COMPONENTS RESPONSIBLE FOR THE HEALTH EFFECTS OF INHALED ENGINE EMISSIONS

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Description of LRRI Engine Health Effects Programs

Objectives:

1. **Compare health hazards of competing existing technologies**

2. **Determine contributions of emissions components to hazards**

3. **Evaluate health benefits of emission reductions**

4. **Evaluate emerging technologies for unintended consequences**

Key activities:

- **Determining the importance of non-PM components**

    Importance of vapor-phase SVOCs

    Vascular effects of non-PM gasoline emissions

    Vascular effects of CO and NO_2

- **Determining the importance of lube oil emissions**

- **Clarifying the importance of particles**

    EC-based "soot"

    Organic nanocondensates

- **Demonstrating the benefits of emission reductions**

    Retrofit (LS fuel + catalyzed trap)

    2007-compliant on-road diesel (ACES)

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DISCOVERED IMPORTANCE OF VAPOR-PHASE SEMI-VOLATILE ORGANIC COMPOUNDS

1. **Collected PM (filter mass) and vapor-phase SVOC (PUF/XAD trap) from "truck" bore of Baltimore harbor traffic tunnel**

2. **Instilled the 2 phases, separately and combined, into rat lungs**

3. **Measured Inflammatory responses in lung 24 hr later**
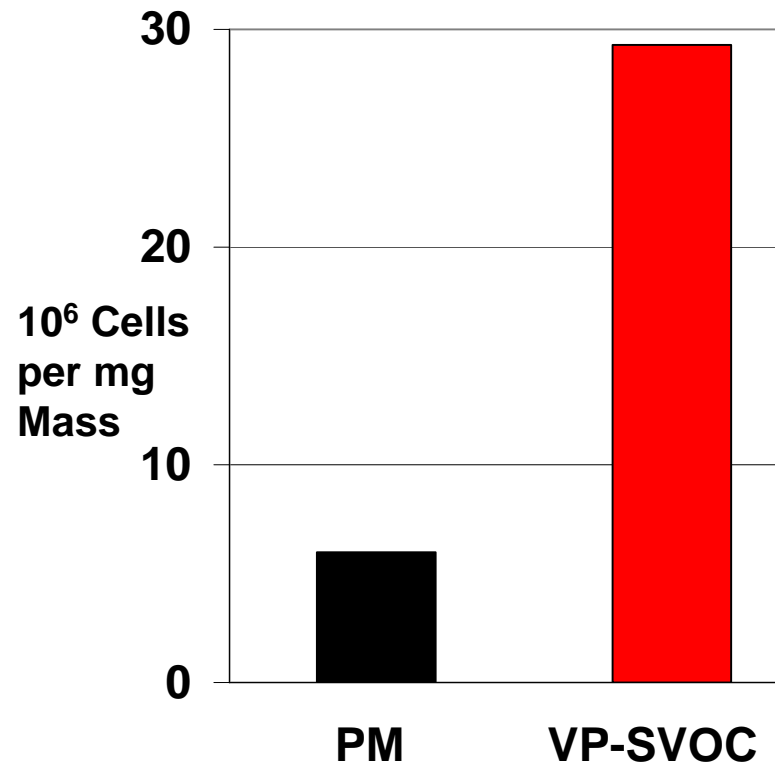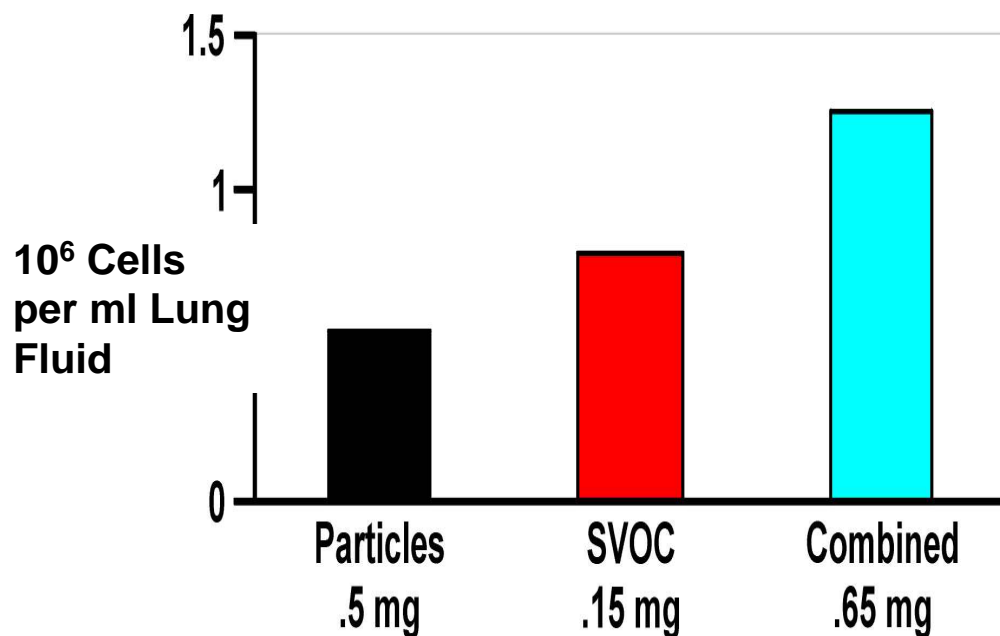
Per unit of mass, the vapor-phase SVOC was ~ 5x more toxic than PM !!

