Diesel Exhaust: Health Effects and Research Needs

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Outline

- What is diesel exhaust?
- How is exposure assessed?
- What exposure levels have been reported?
- What are the potential health effects?
  - Non-cancer
  - Cancer
- Are these health effects unique to diesel or properties of all inhaled (ambient) particles?
- Current research
Diesel Exhaust Composition

- Vapor Phase
  - Carbon Monoxide
  - Carbon Dioxide
  - Sulfur Dioxide
  - Nitrogen Oxides
  - Aldehydes
  - Hydrocarbons
  - PAH Compounds

- Particulate Phase
  - Elemental Carbon
  - Sulfates
  - Hydrocarbons
  - PAH Compounds

PAH= Polycyclic Aromatic Hydrocarbons
Diesel Particle

Solid Carbon Spheres (0.01 - 0.08 μm diameter) form to make Solid Particle Agglomerates (0.05 - 1.0 μm diameter) With Adsorbed Hydrocarbons

Adsorbed Hydrocarbons

Liquid Condensed Hydrocarbon Particles

Sulfate with Hydration

Vapor Phase Hydrocarbons

Soluble Organic Fraction (SOF)/Particle Phase Hydrocarbons

Adsorbed Hydrocarbons

Sulfate (SO₄)
**Particulate Phase**

- Particles have adsorbed hydrocarbons and PAH compounds
- PAH compounds cause mutations/chromosomal alterations in laboratory test systems
- Size favors inhalation and deposition into lower region of the lung
- Older diesel engines produce more particles by weight than newer engines, but newer diesels may produce finer particles
Diesel Exhaust Contributes to Ambient Particulate Matter

- Total Suspended Particulates (TSP)
- Respirable particles: < 10 μm
  - Coarse particles: 2.5-10 μm
  - Fine particles: < 2.5 μm (PM$_{2.5}$)
  - Ultrafine particles: < 1.0 μm (PM$_{1.0}$)
- Diesel exhaust 0.05-1.0 μm

Contribution of diesel to ambient particles varies
Sources of PM$_{2.5}$ - PM$_{10}$

- Crustal particles:
  - Mechanically generated from agriculture, mining, construction, road traffic and related sources

- Organic particles
  - Biological origin
Sources of PM$_{2.5}$

- Combustion particles
  - Motor vehicles (diesel, gas)
  - Burning of coal, fuel oil, wood, food
- Crustal particles
  - Finely ground airborne road dust and soils
How is Diesel Exhaust Exposure Assessed?

- Job title
- \( PM_{10} \) or \( PM_{2.5} \)
- Elemental carbon (EC) in particles < 1.0 \( \mu m \)
- Source apportionment: EC and assessment of associated organic chemical profile
  - Provide estimates of diesel and non-diesel EC
# Measurements in Various Exposure Settings

<table>
<thead>
<tr>
<th>Exposure Setting</th>
<th>Measurement</th>
<th>µg/m³</th>
</tr>
</thead>
<tbody>
<tr>
<td>25 underground metal/non-metal mines 1988-97</td>
<td>Mostly EC</td>
<td>68 - 1835</td>
</tr>
<tr>
<td>11 surface mines (coal/non-coal) 1988-97</td>
<td>Mostly EC</td>
<td>19 – 160</td>
</tr>
<tr>
<td>Railroad workers (various jobs) 1981-83</td>
<td>Respirable*</td>
<td>39 - 130</td>
</tr>
<tr>
<td>Truck terminals/diesel forklifts (1988-1989)</td>
<td>EC</td>
<td>27.2</td>
</tr>
<tr>
<td>Railroad workers (various jobs) 1996</td>
<td>EC</td>
<td>1.9 – 21.0</td>
</tr>
<tr>
<td>Truck mechanics (1988-1989)</td>
<td>EC</td>
<td>12.1</td>
</tr>
<tr>
<td>City truck drivers/Road drivers (1988-1989)</td>
<td>EC</td>
<td>4.0/3.8</td>
</tr>
<tr>
<td>Harlem streets, July 1996</td>
<td>EC, PM&lt;sub&gt;2.5&lt;/sub&gt;</td>
<td>1.5 – 6.2, 36.6 – 47.1</td>
</tr>
</tbody>
</table>

* cigarette adjusted
### Area Levels of Elemental Carbon, $\mu g/m^3$

*Truckling Industry Study, October 1999*

<table>
<thead>
<tr>
<th>Area</th>
<th>Urban</th>
<th>Rural</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>GM</td>
</tr>
<tr>
<td>Terminal Dock*</td>
<td>16</td>
<td>4.2</td>
</tr>
<tr>
<td>Yard (Upwind)</td>
<td>6</td>
<td>2.2</td>
</tr>
</tbody>
</table>

