Advanced Collaborative Emissions Study (ACES)

Cooperative multi-party effort to characterize emissions and possible health effects of new advanced heavy duty engine and control systems and fuels in the market 2007 – 2010.

DOE Merit Review June 2012
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¹Health Effects Institute (HEI), ²Lovelace Respiratory Research Institute, and ³Coordinating Research Council (CRC)

This presentation does not contain any proprietary or confidential information

ID # ACE044
NETL Agreement 13919
PROJECT OVERVIEW

Phases:
1. 2007 Engine Emissions Characterization (Southwest Research Institute® (SWRI®))
   – CRC Technical Leader
2. 2010 Engine Emissions Characterization
   – CRC Technical Leader
3. 2007/2010 Engine Health Effects Testing (Lovelace Respiratory Research Institute (LRRI)
   – Short Term biological screening and Long-Term Health Effects Test on 2007 Engines
   – HEI Technical Leader ; CRC Technical Monitor

Funding
Overall Project: $15.5 million
• Total DOE Contract: $5.95 million (Contractor Share: $3.98 million)
  – FY 11 DOE Funding: $500,000
  – FY 12 DOE Funding: $500,000 (Planned)

Partners
• DOE OVT and NETL
• Engine Manufacturers Association (EMA)
• US Environmental Protection Agency (EPA)
• California Air Resources Board (ARB)
• American Petroleum Institute (API)
• After-treatment Manufacturers
• Coordinating Research Council (CRC)

Overall Project Timeline
Some delays in Phase 2

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<th>2011</th>
<th>2012</th>
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RELEVANCE AND BARRIERS:
Evaluating Emissions of Advanced Technology Diesels

• **DOE OVT MYPP Advanced Combustion R and D**: New Generation diesel is highly fuel efficient and a likely significant contributor to enhanced fuel economy for the next 15 – 20 years
  – IF they gain wide acceptance

• The combination of advanced-technology engines, aftertreatment systems, reformulated fuels, and reformulated oils developed to meet the 2007/2010 emission standards will result in substantially reduced emissions.

• With any new technology it is prudent to conduct research to confirm benefits and to ensure that there are no adverse impacts to public health and welfare.

• **Major Challenge/Barrier:**
  – Substantial public health benefits and enhanced public acceptance and use are expected from these reductions.
  – *Key upcoming reviews of diesel and cancer at International Agency for Research on Cancer (IARC) and U.S. National Toxicology Program*
    – But, must test engines in most realistic way possible to distinguish, with rigorous cycles and top quality science, new diesel technology from old

**Overall Objective**

• to characterize emissions and possible health effects of new advanced heavy duty engine and control systems and fuels in the market 2007 – 2010
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<th>Name</th>
<th>Organization</th>
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<tr>
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<td>Joseph H. Somers</td>
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<td>Hector Maldonado</td>
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<td>M. Matti Maricq</td>
<td>Ford Motor Company</td>
<td>Ken Wright</td>
<td>ConocoPhillips</td>
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ACES Phase I – 2007 Engines

APPROACH

• Quantify the significant reduction in both regulated and unregulated emissions from advanced diesel engines
• Provide regulated and unregulated emissions for this new engine technology,
• Provide initial guidance for ACES Phase 3 health study using the regulated and unregulated emissions information from ACES Phase 1
• Heavy Heavy Duty (Class 8) Engines from: Caterpillar, Cummins, Detroit Diesel, and Volvo

RESULTS

• Regulated PM, CO, and NMHC emissions were at least 90% below the 2007 standard, and NO$_x$ was 10% below standard
• Most unregulated emissions at least 90% below 2004 technology
• Average NO$_2$ emission of 0.68 g/hp-hr was 2 to 7 times higher than the emissions from 2004 engines
  – However, 2010 engine technology NO$_x$ limit of 0.20 g/hp-hr will force NO$_2$ emissions to be substantially lower than both 2007 and 2004 technology engines
• Particle number emissions average was at least 90% below 2004 technology engines, even when DPF regeneration occurred
• Elemental carbon represented only 7% of total PM mass, and the hydrated sulfuric acid determined from measured sulfate was the dominant PM component for the 16-Hour Cycle, 70 percent of total PM mass
• Comprehensive final report issued June 30, 2009; JAWMA Paper April 2011
ACES PHASE 2: APPROACH AND OBJECTIVES
2010 Compliant Engines