Seagrave et al., *Toxicologist* 60:192, 2001

EXAMINED RELATIVE TOXICITY OF DIESEL AND GASOLINE EMISSIONS – AND CAUSAL COMPONENTS

- **Collected PM and VP-SVOC from in-use vehicles on chassis dyno.**

- **Extracted from media** (filters & PUF/XAD) **and analyzed composition**

- **Instilled extracts into rat lungs, and lavaged at 24 hr**

- **Analyzed composition-toxicity relationships by PCA/PLS**

Samples

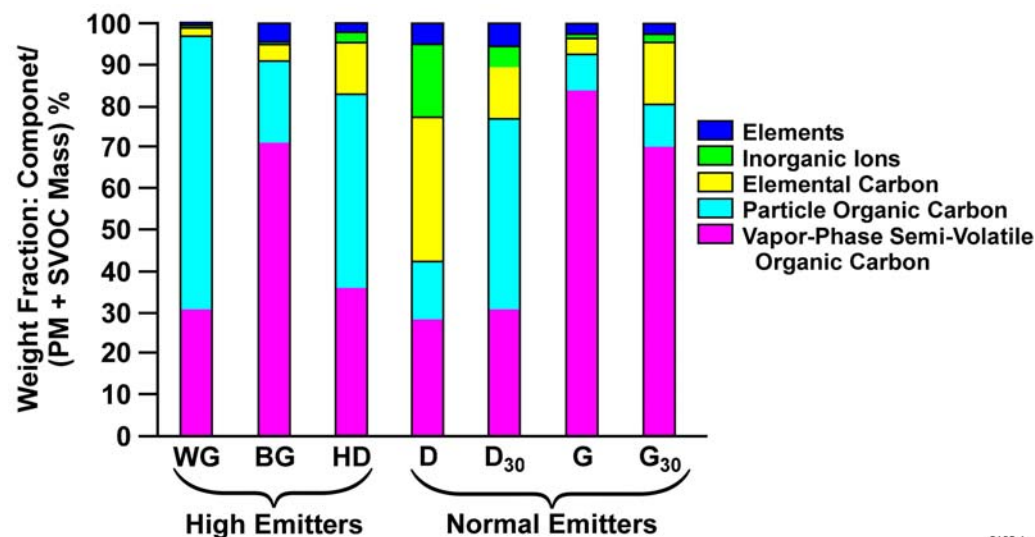|	
Gasoline (5)	**G**
Gasoline 30°	**G$_{30}$**
White smoker gas.	**WG**
Black smoker gas.	**BG**
Diesel (3)	**D**
Diesel 30°	**D$_{30}$**
High-emitter diesel	**HD**

