Ozone Health Effects
A Review of Scientific Literature and Its Influence of the National Ambient Air Quality Standard

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October 5, 2011
History of the Ozone NAAQS

- **1971**: EPA establishes 1-hour NAAQS of 0.08 ppm
- **1979**: EPA revises 1-hour NAAQS to 0.12 ppm
- **1991**: Number of nonattainment counties reaches 371
- **1996**: EPA issues AQCD citing "strong" evidence of health effects below 1979 standard
- **1997**: EPA revises 1979 standard with 8-hour NAAQS of 0.08 ppm
  - 3 states and dozens of industry plaintiffs challenged new standard
- **1999**: DC Circuit Court of Appeals sends 1997 standard back to EPA for further study
  - EPA appeals
- **2001**: Supreme Court unanimously upholds EPA’s 1997 standard
History of the Ozone NAAQS

2002
- EPA begins process by which States and tribes submit which areas should be designated as nonattainment areas

2003
- EPA proposes ozone implementation rule for transition from 1- to 8-hour standard
- States and tribes designate 412 nonattainment counties
- EPA modifies recommended designations to 506 counties in nonattainment

2004
- EPA finalizes designations and ozone implementation rule

2006
- EPA issues second AQCD citing evidence of "sensitive" individuals below 1997 standard

2007
- States submit plans for meeting 1997 standard to EPA
- EPA proposes new revisions to ozone NAAQS
History of the Ozone NAAQS

2008
- EPA revises 1997 standard with 8-hour NAAQS of 0.075 ppm
- CASAC advises new standard not sufficiently protective and recommends standard within range of 0.060–0.070 ppm
- 2008 standard comes under legal challenge

2009
- EPA reconsiders its 2008 decision
- States and tribes submit which areas should be designated as nonattainment areas

2010
- EPA announces plan to reconsider its 2008 decision

2011
- President Obama announces withdrawal of EPA’s re-proposal of 2008 decision, delaying review until at least 2013 (next regularly scheduled review)
- EPA moves ahead with actions required to implement 2008 standard
- Preliminary EPA review of monitoring data shows 52 areas in nonattainment
Ozone Background

- Reactive gas – “Category 1 gas”
- Acute effects – Respiratory irritant
- Chronic effects – Associated with accumulated effects of repeated acute insults
How are People Exposed to Ozone?
Ozone causes **inflammation** of the lungs and respiratory airways.
What symptoms does ozone cause?

- Eye, nose and throat irritation
- Coughing or chest tightness
- Difficulty breathing or wheezing
- Increased allergy symptoms
- Increased asthma symptoms
# Types of Studies about Ozone

<table>
<thead>
<tr>
<th>Experimental</th>
<th>Epidemiological</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Animal or Human Chamber)</td>
<td>(Field/Panel or Population-Based)</td>
</tr>
</tbody>
</table>

## Strengths
- Exposures are controlled
- Sources of variation minimized
- Strong evidence of causality
  - Prospective

## Limitations
- (Usually) Very high dose
- (Usually) Not humans
- Restrictive exposure conditions

## Strengths
- Direct evidence of human effects

## Limitations
- Exposures complex and changing
- Exposures to many chemicals
- Hard to control for confounders
- Shows correlation not cause
  - Retrospective
- Conclusions require weight-of-evidence judgments based on entire body of evidence
Human Chamber Studies

- Observed effects at near-ambient concentrations for one to several hours
  - Spirometry effects (e.g., ↓ FEV1)
  - Bronchoconstriction
  - Airway hyperresponsiveness
  - Lung inflammation
  - Airway cell damage
  - Shallow/rapid breathing pattern
  - Airway irritation with cough/pain on inspiration
# Summary of Ozone-Induced Respiratory Health Effects from Chamber Studies