- 2010 engines will offer substantial improvements in NOx emissions

- Phase 2, in progress, is conducting Emissions Characterization

- Engines are being prepared (degreened) by three manufacturers for availability in Spring 2012

- SwRI® is preparing detailed program plan and test cell for testing to commence in Spring 2012

- Final reporting on Phase 2 characterization study anticipated in Spring 2013
ACES PHASE 3 APPROACH AND OBJECTIVES

Health Bioscreening

Phase 3A: Characterization of emissions and exposure atmospheres
Phase 3B: Conduct of animal bioscreening studies

DOE Funding:
- Characterization of animal exposures
- 3 month mouse pulmonary bioscreening

EPA Funding (leveraged by DOE investment):
- Long-term rat carcinogenesis bioassay
- Pulmonary bioscreening at 1, 3, 12 & 24 mo
**PHASE 3A – TECHNICAL APPROACH**

- 2007-compliant “engine B'” (selected from four candidates)
  - Confirmed that engine/control systems met performance criteria
    
    Steady-state (SS) and Federal Test Procedure (FTP) cycles
    16-hr ACES cycle (4 repeats of 4 hr cycle with cold start)

- Evaluated diluted emissions in empty animal chamber, and
  compared to SwRI® results (using same fuel)
  
  - Emissions = exhaust + crankcase blow-by
  - FTP, SS modes 1, 3 & 5, ACES cycle
  - Constant pressure primary dilution tunnel

- Determined dilutions required to meet targets set by HEI
  
  - Dilutions set to achieve 4.2, 0.8 & 0.1 ppm NO₂
  - Dilutions $\approx 40:1$, 210:1 & 1680:1

- PM levels are very low; study may primarily detect effects of NO₂ if any effects are seen

- *Report published as HEI Communications 17 in February 2012*
PHASE 3A – TECHNICAL APPROACH

Engine and Primary Dilution System

- Engine
- Engine Exhaust Tunnel
- Fuel “Day Tank”
- Combustion and dilution Air Supply
- Engine Cooling heat Exchanger
- Engine Exhaust Injection Point
- Dynomometer

Exhaust Extraction and Secondary Dilution Systems

- Main Exhust Duct
- MAIN SUPPLY AIR DUCT
- MAIN EXHAUST DUCT
- H-2000 EXPOSURE CHAMBER
- Primary Dilution Tunnel
- 2ndary CHAMBER DILUTION
- Muffler
- Air-Vac
- Extraction probe flange
- Compressed air

(Note: Drawing is not to scale)
<table>
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<tr>
<th>Gases:</th>
<th>High Mean</th>
<th>High Stdev</th>
<th>Mid Mean</th>
<th>Mid Stdev</th>
<th>Low Mean</th>
<th>Low Stdev</th>
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<td>NO₂ (ppm)</td>
<td>4.2</td>
<td>0.5</td>
<td>0.91</td>
<td>0.11</td>
<td>0.109</td>
<td>0.013</td>
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<td>NO (ppm)</td>
<td>5.8</td>
<td>1.1</td>
<td>1.40</td>
<td>0.23</td>
<td>0.293</td>
<td>0.160</td>
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<td>NOₓ (ppm)</td>
<td>9.9</td>
<td>1.4</td>
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<td>0.29</td>
<td>0.402</td>
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<td>CO (ppm)</td>
<td>6.8</td>
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<td>THC (ppm)</td>
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<td>SO₂</td>
<td>23.9</td>
<td>4.4</td>
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<th>Inlet (filter)</th>
<th>Chamber</th>
<th>Chamber (filter)</th>
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<td></td>
<td></td>
<td>2</td>
<td>1</td>
<td>12</td>
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PHASE 3A - RESULTS
ATMOSPHERE COMPOSITION