Composition

Seagrave et al. *Toxicol. Sci.* **70:** 212-226, 2002

Zielinska et al., *J. Air Waste Man. Assoc.* **54:** 1138-1150, 2004

THE SAMPLES HAD A 5-FOLD RANGE OF TOXICITY AND TOXICITY WAS LINKED TO COMPOSITION

Relative Inflammatory Potential

Emissions from high-emitters were more toxic per unit of mass !!
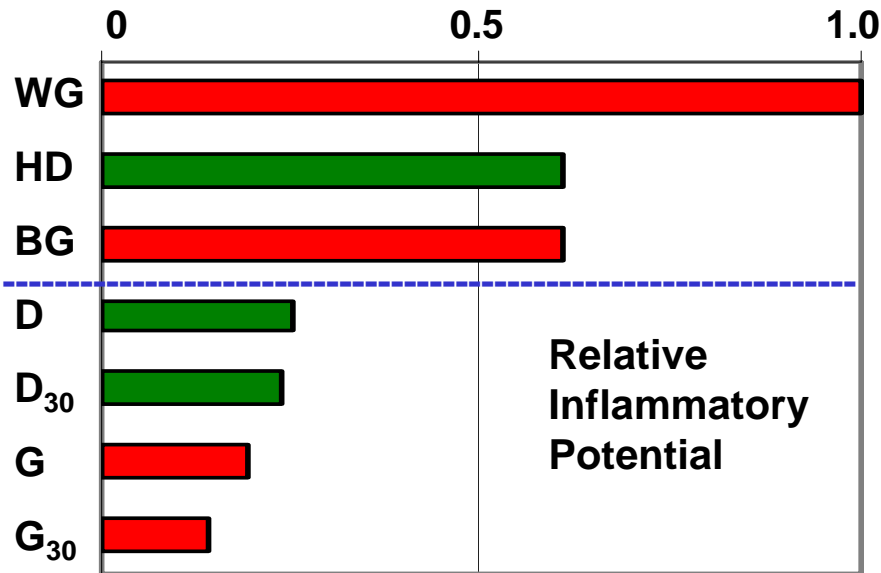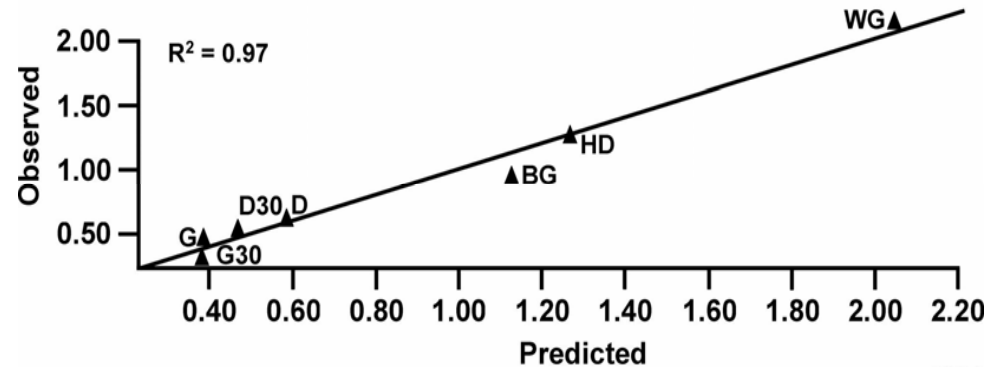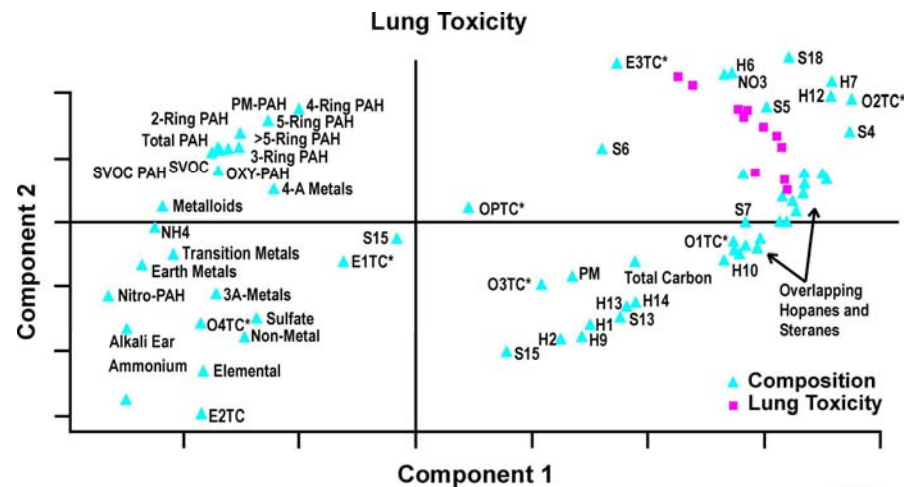

PCA/PLS could model relative toxicity from composition

Hopanes & steranes, markers of <u>crankcase lube oil</u>, were most closely linked to toxicity !!

