Lower Respiratory Illnesses and Diesel Exposure in Tucson

*Health Effects of Common Tucson Air Pollutants*

Paloma Beamer, PhD
Assistant Professor
Environmental Health Sciences

Acknowledgements. Funding provided from a pilot project grant of the Southwest Environmental Health Sciences Center (NIEHS – ES006694). CRS funded by NHLBI (HL 56177).
Literature review: “Air Pollution and Health”

• Anytime
  – 1,810,000 hits

• Since 2009
  – 259,000 hits

• Since 2013
  – 38,400 hits

Disability Adjusted Life Years
Health Effects of CO (US EPA, 1999)

• Acute
  – Reduction of Oxygen
  – Death
  – Coma, seizures
  – Headache, dyspnea
  – Congestive heart failure (ambient)

• Chronic
  – Cardiovascular
  – Neurobehavioral (but not likely at current ambient levels)
  – Developmental (but not likely at current ambient levels)
Health Effects of Ozone (Sousa et al, 2013)

• Acute
  – Inflammation of Airways
    • Decrease lung function
    • Increase respiratory symptoms
    • Asthma and COPD exacerbations

• Chronic
  – Lung function development
  – Less controlled asthma
1. Particle pollution inhaled

2. Microscopic particles evade body’s natural defenses

3. Particles lodge deep in lung’s air sacs

4. Particles damage the lungs
Characteristics of PM

- Size
- Shape
- Composition
  - Metals
  - PAHs
  - Black Carbon
PM Inhalation

**Lungs**
- Inflammation
- Oxidative stress
- Accelerated progression and exacerbation of COPD
- Increased respiratory symptoms
- Effected pulmonary reflexes
- Reduced lung function

**Blood**
- Altered rheology
- Increased coagulability
- Translocated particles
- Peripheral thrombosis
- Reduced oxygen saturation

**Vasculature**
- Atherosclerosis, accelerated progression of and destabilization of plaques
- Endothelial dysfunction
- Vasoconstriction and Hypertension

**Systemic Inflammation**
- Increased CRP
- Proinflammatory mediators
- Leukocyte & platelet activation

**Brain**
- Increased cerebrovascular ischemia

Pope and Dockery (2008)
Health Effects of Particulate Matter

• Acute
  – Increased mortality (especially cardiovascular disease)
  – Increased hospital admissions (CVD)
  – Decreased lung function

• Chronic
  – Cancer
  – Cardiopulmonary mortality
  – Respiratory disease in children
  – Accelerated progression of COPD
  – Lung function development
Results: A decrease of 10 μg/m³ in the concentration of PM$_{2.5}$ was associated with an increase in mean life expectancy of 0.35 years (SD = 0.16 years, $P = 0.033$).
Published in 1997, supported by NIH grant ES 0002

- Wanted to confirm associations of PM and CO with cardiovascular disease in unique location.
  - Reduce correlations between pollutants
  - SO$_2$ not correlated with PM
  - PM highest in winter, when ozone lowest

- Results, pollutant independent associations with hospital admissions for CVD (1988-1990) with:
  - PM: 2.75% (0.52-5.04) per 23 $\mu$g/m$^3$
  - CO: 2.79% (0.59-5.41) per 1.66 ppm
National Data Set (1987-2001)

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Mean</th>
<th>Std Dev</th>
<th>Minimum</th>
<th>10th Pctl</th>
<th>Median</th>
<th>90th Pctl</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO</td>
<td>5114</td>
<td>886.92</td>
<td>400.86</td>
<td>-236.10</td>
<td>459.69</td>
<td>830.75</td>
<td>1409.04</td>
<td>4521.38</td>
</tr>
<tr>
<td>NO2</td>
<td>4096</td>
<td>21.72</td>
<td>6.94</td>
<td>3.37</td>
<td>12.33</td>
<td>23.02</td>
<td>31.29</td>
<td>60.25</td>
</tr>
<tr>
<td>SO2</td>
<td>4591</td>
<td>1.97</td>
<td>1.24</td>
<td>0.00</td>
<td>0.71</td>
<td>1.63</td>
<td>3.26</td>
<td>12.96</td>
</tr>
<tr>
<td>O3</td>
<td>5114</td>
<td>27.74</td>
<td>9.19</td>
<td>5.32</td>
<td>15.88</td>
<td>27.53</td>
<td>40.17</td>
<td>58.43</td>
</tr>
<tr>
<td>PM10</td>
<td>2467</td>
<td>25.65</td>
<td>16.07</td>
<td>-9.87</td>
<td>8.27</td>
<td>23.83</td>
<td>43.31</td>
<td>209.85</td>
</tr>
</tbody>
</table>

Tucson, Total Mortality

[ihapss.jhsph.edu](ihapss.jhsph.edu)
PM$_{10}$ and Mortality in Tucson, AZ

**Graph: PM$_{10}$ and Mortality in Tucson, AZ**

- **Deaths**
- **CVD Deaths**
PM$_{10}$ and Mortality in Phoenix, AZ

![Graph showing the percentage increase per 10 µg/m$^3$ for Deaths and CVD Deaths across different lags (Lag 1 to Lag 7).]
PM$_{10}$ and Mortality in US (108 cities)
Lower Respiratory Illnesses and Diesel Exposure in Tucson

Health Effects of Common Tucson Air Pollutants

Paloma Beamer, PhD
Assistant Professor
Environmental Health Sciences
What is diesel exhaust?

