Bryan Shaw, Ph.D.

Commissioner

Texas Commission on Environmental Quality

EPA is currently considering lowering the existing national eight-hour ozone standard from its current level of 75 parts per billion (ppb) to a much lower range, between 70 and 60 ppb. However, after an in-depth review of the EPA's analysis, as well as a thorough study of the relevant scientific literature, the TCEQ has concluded that there will be little to no public health benefit from lowering the current standard. Surprisingly, the EPA's own modeling in twelve cities across the country indicates the net result will be *increased* mortality in some areas, including Houston and Los Angeles. The EPA did not do the analysis for other cities in Texas.

There is no doubt that, at some higher level, ground level ozone is harmful to human health. The question is, has the EPA adequately demonstrated that lowering the ozone standard to 70 to 60 ppb would actually have health benefits. We think that EPA's process of setting ozone standards has not scientifically proven this, and that further lowering of the ozone standard will fail to provide any measurable increase in human health protection.

The EPA's own modeling in their Health Risk and Exposure Assessment (HREA) indicates that lowering ozone concentrations would actually result in more deaths in Houston (Appendix 7, page 7B-2 of the HREA). Either this indicates that lowering ozone standards defeats its stated purpose of protecting human health, or it indicates that something is wrong with the EPA's methodology. Either way, it's not a good argument for lowering ozone standards. Further, EPA is not very forthcoming about the increased deaths. It's not mentioned in the executive summary of their policy analysis, but it's found on page 115 of Chapter 3, more than one third of the way through the 597 page document.

The EPA's proposed lower ozone standard derives much of its claimed benefits from associating ozone with worsening asthma. The problem with this association is that asthma diagnoses are increasing in the U.S., yet nationwide, air quality is improving. If asthma were actually tied to ozone, you would expect to see the instances of asthma decreasing, not increasing. In fact, data from Texas hospitals show that asthma admissions are actually highest in the winter, when ozone levels are the lowest.

Below are a few simplified facts and explanations of the TCEQ's conclusion that a lower ozone standard is not justified.

Sensitivity of asthmatics to ozone:

- Exposure of human volunteers to ozone showed similar lung effects in asthmatics as in non-asthmatic subjects.

Mortality caused by long-term exposure to ozone:

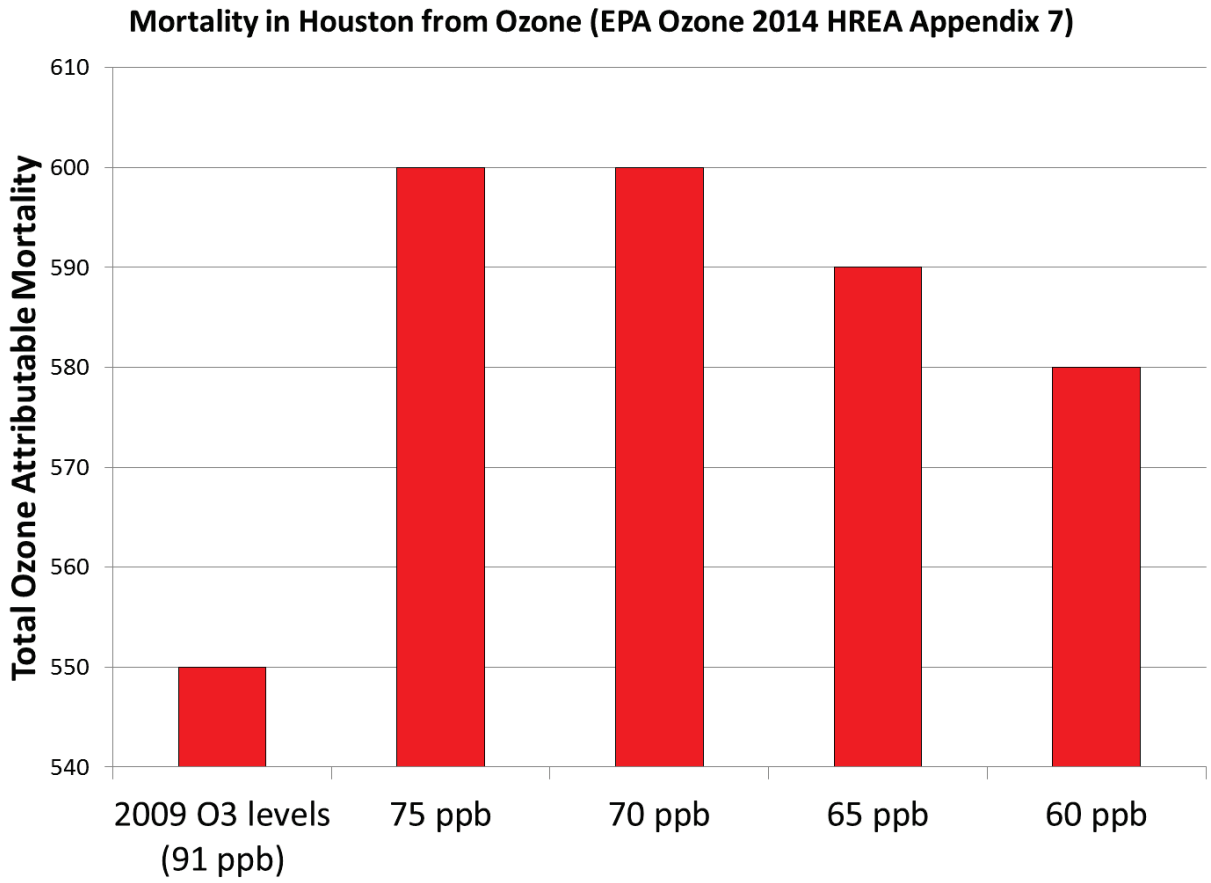
- Only 1 out of 12 studies showed an association between long-term exposure to ozone and early death (after considering other pollutants). This single study is used by the EPA as evidence that long-term exposure to ozone causes mortality. Interestingly, this study did not show higher mortality in Southern California, where some of the highest ozone levels in the country are measured.
- Laboratory animals have been used for decades to assess the health effects of pharmaceuticals, cosmetics, household chemicals, and environmental contaminants. Despite many years of exposing laboratory animals to high doses of ozone for long periods of time, none die from ozone exposure. Therefore, does ozone actually kill people?

Ambient ozone concentrations don't represent real-world conditions:

- Ozone is an outdoor air pollutant, because systems such as air conditioning remove it from indoor air. Since most people spend more than 90% of their time indoors, we (and the people in the epidemiology studies used to justify lowering the standard) are rarely exposed to significant levels of ozone.
- Ozone concentrations under shade trees are lower than concentrations in direct sunlight, where ozone monitors are located.
- For ozone to cause a slight change in lung function in clinical studies, people need to be exposed to outdoor levels of ozone for hours while vigorously exercising (e.g. 6 hours of bicycling). These changes in lung function are often so small that they are within a person's *normal* daily variation.
- Epidemiology studies show an association between a person's likelihood of dying, and the outdoor concentrations of ozone in the days before (or the day of) a person's death. However, we spend most of our time indoors (particularly people who are near death), so we are exposed to levels of ozone that are far below those that cause any clinical effect.

Problems with implementing a new ozone standard:

- Some places in the US have background levels of ozone that account for up to 80% of total ozone. Background ozone occurs naturally, or is transported from other countries. The EPA does not take this into account when making the rule.
- Ozone is not produced directly, but instead is made when other chemicals (particularly nitric oxides) react with sunlight. However, ozone chemistry is complicated, and the same nitric oxides that produce ozone can also react with ozone to remove it from the air. So places that have high nitric oxide production (such as near roads), often have lower ozone levels.
- Because ozone chemistry is complex, lowering ozone-producing chemicals in the cities would decrease ozone in the suburbs, but could increase ozone in the inner-cities (because there will be a decrease in the nitric oxides that can remove ozone). This means that, according to EPA predictions, those living in the inner cities could bear more health burdens, while people on the outskirts enjoy the predicted benefits.