Same results from high-emitter, normal-emitter, and new technology CNG buses

McDonald et al., *Environ Health Perspect.* 112: 1527-1538, 2004

Seagrave et al., *Toxicol. Sci.* 87:323-241, 2005

EXAMINING THE HEALTH IMPORTANCE OF OIL-DERIVED AND SULFATE CONDENSATE NANOPARTICLES

- **Aerosolized by vaporization-condensation**

  1. **New and used diesel crankcase oil**

     Shell Rotella-T® 10W-40

     2000 Cummins 5.9L ISB on HD cert. cycle

  2. **Sulfate**

     Sulfuric acid

- **Exposed mice by inhalation**

  6 hr/day x 7 days

  15-25 nm particles at 10^6 particles/cc
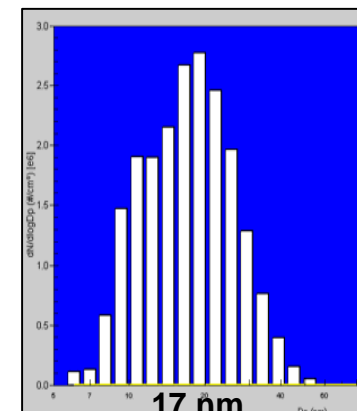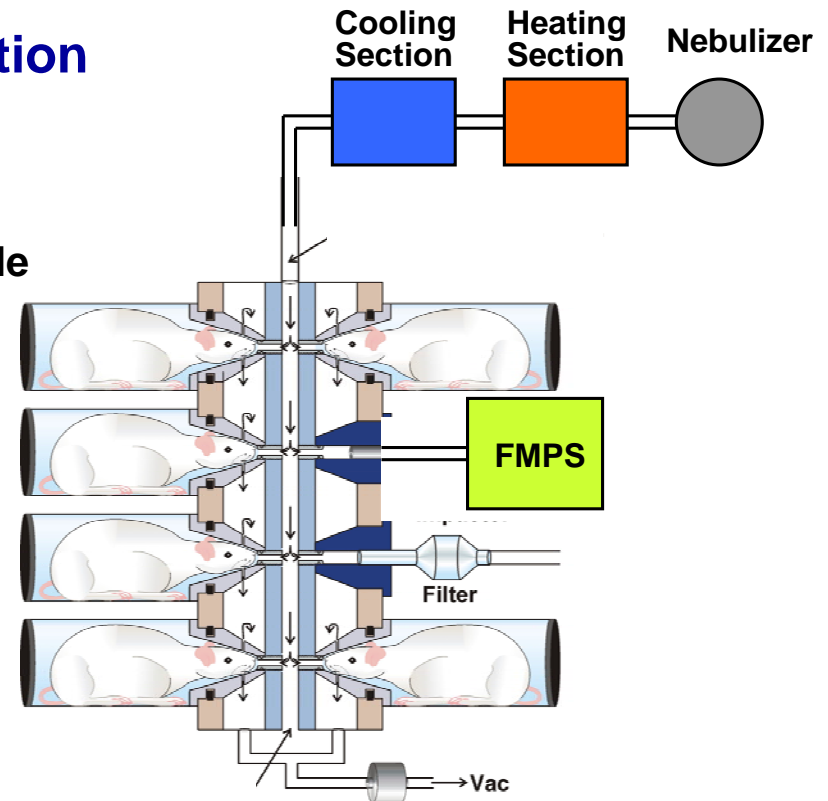
- **Measured:**

  Lung inflammation

  Lung tissue oxidative stress

  Lung histopathology

  Function of systemic immune system

  Cell proliferation

  Antibody formation

Cooling Section **Heating Section** **Nebulizer**

FMPS

Filter

→ Vac

17 nm

McDonald et al. *Toxicologist* 96:230, 2007

RESPONSES TO HIGH LEVELS OF "NANOCONDENSATES"

Exposure did <u>not</u> cause:

Significant indications of lung inflammation

Although some indicators showed small effects

Detectable lung pathology

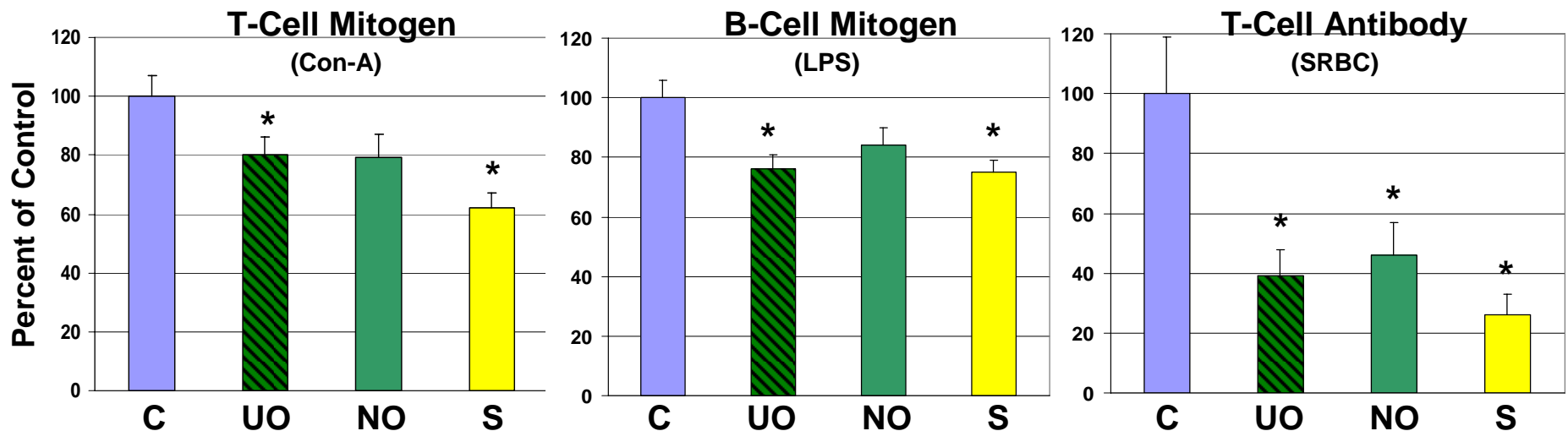**Indications of oxidative stress in plasma**