<table>
<thead>
<tr>
<th>Health Effect</th>
<th>Exercise Level</th>
<th>Prolonged Exposure</th>
<th>Short-term Exposure</th>
<th>Lowest Ozone Effect Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary Function</td>
<td>Moderate</td>
<td>6.6 hr</td>
<td></td>
<td>0.06 ppm</td>
</tr>
<tr>
<td>Decrement</td>
<td>Moderate</td>
<td>6.6 hr</td>
<td></td>
<td>0.08 ppm</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>4.6 hr</td>
<td>1 hr</td>
<td>0.10 ppm</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>3.0 hr</td>
<td>1-3 hr</td>
<td>0.12 ppm</td>
</tr>
<tr>
<td></td>
<td>Competitive</td>
<td></td>
<td></td>
<td>0.12-0.14 ppm</td>
</tr>
<tr>
<td></td>
<td>Very Heavy</td>
<td></td>
<td></td>
<td>0.16 ppm</td>
</tr>
<tr>
<td></td>
<td>Heavy</td>
<td></td>
<td></td>
<td>0.18 ppm</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td></td>
<td></td>
<td>0.30 ppm</td>
</tr>
<tr>
<td></td>
<td>Light</td>
<td></td>
<td></td>
<td>0.37 ppm</td>
</tr>
<tr>
<td></td>
<td>At rest</td>
<td></td>
<td></td>
<td>0.50 ppm</td>
</tr>
<tr>
<td>Increased Respiratory</td>
<td>Moderate</td>
<td>6.6 hr</td>
<td></td>
<td>0.06 ppm</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Moderate</td>
<td>6.6 hr</td>
<td></td>
<td>0.08 ppm</td>
</tr>
<tr>
<td></td>
<td>Very Heavy</td>
<td></td>
<td>1-3 hr</td>
<td>0.12 ppm</td>
</tr>
<tr>
<td>Airway Responsiveness</td>
<td>Moderate</td>
<td>6.6 hr</td>
<td>1-3 hr</td>
<td>0.08 ppm</td>
</tr>
<tr>
<td></td>
<td>Very Heavy</td>
<td></td>
<td>1-3 hr</td>
<td>0.18 ppm</td>
</tr>
<tr>
<td></td>
<td>At rest</td>
<td></td>
<td></td>
<td>0.40 ppm</td>
</tr>
<tr>
<td>Respiratory Inflammation</td>
<td>Moderate</td>
<td>6.6 hr</td>
<td>1-3 hr</td>
<td>0.08 ppm</td>
</tr>
<tr>
<td></td>
<td>Very Heavy</td>
<td></td>
<td></td>
<td>0.20 ppm</td>
</tr>
<tr>
<td>Changes in Host Defenses</td>
<td>Moderate</td>
<td>6.6 hr</td>
<td></td>
<td>0.08 ppm</td>
</tr>
<tr>
<td>Decreased Exercise</td>
<td>Competitive</td>
<td></td>
<td>1 hr</td>
<td>0.18 ppm</td>
</tr>
<tr>
<td>Performance</td>
<td></td>
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</tr>
</tbody>
</table>
Field/Panel Studies

- 80’s/90’s Camp Studies
  - >600 children, mostly healthy, non-asthmatic
  - Multiple measurements per child
  - Significant ↓ FEV1 with increased ozone
  - Ozone predominant ambient pollutant associated with health endpoint
  - Mean levels - 0.053 to 0.123ppm
  - Maximum levels - 0.095 to 0.245 ppm
Population-Based Studies

- Acute (often more severe) endpoints assessed via daily time-series analysis
  - Emergency visits, hospitalizations, death
- Large sample sizes
- Consistent statistical design across studies
- Uses area/regional monitor exposure data
- Confounders are important consideration
  - Temperature, season, co-pollutants, other exposures (e.g., ETS), socioeconomic status
Summary of Population-Based Studies

% increase in risk per standard increment

Respiratory Emergency Department Visits

Respiratory Hospital Admissions

Respiratory Mortality

Asthma
COPD
Pneumonia
Respiratory
Infection

all year

warm season only
Daily Mortality Studies

- Population-based times series
- Estimate associations between mortality and daily variations in population average exposures
- Lag time between exposure and death one to several days
- Persistent effect after adjusting for co-pollutants
Daily Mortality Studies

- Robust association between daily mortality and increased ozone
- Pooled effect estimate robust to PM
- Larger risks in analyses limited to warm season
- Cardiovascular mortality somewhat stronger and more consistent than respiratory