*PM$_{2.5}$ = 53 $\mu g/m^3$ (mean) on dock*
Marine Dock Exposure

- Ro-ro ferry, Great Britain (Groves and Cain, 2000)
  - Elemental Carbon GM = 37 µg/m³ (GSD 2.5)
  - 20 personal samples

- Ro-ro ship stevedores, Sweden (Dahlqvist and Ulfvarson, 1996)
  - Study conducted in 1987; respirable dust (AM/SD)
  - Ship 1: 80 (+/-20) µg/m³
  - Ship 2: 250 (+/-40) µg/m³
Human Health Effects Overview

- Odor and Irritant
- Allergic Response
- Respiratory Symptoms: Acute and Chronic
- Pulmonary Function: Acute Exposure
- Pulmonary Function: Chronic Exposure
- Lung Cancer (best studied)
- Other Cancers (weak evidence)
Early Diesel Research

The SNIFFMOBILE
PUBLIC OPINION
ODOR TESTING SURVEY
Odor and Irritant Effects

- Odor: focus of early research
- Old diesel: black smoke with characteristic odor
- Reports in miners, bus garage workers, stevedores
- Specific constituent unknown
- Newer diesels: less black smoke, but also depends on maintenance and operating characteristics
Highway Diesel Exhaust
Methods Allergic and Lung Inflammatory Response: Experimental

- Nasal studies in humans:
  - Particles with and without pollen sprayed in nose
  - Washings to assess chemical mediators of allergy

- Lung studies in humans:
  - Controlled diesel exposure followed by lung lavage
  - Assess cellular and chemical response in lavage fluid

- Studies with cultured human airway cells
Assessment of Lung Function and Respiratory Symptoms: Experimental Methods

- **Pulmonary Function**
  - $\text{FEV}_1 = \text{Volume exhaled in 1 second}$
  - $\text{FVC} = \text{Volume between total lung capacity and residual volume}$

- **Standardized respiratory symptom questionnaire**

- **Acute effects**: Assess work shift changes

- **Chronic effects**: Assess lung function attributable to long-term exposures
**Allergic Response: Summary**

- Grass pollen can bind to diesel particles
- Diesel particles sprayed in nose results in production of nasal IgE
- Ragweed + diesel particles = greater ragweed response than ragweed alone
- Nasal challenge with diesel particles result in nasal inflammatory response
- Particles without organic compounds = less response
- Diesel particles result in release of markers of inflammation from cultured human airway cells
**Pulmonary Function: Bronchial Changes**

- 15 nonsmoking volunteers, no asthma or allergies
- 300 µg/m³ whole diesel exhaust for 1 hour
- No change in FEV₁ and FVC
- Lavage: increase in inflammatory cells and other inflammatory markers
- Bronchial biopsies: inflammatory changes

Acute Health Effects: Bus Garage

- 232 male workers in four garages
- Workers ≥ 310 µg/m³ respirable particles reported more cough, itchy or burning eyes, chest constriction, wheeze, difficulty breathing
- No significant shift related changes in FEV₁
- Smoking and age adjusted

**Acute Health Effects: Miners**

- 150 underground coal miners - diesel/non-diesel
- Diesel mine respirable dust = $2.0 \pm 1.7 \text{ mg/m}^3$
- Non-diesel mine respirable dust = $1.4 \pm 1.5 \text{ mg/m}^3$
- Similar shift related changes in both groups

Acute Health Effects: Stevedores

- 23 stevedores exposed to diesel truck exhaust after 10 days off work
- No control group
- Respirable dust 130 - 590 µg/m³ (contribution of diesel)
- Shift related changes in FEV₁ and FVC

Acute Health Effects: Children

- Lung function testing in 1,092 children who lived near highways
- 4% reduction in FEV$_1$ and FVC per 10,000 trucks/24 hours - within 300 meters
- Weaker relationship between reduced function and automobile traffic
- Adjusted for parental smoking, gas cooking

Chronic Health Effects

- Five studies in surface and underground coal, potash, and salt miners, and bus garage workers
  - Cross sectional studies of active workers, many with short-term exposure
  - One five year longitudinal study
- Diesel exhaust not specifically measured
- No consistent association between exposure and pulmonary function
Lung Cancer
Summary Of Results In Animals

- Dose related increase in lung tumors at high levels of diesel exhaust exposure (3500 µg/m³) in rats
- Results can be reproduced by inhalation of other insoluble particles without associated organics
- Relevance to humans occupationally exposed at lower concentrations is uncertain
Lung Cancer

Epidemiological Studies in Humans

- >30 studies
- Truck Drivers
- Railroad Workers
- Bus Garage And Transport Workers
- Dock Workers
- General Population Groups
solid circle = smoking adjusted  open circle = smoking unadjusted
solid circle = smoking adjusted  open circle = smoking unadjusted
Epidemiological Studies With Quantitative Exposure Assessments

