Interesting Facts About Ozone

The EPA is considering lowering the national ozone standard. This is based primarily on two health effects: a decrease in lung function, and premature mortality (dying sooner than you should). The EPA sets standards that are protective of public health, including presumed sensitive subpopulations, such as people (particularly children) with asthma. The following interesting facts address the relationship between ozone and these health effects. For general information about ozone and ozone monitoring go to https://www.tceq.texas.gov/airquality/monops/ozonefacts.html.

FEV1 and Lung Function:

- The EPA is considering lowering the ozone standard to 60 ppb averaged over an 8 hour period[1]. This is based in part on data from studies that exposed human volunteers to 60 ppb ozone, while they were exercising at high intensity for 50 minutes of every hour for 6.6 hours. In one study (Adams 2006) these volunteers had an average 2.8% decrease in forced expiratory volume for 1 second (FEV_1) with ozone exposure, and in another study, the volunteers had a 1.75% decrease in FEV_1 (Kim, 2011). However, the American Thoracic Society and the European Respiratory Society (ATS/ERS) together published that the daily variation in FEV_1 for a healthy person is 5% (Pellegrino et al 2005). Therefore, the effects of ozone at 60 ppb were *within normal variation* and cannot be characterized as adverse.

- In addition, the ATS/ERS states that changes in FEV_1 correlate "poorly with symptoms and may not, by itself, accurately predict clinical severity or prognosis for individual patients." This group requires that reversible loss of lung function *in conjunction with* symptoms (such as coughing and pain with deep inhalation) should be considered adverse. The EPA should consider both FEV_1 and symptoms when judging effects on lung function, but in recent reviews has only used FEV_1.

- Based on computer-generated models, the EPA estimates that < 20% of children in urban areas will be exposed at least once per year to 60 ppb ozone for 8 hours, and few if any will be exposed to at least one 8 hr period of 70 or 80 ppb ozone (EPA ozone Health Risk and Exposure Assessment). This is true even if the current 75 ppb ozone standard is maintained:

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Figure 1. *Percent of children in Houston who will be exposed at least once per year to 60 (red line), 70 (green line) or 80 (blue line) ppb ozone for 8 hours while exercising. This was modeled using the data from 2006 – 2010, assuming that the city met the current ozone standard (75 ppb) or one of the alternate standards (70, 65 or 60 ppb).*

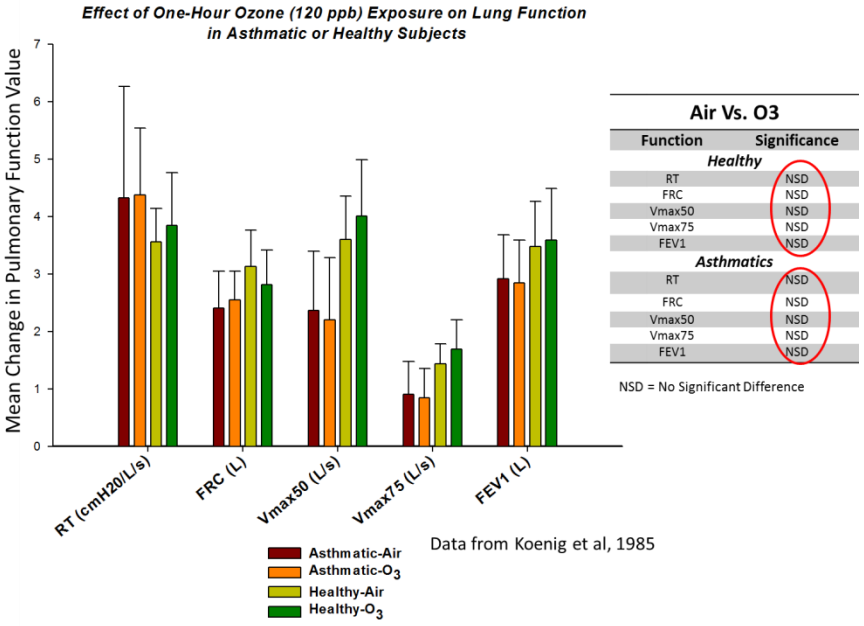
[1] The standard will take the form of the annual fourth-highest daily maximum 8-hour concentration, averaged over 3 years.

