

Good morning, Mr. Chairman and members of the committee. I am Dr. Michael Honeycutt, director of the Toxicology Division at the Texas Commission on Environmental Quality (the TCEQ). I lead a division of 14 toxicologists who are responsible for evaluating a broad spectrum of environmental quality issues, including deriving acceptable levels of air contaminants.

The TCEQ has derived acceptable air contaminant levels for many thousands of air contaminants over the last 30+ years and our current team of toxicology risk assessors has over 280 combined years of experience in these fields. We derive these levels using a scientific, peer-reviewed method and many of these levels and their derivation process have been published in independent scientific journals [1-14]. Other state governments, federal government agencies including the EPA, and other countries, use the TCEQ's acceptable air contaminant values.

On October 1 of this year, the EPA decreased the level of the ozone standard from an annual fourth-highest daily maximum 8-hour concentration of 75 ppb, to 70 ppb. Today I will address considerations of overall health risk, but first, I would like to set the record straight on the ozone science.

Based on our extensive background in deriving acceptable air contaminant levels, we independently reviewed thousands of studies on ozone, including studies the EPA reviewed as a part of setting the final standard.

Ozone is a simple oxidizing chemical that, at high enough concentrations, can cause inflammation in the lungs, and it can reversibly limit the body's ability to inhale and exhale a normal volume of air. However, there remains large uncertainty and variability in the scientific literature. With regard to changes in lung function and asthma exacerbations,

- 8 out of 9 studies investigating lung function changes caused by ozone showed no difference between asthmatics and healthy individuals [15-23].
- As we stated in our comments to the EPA, the dose a person would be expected to receive at 75 ppb is almost no different than at 70 ppb, or even 65 ppb – see Figure 1. Consistent with this finding, the EPA does not predict that a decrease in the ozone standard will cause a statistically significant decrease in asthma exacerbations (attacks) – see Figure 2 [24].
- The basis for setting the standard at 70 ppb was to make it lower than the lowest exposure concentration where adverse effects were observed in human controlled exposure studies, which was 72 ppb [25]. However, in order to observe any effects at this low ozone concentration, the study authors had to expose the human subjects to ozone while they were exercising at moderate to heavy exertion for 50 minutes out of every hour for 6.6 hours [25-28]. This is an unrealistic exposure scenario for the general public, much less for sensitive groups. Therefore, it would take higher concentrations to have the same effect noted in the study.

Although asthma exacerbations and changes in lung function are the most important and biologically relevant effects, most of the monetary benefits that EPA ascribes to reductions in ozone are from reductions in premature mortality [24]. They do this despite the fact that, from a toxicology standpoint, there is no explanation for how 8 hours of ozone exposure at ambient, present-day concentrations on one day causes premature mortality the next day. In addition, the

EPA Administrator has expressed a lack of confidence in the studies associating ozone with premature mortality due to inherent study uncertainties [29]. The results from these studies are also contradictory and inconsistent. For example, in the main mortality study that the EPA uses, Smith et al. (2009) [30] showed that only 7 out of 98 US cities have a significant association between 8 hour ozone concentrations and mortality. Also, astonishingly, the EPA's analysis shows mortality **increasing** in certain cities, including Detroit and Houston when **decreasing** the ozone standard from 75 ppb to 70 ppb [31].

Some inconsistency between study findings is not uncommon. Scientists who are experienced in risk assessment can incorporate these disparate pieces of information into a cohesive characterization of health risk. The EPA would be better advised and critiqued on their risk assessment if a risk assessor was included on the Clean Air Scientific Advisory Committee, or CASAC. A chemical risk assessor is essential to put the potential risks highlighted by the other CASAC experts into context with the inherent background risk present in our daily lives. The Clean Air Act does not require that risks are reduced to zero, and risk assessment with uncertainty analysis can demonstrate the reduction in risk, or lack thereof, from a reduction in a regulatory standard.