Exposure <u>did</u> cause:

Oxidative stress to lung tissue

Reduced function of immune cells <u>outside</u> the lung

Reduced cell division in response to stimulus

Reduced formation of antibody to foreign protein

Mitchell et al. New Data, and McDonald et al. *Toxicologist* 96:230, 2007

ISSUES AND NEXT STEPS FOR NANOCONDENSATES

Issues:

1. **Would these effects occur at lower exposure levels?**

2. **What is the significance of the immune effects?**
    Imply suppression of immune function
    Tend to reduce defenses against infections

Next Steps:

1. **Repeat at lower exposure level**
    Indicators showing significant effects
    May not have funds to do this

2. **Evaluate mechanism of immune suppression**
    Trying to do this in another program

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DISCOVERED THAT NON-PM EMISSIONS FROM GASOLINE ENGINES CAN HAVE CARDIOVASCULAR EFFECTS

LA92 "Unified" Dynamometer Driving Schedule

Sample Period = 1435 seconds - Distance = 9.82 miles - Average Speed = 24.61 mph

1996 4.3 L General Motors V-6 engines

- 3 in-use Chevrolet S-10 pickup trucks
- Mid-range mileage (40-70k miles)
- Normal emissions

(California) Unified Driving Cycle

- 3-phase cycle mapped from chassis dynamometer to engine stand
- 2 engines used for 2 cold starts/day

Gasoline blended to 2002 U.S. national average regular unleaded

- No added oxygenates
- Reid vapor pressure = 10.3 psia
- 275 ppm sulfur, 30% aromatics

Pennzoil® 10w-30 lube oil
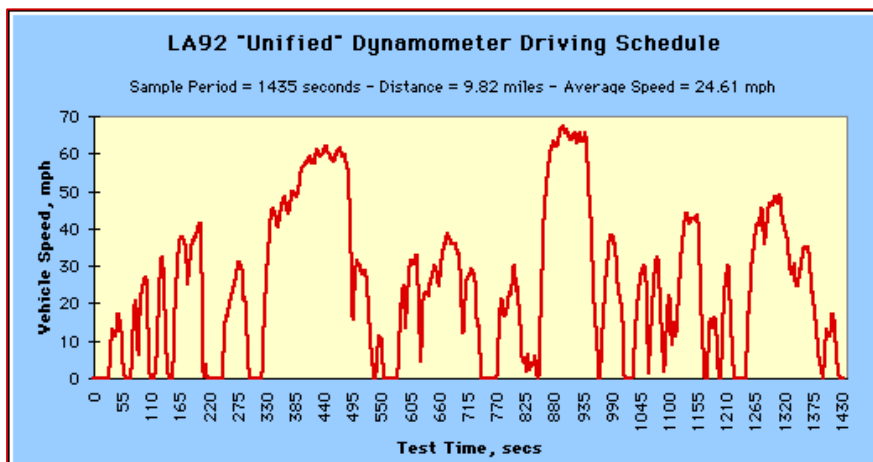
Exposed animals at 1:110 to 1:12 dilutions

- PM $\approx$ 7, 30 & 60 µg/m^3
- CO $\approx$ 8, 50, & 80 ppm
- NOx $\approx$ 2, 10 & 20 ppm

Also <u>filtered</u> emissions at high level

www.nercenter.org

FOUND EFFECTS IN VESSELS NEAR HEART

- **Fed ApoE$^{-/-}$ mice high-fat diet and exposed daily for 7 weeks**
- **Measured indicators of oxidative stress and pro-atherosclerotic remodeling in aorta near heart**