Mortality in Houston from Ozone (EPA Ozone 2014 HREA Appendix 7)
Total Ozone Attributable Mortality
610
600
590
580
570
560
550
540
2009 O3 levels (91 ppb)
75 ppb
70 ppb
65 ppb
60 ppb

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:

EPA Ozone HREA 2nd Draft, pg 5-30

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).*

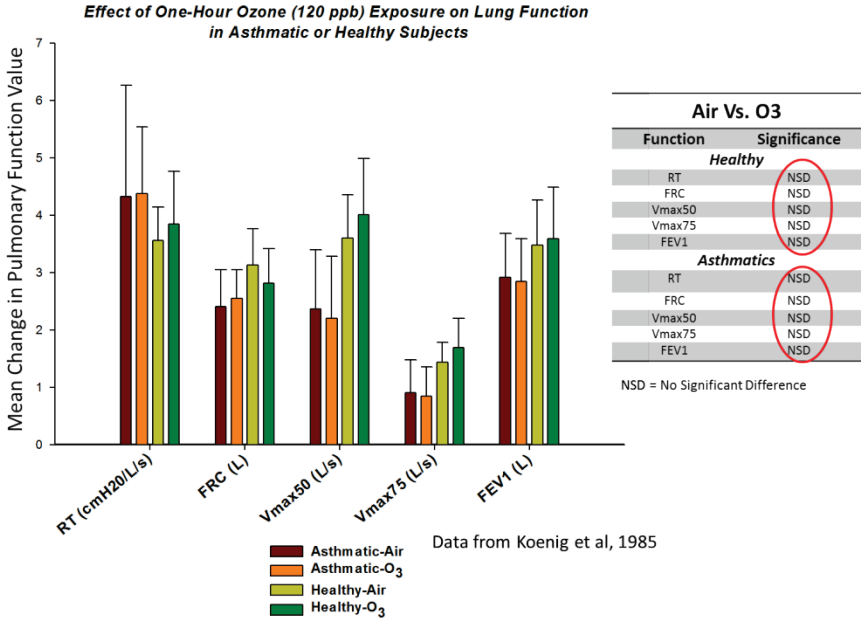
[1] The standard will take the form of the annual fourth-highest daily maximum 8-hour concentration, averaged over 3 years.

- 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:

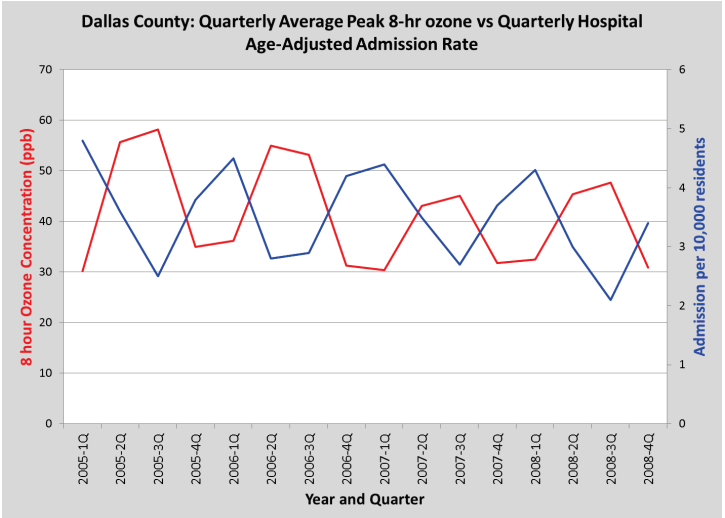
- A one hour exposure to 120 ppb ozone does not cause any significant respiratory effects in healthy or asthmatic adolescents (Koenig et al 1985). Other studies have found similar results (Holz et al 1999, Chen et al 2004). **This means that asthmatics are not necessarily more sensitive to ozone than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

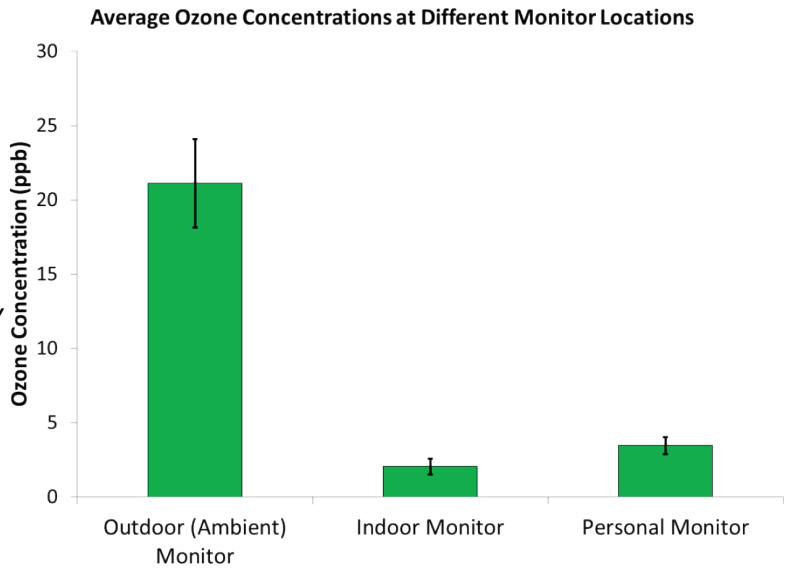


- Over the last ten years, the incidence of asthma has increased, whereas the ambient concentrations of ozone have decreased. If asthma incidence was associated with ozone concentrations, then the incidence should be going down, not up.
- Altogether, there is very little evidence that people with asthma 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:

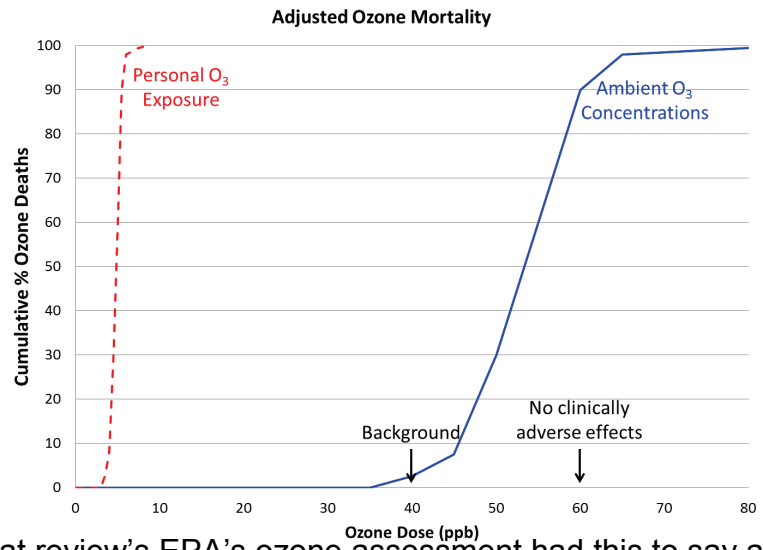
Figure 4. *Concentrations of ozone in Tennessee measured at an outdoor ambient monitor, measured using an indoor monitor, or measured using a personal monitor being worn by the study subjects.*



- 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:

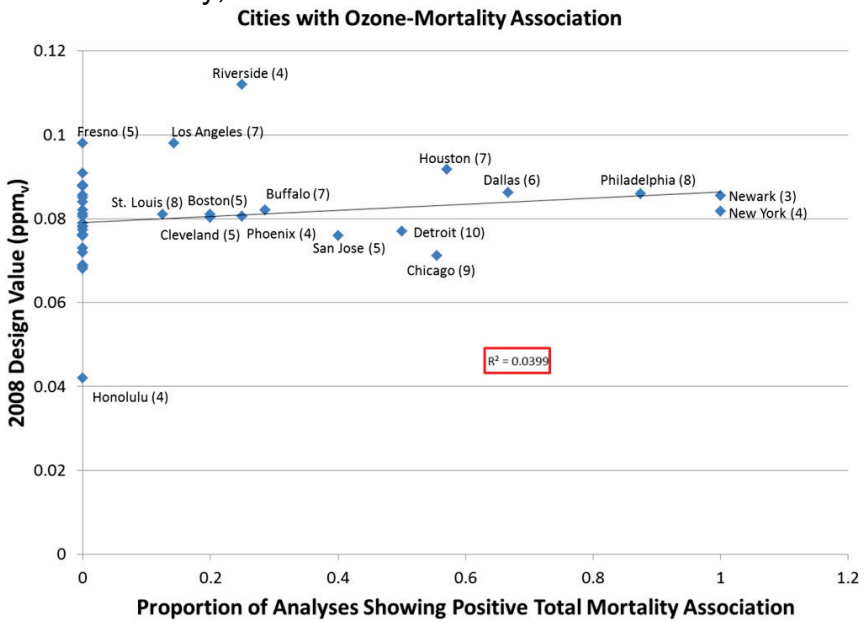


Figure 6. *Graph comparing the proportion of studies that have shown a positive association between ozone and mortality for a particular city (number of studies shown in parentheses next to the city name), compared to the 2008 ambient concentrations of ozone in those cities.*