Real-time particle mass

Real-time particle number
PHASE 3A – RESULTS

EXPOSURE COMPOSITION

Exposure Atmosphere Compositions

Particle Mass
Nitrogen Oxide
Nitrogen Dioxide
Carbon Monoxide
Sulphur Dioxide
Volatile Organic Compounds

Volatile Organics

Aromatic
Alkynes
Alkenes
Alkanes

Particle Composition

Elements
Nitrate
Sulphate
Ammonium
Organic Carbon
Elemental Carbon

Polars (Semi-Volatile Organics)

Sugar
Sterol
Phenol
Acids

Semi-Volatile Organics

Hopane & Steranes
Nitro-PAH
PAH
Alkanes
Polars
PHASE 3B – APPROACH AND STATUS
CORE BIOSCREENING STUDY

APPROACH

3-Month Exposure of C57BL/6 Mice at LRRI:
• Expose 120/group 16 hr/day, 5 days/wk for 3 months (13 wk)

Chronic Carcinogenicity Bioassay of Wistar Han Rats at LRRI:
• Expose 288/group 16 hr/day, 5 days/wk for 24-30 months

Accommodate ancillary biological studies of rats and mice
• Markers of potential Cancer, vascular inflammation effects

STATUS

• 1- and 3-month exposures of both rats and mice complete; Rats now in Month 22 of long term exposure
• 1- and 3-month Health Evaluations complete at LRRI and all Ancillary Study Sites
• Some 12-month Health Evaluations complete at LRRI
• Revised reports reviewed and now in press (3 KEY reports to be released in April 2012, (in time for IARC); 4th report in September)
• Mice and rats generally healthy and gaining weight as expected
  • Chronic study expected to be extended to 30 months
PHASE 3B - RESULTS
Key Findings at LRRI

The majority of the analyses showed no difference between diesel exhaust exposure and clean air control.

**Histopathology** analysis revealed mild/minimal exposure-related hyperplasia in the rats after 3 months of exposure, but not in mice. The hyperplasia increased at 12 months, but was still considered mild/minimal severity.

Statistically significant findings were noted for several indicators of oxidative stress and inflammation in rats and mice at 3 months (fewer findings in mice).

**Pulmonary function** assessments in rats showed slight differences in exposed rats compared with control after 3 and 12 months of exposure.

*Note*: When designing the study, it was expected that at the high concentration (at 4.2 ppm NO₂) some NO₂-related effects may be observed. Results so far are not inconsistent with that.
### PHASE 3B - RESULTS
### HISTOPATHOLOGY IN MALE RATS AT 3 AND 12 MONTHS

**Incidence and Types of Findings**  (Note all findings considered minimal/mild severity (~1 on 1-4 scale))

#### Males 3 Month

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<tr>
<th>Lung</th>
<th>Control</th>
<th>Low</th>
<th>Mid</th>
<th>High</th>
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<td>Hyperplasia Epithelium Periacinar</td>
<td>0/10</td>
<td>0/10</td>
<td>0/10</td>
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<tr>
<td>Accumulation Macrophage</td>
<td>0/10</td>
<td>0/10</td>
<td>0/10</td>
<td>3/10</td>
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<tr>
<td>Fibrosis Interstitial</td>
<td>0/10</td>
<td>0/10</td>
<td>0/10</td>
<td>4/10</td>
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#### Males 12 Month

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<th>High</th>
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<tr>
<td>Hyperplasia Epithelium Periacinar</td>
<td>0/10</td>
<td>0/10</td>
<td>0/10</td>
<td>10/10</td>
</tr>
<tr>
<td>Accumulation Macrophage</td>
<td>0/10</td>
<td>0/10</td>
<td>0/10</td>
<td>Not evaluated yet</td>
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<tr>
<td>Fibrosis Interstitial</td>
<td>0/10</td>
<td>0/10</td>
<td>0/10</td>
<td>10/10</td>
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Initial read shows similar incidence trend at 12 months for Hyperplasia. However, increased fibrotic tissue found.
PHASE 3B - RESULTS

