COMPONENTS RESPONSIBLE FOR THE HEALTH EFFECTS OF INHALED ENGINE EMISSIONS

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HEALTH IMPACTS ACTIVITY
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KEY ISSUES

- PM vs. non-PM
- Lube oil
- Nano-PM
- Benefits of emission reductions
DISCOVERED IMPORTANCE OF VAPOR-PHASE SEMI-VOLATILE ORGANIC COMPOUNDS

1. PM and vapor-phase SVOC from truck bore of Baltimore harbor tunnel
2. Instilled into rat lungs
3. Measured inflammatory responses in lung

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

- PM and VP-SVOC from in-use vehicles on chassis dyno.
- Analyzed composition
- Instilled combined fractions into rat lungs
- Analyzed composition-toxicity relationships

**Samples**

- Gasoline (5) G
- Gasoline 30° G₃₀
- White smoker gas. WG
- Black smoker gas. BG
- Diesel (3) D
- Diesel 30° D₃₀
- High-emitter diesel HD

**Composition**

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

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

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

Same results from CNG buses

McDonald et al., Environ Health Perspect. 112: 1527-1538, 2004
Seagrave et al., Toxicol. Sci. 87:323-241, 2005
DISCOVERED THAT NON-PM EMISSIONS FROM GASOLINE ENGINES CAN HAVE CARDIOVASCULAR EFFECTS

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 ≈ 7, 30 & 60 µg/m³
CO ≈ 8, 50, & 80 ppm
NOx ≈ 2, 10 & 20 ppm

Also **filtered** emissions at high level

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FOUND EFFECTS IN VESSELS NEAR HEART

- Exposed ApoE\textsuperscript{-/-} mice for 7 weeks
- Measured responses in aorta near heart

1. Effects in vessels outside the lung
2. Non-PM components drove these effects

What caused it – NO\textsubscript{x}? CO? VOCs?

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

Exposed mice to these gases, alone and in combination at the high and low concentrations in the gasoline study

1. CO and NO did reproduce effects on some responses (ET-1, HO-1, MMP-9)

2. NO₂ was not a cause

3. CO and NOx did not reproduce effects on other responses (TBARS, TIMP-2)

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

Campen, Lund et al., new unpublished results
EXAMINED THE HEALTH IMPORTANCE OF OIL-DERIVED AND SULFATE 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

McDonald et al. Toxicologist 96:230, 2007
RESPONSES TO HIGH LEVELS OF “NANOCONDENSATES”

Exposure did not cause:
- Significant indications of lung inflammation
- Detectable lung pathology
- Indications of oxidative stress in plasma

Exposure did cause:
- Some stress to lung tissue
- Reduced function of immune cells outside the lung
- Reduced cell division in response to stimulus
- Reduced formation of antibody to foreign protein

T-Cell Mitogen (Con-A)
- C
- UO
- NO
- S

B-Cell Mitogen (LPS)
- C
- UO
- NO
- S

T-Cell Antibody (SRBC)
- C
- UO
- NO
- S

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 same load and dilution
- Exposed 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])

Health effects were eliminated or reduced to non-significant levels

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 in Phase 1 at SwRI

- **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
The problem is emissions - not diesel emissions

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

There is no single “magic bullet”

- Particles, VP-SVOCs, and gases can all have effects
- Lube oil emissions are important
- Nanoparticles can have effects
- Hazards are not all in the lung

Cleaning up diesels is undoubtedly reducing health impacts

- Retrofits help
- 2007-2010 “on-road” emissions ???