- 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:

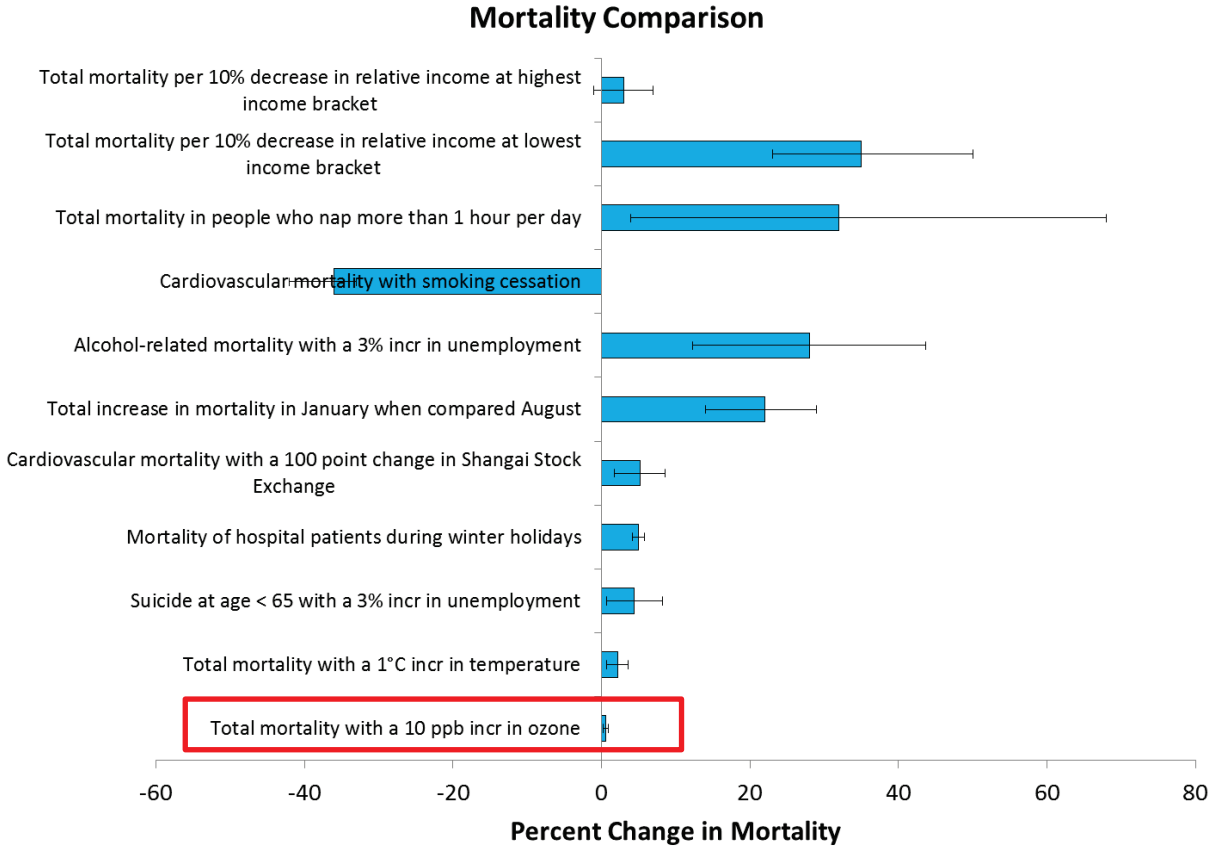


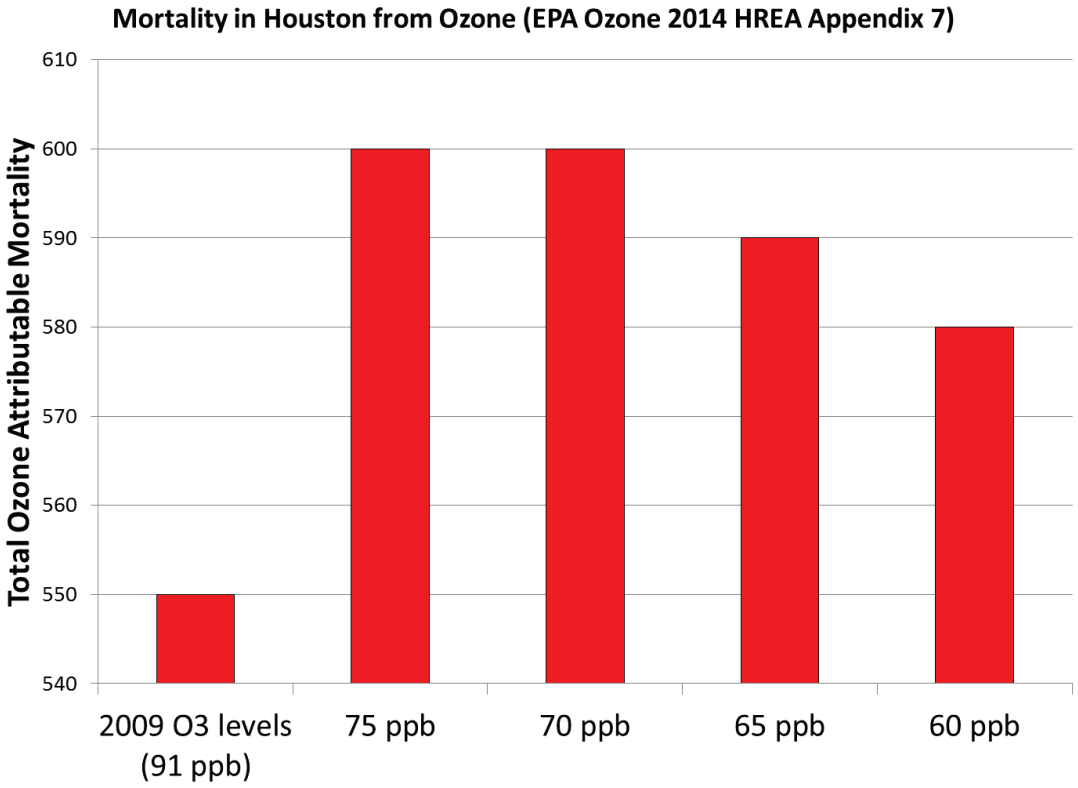
Figure 7: *Graph comparing the percent change in mortality caused by different stimuli. The reference table for this figure is at the end of this document.*

- 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

- Here is a graphical representation of the above table:



- 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).

