissions, as the underlying assumption of the policy is incorrect.

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$^{1-5}$. Measurements in Phoenix, for example, indicate that peak and mean CO$_2$ in the city center are 75% and 38-43% higher, respectively, than in surrounding rural areas$^2$. Many recent studies have examined the impact of global greenhouse gases on air pollution$^7$-$^{14}$. However, no study has isolated the impact of locally-emitted CO$_2$ on local air pollution, health, or climate. 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 CO₂ emissions, regardless of whether other air pollutants are reduced locally or whether other locations reduce CO₂.

For this study, the nested global-through-urban 3-D model, GATOR-GCMOM\textsuperscript{15-20} was used to examine the effects of locally-emitted CO₂ on local climate and air pollution on two scales, California as a whole and the Los Angeles basin. Three pairs of baseline and sensitivity simulations were run: one pair nested from the globe to California for one year and two pairs nested from the globe to California to Los Angeles, each for three months (Aug-Oct; Feb-Apr). In each sensitivity simulation, only anthropogenic CO₂ emissions (emCO₂) were removed from the finest domain. Initial ambient CO₂ was the same in all domains of both simulations and emCO₂ was the same in the parent domains of both. As such, all resulting differences were due solely to locally-emitted (in the finest domain) CO₂.

The model and comparisons with data have been described over 16 years, including recently\textsuperscript{15-20}. Figure 1 further compares modeled O₃, PM\textsubscript{10}, and CH₃CHO from August 1-7 of the baseline (with emCO₂) and sensitivity (no emCO₂) simulations from the Los Angeles domain with data. The comparisons indicate very good agreement with respect to ozone in particular and that emCO₂ increased O₃, PM\textsubscript{10}, and CH₃CHO almost immediately, during day and night.

Figure 2a shows the modeled contribution to surface CO₂ of California’s CO₂ emissions. The CO₂ domes over Los Angeles, the San Francisco Bay Area, and parts of the Central Valley are evident. The largest CO₂ increase (5%, or 17.5 ppmv) was lower than observed increases in cities (1) since the resolution of the California domain was coarser than the resolution of measurements. As shown for Los Angeles shortly, an increase in model resolution increases the magnitude of the CO₂ dome. Whereas the population-weighted (PW) and domain-averaged (DA) increases in surface CO₂ due to emCO₂ were 7.4 ppmv and 1.3 ppmv, respectively, the corresponding increases in column CO₂ were 6.0 g/m\textsuperscript{2} and 1.53 g/m\textsuperscript{2}, respectively, indicating that changes in column CO₂ were spread horizontally more than were changes in surface CO₂. This is because local emCO₂ starts mixing with
the larger scale soon after emissions, but the losses are quickly replaced with more local \( \text{CO}_2 \) emissions.

The \( \text{CO}_2 \) increases in California increased the PW air temperature by about 0.0063 K, more than it changed the domain-averaged air temperature (+0.00046) (Fig. 2b). Thus, \( \text{CO}_2 \) domes had greater temperature impacts where the \( \text{CO}_2 \) was emitted and where people lived than they had in the domain average. This result holds for the effects of \( \text{emCO}_2 \) on column water vapor (Fig. 2c - PW: +4.3 g/m\(^2\); DA: +0.88 g/m\(^2\)), ozone (Fig. 2d – PW: +0.06 ppbv; DA: +0.0043 ppbv), \( \text{PM}_{2.5} \) (Fig. 2f – PW: +0.08 µg/m\(^3\); DA: -0.0052 µg/m\(^3\)), PAN (Fig. 2h – PW: +0.002 ppbv; DA: -0.000005 ppbv) and particle nitrate (Fig. 2i – PW: +0.030 µg/m\(^3\); DA: +0.00084 µg/m\(^3\)).

Figure 3 elucidates correlations between changes in local ambient \( \text{CO}_2 \) caused by \( \text{emCO}_2 \) and changes in other parameters. Modeled temperature, water vapor, ozone, and \( \text{PM}_{2.5} \) increased more in grid cells with larger ambient \( \text{CO}_2 \) increases than in cells with smaller ambient \( \text{CO}_2 \) increases. In other words, increases in ozone and \( \text{PM}_{2.5} \) correlated spatially with local \( \text{CO}_2 \) increases. Figure 2 shows further that ozone increases correlated spatially with temperature and water vapor increases, both of which increase ozone particularly at high ozone\(^{15}\).