- Therefore, even if the ozone standard is not changed, few if any children will be exposed to 80 ppb ozone for 8 hours (a dose that shows some adverse clinical effect). A few children will be exposed to 70 ppb ozone for 8 hours (there is debatable evidence for clinically adverse effects at this dose), and some children could be exposed to 60 ppb for 8 hours, but (as stated above) there is no concrete evidence that this dose causes any adverse effects on lung function.

Asthma:

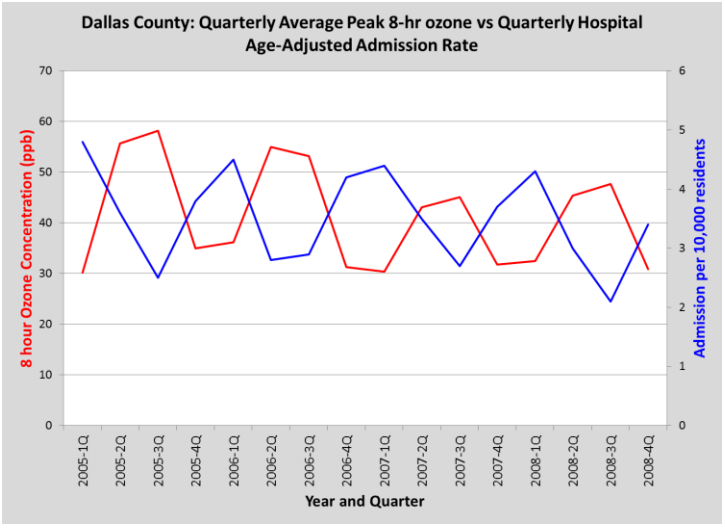
- A one hour exposure to 120 ppb ozone does not cause any significant respiratory effects in healthy or asthmatic adolescents (Koenig et al 1985). Other studies have found similar results (Holz et al 1999, Chen et al 2004). **This means that asthmatics are not necessarily more sensitive to ozone than non-asthmatics.**

***Figure 2**. Changes in different pulmonary values after healthy or asthmatic subjects were exposed to 120 ppb ozone for one hour. The figure to the right shows that there is no statistically significant difference between people exposed to filtered air or to ozone, or between healthy and asthmatic subjects.*



- There is mixed evidence that high ozone days increase the number of hospitalizations for asthma, and several multi-city studies show no relationship: Schildcrout et al 2006 and O'Connor et al 2008. And in fact, analysis in Texas shows that there is an increase in asthma hospitalizations (blue line) in the winter, when ambient ozone concentrations (red line) are low:

***Figure 3**. Plot of time in yearly quarters from 2005 – 2008, against 8 hour ozone (left y-axis, red line) and hospital admissions per 10,000 residents (right y-axis, blue line). It shows that in quarters where mortality is high (during the winter), ozone is low.*

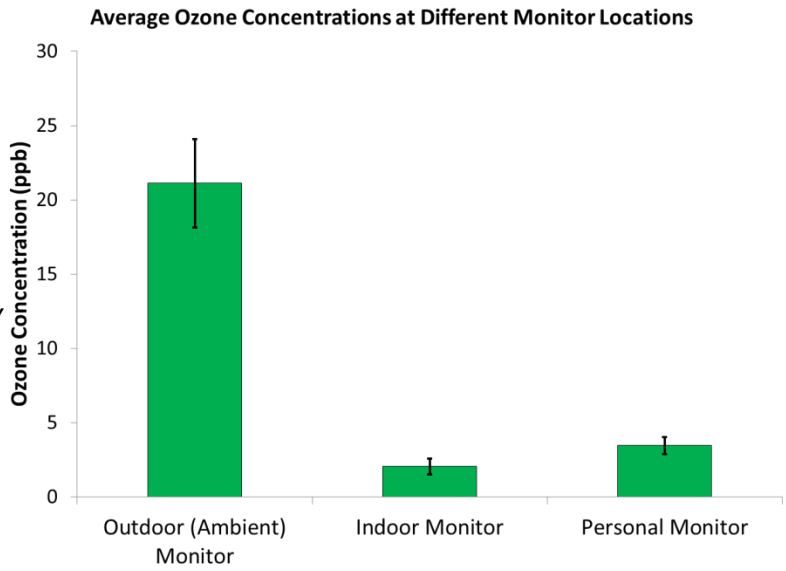


- Over the last ten years, the incidence of asthma has increased, whereas the ambient concentrations of ozone have decreased. If asthma incidence was associated with ozone concentrations, then the incidence should be going down, not up.
- Altogether, there is very little evidence that people with asthma are more sensitive to ozone. However, there are many other known triggers for asthma, including cold dry air, allergens, tobacco smoke, dust mites and mold. The Centers for Disease Control have information about these triggers: http://www.cdc.gov/asthma/triggers.html.