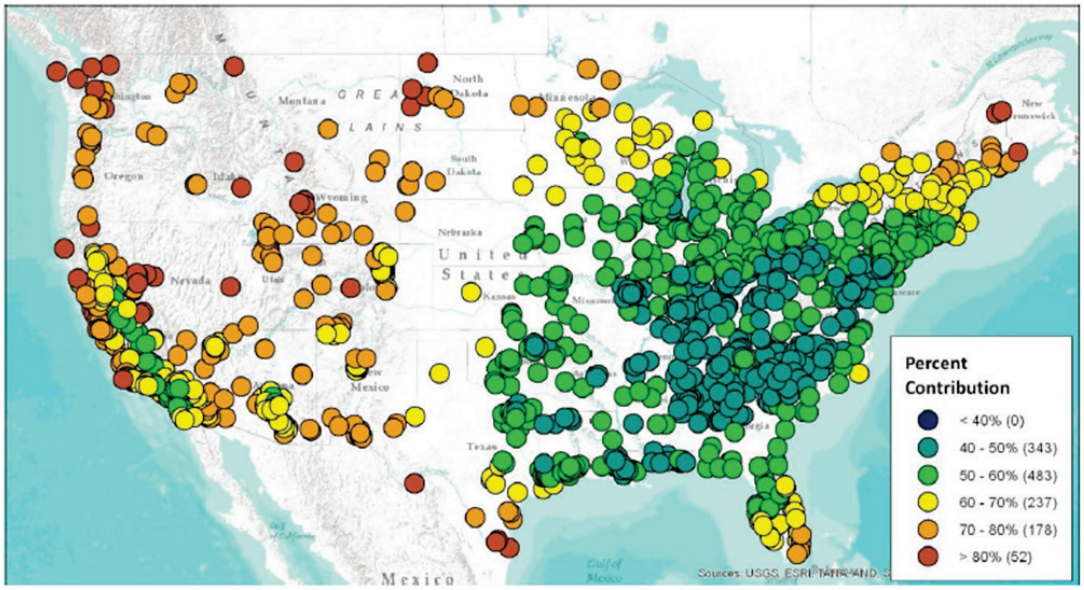


Figure 2-12. Map of apportionment-based U.S. background percent contribution to seasonal mean O_3 based on 2007 CAMx source apportionment modeling.

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).

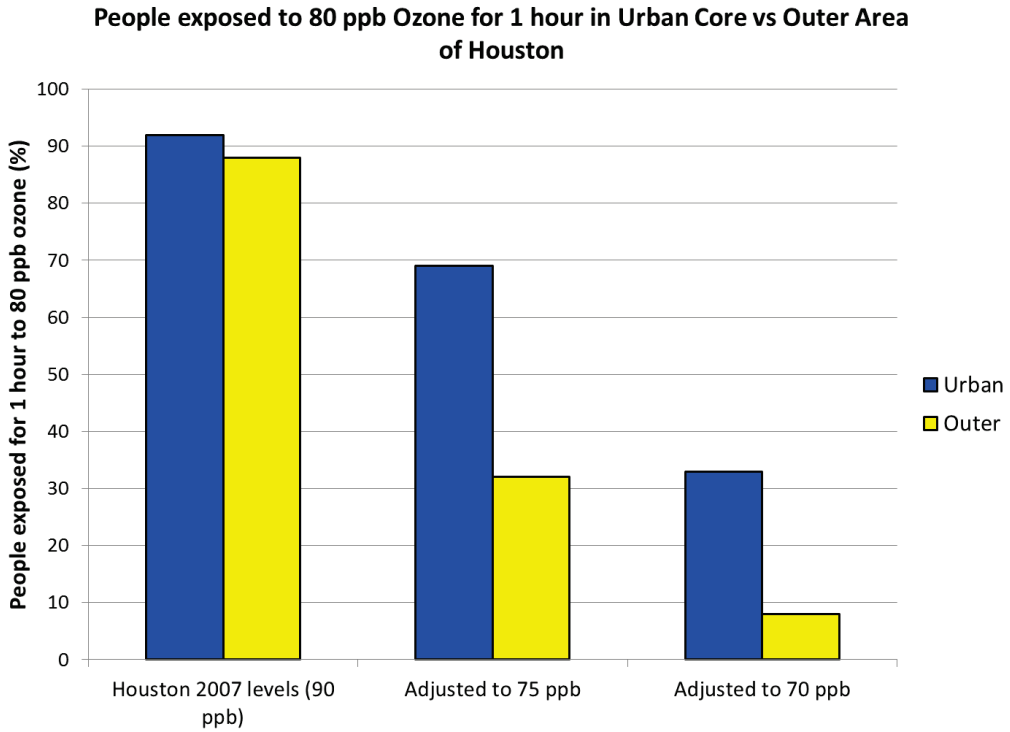


Figure 9. *Ozone levels are proportionally higher in urban compared to outlying areas, and this increases with a decreasing ozone standard. Based on Ozone HREA (Appendix 9).*

- 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

How dose response curves derived from clinical ozone exposures can inform public policy

S.S. Lange[1], L.R. Rhomberg[2], M. Dourson[3], G .Tao[2], J.E. Goodman[2], and M. Honeycutt[1]

[1]Toxicology Division, Texas Commission on Environmental Quality, Austin, TX; [2]Gradient, Cambridge, MA; [3]Toxicology Excellence for Risk Assessment, Cincinnati, OH

Abstract

Ozone is one of the 6 criteria air pollutants whose levels are set by the US EPA through the National Ambient Air Quality Standards. Data from animal, human clinical and epidemiology studies are used to decide at which level to set the standard. The purpose of our work is to use data from human clinical studies to inform policy decisions about a protective ambient ozone level. Many studies have been conducted and can be applied to generate ozone dose-response curves, using ozone total inhaled dose (which is calculated from ozone concentration, duration of exposure and ventilation rate) and forced expiratory volume (FEV_1) decrements. Outside of modeling done by the EPA, these dose response curves have not been utilized as tools to inform the choice of a protective ambient ozone concentration. In this work we plotted mean FEV_1 response versus total inhaled ozone dose from clinical studies of varying durations (1 – 8 hours). Mode of action (MOA) information was incorporated as appropriate. The initial plot used data from healthy young adults, and additional analyses incorporated data from children and asthmatics to determine whether they differed from the healthy adult curve. The trend line from this data was employed to make tables demonstrating the ozone concentrations required to produce a given FEV_1 decrement at different exposure times and ventilation rates (i.e. exercise levels). We also plotted ozone doses at which other relevant clinical effects occur (e.g. inflammation) although the variability in technique and lack of consistent quantification makes these difficult to model in a similar way as FEV_1. We think that this type of analysis is crucial for deciding on a protective ambient ozone concentration, because differing levels have significant societal and economic implications. Clinical data provides quantifiable and confident endpoints that can be justifiably used for well-reasoned and scientifically credible rule making.

Introduction

- Ozone (O_3) is one of 6 air pollutants regulated by the National Ambient Air Quality Standards (NAAQS).
- The level of the O_3 NAAQS is currently 75 ppb, and the EPA is proposing to lower it into the range of 65 – 70 ppb.
- In clinical studies volunteers were exposed to O_3 at different concentrations and ventilation rates (ie. exercise levels), for different times; these studies measure respiratory endpoints.
- Other groups have used this data to make ozone dose-response curves, using ozone total inhaled dose and decrements in forced expiratory volume in 1 second (FEV_1)[18].
- Outside of the EPA, dose response curves have not been used as a tool to inform the choice of a protective ambient ozone concentration.
- This is important because choosing a protective ambient ozone concentration has societal and economic implications, and clinical data can provide quantifiable endpoints that can be used for rule making.

Ozone Mode of Action

- O_3 is an oxidant which can be scavenged by antioxidants (such as uric acid, glutathione and ascorbic acid) in the extracellular lining fluid of the respiratory tract.
- O_3 in the nasal cavity activates bronchial C-fibers, which initiates a neural reaction, leading to spirometric responses (e.g. FEV_1 decrements).
- O_3 initiates inflammation in all areas of the respiratory tract, measured by influx of neutrophils; this is considered more detrimental than spirometric responses.
- O_3 can impair epithelial barrier function of the respiratory epithelia.
- O_3 increases airway hyper-responsiveness to bronchoconstrictive stimuli, and this may be worse in those with compromised airways.
- None of these effects are correlated with spirometric responses – that is, people with heightened spirometric responses do not necessarily show increased inflammation, loss of epithelial barrier function or airway hyper-responsiveness.
- The ozone mode of action is thoroughly reviewed in the most recent EPA ozone Integrated Science Assessment (2013)[22]

Methods

- Ozone concentration (in ppm), time of exposure (in min) and ventilation rates (in L/min) were extracted from 11 publications[1-5,8,10,13,14,19,20]. These were multiplied to produce **total inhaled dose** (in ppm×L). The associated **mean change in FEV_1** (in % change from baseline) for the group of study subjects was also used.
- The main curve was made using data derived from healthy young adults. We also plotted data from 3 additional studies using mild asthmatics as volunteers[8,11,12], one study using children aged 8-11 as volunteers[15], and one study that exposed elite athletes at very high exercise levels in a hot environment[7].
- Non-linear dose-response curves were fit to the short exposure (≤ 3 hours) and long exposure (> 6 hour) data, using the following sigmoid response model:

$$\%\Delta FEV_1 = \delta + \frac{\alpha - \delta}{1 + \exp\{\beta \ln(\text{Total dose} / \lambda_{50})\}}$$

Where $\%\Delta FEV_1$ is the percent change in FEV_1 after the ozone exposure compared to the pre ozone exposure, "Total dose" is the total ozone dose defined as ventilation (L/min) × time (min) × ozone (ppm), δ is the top plateau of FEV_1 decrements at minimal dose, α is the bottom plateau of FEV_1 decrements at high dose, β is the slope parameter that defines the steepness of the curve, λ_{50} is the dose at which the response is halved, and δ, α, β and λ_{50} are parameters of the model and can be estimated from observed data.