Oxidative stress

Vascular remodeling

1. **Clear evidence of effects in vessels outside the lung**

2. **Non-PM components drove these effects**

3. **Less effects at typical exposure levels**
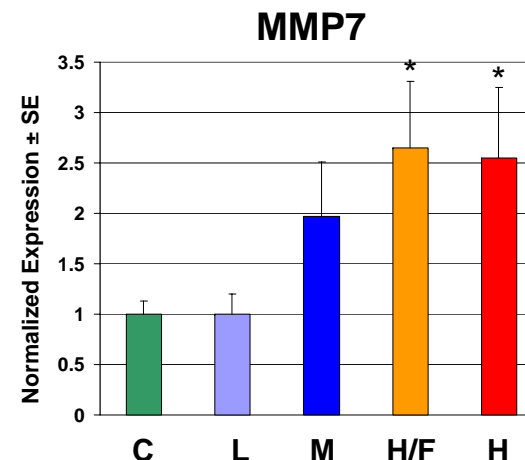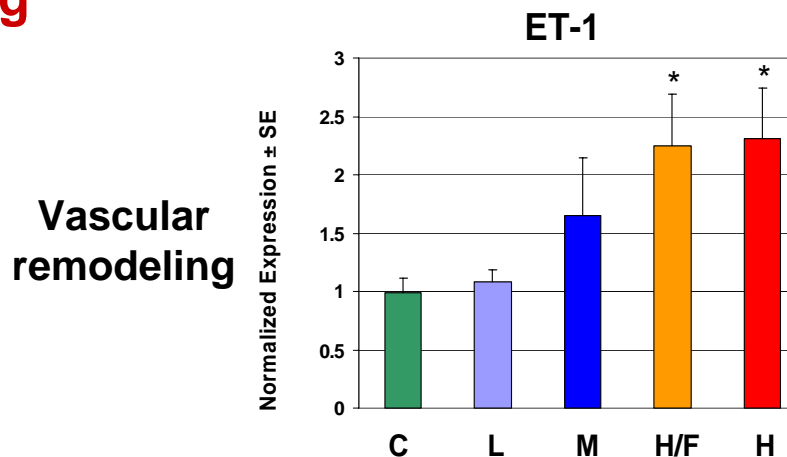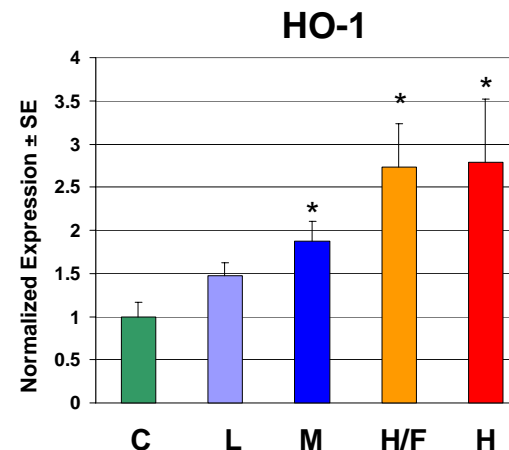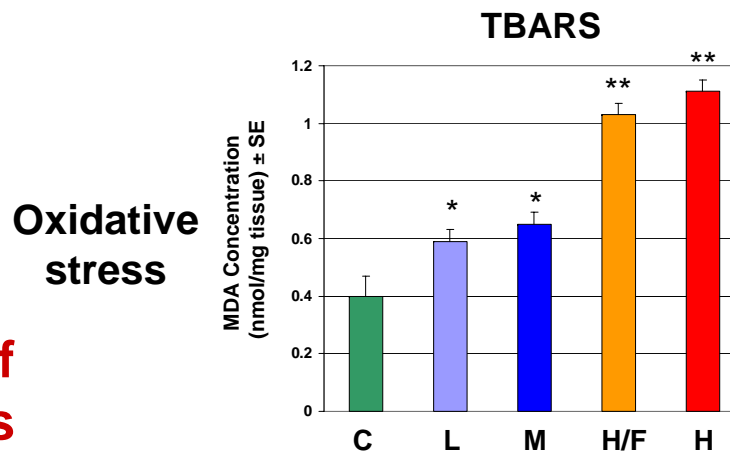
What caused it – NOx? CO? VOCs?