Personal Exposure:

- Personal exposure to ozone (the amount of ozone that a person actually breathes) is much lower than the ozone concentrations measured at monitors, because people spend most of their time indoors, and indoor ozone concentrations are very low. This has been shown by a number of studies, including Lee et al 2012, who measured the levels of outdoor, indoor and personal ozone concentrations for a group of children in Tennessee:

Figure 4. *Concentrations of ozone in Tennessee measured at an outdoor ambient monitor, measured using an indoor monitor, or measured using a personal monitor being worn by the study subjects.*



- Several national studies have shown that actual personal exposure is much lower than the concentrations of ozone that the EPA is considering for a new, lower standard (Meng et al 2012). This is also true for outdoor workers. For example, a study by O'Neill et al 2003 reported that outdoor workers in Mexico City experienced average personal ozone exposures that were 60% lower than ambient monitor levels. In addition, there is a protective ozone standard already in place for outdoor workers in the United States.

- Epidemiological studies that connect ozone and mortality assume that people are exposed to outdoor levels of ozone all the time. If personal exposure were used instead, all of the mortality would occur at levels of ozone that are well below background. Therefore, the mortality seen in these studies is likely attributable to another cause, or to natural random variation in daily mortality rates.

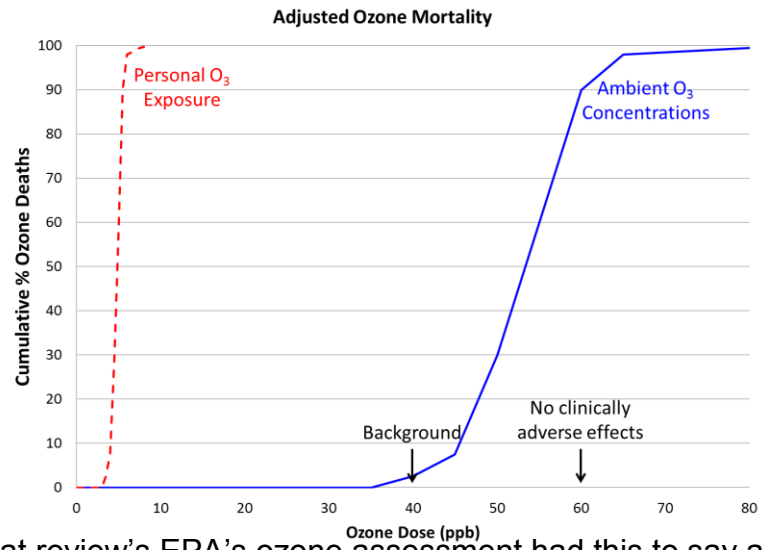


***Figure 5**. Concentration-Response curve for short-term mortality of ozone based on ambient monitoring data (blue line), or personal exposure data (red line). Exposure to 60 ppb ozone does not cause adverse respiratory effects and 40 ppb ozone is considered to be background.*

- The scientific advisory committee that review's EPA's ozone assessment had this to say about mortality and personal exposure:

  "The Ozone Staff Paper should consider the problem of exposure measurement error in ozone mortality time-series studies. It is known that personal exposure to ozone is not reflected adequately, and sometimes not at all, by ozone concentrations measured at central monitoring sites….Therefore, it seems unlikely that the observed associations between short-term ozone concentrations and daily mortality are due solely to ozone itself." CASAC ozone review panel – June 5, 2006

Mortality:

- The relationship between long-term ozone exposure and mortality has been investigated in at least 12 epidemiology studies. When considering other potential causes of mortality, such as other air pollutants, ***only one*** of those studies showed a statistically significant (but very small) effect of ozone on mortality.