- Ozone concentration matrix:
  - Exposures ≤ 4 hours: Using the short exposure time curve, the doses at which the mean curve crossed -10% FEV_1 were taken, and then the ozone concentrations were calculated based on the different exposure times and ventilation rates.
  - Exposures > 4 hours: Using the longer exposure time curve, the doses at which the mean curve crossed -10% FEV_1 were taken, and then the ozone concentrations were calculated based on the different exposure times and ventilation rates.

Results

Dose Response Curve from O_3 Clinical Data

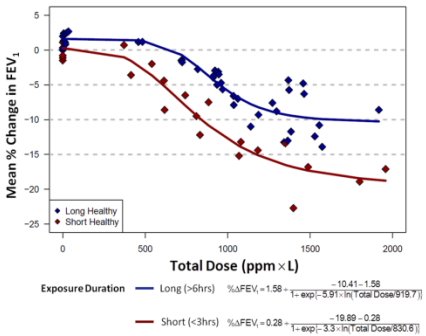


Figure 1: Plot of total inhaled dose (in ppm×L) versus percent change in mean FEV_1; data is derived from mean FEV_1 change of healthy young adults exposed for ≤3 hours (short) or > 6 hours (long) to ozone while exercising. Below the plot are the equations associated with each curve.

Dose Response Curve Characteristics

- Sigmoid-shaped curve:
  - FEV_1 barely changes at low doses (<500 ppm×L)
  - FEV_1 decreases as ozone dose increases and the decreasing rate increases (500-1000 ppm×L) and then decreases (1000-1500 ppm×L) at medium doses
  - the FEV_1 decrements reach a plateau at high total doses (>1500 ppm×L)
- Using regression analysis, there is a significant difference in response rate between long exposure experiments and short exposure experiments.

Dose Response Curve with Sensitive Populations

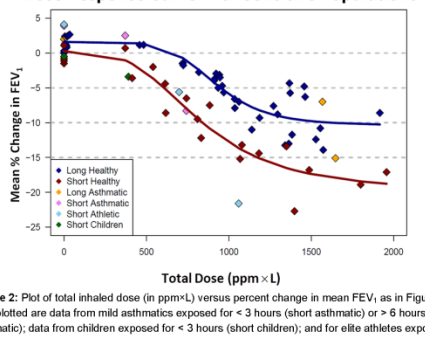


Figure 2: Plot of total inhaled dose (in ppm×L) versus percent change in mean FEV_1 as in Figure 1; also plotted are data from mild asthmatics exposed for < 3 hours (short asthmatic) or > 6 hours (long asthmatic); data from children exposed for < 3 hours (short children); and for elite athletes exposed for 1 hour (short athletic).

Inverse of the dose-response curve for identifying benchmark doses

$$\text{Total dose} = \lambda_{50} \times \exp\left\{\ln\left(\frac{\alpha - \%\Delta FEV_1}{\%\Delta FEV_1 - \delta}\right) / \beta\right\}$$

Table 1: Doses associated with mean changes in FEV_1, derived from long & short dose response curves

Mean % Change in FEV_1	Short exposure dose (ppm×L)	Long exposure dose (ppm×L)
0	228.2	668.5
- 5	606.7	950.7
- 10	**840.4**	**1618.6**
- 15	1173.1	N/A

Exercise Ventilation Rates

Table 2: Ventilation rates in L/min and m^3/day for different exercise levels

Source	Population	Exercise	Ventilation (L/min)	Ventilation (m^3/day)
EPA O_3 ISA 2013[22]	Children (6-11)	Sedentary	4.8	6.9
		Light Intensity	11	15.8
		Moderate Intensity	22	31.7
		High Intensity	42	60.5
	Young adult (21-31)	Sedentary	5.3	7.6
		Light Intensity	12	17.3
		Moderate Intensity	26	37.4
		High Intensity	50	72
Zuurbier 2003[23]	Adult	Commuting by bicycle	23.5	33.8
Samet 1993[18]	Child	Outdoor play	16	23
	Child	Bicycling	27	38.9
	Adult male	Bicycling	65	93.6
TCEQ Guidance 2012[21]	Adult worker	Occupational (8 hour day)	22	10
	General Population	Non-Occupational (24 hour day)	14	20

Table 3: Concentrations of O_3 at which a population would be expected to experience an FEV_1 decrement of 10%, given different exposure times and ventilation rates (V_E - ie. exercise levels)

FEV_1 Decrement =10%

			Ozone Concentration (ppb)									
			Time (hrs)									
Source	Population & Exercise	V_E (L/min)	1	2	3	4	5	6	7	8	12	24
EPA[22]	Sedentary Child	5	2917	1458	972	729	1124	937	803	703	468	234
EPA	Sedentary Adult	5	2642	1321	881	660	1018	849	727	636	424	212
EPA	Light Int Child	11	1273	636	424	318	491	409	350	307	204	102
EPA	Light Int Adult	12	1167	583	389	292	450	375	321	281	187	94
TCEQ[21]	General Pop (24 hr)	14	1000	500	333	250	385	321	275	241	161	80
Samet[18]	Child Outdoor Play	16	875	438	292	219	337	281	241	211	141	70
EPA	Med Int Child	22	636	318	212	159	245	204	175	153	102	51
TCEQ	Adult Worker (8 hr)	22	636	318	212	159	245	204	175	153	102	51
Zuurbier[23]	Adult Bicycle Commute	24	596	298	199	149	230	191	164	144	96	48
EPA	Med Int Adult	26	538	269	179	135	208	173	148	130	86	43
Samet	Child Bicycling	27	519	259	173	130	200	167	143	125	83	42
EPA	High Int Child	42	333	167	111	83	128	107	92	80	54	27
EPA	High Int Adult	50	280	140	93	70	108	90	77	67	45	22
Samet	Adult Male Bicycling	65	215	108	72	54	83	69	59	52	35	17

Note: The highlighted 8 hour time point is the averaging time of the O_3 NAAQS; grey numbers indicate times and ventilation rate combinations that are unlikely to occur.

Threshold Doses of Ozone

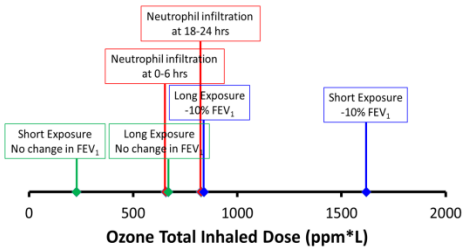


Figure 3: Threshold ozone doses after which one would expect to see various respiratory effects

Uncertainties in Ozone Dose-Response Data

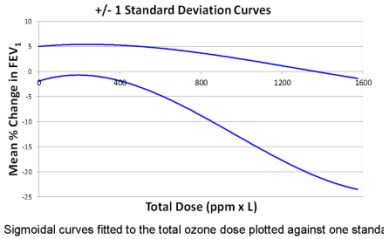


Figure 4: Sigmoidal curves fitted to the total ozone dose plotted against one standard deviation higher than the mean FEV_1 response and one standard deviation lower than the mean FEV_1 response.