\( \text{PM}_{2.5} \) correlated slightly negatively (R=0.017) with higher temperature but more strongly positively (R=0.23) with higher water vapor (Fig. 2). Higher temperature decreased \( \text{PM}_{2.5} \) by increasing vapor pressures thus PM evaporation and by enhancing precipitation in some locations. Some \( \text{PM}_{2.5} \) decreases from higher temperatures were offset by biogenic organic emission increases from higher temperatures followed by biogenic oxidation to organic PM. But in California, biogenic emissions are lower than in the southeast U.S. Some \( \text{PM}_{2.5} \) decreases were also offset by slower winds caused by enhanced boundary-layer stability from \( \text{CO}_2 \). While higher temperatures slightly decreased \( \text{PM}_{2.5} \), higher water vapor due to \( \text{emCO}_2 \) increased \( \text{PM}_{2.5} \) by increasing aerosol water content, increasing nitric acid and ammonia gas dissolution, forming more particle nitrate (Fig. 2i) and ammonium. Higher ozone from higher water vapor also increased oxidation of organic gases to organic PM. Since \( \text{PM}_{2.5} \) increased overall due to \( \text{emCO}_2 \), water vapor increases of PM exceeded temperature decreases.
Health effect rates ($y$) due to pollutants in each model domain were determined from

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

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, $\beta$ 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 $P$, $\beta$, $y_0$, and $x_{th}$.

California’s local CO$_2$ resulted in ~13 (6-19) additional ozone-related deaths/year (Fig. 2e), or 0.3% above the baseline 4600 (2300-6900) deaths/year (Table 1). Higher PM$_{2.5}$ due to emCO$_2$ contributed another ~39 (13-60) deaths/year (Fig. 2g), 0.2% above the baseline death rate of 22,500 (5900-42,000) deaths/year. Changes in cancer due to emCO$_2$ were relatively small (Table 1).

Simulations for Los Angeles echo results for California but allowed for a higher-resolution, more accurate picture of the effects of emCO$_2$. Figure 4 (Feb-Apr) indicates that the CO$_2$ dome that formed over Los Angeles peaked at about 34 ppmv, twice that over the coarser California domain. The column difference 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 (Fig. 4, Table 1) and other health effects (Table 1). The causes of such increases, however, differed with season. From Feb-Apr, emCO$_2$ increased surface temperatures and water vapor over the Los Angeles basin (Fig. 4). This slightly enhanced ozone and PM$_{2.5}$, but the increase in the land-ocean temperature gradient also increased sea-breeze wind speeds, increasing resuspension of road and soil dust and moving particulate matter more to the eastern basin. From Aug-Oct, emCO$_2$ increased temperatures aloft, increasing the land-sea temperature gradient and wind speed aloft, increasing the flow of moisture from the ocean to land aloft, increasing water vapor and clouds over land, decreasing surface solar radiation, causing a net decrease in local ground temperatures and UV radiation but a net increase in water vapor at all altitudes due to the vertical diffusion of water vapor aloft to the surface. The higher water vapor triggered higher ozone and relative humidities, which increased...
aerosol particle swelling, increasing gas growth onto aerosols, and reducing particle evaporation. In
sum, emCO$_2$ increased ozone and PM$_{2.5}$ and their corresponding health effects in both seasons,
increasing air pollution deaths in California and Los Angeles by about 50-100 per year (Fig. 4, Table
1). Death rates for Los Angeles were similar or higher than those for California due to the greater
accuracy of higher resolution (Los Angeles) simulations, as shown in Table 2 of Ref 17; thus, these
results are likely to be conservative for California as a whole.

The California mortality increase compares with a U.S. death rate increase of about 1000/yr
per 1 K rise due to all globally-emitted anthropogenic CO$_2$, with about 300 deaths/yr occurring in
California$^{15}$, which has 12% of the U.S. population. The greater death rates in California versus the
rest of the U.S. are due to the fact that higher temperatures and water vapor due to CO$_2$ enhance air
pollution the most where it is already high, and California has 6 of the top 10 polluted cities in the
U.S.

Worldwide, emissions of many pollutants (e.g., NO$_x$, HCs, CO, PM) that cause local air
pollution are regulated. The few CO$_2$ emission regulations proposed to date have been justified based
on the large-scale climate effects and resulting feedbacks to sea levels, water supply, and global air
pollution that such emissions cause. However, 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$^{6}$. The
result here suggests that reducing local CO$_2$ will reduce 50-100 California air pollution deaths/yr
even if CO$_2$ in adjacent regions is not controlled. Thus, CO$_2$ emission controls are 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 areas. As such, CO$_2$ cap and trade, if done,
should consider the location of emissions to avoid additional health damage.