Table 1: Studies examining the relationship between long-term ozone exposure and mortality, while considering other air pollutants

Statistically Significant Effect	NO Statistically Significant Effect
Jerrett et al 2009	Dockery et al 1993
	Abbey et al 1999
	Lipfert et al 2000
	Pope et al 2000
	Chen et al 2005
	Jerrett et al 2005
	Lipfert et al 2006a
	Lipfert et al 2006b
	Krewski et al 2009
	Smith et al 2009
	Wang et al 2009

- Different cities have different associations between short-term exposure to ozone and mortality, and very few of those associations are positive. This has been shown by many studies (Smith et al 2009, Bell et al 2004, Bell et al 2005, Zanobetti & Schwartz 2008). Of those cities that do show an association with mortality, there is no correlation between a positive association of ozone with mortality, and the ambient concentrations of ozone in that city:

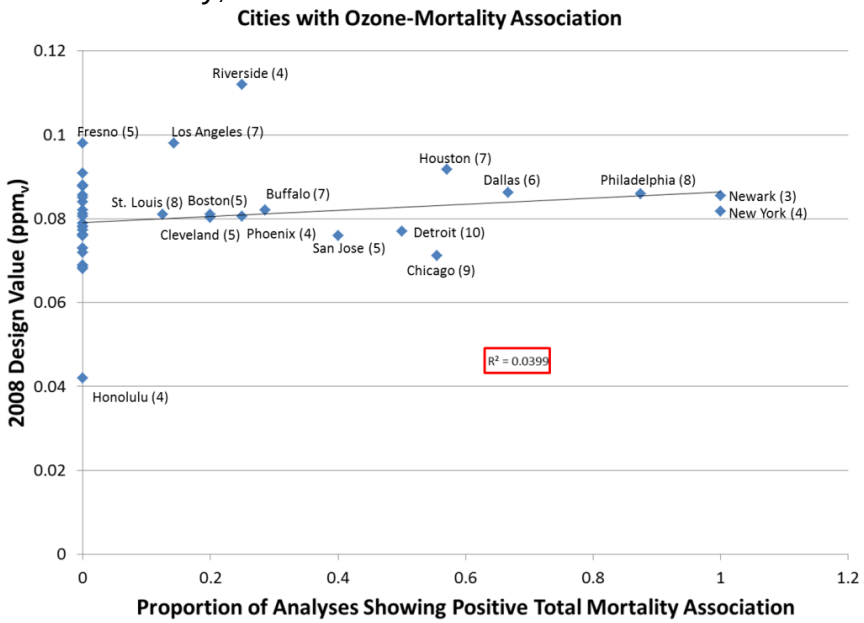


Figure 6. *Graph comparing the proportion of studies that have shown a positive association between ozone and mortality for a particular city (number of studies shown in parentheses next to the city name), compared to the 2008 ambient concentrations of ozone in those cities.*

- Even when a positive association is observed between short-term mortality and ozone concentration, that association is very small when considering other factors that affect mortality, such as socioeconomic status, temperature, time of year, and even napping:

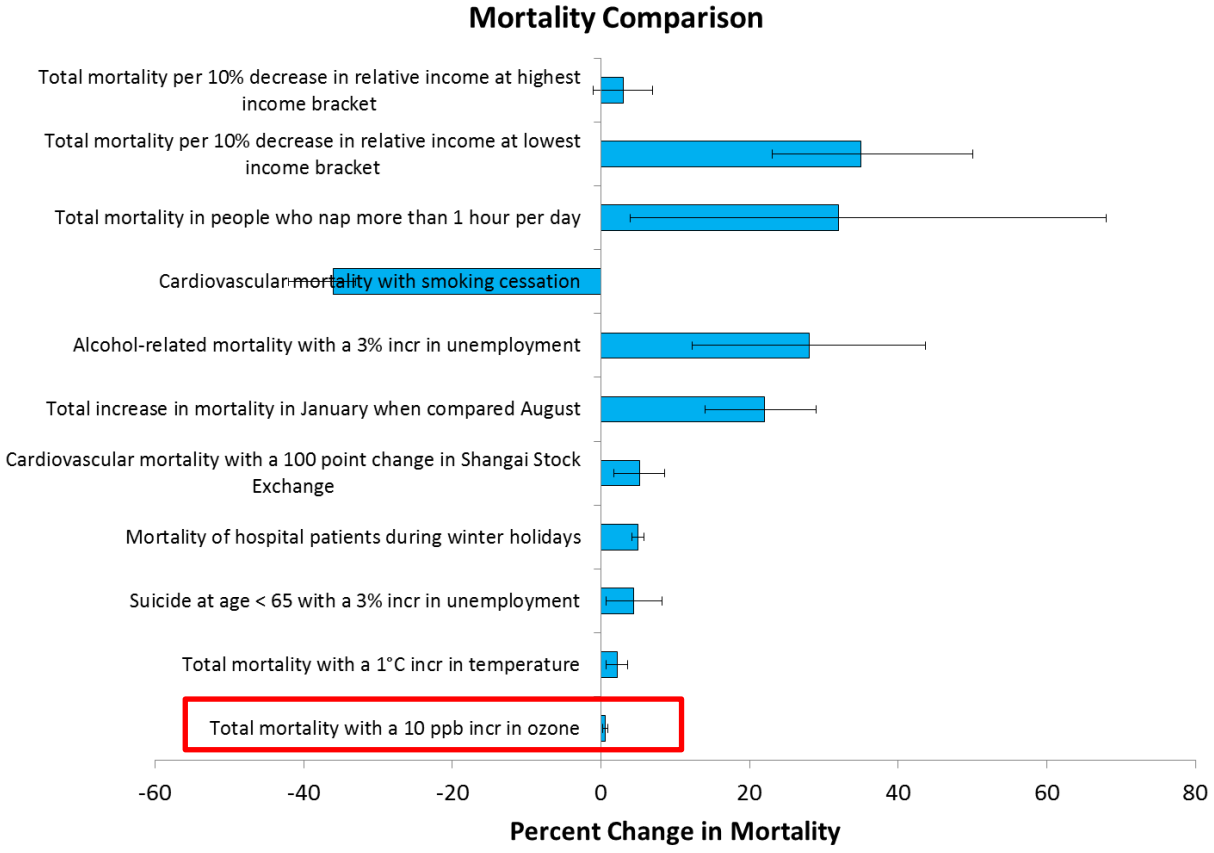


Figure 7: *Graph comparing the percent change in mortality caused by different stimuli. The reference table for this figure is at the end of this document.*

- Based on the EPA's analysis, lowering the ozone standard would ***increase*** overall mortality in certain U.S. cities, including Houston. These numbers were not presented in the main text, but could be found in Appendix 7 of the EPA Ozone HREA (see Final HREA, Appendices 7-9). This result is not discussed in the executive summary for the EPA Ozone Policy Assessment, although it was briefly mentioned on page 3-115 of that document, as well as on pages 7-69 – 7-70 of the HREA. The idea that mortality increases with decreasing ozone doesn't make logical sense, and shows that the EPA models and assumptions are flawed.

Table 2: Number of Premature Mortalities Predicted by EPA to Occur in Houston (2009 simulation year, mortality per 100,000 people)		
	Presented by EPA in Chapter 7	Based on Full Analysis found in Appendix 7 going from 2009 ozone levels to standard level
Meeting Current Standard (75 ppb) from Present Day Ozone Levels	*Not presented*	47 more deaths
Going from 75 ppb to 70 ppb	1 more death	48 more deaths
Going from 75 ppb to 65 ppb	3 fewer deaths	44 more deaths
Going from 75 ppb to 60 ppb	12 fewer deaths	35 more deaths

- Because mortality has little connection to ozone concentration (and doesn't take into account personal exposure), it should not be the basis of a new, lower national standard for ozone.

Difficulties with Implementing the Ozone Standard:

- Background ozone is ozone produced naturally, or transported from other countries. These background ozone levels can be greater than 40 ppb, and can contribute >80% of the measured ozone in an area (from the EPA Ozone Policy Assessment).

