

Diesel Exhaust: Health Effects and Research Needs

Eric Garshick, MD, MOH
Assistant Professor of Medicine
VA Boston Healthcare System
Channing Laboratory, Brigham and Womens
Hospital
Harvard Medical School
Boston, MA

Co-Investigators

Francine Laden, ScD

Frank E. Speizer, MD

■ Channing Laboratory,
Brigham and Womens
Hospital, Harvard
Medical School

Thomas J. Smith, PhD

Douglas Dockery, ScD

■ Harvard School of
Public Health

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

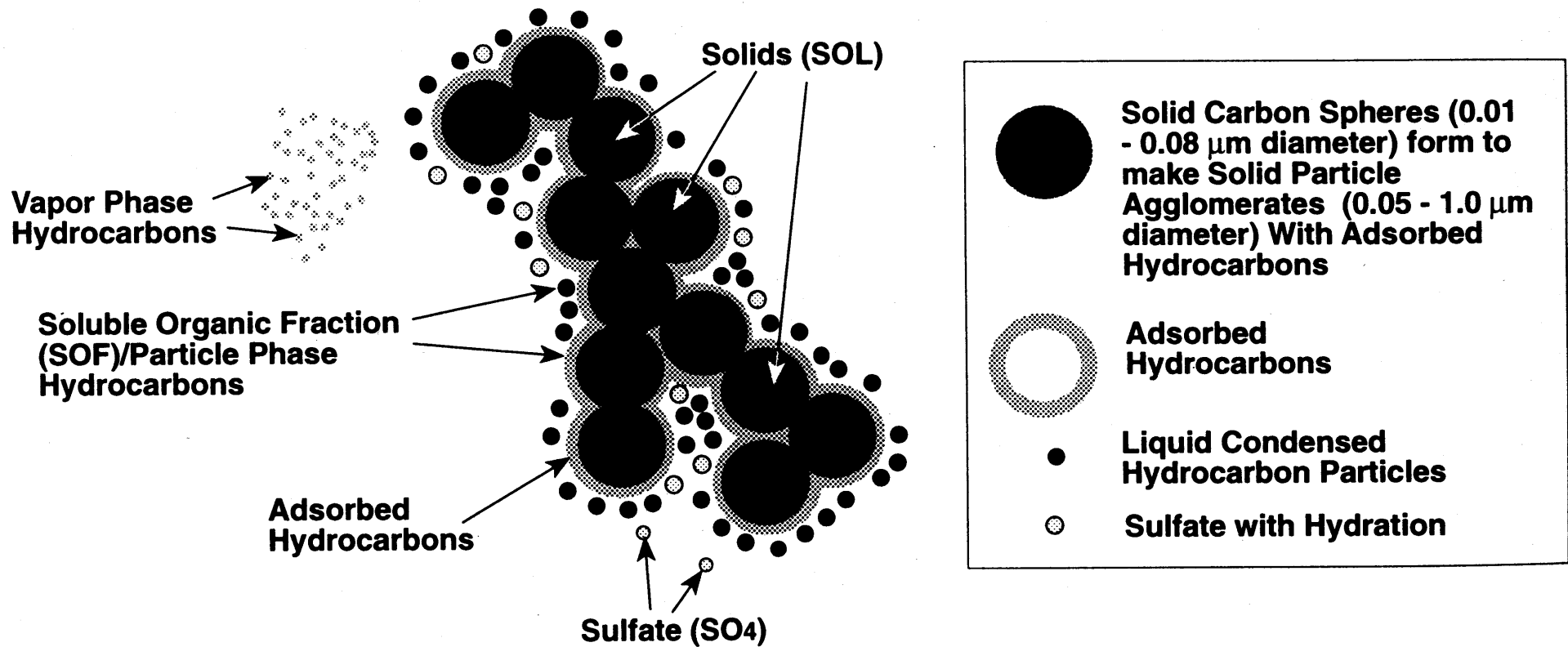
Sulfates

Hydrocarbons

PAH Compounds

PAH= Polycyclic Aromatic Hydrocarbons

Diesel Particle



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: $< 10 \mu\text{m}$
 - ◆ Coarse particles: $2.5\text{-}10 \mu\text{m}$
 - ◆ Fine particles: $< 2.5 \mu\text{m}$ ($\text{PM}_{2.5}$)
 - ◆ Ultrafine particles: $< 1.0 \mu\text{m}$ ($\text{PM}_{1.0}$)
 - ◆ Diesel exhaust $0.05\text{-}1.0 \mu\text{m}$
- 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
- PM₁₀ or PM_{2.5}
- Elemental carbon (EC) in particles < 1.0 μm
- Source apportionment: EC and assessment of associated organic chemical profile
 - ◆ Provide estimates of diesel and non-diesel EC