Summary & Conclusions

- O_3 clinical exposure data can be used to derive dose-response curves.
- O_3 dose-response is dependent on the rapidity of exposure, consistent with a mechanism of antioxidant protection against ozone in the respiratory tract.
- Sensitive populations such as asthmatics and children show similar responses to O_3 as healthy young adults.
- These dose-response relationships can be used to create a tool to provide guidance as to how long populations can be exposed, at what exercise level and O_3 concentration, before experiencing a given FEV_1 decrement.
- This tool can be used by policy makers to help make an informed decision about setting the level of the O_3 standard, based on time activity data that will inform choices about exposure times and ventilation rates.

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Proposed 2015 Eight-Hour Ozone Standard

OVERVIEW

On November 25, 2014, the United States Environmental Protection Agency (EPA) proposed a more stringent National Ambient Air Quality Standard (NAAQS) for ground-level ozone. The EPA is proposing to revise both the primary ozone standard to protect public health, and the secondary standard to protect public welfare (e.g., crops and vegetation).

- Both standards are proposed to be eight-hour standards set within a range of 0.065 to 0.070 parts per million (ppm), which is often expressed as 65 to 70 parts per billion (ppb). The form of the standard will remain as the annual fourth highest daily maximum eight- hour average concentration, averaged over three years.
- The current standards were set in 2008 at a level of 0.075 ppm measured over eight-hours.
- The EPA is taking comment on a primary standard as low as 0.060 ppm as well as retaining the current standard.
- The EPA is taking comment on a secondary standard based on the weighted (W126) metric within a range of 13 to 17 ppm-hours averaged over three years and on defining a target protection level in terms of a W126 index value as low as 7 ppm-hours.
- In addition to the proposed revisions to the NAAQS, the EPA is proposing revisions to requirements for ambient air monitoring and permitting for ozone.
- The EPA estimates a cost of $3.9 billion for 70 ppb and $15 billion for 65 ppb by 2025. California is expected to have an attainment date and costs incurred after 2025 and an additional cost of $0.8 – $1.6 billion for 70 or 65 ppb, respectively.
- The EPA estimates benefits of $7.5-$15 billion for a 70 ppb standard and $21 - $42 billion for a 65 ppb standard (includes California benefits after 2025).

NOTE: After an in-depth review of the EPA's analysis, as well as a thorough study of the relevant scientific literature, the TCEQ has concluded that there will be little to no public health benefit from lowering the current standard. The EPA's own modeling in 12 cities across the country indicates the net result will be increased mortality in some areas, including Houston and Los Angeles.

The EPA's proposed lower ozone standard derives much of its claimed benefits from associating ozone with worsening asthma. However, asthma diagnoses are increasing in the U.S., yet nationwide, air quality is improving. Data from Texas hospitals show that asthma admissions are actually highest in the winter, when ozone levels are the lowest.

TIMELINE

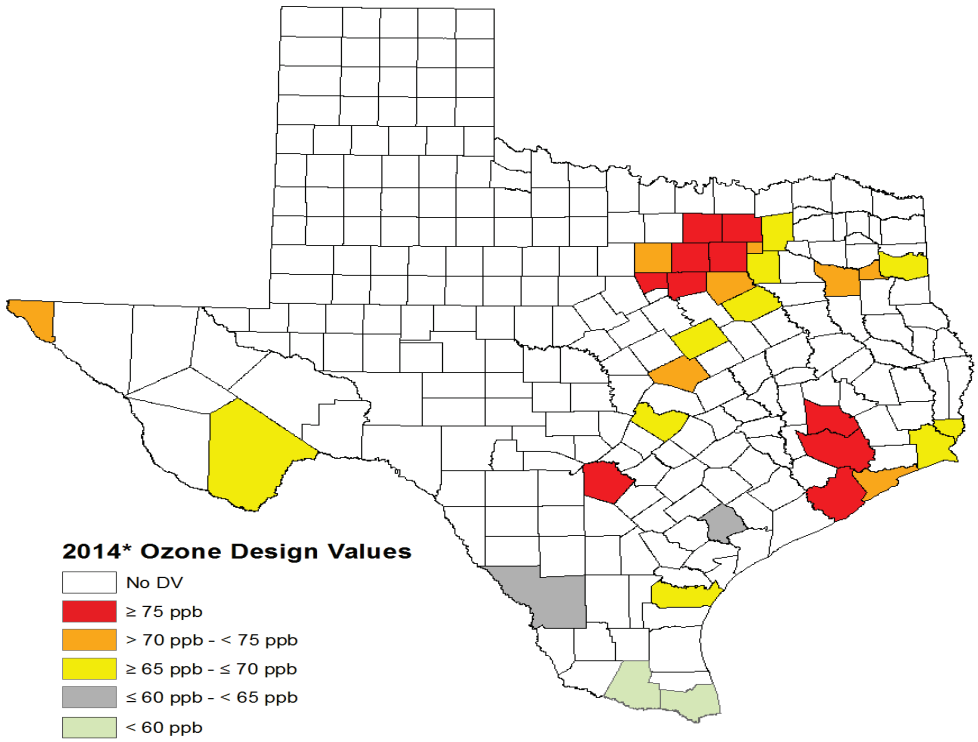
- **October 1, 2015** - The EPA is under consent decree to finalize the standard by this date.
- **October 1, 2016** – Upon finalization, states will have one year to submit designation recommendations to the EPA regarding the attainment status of all areas within the state. The state's recommendation will likely be based on air monitoring data from 2013, 2014, and 2015.
    - Nonattainment – the area does not meet or contributes to an area that does not meet the standard
    - Attainment – the area meets the standard

- Unclassifiable – there is not enough information to determine whether an area meets the standard

- **October 1, 2017** – The EPA has two years after the standard is promulgated to finalize designations based on recommendations made by the states. The EPA's final designations will likely consider air monitoring data from 2014, 2015, and 2016.
  - The Federal Clean Air Act (FCAA) requires the EPA to notify states 120 days prior to making final designations if the Administrator intends to finalize designations that differ from the state's recommendation.
- **2020** – States would be required to submit revisions to the State Implementation Plan (SIP) to demonstrate how the state will meet the revised standard to the EPA.
- **2020 to 2037** – Nonattainment area deadlines fall within this range depending on the area's classification.