- > 400 chemicals
  - 40 toxic air pollutants
- Gases, Vapor and Particulate Matter
- Greater proportion:
  - NOx/CO ratio
  - Elemental/Organic carbon ratio
Lower Respiratory Illnesses (LRIs)

- LRIs cause of 1/5th of all deaths worldwide in children under age 5 years
- LRIs are a risk factor for:
  - asthma
  - chronic obstructive pulmonary disorder
  - decreased lung function development
  - alterations in cytokine response

- Associated with:
  - tobacco smoke
  - smoke from indoor cooking
  - outdoor air particulates
  - polyaromatic hydrocarbons
  - In Tucson:
    - tobacco smoke
    - evaporative cooling
    - perception of neighborhood dustiness

Jones et al., 2011; Mehta et al., 2011; Smith et al. 2004; Piccioto et al. 2007; Aldous et al. 1996; Wright et al. 1991
Tucson Children’s Respiratory Study (CRS)

• Non-selected longitudinal birth cohort
• 1,246 subjects enrolled between 1980-1984
• LRIs are MD diagnosed and assessed for wheeze and viruses

Number of index subjects with data at each evaluation

<table>
<thead>
<tr>
<th>Age of Subjects</th>
<th>Birth to 3y</th>
<th>6y</th>
<th>11y</th>
<th>16y</th>
<th>20y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory questionnaires</td>
<td>1055</td>
<td>1025</td>
<td>955</td>
<td>767</td>
<td>735</td>
</tr>
<tr>
<td>LRI evaluations through 3y</td>
<td>888</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin prick tests</td>
<td></td>
<td>762</td>
<td>709</td>
<td>536</td>
<td>479</td>
</tr>
<tr>
<td>Serum IgE</td>
<td>1120 (at birth)</td>
<td>534</td>
<td>601</td>
<td>404</td>
<td>397</td>
</tr>
<tr>
<td>Peripheral blood eosinophils</td>
<td>880 (at 9 mo)</td>
<td>522</td>
<td>596</td>
<td>467</td>
<td>397</td>
</tr>
<tr>
<td>Pulmonary function studies</td>
<td>176 (at 4 mo)</td>
<td>676</td>
<td>627</td>
<td>485</td>
<td>456</td>
</tr>
<tr>
<td>Bronchial hyperresponsiveness</td>
<td>368</td>
<td>397</td>
<td>398</td>
<td>342</td>
<td></td>
</tr>
</tbody>
</table>
Methods: Exposure Assessment

• US EPA National-Scale Air Toxics Assessment
  – Average hazardous air pollutant exposure by census tract
  – Used in studies since 2006, e.g., children’s respiratory hospital admissions (Grineski et al., 2012)

• Used 1996 data for Diesel Particulate Matter (DPM) exposure
  – Spatial correlation
    • DPM 1996 vs. 2002 (p=0.006)
    • Traffic Volumes 1980 vs. 1996 (p<0.0001)
Infant Characteristics by DPM Exposure

No relation with gender, season of birth, maternal smoking, duration of breastfeeding, household cooling method and day care by 6 months.
Association of DPM Exposure to LRIs by type

- Any LRI vs. no LRI – no relation with DPM exposure

*adjusted for child ethnicity and care outside of home at 6 months
LRI Spatial Clusters

Wheezing LRIs

Viral LRIs
Diesel PM$_{2.5}$ Concentration

1980 Fall

DPM 2.5 (µg/m$^3$) |
---
0 – 0.18
0.19 – 0.533
0.534 – 1.0
1.01 – 2.69

Elevation (m)  |
---
2,789
465

2006 Fall

DPM 2.5 (µg/m$^3$) |
---
0 – 0.18
0.19 – 0.533
0.534 – 1.0
1.01 – 3.1
Conclusions

• Air pollution is a significant risk factor in global burden of disease

• Here in Tucson, associations with:
  – PM and cardiovascular disease
  – Diesel PM and wheezing lower respiratory illnesses in children
Questions

Paloma Beamer
pbeamer@email.arizona.edu
Diesel PM Emission Factors by Vehicle Type

- HDDV4
- HDDV5
- HDDV6
- HDDV7
- 8A
- 8B
- Urban Bus
- School Bus

Emissions (g/mile)

- 1980-Running
- 2006-Running