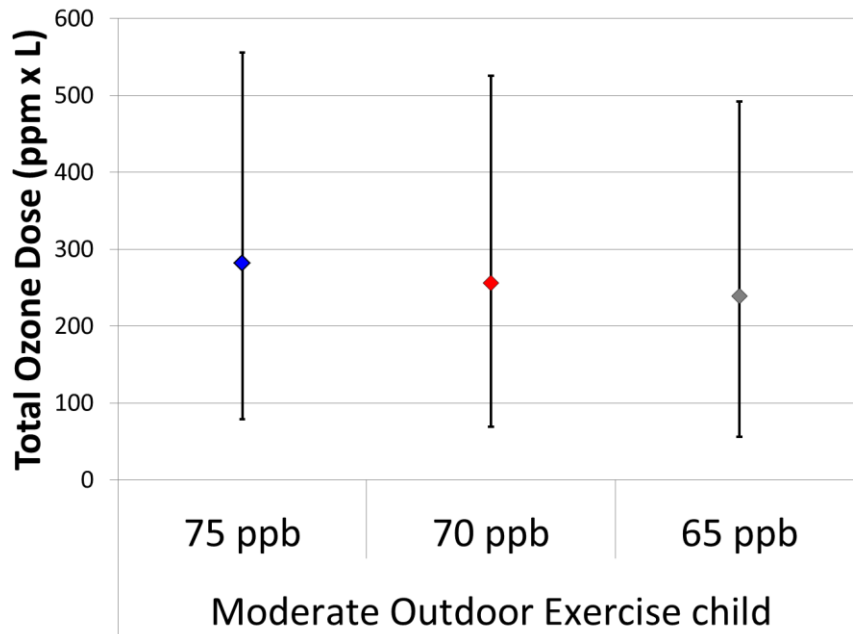
The lack of consideration of overall risk is perhaps most apparent when reviewing the revisions to the EPA's Air Quality Index (AQI) in the final ozone rule. According to the new category breakpoints:

- Sensitive members of the public will now be cautioned to consider reducing prolonged or heavy outdoor exertion at 55 ppb ozone, a number that has no experimental basis.
- Beginning at 71 ppb, the EPA advises the public to keep their asthma inhalers handy. Anecdotally, we are told that some schools in Texas will cancel recess when they receive this alert. The problem is that this is based on a single study that showed a mild lung function effect after exposure to 72 ppb ozone for 6.6 hours with vigorous exercise. And it is being used to cancel a 20 minute recess. In the Dallas-Fort Worth area of Texas in 2014, there would have been 23 such days that children might not have been able to play outdoors.

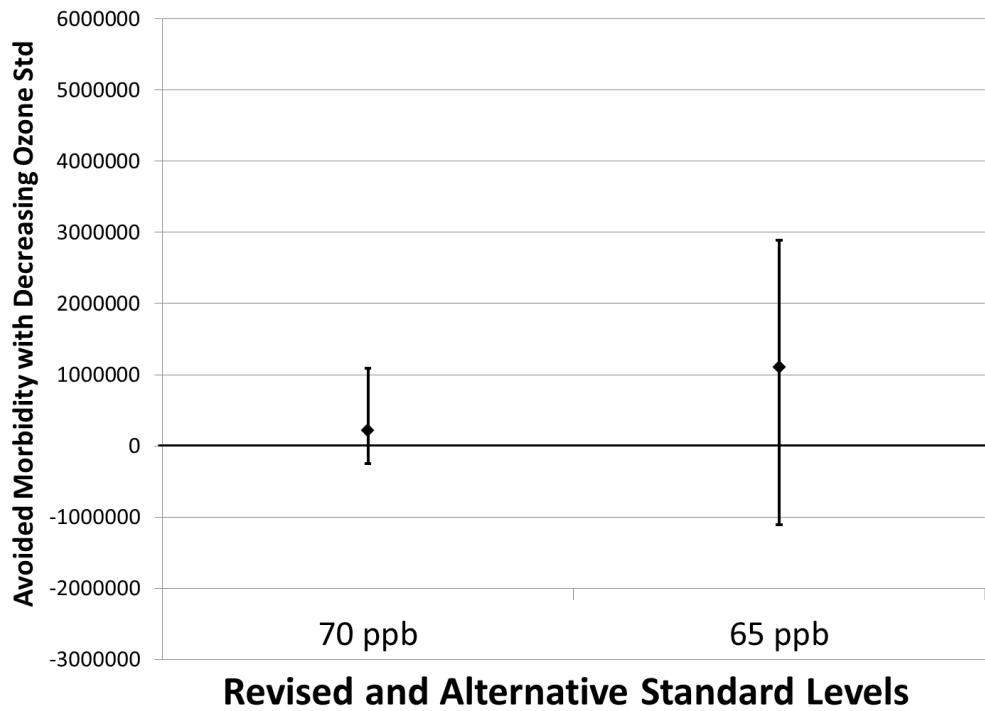
These health messages and the new frequency with which they will be released (see Table 1) can lead to growing public concern over air quality that is actually getting better, and can lead to keeping our children and ourselves from the well-documented physical and psychological benefits of outdoor exercise.