IMPACTS TO TEXAS

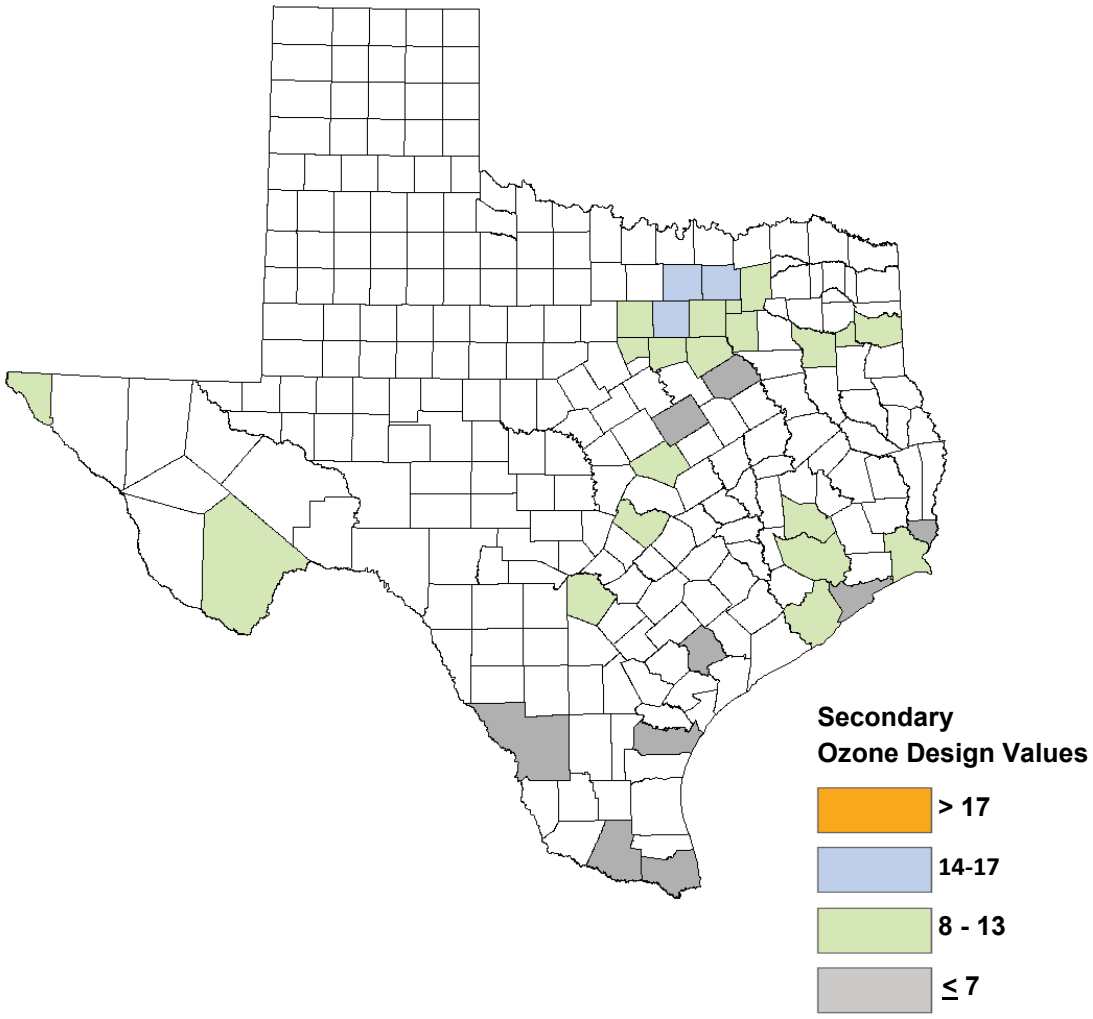
Under the current ozone standard, two areas are designated nonattainment: Dallas-Fort Worth and Houston-Galveston-Brazoria. If the EPA finalizes a standard below 0.075 ppm, several additional areas could be designated nonattainment. The map below shows where counties with ozone monitors stand with regard to the range of potential primary standards the EPA has proposed based on the most recent monitoring data (2012, 2013, and 2014 as of December 4).



*2014 data as of December 1, 2014 and subject to change. Counties in white indicate that the county does not have enough data for a 2014 design value or that there is no monitor located within that county. Only regulatory data shown.

Because the EPA's most recent guidance begins with a presumed nonattainment area based on Core Based Statistical Areas (CBSAs) or Combined Statistical Areas (CSAs), several additional counties would likely be designated nonattainment.