Lund et al. *Toxicol. Sci.* 95:485-494, 2007

FOUND THAT NO AND CO CAUSE SOME, BUT NOT ALL EFFECTS

- **Plausible biological mechanisms suggested that NO, NO_2, or CO might be the primary cause of the vascular effects**
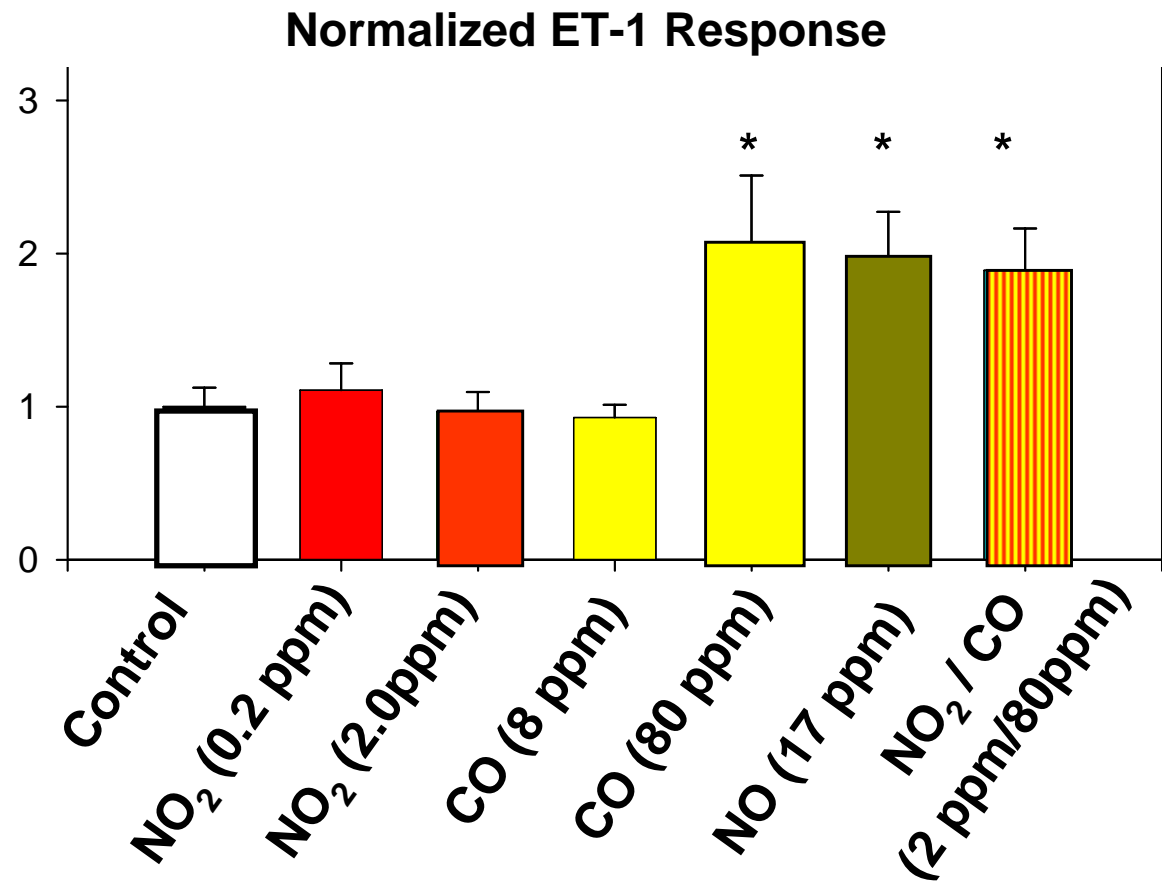
- **Exposed mice to these gases, alone and in combination, for 7 days at the high and low concentrations in the gasoline study**

1. **CO and NO <u>did</u> reproduce effects on some responses**
   (ET-1, HO-1, MMP-9)

2. **NO_2 was <u>not</u> a cause**

3. **CO and NOx <u>did not</u> reproduce effects on other responses**
   (TBARS, TIMP-2)