Measurements in Various Exposure Settings

Exposure Setting	Measurement	$\mu\text{g}/\text{m}^3$
25 underground metal/non-metal mines 1988-97	Mostly EC	68 - 1835
11 surface mines (coal/non-coal) 1988-97	Mostly EC	19 - 160
Railroad workers (various jobs) 1981-83	Respirable*	39 - 130
Truck terminals/diesel forklifts (1988-1989)	EC	27.2
Railroad workers (various jobs) 1996	EC	1.9 - 21.0
Truck mechanics (1988-1989)	EC	12.1
City truck drivers/Road drivers (1988-1989)	EC	4.0/3.8
Harlem streets, July 1996	EC	1.5 - 6.2
	PM _{2.5}	36.6 - 47.1

*cigarette adjusted

Area Levels of Elemental Carbon, $\mu\text{g}/\text{m}^3$

Trucking Industry Study, October 1999

Area	Urban		Rural	
	N	GM	N	GM
Terminal Dock*	16	4.2	8	0.6
Yard (Upwind)	6	2.2	7	0.3

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

Marine Dock Exposure

- Ro-ro ferry, Great Britain (Groves and Cain, 2000)
 - ◆ Elemental Carbon GM = $37 \mu\text{g}/\text{m}^3$ (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: $80 (+/-20) \mu\text{g}/\text{m}^3$ Ship 2: $250 (+/-40) \mu\text{g}/\text{m}^3$

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



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

- ◆ FEV_1 = Volume exhaled in 1 second
- ◆ FVC = 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 $\mu\text{g}/\text{m}^3$ 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

Salvi et al. *Am J Respir Crit Care Med*
1999; 159: 702-709.

Acute Health Effects: Bus Garage

- 232 male workers in four garages
- Workers $\geq 310 \mu\text{g}/\text{m}^3$ respirable particles reported more cough, itchy or burning eyes, chest constriction, wheeze, difficulty breathing
- No significant shift related changes in FEV_1
- Smoking and age adjusted

Gamble J et al. *Environ Res* 1987; 42:201-214.

Acute Health Effects: Miners

- 150 underground coal miners - diesel/non-diesel
- Diesel mine respirable dust = 2.0 ± 1.7 mg/m³
- Non-diesel mine respirable dust = 1.4 ± 1.5 mg/m³
- Similar shift related changes in both groups

Ames RG et al. *Am Rev Respir Dis* 1982; 125: 39-42.

Acute Health Effects: Stevedores

- 23 stevedores exposed to diesel truck exhaust after 10 days off work
- No control group
- Respirable dust 130 - 590 $\mu\text{g}/\text{m}^3$ (?contribution of diesel)
- Shift related changes in FEV_1 and FVC

Ulfvarson et al. *Scand J Work Environ Health*
1987; 13: 505-512.

Acute Health Effects: Children

- Lung function testing in 1,092 children who lived near highways
- 4% reduction in FEV₁ 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

Brunekreef et al. *Epidemiology* 1997; 8: 298-303.