2014* Secondary Ozone
W126 Design Values by County

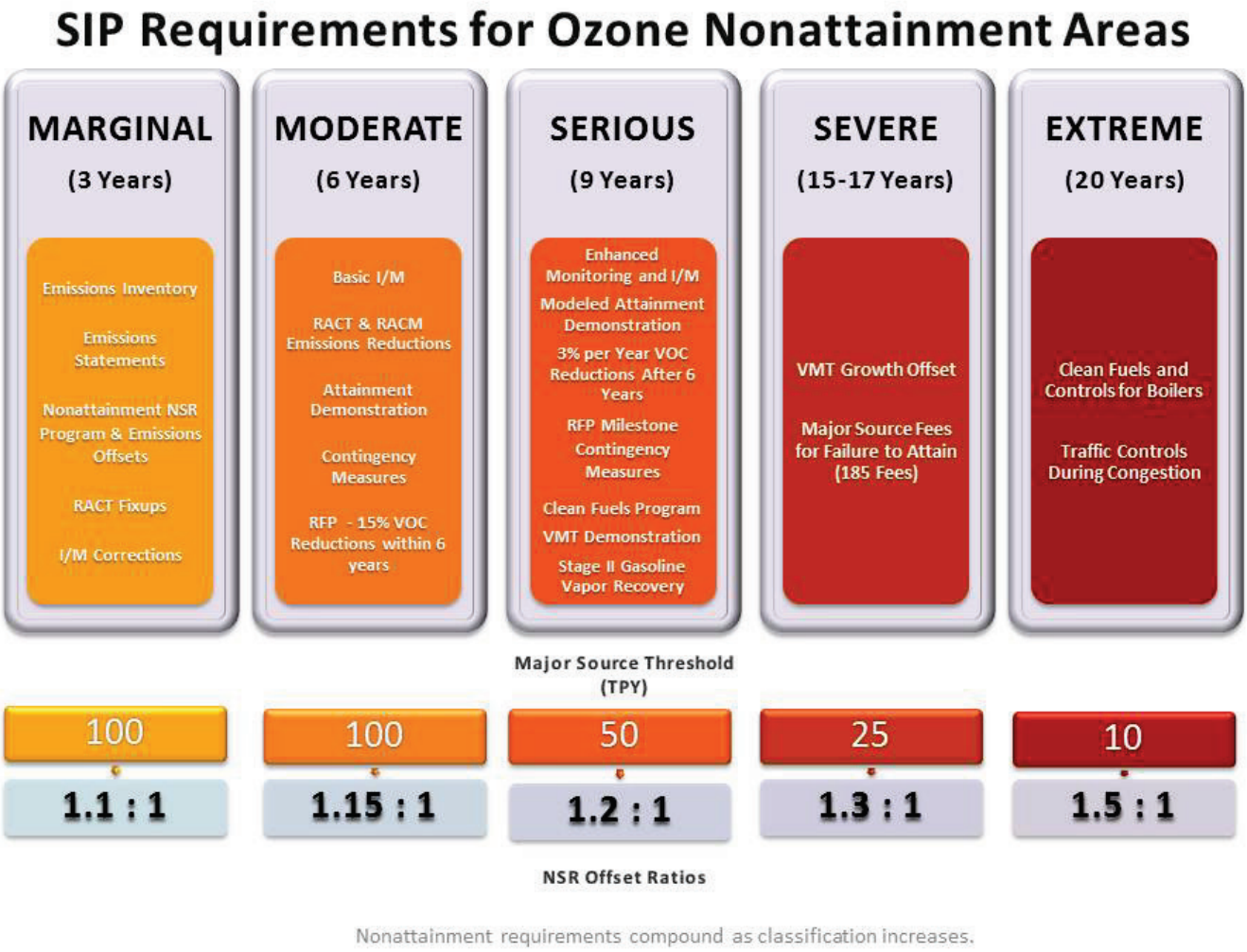


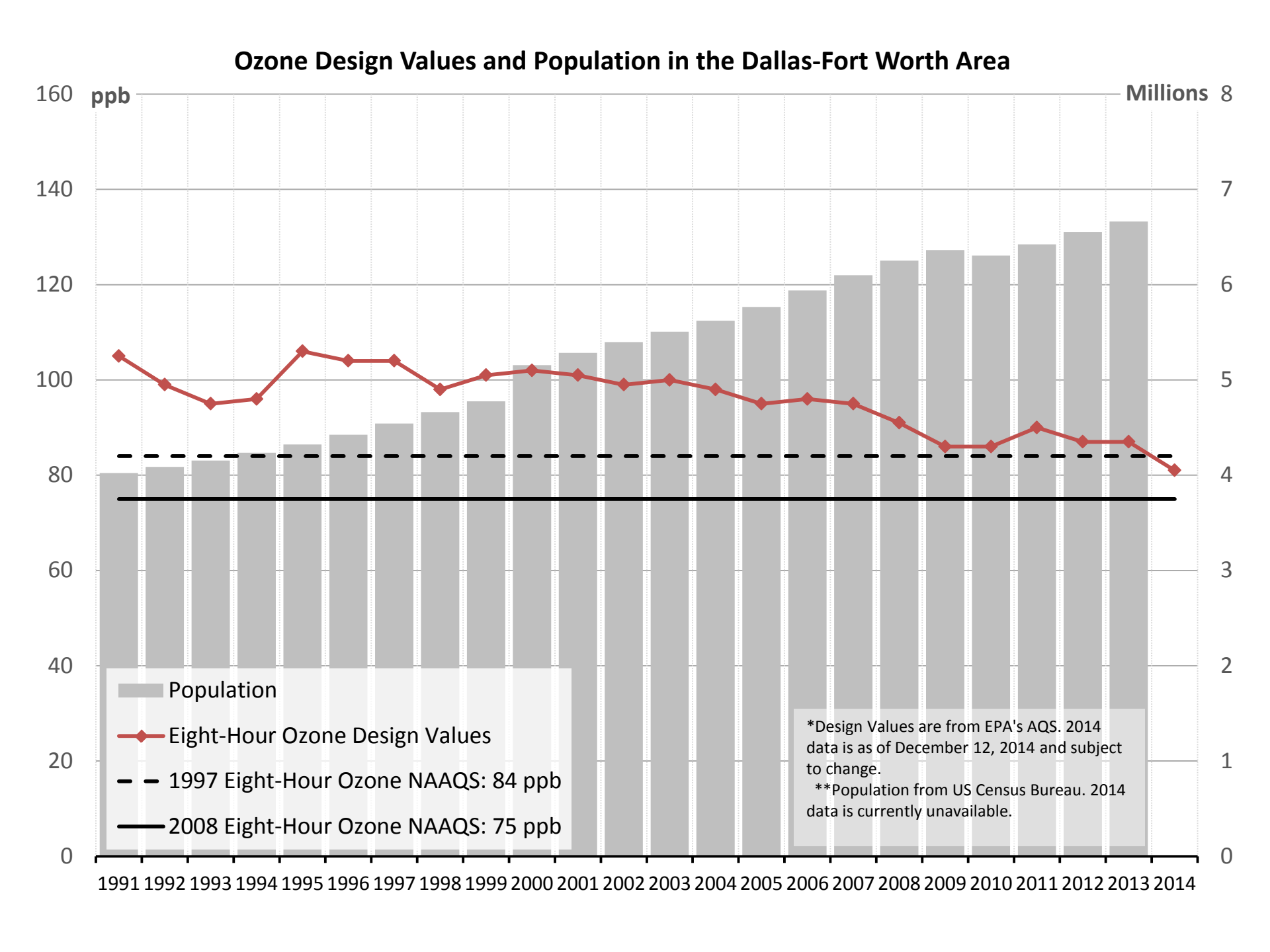
*2014 W126 Design Values are preliminary and are subject to change.

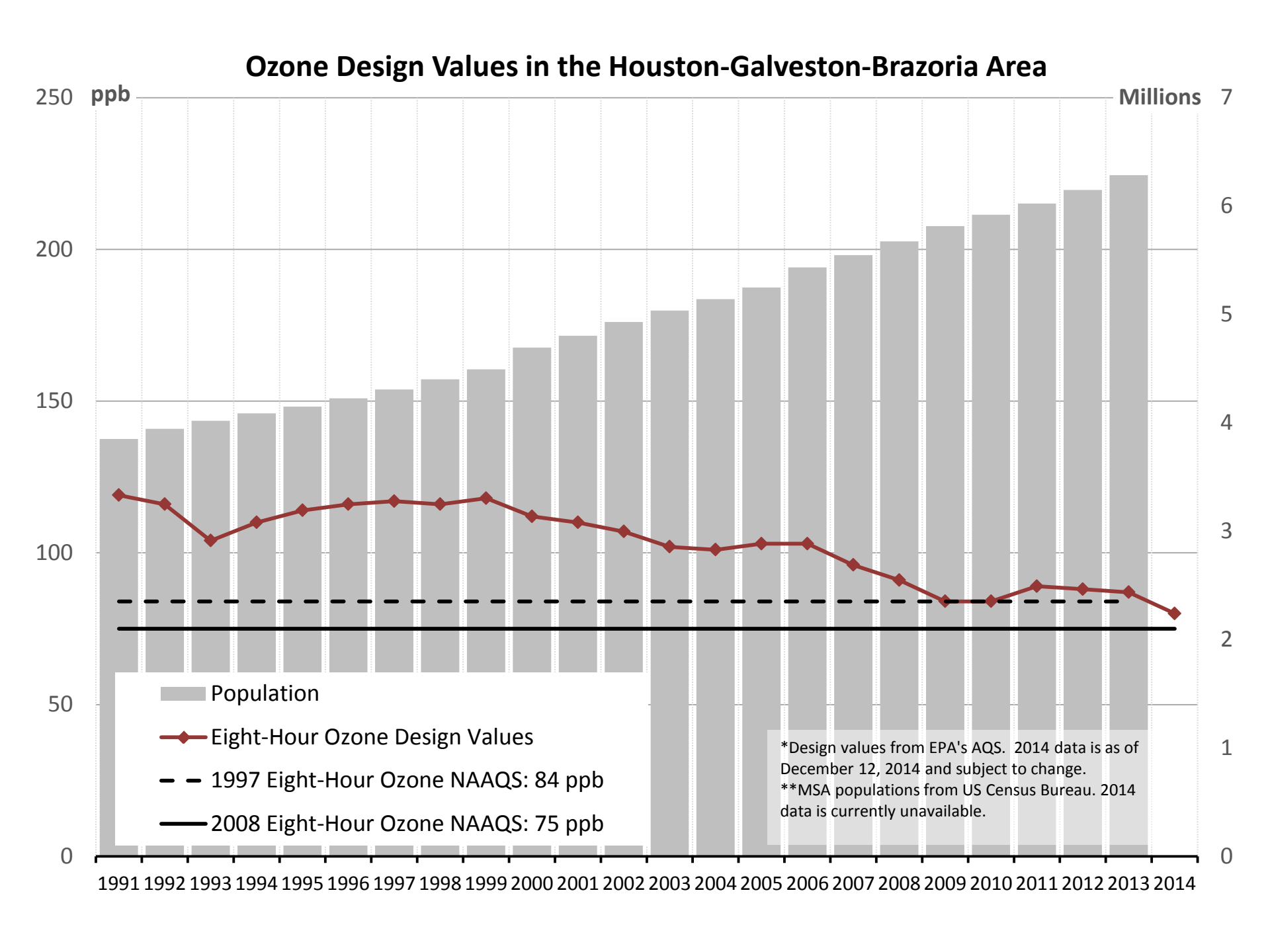
NONATTAINMENT AREA REQUIREMENTS

The EPA classifies areas designated nonattainment based on the area's design value at the time of designations. In order of severity, the classifications are: marginal, moderate, serious, severe, and extreme. Requirements for nonattainment areas become more stringent as the classification increases, and requirements are cumulative as the classification increases.

States with areas that are classified as moderate and above are required to submit a SIP revision that demonstrates to the EPA that the area will meet its attainment deadline and how, including all applicable FCAA requirements for its classification. The figure below includes FCAA nonattainment area requirements by classification.




Ozone Design Values and Population in the Dallas-Fort Worth Area
ppb
Millions
160
140
120
100
80
60
40
20
0
8
7
6
5
4
3
2
1
0
1991 1992 1993 1994 1995 1996 1997 1998 1999 2000 2001 2002 2003 2004 2005 2006 2007 2008 2009 2010 2011 2012 2013 2014
Population
Eight-Hour Ozone Design Values
1997 Eight-Hour Ozone NAAQS: 84 ppb
2008 Eight-Hour Ozone NAAQS: 75 ppb
*Design Values are from EPA's AQS. 2014 data is as of December 12, 2014 and subject to change.
**Population from US Census Bureau. 2014 data is currently unavailable.


Ozone Design Values in the Houston-Galveston-Brazoria Area
ppb
Millions
250
200
150
100
50
0
7
6
5
4
3
2
1
0
1991 1992 1993 1994 1995 1996 1997 1998 1999 2000 2001 2002 2003 2004 2005 2006 2007 2008 2009 2010 2011 2012 2013 2014
Population
Eight-Hour Ozone Design Values
1997 Eight-Hour Ozone NAAQS: 84 ppb
2008 Eight-Hour Ozone NAAQS: 75 ppb
*Design values from EPA's AQS. 2014 data is as of December 12, 2014 and subject to change.
**MSA populations from US Census Bureau. 2014 data is currently unavailable.