- **Other non-PM components (VOCs?) are also important**

Normalized ET-1 Response

Bar chart — Normalized ET-1 Response. Y-axis 0 to 3. Categories: Control (~1.0), NO_2 (0.2 ppm) (~1.1), NO_2 (2.0ppm) (~1.0), CO (8 ppm) (~0.9), CO (80 ppm) (~2.1, *), NO (17 ppm) (~2.0, *), NO_2 / CO (2 ppm/80ppm) (~1.9, *).
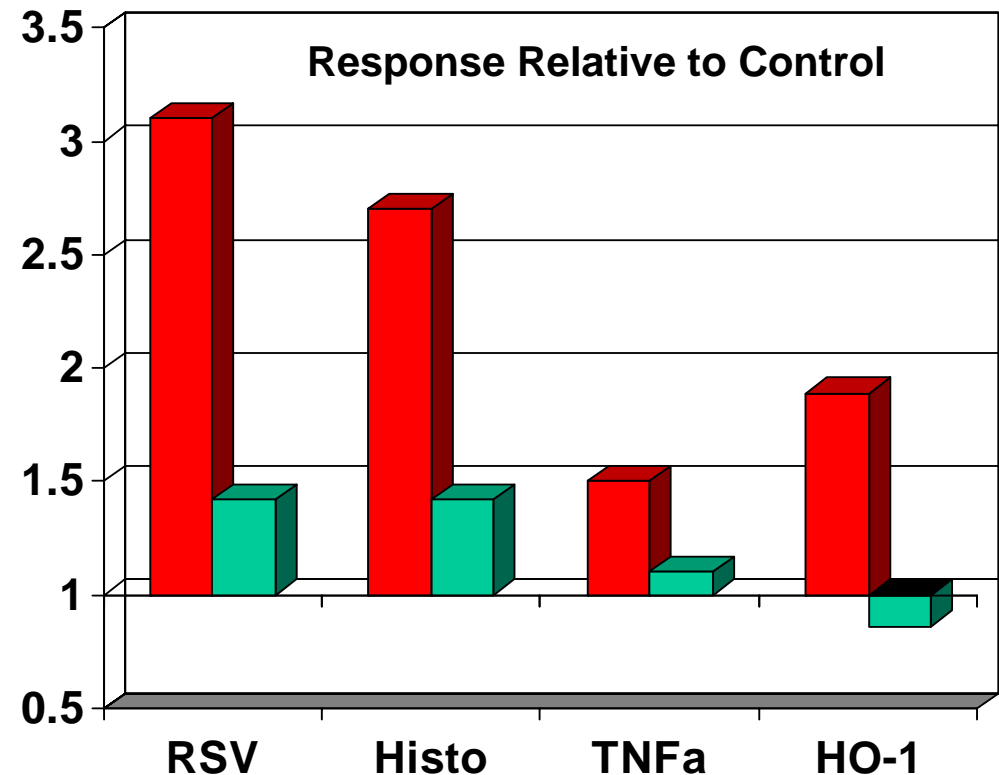
DEMONSTRATED HEALTH BENEFITS OF DIESEL EMISSIONS REDUCTION BY RETROFIT

Evaluated benefits of simulated retrofit by comparing effects of:

350 ppm S pre-2007 cert. fuel
No after-treatment

15 ppm S fuel (BP-15)
Catalyzed PM trap

- **Yanmar YDG5500E diesel generator at constant (full) load and dilution**

- **Exposed C57BL/6 mice 6 hr/d x 7d**

- **Measured:**

  Clearance of Respiratory Syncytial Virus (RSV)

  Histopathology (HISTO)

  Pro-inflammatory cytokine (TNFα)

  Indicator of oxidative tissue stress (heme-oxygenase-1 [HO-1])

Response Relative to Control — bar chart with y-axis from 0.5 to 3.5, categories: RSV, Histo, TNFa, HO-1

Health effects were eliminated or reduced to non-significant levels !!

McDonald et al. *Aerosol Sci. Technol.* 38: 62-78, 2004
McDonald et al., *Environ. Health Perspect.* 112: 1307, 2004

INITIATING LONG-TERM STUDY OF 2007-COMPLIANT HD DIESEL EMISSIONS (ACES)

Exposure:

- **2007-compliant ~400-450 hp engine/after-treatment system**
    - **Engine to be selected from 4 candidates currently being evaluated**
- **Variable-duty cycle on AC dynamometer**
- **2007-compliant** (ultra low sulfur) **petroleum-based fuel**
- **Whole emissions diluted to 3 concentrations** (not yet specified) **+ control**
- **Expose 6 hr/day, 5 days/wk**

Evaluation of Biological Effects

- **2 yr exposure of 166 Wistar rats/group for carcinogenicity** (begin in fall of '08)
- **Interim evaluation of 20 Wistar rats/group at 1, 3, 12, and 24 mo**
    - **Respiratory function**
    - **Bronchoalveolar lavage**
    - **Histopathology and lung cell proliferation**
    - **Additional measurements selected by HEI**
- **3 mo exposure of 120 C57BL/6 mice/group, with evaluation at 1 & 3 mo**
    - **Bronchoalveolar lavage**
    - **Lung histopathology and cell proliferation**
    - **Additional measurements selected by HEI**

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BOTTOM LINES

All combustion emissions cause biological response

- **All competing combustion technologies emit pollutants that can be hazardous at <u>some</u> level**
- **Small differences among current technologies at equal masses**
- **High emitters of all technologies are the main problem**

There is no single "magic bullet"(not even nanoparticles!)

- **Particles, VP-SVOCs, and gases can <u>all</u> have effects**
- **Lube oil emissions are important**
- **Nanoparticles can have effects, but do not drive ALL effects**
- **Hazards are not all in the lung**

Cleaning up diesels is undoubtedly reducing health impacts

- **Retrofits help**
- **2007-2010 on-road emissions are anticipated to have much reduced impact**