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

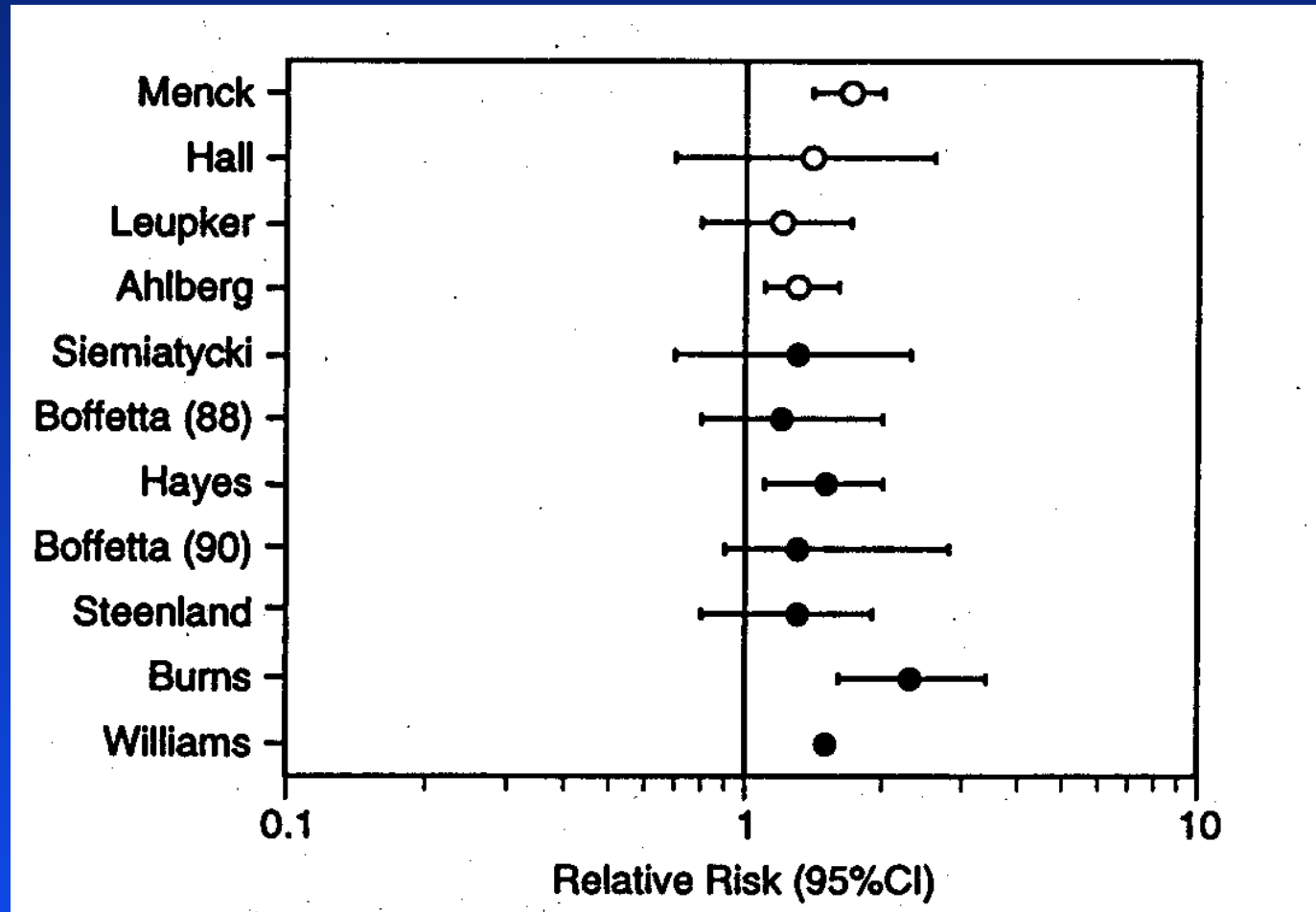
- Dose related increase in lung tumors at high levels of diesel exhaust exposure ($3500 \mu\text{g}/\text{m}^3$) in rats
- Results can be reproduced by inhalation of other insoluble particles without associated organics
- Relevance to humans occupationally exposed at lower concentrations is uncertain