References


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Figure Captions

**Figure 1.** (a) Paired-in-time-and-space comparisons of modeled baseline (solid lines), modeled no-emCO$_2$ (dashed lines), and data$^{21}$ (dots) for ozone, sub-10-µm particle mass, and acetaldehyde from the Los Angeles domain for August 1-7, 2006. The resolutions of the global, California, and Los Angeles domains were 4° SN x 5° WE, 0.20° SN x 0.15° WE, and 0.45° SN x 0.05° WE, respectively. The global domain included 47 sigma-pressure layers up to 0.22 hPa (≈60 km), with very 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 run without data assimilation or model spinup.

**Figure 2.** Modeled annually averaged difference for several surface or column parameters when two simulations (with and without emCO$_2$) were run. The numbers in parentheses are population-weighted changes.

**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.

**Figure 4.** Same as Fig. 2., but for the Los Angeles domain and for Feb-Apr and Aug-Oct. Also shown are scatter plots for Aug-Oct similar to those for Fig. 3.
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. 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 California and Los Angeles. The domain summed populations in the Los Angeles and California domains were 17.268 million and 35.35 million, respectively. All concentrations are near-surface values weighted spatially by population. Los Angeles results were an average of Feb-Apr and Aug-Oct results.

<table>
<thead>
<tr>
<th></th>
<th>Annual base Calif.</th>
<th>Base minus no emCO$_2$ Calif.</th>
<th>Annual base LA</th>
<th>Base minus no emCO$_2$ LA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ozone ≥ 35 ppbv (ppbv)</td>
<td>47.4</td>
<td>+0.060</td>
<td>44.7</td>
<td>+0.12</td>
</tr>
<tr>
<td>PM$_{2.5}$ ($\mu$g/m$^3$)</td>
<td>50.0</td>
<td>+0.08</td>
<td>36</td>
<td>+0.29</td>
</tr>
<tr>
<td>Formaldehyde (ppbv)</td>
<td>4.43</td>
<td>+0.0030</td>
<td>4.1</td>
<td>+0.054</td>
</tr>
<tr>
<td>Acetaldehyde (ppbv)</td>
<td>1.35</td>
<td>+0.0017</td>
<td>1.3</td>
<td>+0.021</td>
</tr>
<tr>
<td>1,3-Butadiene (ppbv)</td>
<td>0.11</td>
<td>-0.00024</td>
<td>0.23</td>
<td>+0.0020</td>
</tr>
<tr>
<td>Benzene (ppbv)</td>
<td>0.30</td>
<td>-0.00009</td>
<td>0.37</td>
<td>+0.0041</td>
</tr>
</tbody>
</table>

Cancer
- USEPA cancers/yr
  - 44.1
  - 0.016
  - 22.0
  - +0.28
- OEHHA cancers/yr
  - 54.4
  - -0.038
  - 37.8
  - +0.39

Ozone health effects
- High O$_3$ deaths/yr
  - 6860
  - +19
  - 2140
  - +20
- Med. O$_3$ deaths/yr
  - 4600
  - +13
  - 1430
  - +14
- Low O$_3$ deaths/yr
  - 2300
  - +6
  - 718
  - +7
- O$_3$ hospitalizations/yr
  - 26,300
  - +65
  - 8270
  - +75
- Ozone ER visits/yr
  - 23,200
  - +56
  - 7320
  - +66

PM health effects
- High PM$_{2.5}$ deaths/yr
  - 42,000
  - +60
  - 16,220
  - +147
- Medium PM$_{2.5}$ deaths/yr
  - 22,500
  - +39
  - 8500
  - +81
- Low PM$_{2.5}$ deaths/yr
  - 5900
  - +13
  - 2200
  - +22