Used to validate exposure assignments

- Teamsters Union (Steenland et al. 1990)
  - Case-control study of retired workers who died in 1982-1983
  - Elevated risk in long haul drivers (mainly diesel) and pick-up and delivery (gas) drivers
  - Extent of diesel exposure uncertain

- U.S. Railroad Workers (Garshick et al. 1987, 1988)
  - Case-Control Study - 1981-1982
  - Retrospective Cohort Study – 55,407 workers 1959-1976
  - RR exposures 1950’s-1970’s
Railroad Workers Studies

- **Case-control study**
  - Odds ratio = 1.41 (1.06, 1.88) for 20 years of work
  - Smoking adjusted

- **Retrospective cohort study**
  - Relative risk = 1.45 (1.11, 1.89) in workers with greatest potential for exposure
  - Dose – response controversial
  - Mortality update through 1996 nearly complete
**Conclusions: Lung Cancer Epidemiological Studies**

- Consistently elevated lung cancer risk
- Risk unlikely explained by cigarette smoking
- Exposure Related Uncertainties
  - Job title and exposure not directly linked to job title
  - Dose and duration of exposure uncertain
- Study Design Related Uncertainties
  - Few studies with long-term follow-up and none with well characterized exposure over years
Health Effects of PM*

Effects associated with a 10 µg/m³ rise in \( PM_{10} \)

- >150 studies
- Cardiovascular mortality: 1.4%
- Cardiovascular hospital admissions 0.8%
- Respiratory mortality: 3.4%
- Respiratory hospital admissions
  - Asthma: 2%  COPD: 2.5%  Pneumonia: 1.5%
- Effect driven by PM\(_{2.5}\) rather than by larger particles
- Biologic mechanism under study

*Adapted from Pope & Dockery, 1999
## Chronic Exposure and Mortality

*Most vs Least Polluted City, Smoking Adjusted*

<table>
<thead>
<tr>
<th></th>
<th>Six Cities ( \text{PM}_{2.5} )</th>
<th>ACS ( \text{PM}_{2.5} )</th>
<th>ACS ( \text{SO}_4 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Mortality</td>
<td>1.26 ( (1.08-1.47) )</td>
<td>1.17 ( (1.09-1.26) )</td>
<td>1.15 ( (1.09-1.22) )</td>
</tr>
<tr>
<td>Cardio-pulmonary</td>
<td>1.37 ( (1.11-1.68) )</td>
<td>1.31 ( (1.17-1.46) )</td>
<td>1.26 ( (1.16-1.37) )</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>1.37 ( (0.81-2.31) )</td>
<td>1.03 ( (0.80-1.33) )</td>
<td>1.36 ( (1.11-1.66) )</td>
</tr>
<tr>
<td>All others</td>
<td>1.01</td>
<td>1.07</td>
<td>1.01</td>
</tr>
</tbody>
</table>
## Association of Fine Particulate Matter from Different Sources with Daily Mortality in Six U.S. Cities

<table>
<thead>
<tr>
<th>Source</th>
<th>% Increase in Daily Deaths (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crustal (Si)</td>
<td>-2.3 (-5.8, 1.2)</td>
</tr>
<tr>
<td>Motor (Pb)</td>
<td>3.4 (1.7, 5.2)</td>
</tr>
<tr>
<td>Coal (Se)</td>
<td>1.1 (0.3, 2.0)</td>
</tr>
</tbody>
</table>

Cardiovascular Effects of PM

- Preliminary studies suggest PM$_{2.5}$ related to:
  - Implantable defibrillator discharge
  - Abnormal heart rate control
  - Increased risk of myocardial infarction

- Efforts underway to understand biologic mechanisms
Health Effect Summary

- **Diesel Studies**
  - Potential to influence the development of allergy
  - May induce inflammatory changes in human airway
  - Probable elevated lung cancer risk
  - Dose-response and mechanisms uncertain

- **PM Studies**
  - Elevated mortality/morbidity due to respiratory/cardiac causes
  - Fine particles associated with lung cancer mortality
  - Mortality related to PM$_{2.5}$ from gasoline powered sources in one study
Conclusions

- Considerable data indicates adverse health potential of diesel exhaust and PM
- Effects of low level exposures to diesel difficult to characterize
- Diesel contributes to PM
- Studies characterizing fine and ultrafine PM and associated health effects are needed
Research Study: Trucking Industry Particle Study

- Cooperative efforts of 4 national LTL carriers and Teamsters Union
- Characterize current exposure
  - PM$_{2.5}$, EC, source apportionment
- Estimate previous exposures
- Funded by National Cancer Institute 2001-2006