Let me be clear. Under certain circumstances, such as at high concentrations, ground-level ozone can have negative implications for respiratory health. But our investigations conclude that at low concentrations the risks to respiratory health are small, and are not significantly diminished by the decreased ozone standard or activity advice in the revised AQI categories.

I have a tremendous amount of respect for the intent of the Clean Air Act, the EPA, and the role that science plays in setting meaningful policy to protect the health of all Americans. I thank you for the opportunity to be here and I am happy to answer any questions you may have.



**Figure 1.** Total dose of ozone changes very little with decreasing the ozone concentration from 75 ppb to 65 ppb



**Figure 2.** A lower ozone standard will not result in a statistically significant decrease in asthma exacerbations (attacks).

**Table 1.** The 2015 Air Quality Index categorization scheme will increase the number of days with an air quality alert, even though ambient concentrations remain the same.

<i>Air Quality Index Category</i>	<i>Houston-Galveston-Brazoria</i>								<i>Dallas-Fort Worth-Arlington</i>							
	2012		2013		2014		2015**		2012		2013		2014		2015**	
	2008*	2015	2008	2015	2008	2015	2008	2015	2008	2015	2008	2015	2008	2015	2008	2015
Good	283	259	302	274	307	273	179	164	259	233	271	240	288	252	191	179
Moderate	51	60	44	66	52	79	23	33	73	78	62	80	65	88	12	21
Unhealthy for Sensitive Groups	28	36	18	20	6	12	9	10	31	43	31	40	12	23	9	11
Unhealthy	3	9	1	5	0	1	1	4	3	9	1	5	0	2	0	1
Very Unhealthy	1	2	0	0	0	0	0	1	0	3	0	0	0	0	0	0

\* Air Quality Index categorization scheme based on the 2008 or 2015 ozone national ambient air quality standard.

\*\* 2015 data only includes January through July.