(+) USEPA and OEHHA 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, then dividing by the population of the model domain and by 70 yr. USEPA CURES are $1.3\times10^{-5}$ (formaldehyde), $2.2\times10^{-6}$ (acetaldehyde), $3.0\times10^{-5}$ (butadiene), $5.0\times10^{-6}$ (=average of $2.2\times10^{-6}$ and $7.8\times10^{-6}$) (benzene) (www.epa.gov/IRIS/). OEHHA CURES are $6.0\times10^{-6}$ (formaldehyde), $2.7\times10^{-6}$ (acetaldehyde), $1.7\times10^{-4}$ (butadiene), $2.9\times10^{-5}$ (benzene) (www.oehha.ca.gov/risk/ChemicalDB/index.asp).
(*) High, medium, and low deaths/yr, hospitalizations/yr, and emergency-room (ER) visits/yr due to short-term $O_3$ exposure were obtained from Equation 1, assuming a threshold of 35 ppbv. The baseline 2003 U.S. death rate ($y_0$) was 833 deaths/yr per 100,000. The baseline 2002 hospitalization rate due to respiratory problems was 1189 per 100,000. The baseline 1999 all-age emergency-room visit rate for asthma was 732 per 100,000. The fractional 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. These were multiplied by 1.33 to convert the risk associated with a 10 ppbv increase in 1-hr maximum $O_3$ to that associated with a 10 ppbv increase in 8-hour average $O_3$. 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 $O_3$ (3.2% per 10 ppbv increase in 8-hour $O_3$).

(*) The death rate due to long-term PM$_{2.5}$ exposure was calculated from Equation 1. Increased death risks to those ≥30 years were 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 here were assumed ≈¼ those >8 µg/m$^3$ to account for reduced risk near zero PM$_{2.5}$. The all-cause 2003 U.S. death rate of those ≥30 years was 809.7 deaths/yr per 100,000 total population.
Figure 1

Acetaldehyde (ppbv)

PM (µg/m³)

Ozone (ppbv)

Ozone (ppbv)

GMT hour of simulation (starting 12 GMT Aug. 1, 2006)

34.1992° N, 118.5328° W (1211)

34.3834° N, 118.5284° W (1241)

34.1037° N, 117.6291° W (1456)

34.1760° N, 118.3171° W (1202)

34.5100° N, 117.3306° W (1452)

34.0837° N, 117.6291° W (1456)
Figure 2

a) Δ CO₂ (ppbv) w/o emCO₂ (+7400)

b) Δ Air temp. (K) w/o emCO₂ (+0.0063)

c) Δ Column H₂O (g/m²) w/o emCO₂ (+4.3)

d) Δ Ozone (ppbv) w/o emCO₂ (+0.060)

e) Δ Ozone 8-h deaths/yr w/o emCO₂ (+13)

f) Δ PM₁₀ deaths/yr w/o emCO₂ (+39)

h) Δ PAN (ppbv) w/o emCO₂ (+0.002)

i) Δ NO₃ (ng/m³) w/o emCO₂ (+0.030)
Figure 3

- \( \Delta T = -0.00040315 + 0.000000749 \Delta CO_2 \)  
  \( R = 0.050 \)

- \( \Delta H_2O = 173.59 + 0.69162 \Delta CO_2 \)  
  \( R = 0.0755 \)

- \( \Delta O_3 = -0.0050819 + 0.00000729 \Delta CO_2 \)  
  \( R = 0.200 \)

- \( \Delta H_2O = 173.59 + 0.69162 \Delta CO_2 \)  
  \( R = 0.0755 \)

- \( \Delta O_3 = -0.0050819 + 0.00000729 \Delta CO_2 \)  
  \( R = 0.200 \)
Figure 4

February-April

August-October

\[ \Delta \text{Near-surface } \text{CO}_2 \text{ (ppbv) w-w/o emCO}_2 \ (\pm 8800) \]

\[ \Delta \text{Column } \text{CO}_2 \ (g/m}^2 \text{) w-w/o emCO}_2 \ (\pm 10.1) \]

\[ \Delta \text{Surface air temp. (K) w-w/o emCO}_2 \ (\pm 0.026) \]

\[ \Delta \text{Column } \text{H}_2\text{O (g/m}^2 \text{) w-w/o CO}_2 \ (\pm 19.7) \]

\[ \Delta \text{8-hr ozone deaths/yr w-w/o emCO}_2 \ (\pm 17) \]

\[ \Delta \text{PM}_{2.5} \text{ deaths/yr w-w/o emCO}_2 \ (\pm 54) \]

\[ \Delta \text{8-hr ozone deaths/yr w-w/o emCO}_2 \ (\pm 10) \]

\[ \Delta \text{PM}_{2.5} \text{ deaths/yr w-w/o emCO}_2 \ (\pm 108) \]

\[ \Delta \text{O}_3=0.0228+0.00000572\Delta \text{CO}_2 \quad R=0.139 \]

\[ \Delta \text{PM}_{2.5}=0.00136+0.0000166\Delta \text{CO}_2 \quad R=0.242 \]