Chronic Respiratory Effects

- 1996 AQCD
  - Repeated exposure studies in animals show irreversible lung damage
  - Human epidemiology data too limited to conclude whether chronic effects occur

- More recent epidemiology studies focus on asthma development & lung-function growth
  - Lung-function growth in children inversely related to summer ozone concentrations
  - Limited evidence of recovery in lung-function parameters after ozone season
Children’s Health Study

- Long-term cohort from 12 southern CA communities
- Self-reported asthma prevalence, cough, bronchitis and wheeze unrelated to max 1hr ozone
- Reduced baseline lung function in females related to annual average ozone
- Reduced lung function growth related to ozone levels
  - Weaker than PM$_{10}$ effects
- No increased risk of developing asthma high ozone to communities
- Within high ozone RR = 3.3 asthma if sports
College Freshman Studies

• Yale
  ◦ Reduced lung function in cohort from high vs. low ozone communities
  ◦ Stronger effect in males than females
  ◦ No co-pollutant analysis

• UC Berkeley
  ◦ Reduced lung function comparing students from LA to San Francisco areas
  ◦ Adjusts for intrinsic airway diameter
  ◦ Robust effect in multi-pollutant models
Panel Studies: Acute Cardiovascular Effects

- **Myocardial Infarction – MONICA**
  - Current-day ozone associated with acute MI
  - No PM co-pollutant analysis

- **Myocardial Infarction – Peters, et al. (2001)**
  - Non-significant trend toward increased risk with increased ozone in previous 2 hours
  - PM effects were stronger

- **Heart rhythm disturbances – ARIC, NAS, HSPH**
  - Ozone associated with reduced heart rate variability and ventricular arrhythmias
  - PM effects on heart rate generally larger and more consistent
  - Ozone effect on arrhythmias robust in two-pollutant models
### 1996
- **Most conclusive evidence**
  - Controlled acute human exposures
    - Acute lung function deficits
  - Field/Panel studies
    - Acute lung function deficits
- **Highly suggestive evidence**
  - Associations with respiratory ED visits and hospitalizations
  - Co-pollutant interactions and chronic effects unclear

### 2006
- **Further evidence from controlled exposures**
  - Suggestion of sensitive individuals below 0.08 ppm
- **Much larger epidemiology database**
  - Acute respiratory effects considered causal
  - Strong evidence of association with daily mortality
  - Cardiovascular effects suggestive, but evidence inconclusive
  - Chronic respiratory effects inconclusive
  - Lack of conclusive evidence for several other endpoints (lung cancer, developmental effects)

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**EPA’s Ozone Air Quality Criteria Document (AQCD)**
March 2011

- Framework for causal determination offered
- Likely to be causal
  - Daily mortality
  - Acute and long-term respiratory effects
- Suggestive of causal
  - Cardiovascular – acute and long-term effects
  - Central nervous system – acute and long-term effects
  - Reproductive – long-term effects

EPA’s Ozone Integrated Science Assessment (ISA)
Air Pollution Mitigation Study: 1996 Atlanta Olympics

- Intervention: around-the-clock public transportation
  - 1,000 buses added
  - Downtown city streets closed to private cars
  - Downtown delivery schedules altered
  - Flexible and telecommuting work schedules encouraged

Mean Levels of Major Pollutants Before, During, and After the 1996 Summer Olympic Games as a Percentage of the NAAQS

- Weekday morning traffic counts dropped 22.5%
- Peak daily ozone concentrations decreased 27.9%
# Reduction in Number of Asthma Claims During 1996 Atlanta Olympics

<table>
<thead>
<tr>
<th>Type of claim</th>
<th>% change in mean number of asthma claims/day</th>
<th>% change in mean number of non-asthma claims/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medicaid Hosp and ED Visits</td>
<td>-41.6%</td>
<td>-3.1%</td>
</tr>
<tr>
<td>HMO ED, Urgent Visit, Hosp</td>
<td>-44.1%</td>
<td>+1.3%</td>
</tr>
</tbody>
</table>
Section 7408…
Protect the Health of
Sensitive or Susceptible
Individuals

Section 7409…With
an Adequate Margin
of Safety

QUESTIONS?