INFLAMMATORY/OXIDANT RESPONSE IN RATS

**Total Protein (mg/dL)**
- Control: 5 mg/dL
- Low: 5 mg/dL
- Mid: 10 mg/dL
- High: 15 mg/dL

*p<0.01

**Albumin (µg/mL)**
- Control: 20 µg/mL
- Low: 20 µg/mL
- Mid: 40 µg/mL
- High: 80 µg/mL

*p<0.01

**TEAC (µM)**
- Control: 40 µM
- Low: 40 µM
- Mid: 40 µM
- High: 40 µM

*p<0.01

**HO-1 (ng/mg Protein)**
- Control: 0.5 ng/mg Protein
- Low: 0.5 ng/mg Protein
- Mid: 1.5 ng/mg Protein
- High: 2.0 ng/mg Protein

*p<0.05

**p<0.01

**p<0.01

16
PHASE 3B - RESULTS
RESPIRATORY FUNCTION IN RATS

Significant (p<0.05) trend observed for DLCO/kg at 3 and 12 months

Only reached significance when genders were combined in analysis

No statistically significant findings for Other parameters at 12 months (FVC and MMEF decreased slightly at 3 months only).
Role of NO$_2$ in Observed Effects?

When HEI designed the study, it was expected that at the high concentration (16 hr/day 4.2 ppm NO$_2$) some NO$_2$-related effects may be observed. This was based on results of previous studies, including:

HEI Study (Mauderly et al., 1989) F344 rats exposed (7hr/day, 5 days/week) to 9.5 ppm NO$_2$

- Pulmonary function, histopathology, and, immune response assessed after 12, 18, 24 mo (1820, 2730, 3640 hr) of exposure

**Findings:** NO$_2$ caused epithelial hyperplasia, thickening of walls of terminal bronchioles, inflammation, and oxidative stress. There was little effect on respiratory function.

Effects at 12 mo not significantly different than at 24 months

How do the NO$_2$ “doses” compare at 12 mo?

- Mauderly et al: 17,290 ppm-hr.
- ACES: 17,472 ppm-hr
**PHASE 3B - RESULTS**

**GENOTOXICITY STUDIES - BEMIS**

**Endpoint:** Micronucleus formation.

**Result:** No significant changes observed in mice and rats exposed for 1 or 3 months.

![Graphs showing micronucleus formation](image)

Figure 4. Mean frequencies of micronucleated reticulocytes (MN-RET) and micronucleated normochromic erythrocytes (MN-NCE) in male (gray bar) and female (black bar) mice exposed to diesel exhaust. Error bars are standard error of the mean.
PHASE 3B - RESULTS
GENOTOXICITY STUDIES – HALLBERG

**Endpoints:** DNA Damage (Comet assay) and Oxidative Stress (T-BARS)

**Result:** No significant changes observed in mice and rats exposed for 1 or 3 months.
FUTURE WORK

• Phase 2
  – Testing of Three Engines Summer 2012
  – Final Report Spring 2013

• Phase 3
  – Publication of Phase 3B Short term effects Report (April 2012)
  – Completion of 30-month exposures (December 2012)
  – Final Report late 2013

• IARC/NTP
  – Completion and Dissemination of Phase 3 B results by June 2012 IARC meeting
    • Key Question: how will they treat New technology Diesel?
  – Comments to NTP (February 2012; tracking process for review of US Report on carcinogens
SUMMARY

- The progressive changes in the composition and decreases in magnitude of emissions from new technology diesel is changing the landscape of the debate over potential health effects of diesel exhaust.

- 2007 compliant diesel exhaust studied in the ACES program shows low particle emissions. The only measurable particle emissions occur during trap regeneration. The emissions have low volatile and semivolatile organics, and an increase in the proportion of NO$_2$ compared with older technologies.

- Exposures produced no effects in most tests; mild to no response in the mice and minimal inflammatory, tissue remodeling and respiratory function changes in rats.
  - Tissue injury was considered minimal (1 on scale of 1-4)
  - Statistically significant findings observed primarily at high level
  - Respiratory function effects were trends only, and only significant when genders were pooled to enhance statistical power

- No genotoxic effects were observed in mice or rats.

- Communication to key decision forums underway.
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