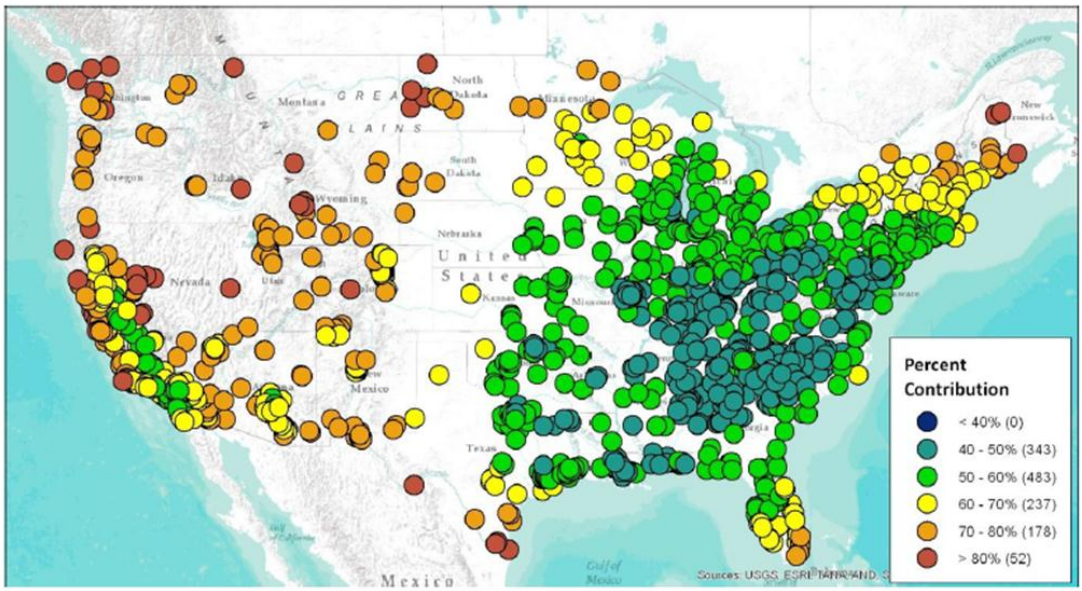


Figure 2-12. Map of apportionment-based U.S. background percent contribution to seasonal mean O_3 based on 2007 CAMx source apportionment modeling.

Figure 8. *Map of the contribution of background ozone to total ozone across the United States (based on 2007 data and modeling).*

- When making judgments about the risks of ozone and the benefits of reducing ozone, the EPA used calculations that unrealistically assume that ozone could be reduced to 0 ppb. Instead, they should use background ozone levels as their baseline, because it is not possible to control or regulate background ozone levels.

- There is a large variation in background ozone levels in different areas of the U.S. (Figure 8 above, from the EPA ozone policy assessment). Therefore, it makes more sense to set an ozone standard that is different for different regions, and not a single national standard that doesn't consider background ozone levels.

- The chemistry of ozone is complex. Ozone is not produced directly, but instead is made when nitrogen oxides (NO_x) and volatile organic compounds (VOCs) react with sunlight. However, NO_x can also chemically remove ozone, and so ozone levels actually decrease the closer you get to a road (vehicles are a major producer of NO_x).

- Because ozone chemistry is so complicated, decreasing NO_x can actually increase the amount of ozone in areas that are close to where the NO_x is being produced (such as in the inner cities). Conversely, areas that are far from major NO_x sources (such as the suburbs) will experience a decrease in ozone when NO_x decreases. This means that a lower ozone standard can lead to disproportionate benefits for those who live outside the cities, compared to those in the inner cities (Figure 9).