Lung Cancer

Epidemiological Studies in Humans

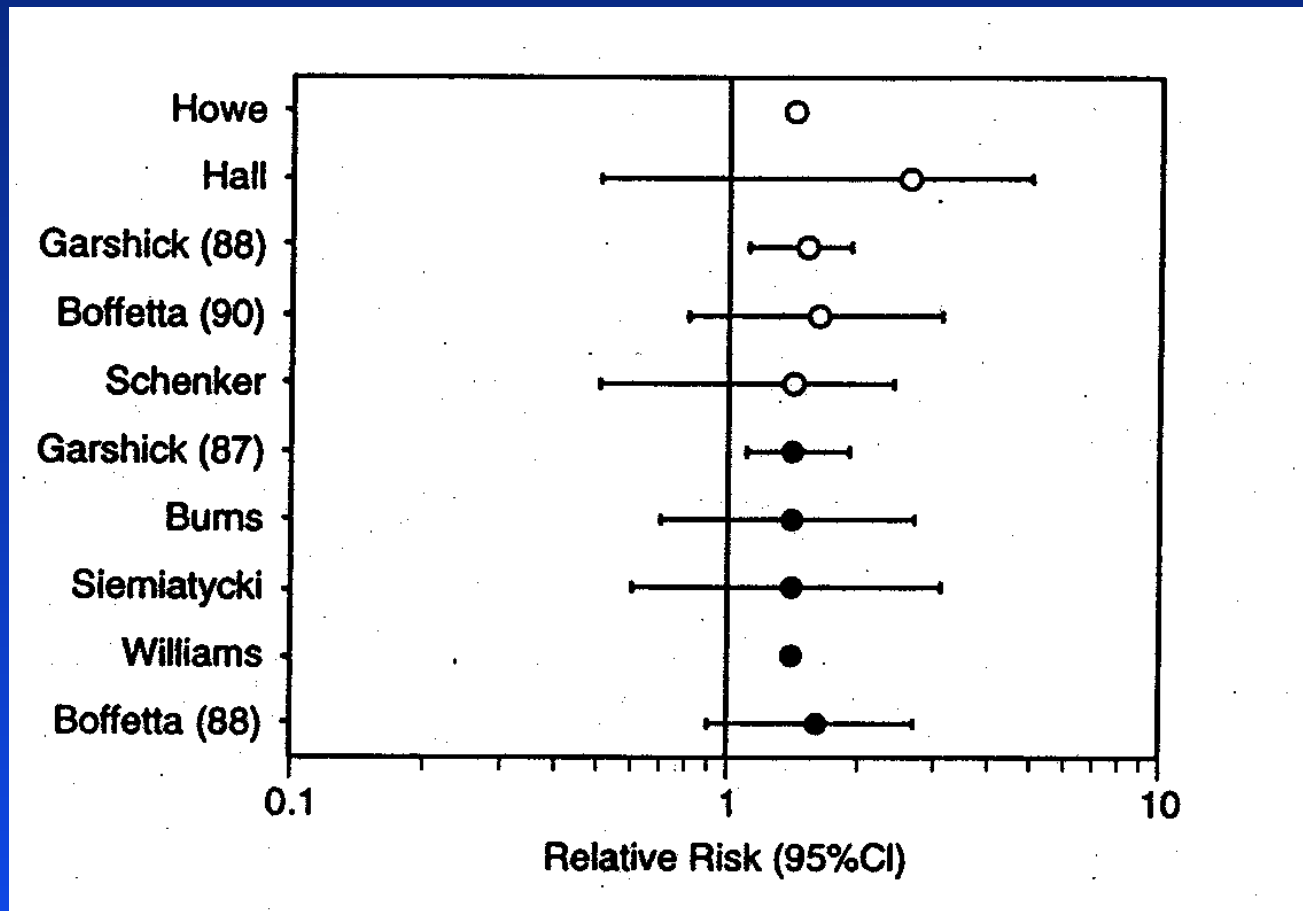
- >30 studies
- Truck Drivers
- Railroad Workers
- Bus Garage And Transport Workers
- Dock Workers
- General Population Groups

Truck Driver Studies



solid circle = smoking adjusted open circle = smoking unadjusted

Railroad Worker Studies



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 $\mu\text{g}/\text{m}^3$ 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 $\text{PM}_{2.5}$ rather than by larger particles
- Biologic mechanism unclear* Adapted from Pope & Dockery, 1999

Chronic Exposure and Mortality

Most vs Least Polluted City, Smoking Adjusted

	Six Cities PM _{2.5}	ACS PM _{2.5}	ACS SO ₄
All Mortality	1.26 (1.08-1.47)	1.17 (1.09-1.26)	1.15 (1.09-1.22)
Cardio-pulmonary	1.37 (1.11-1.68)	1.31 (1.17-1.46)	1.26 (1.16-1.37)
Lung cancer	1.37 (0.81-2.31)	1.03 (0.80-1.33)	1.36 (1.11-1.66)
All others	1.01	1.07	1.01

Association of Fine Particulate Matter from Different Sources with Daily Mortality in Six U.S. Cities

*% Increase in Daily Deaths (95% CI)
with 10 $\mu\text{g}/\text{m}^3$ Increase in $\text{PM}_{2.5}$*

Crustal (Si) -2.3 (-5.8, 1.2)

Motor (Pb) 3.4 (1.7, 5.2)

Coal (Se) 1.1 (0.3, 2.0)

Laden et al. *Environ Health Perspect* 108:941-947, 2000.

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
- Assess lung cancer mortality in 55,750 trucking company workers 1985-2000
- Characterize current exposure
 - ◆ $PM_{2.5}$, EC, source apportionment
- Estimate previous exposures
- Funded by National Cancer Institute 2001-2006