**Table 2.** Air Quality Index categories and suggested ozone-related health messages.

<i>Air Quality Index</i>	<i>8-Hour Ozone Concentration (ppb)</i>	<i>Activity Advice*</i>
Good (0-50)	0-54	“It’s a great day to be active outside.”
Moderate (51-100)	55-70	“Unusually sensitive people: <i>Consider reducing</i> prolonged or heavy outdoor exertion. Watch for symptoms such as coughing or shortness of breath. These are signs to take it a little easier. Everyone else: It’s a good day to be active outside.”
Unhealthy for Sensitive Groups (101-150)	71-85	“Sensitive groups: <i>Reduce</i> prolonged or heavy outdoor exertion. Take more breaks, do less intense activities. Watch for symptoms such as coughing or shortness of breath. Schedule outdoor activities in the morning when ozone is lower. People with asthma should follow their asthma action plans and keep quick relief medicine handy.”
Unhealthy (151-200)	86-105	“Sensitive groups: <i>Avoid</i> prolonged or heavy outdoor exertion. Schedule outdoor activities in the morning when ozone is lower. Consider moving activities indoors. People with asthma, keep quick-relief medicine handy. Everyone else: <i>Reduce</i> prolonged or heavy outdoor exertion. Take more breaks, do less intense activities. Schedule outdoor activities in the morning when ozone is lower.”
Very Unhealthy (201-300)	106-200	“Sensitive groups: <i>Avoid all</i> physical activity outdoors. Move activities indoors or reschedule to a time when air quality is better. People with asthma, keep quick-relief medicine handy. Everyone else: <i>Avoid</i> prolonged or heavy outdoor exertion. Schedule outdoor activities in the morning when ozone is lower. Consider moving activities indoors.”
Hazardous (301-500)	201-Significant Harm Level**	“Everyone: <i>Avoid all</i> physical activity outdoors.”

\* Source: US Environmental Protection Agency, *Air Quality Guide for Ozone*, 2015: August. EPA-456/F-15-006

\*\* 2-hour average of 600 ppb

## REFERENCES

1. Erraguntla, N.K. and R.L. Grant, *Health- and vegetative-based effect screening values for ethylene*. Chem Biol Interact, 2015.
2. Erraguntla, N.K., et al., *An updated inhalation unit risk factor for arsenic and inorganic arsenic compounds based on a combined analysis of epidemiology studies*. Regul Toxicol Pharmacol, 2012. **64**(2): p. 329-41.
3. Grant, R.L., et al., *Development of a unit risk factor for 1,3-butadiene based on an updated carcinogenic toxicity assessment*. Risk Anal, 2009. **29**(12): p. 1726-42.
4. Grant, R.L., et al., *A chronic reference value for 1,3-butadiene based on an updated noncancer toxicity assessment*. J Toxicol Environ Health B Crit Rev, 2010. **13**(6): p. 460-75.
5. Grant, R.L., et al., *Evaluation of acute inhalation toxicity for chemicals with limited toxicity information*. Regul Toxicol Pharmacol, 2007. **47**(3): p. 261-73.
6. Haney, J., Jr., *Implications of dose-dependent target tissue absorption for linear and non-linear/threshold approaches in development of a cancer-based oral toxicity factor for hexavalent chromium*. Regul Toxicol Pharmacol, 2015. **72**(2): p. 194-201.
7. Haney, J., Jr., *Use of dose-dependent absorption into target tissues to more accurately predict cancer risk at low oral doses of hexavalent chromium*. Regul Toxicol Pharmacol, 2015. **71**(1): p. 93-100.
8. Haney, J.T., Jr., et al., *Development of a cancer-based chronic inhalation reference value for hexavalent chromium based on a nonlinear-threshold carcinogenic assessment*. Regul Toxicol Pharmacol, 2012. **64**(3): p. 466-80.
9. Haney, J.T., Jr., et al., *Development of an inhalation unit risk factor for hexavalent chromium*. Regul Toxicol Pharmacol, 2014. **68**(2): p. 201-11.
10. Haney, J.T., Jr., et al., *Development of a unit risk factor for nickel and inorganic nickel compounds based on an updated carcinogenic toxicity assessment*. Regul Toxicol Pharmacol, 2012. **62**(1): p. 191-201.
11. Hofelt, C.S., et al., *Development of a metabolism factor for polycyclic aromatic hydrocarbons for use in multipathway risk assessments of hazardous waste combustion facilities*. Regul Toxicol Pharmacol, 2001. **33**(1): p. 60-5.
12. Kirman, C.R. and R.L. Grant, *Quantitative human health risk assessment for 1,3-butadiene based upon ovarian effects in rodents*. Regul Toxicol Pharmacol, 2012. **62**(2): p. 371-84.
13. Myers, J.L. and R.L. Grant, *Development of a chronic inhalation reference value for hexamethylenediamine using an exposure model based on the dihydrochloride salt*. Inhal Toxicol, 2015. **27**(9): p. 440-9.
14. Rhomberg, L.R., et al., *A survey of frameworks for best practices in weight-of-evidence analyses*. Crit Rev Toxicol, 2013. **43**(9): p. 753-84.
15. Linn, W.S., et al., *Effects of prolonged, repeated exposure to ozone, sulfuric acid, and their combination in healthy and asthmatic volunteers*. Am J Respir Crit Care Med, 1994. **150**(2): p. 431-40.
16. Balmes, J.R., et al., *Effects of ozone on normal and potentially sensitive human subjects. Part I: Airway inflammation and responsiveness to ozone in normal and asthmatic subjects*. Res Rep Health Eff Inst, 1997(78): p. 1-37; discussion 81-99.