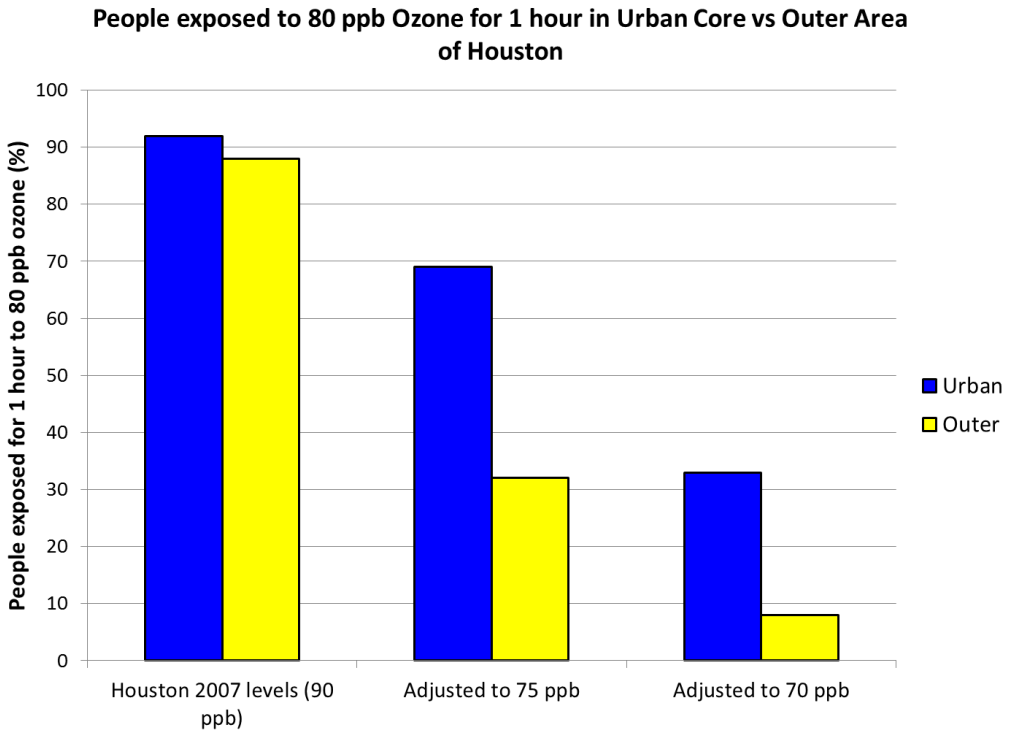


Figure 9. *Ozone levels are proportionally higher in urban compared to outlying areas, and this increases with a decreasing ozone standard. Based on Ozone HREA (Appendix 9).*

- It will be very difficult to implement a new lower ozone standard, particularly if it is set at 60 ppb. The EPA itself can only predict 1/3 of the emissions decreases, meaning that 2/3 of the pollutant decreases will have to be attained by as-yet-unknown technology. Because of this, achieving this decrease in ozone could be very expensive, and in fact is anticipated to cost $270 billion of gross-domestic product nationally, *per year*. There would also be increases in electricity costs, and an effective loss of >$1000 in household income per year in Texas.

References for Mortality Comparison Graph

Statistic	Reference (s)	Pubmed ID
Total mortality per 10% decrease in relative income at highest income bracket	Elstad et al 2006. Eur J Public Health. Associations between relative income and mortality in Norway: a register-based study	16476681
Total mortality per 10% decrease in relative income at lowest income bracket	Elstad et al. 2006	16476681
Total mortality in people who nap more than 1 hour per day	Leng et al 2014. Am J Epidemiol. Daytime napping and the risk of all-cause and cause-specific mortality: a 13-year follow-up of a British population	24685532
Cardiovascular mortality with smoking cessation	Critchley & Capewell 2003. JAMA. Mortality risk reduction associating with smoking cessation in patients with coronary heart disease: a systematic review	12837716
Alcohol-related mortality with a 3% incr in unemployment	Stuckler et al 2009. Lancet. The public health effect of economic crises and alternative policy response in Europe: an empirical analysis	19589588
Total increase in mortality in January when compared August	Van Rossum et al 2001 Int J Epidemiol. Seasonal variation in cause-specific mortality: are there high-risk groups? 25-year follow-up of civil servants from the first whitehall study	11689530
Cardiovascular mortality with a 100 point change in Shangai Stock Exchange	Ma et al. 2011 Eur Heart J. Stock volatility as a risk factor for coronary heart disease	21196446
Mortality of hospital patients during winter holidays	Phillips et al 2004. Circulation. Cardiac mortality is higher around Christmas and New Year's than at any other time: the holidays as a risk factor for death	15596560
Suicide at age < 65 with a 3% incr in unemployment	Stuckler et al 2009	19589588
Total mortality with a 1°C incr in temperature	Martiello & Giacchi 2010. Scand J Public Health. High temperatures and health outcomes: a review of the literature	20688791
Total mortality with a 10 ppb incr in ozone	All year studies referenced in Table 6-27 (page 6-222) from the EPA Ozone 2013 Integrated Science Assessment	http://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=247492