17. Koenig, J.Q., et al., *The effects of ozone and nitrogen dioxide on pulmonary function in healthy and in asthmatic adolescents*. Am Rev Respir Dis, 1987. **136**(5): p. 1152-7.
18. Koenig, J.Q., et al., *Acute effects of 0.12 ppm ozone or 0.12 ppm nitrogen dioxide on pulmonary function in healthy and asthmatic adolescents*. Am Rev Respir Dis, 1985. **132**(3): p. 648-51.
19. Stenfors, N., et al., *Effect of ozone on bronchial mucosal inflammation in asthmatic and healthy subjects*. Respir Med, 2002. **96**(5): p. 352-8.
20. Holz, O., et al., *Ozone-induced airway inflammatory changes differ between individuals and are reproducible*. Am J Respir Crit Care Med, 1999. **159**(3): p. 776-84.
21. Nightingale, J.A., D.F. Rogers, and P.J. Barnes, *Effect of inhaled ozone on exhaled nitric oxide, pulmonary function, and induced sputum in normal and asthmatic subjects*. Thorax, 1999. **54**(12): p. 1061-9.
22. Basha, M.A., et al., *Bronchoalveolar lavage neutrophilia in asthmatic and healthy volunteers after controlled exposure to ozone and filtered purified air*. Chest, 1994. **106**(6): p. 1757-65.
23. Horstman, D.H., et al., *Comparison of pulmonary responses of asthmatic and nonasthmatic subjects performing light exercise while exposed to a low level of ozone*. Toxicol Ind Health, 1995. **11**(4): p. 369-85.
24. USEPA, *Regulatory Impact Analysis of the Final Revisions to the National Ambient Air Quality Standards for Ground-Level Ozone*, 2015: September. p. 480 p.
25. Schelegle, E.S., et al., *6.6-hour inhalation of ozone concentrations from 60 to 87 parts per billion in healthy humans*. Am J Respir Crit Care Med, 2009. **180**(3): p. 265-72.
26. Adams, W.C., *Comparison of chamber and face-mask 6.6-hour exposures to ozone on pulmonary function and symptoms responses*. Inhal Toxicol, 2002. **14**(7): p. 745-64.
27. Adams, W.C., *Comparison of chamber 6.6-h exposures to 0.04-0.08 PPM ozone via square-wave and triangular profiles on pulmonary responses*. Inhal Toxicol, 2006. **18**(2): p. 127-36.
28. Kim, C.S., et al., *Lung function and inflammatory responses in healthy young adults exposed to 0.06 ppm ozone for 6.6 hours*. Am J Respir Crit Care Med, 2011. **183**(9): p. 1215-21.
29. USEPA, *National ambient air quality standards for ozone (Proposed rule)*. 2014. p. 40 CFR Parts 50, 51, 52, 53 and 58.
30. Smith, R.L., B. Xu, and P. Switzer, *Reassessing the relationship between ozone and short-term mortality in U.S. urban communities*. Inhal Toxicol, 2009. **21 Suppl 2**: p. 37-61.
31. USEPA, *Health Risk and Exposure Assessment for Ozone (Final Report)*, 2014: August. p. 502 p.