Advanced Collaborative Emission Study (ACES)

MDC Webinar: Health Effects from Diesel Exhaust
May 21, 2015

Rashid Shaikh, Maria Costantini, and Geoffrey Sunshine
HEATH EFFECTS INSTITUTE
Outline of the Presentation

• Introduction to HEI and to ACES
  – Rashid Shaikh

• Results of ACES Emissions Characterization (Phases 1 and 2)
  – Maria Costantini

• Results of the ACES Health Study (Phase 3)
  – Geoffrey Sunshine

• Overall Conclusions
  – Rashid Shaikh
HEI - The Original Idea

• 1977 Clean Air Act §202(a)(4): Car manufacturers required to test health effects of emissions. However,
  – EPA-Industry relationship was poor, and
  – Science contentious, litigious, expensive, and discredited

• Industry CEOs and EPA Administrator:
  “There must be a better way to produce science …
  [that] we can trust…”

➢ Health Effects Institute:
  □ Credible science for decision Making
HEI: Overview

- HEI is structured to maintain credibility and transparency on scientific issues germane to policy questions:
  - Independent Board of Directors and Expert Science Committees
  - Balanced government and industry funding (EPA, motor vehicle industry, and other government and private groups)
  - All results – both positive and negative – reviewed, reported and commented upon.
  - Focus on science: HEI does not take policy positions
Organizational Structure

• **Board of Directors**
  – Respected senior leaders recognized for integrity, scientific and/or public policy accomplishments
  – Provide overall guidance and establish HEI’s policies

• **Research Committee**
  – Interdisciplinary group of prominent experts
  – Helps HEI with research planning
  – Selects studies for funding and oversees quality and timeliness of research

• **Review Committee**
  – Interdisciplinary group of prominent experts
  – Independently peer-reviews all HEI-funded studies and prepares detailed comments on how research contributes to state of knowledge and implications for policy
  – *Process more intensive than scientific journals*
Key Attributes of HEI

• Sponsors: Joint and balanced funding by government (EPA) and industry (automotive), and other partners
  – Long-term commitment to support research and review activities, agreed to in the “HEI Strategic Plan”

• Sponsors consultations: Research planning (Strategic Plan and specific RFAs, workshops and conferences)
  – Sponsors do not select or oversee studies, or conduct or review results

• Science: Rigorous processes to assure quality, transparency, and independence
Overall Science Strategies

• HEI accomplishes its mission through five principal strategies:
  – Identifies the highest priority research and review needs
  – Funds and oversees high-quality research on priority relevant topics
  – Provides independent review of HEI research; reanalyze data if needed
  – Communicates all results (positive and negative) to public and private decision makers and the scientific community, along with well-considered commentary
  – Develops timely reviews of state of knowledge in critical areas

• Latest “Strategic Plan 2015 – 2020” for recent accomplishments and our five-year plans. Available at www.healtheffects.org
Diesel Engines

• Dominate heavy-duty applications worldwide
• Advantages compared to spark ignition engines:
  – More efficient
  – More durable
  – Less CO₂, CO and hydrocarbons
• Disadvantages:
  – Emissions of soot/particulate matter
  – More NOx (NO₂ and NO)
  – Emissions of other toxic compounds, e.g., PAHs
Toxicity of Diesel Emissions

• 1970s and 1980s:
  – In vitro studies with PM and its extracts ⇒ Mutagenicity
  – Rat inhalation studies with PM ⇒ Carcinogenicity (lung)
  – Epidemiology Studies ⇒ Suggestive of Carcinogenicity (lung)

• World Health Organization (WHO) : International Agency for Research on Cancer (IARC)
  – 1988 Panel: DE is “probably carcinogenic to humans (category 2A)
  – 2012 Panel: DE is a “known human carcinogen” (category 1)

• Other national and regional actions
Regulation of Diesel Emissions

EPA Heavy-Duty Engine Emission Standards

- Steady State Test
- NOx (Unregulated)
- NOx + HC
- Transient Test
- NOx
- PM (Unregulated)
- Urban Bus PM
- PM
- NOx + HC

ACES engines

Model Year

Oxides of Nitrogen (g/bhp-hr)

Particulate Matter (g/bhp-hr)
Emission Control Systems in Modern Diesel Engines

2004:
• Exhaust gas recirculation (EGR) – reduces NOx emissions
• Diesel Oxidation Catalyst (DOC) – reduces PM, NOx, CO, organics, but increases NO₂

2007
• Diesel particulate filter DPF – removes PM by filtering and oxidation; accumulated PM needs to be cleaned or “regenerated”; increases NO₂

2010
• Selective Catalytic Reduction SCR: reduces NO₂ using urea (NH₃)
• Ammonia oxidative catalyst (AMOX): removes any remaining ammonia
Rationale for ACES Study (2006)

• The new developments motivated HEI’s automotive and government sponsors, and others, to ask HEI to undertake a research program:
  – The combination of advanced-technology, diesel engines, after-treatment systems, reformulated fuels and reformulated oils developed to meet the 2007/2010 emission standards will result in substantially reduced emissions
  – Substantial public health benefits are expected from these reductions
  – Most pollutants will decrease, but new species may be formed.
  – Although effects are expected to be reduced, new technologies should be evaluated before widespread introduction

• The research program designed to:
  – Evaluation and characterization of emissions from 2007 and 2010 heavy duty diesel engines
  – Health effects of exposure to emissions from the new engines
Design of ACES Study

- **Phase 1:** Characterization of emissions from four heavy-duty on-road diesel engines (HDDEs) that met the 2007 PM standards
- **Phase 2:** Characterization of emissions from a group of HHDE that met the 2010 NOx standards

Overseen by the Coordinated Research Council (CRC) and funded by the DOE Office of Vehicle Technologies, the Truck and Engine Manufacturers Association (EMA), CARB, and the American Petroleum Institute (API).

- **Phase 3:** Assessment of health effects in rodents - a chronic study assessing cancer and non-cancer effects in rats, and a shorter term study in mice – of inhalation of NTDE from a 2007-technology HDDE that was among the four tested in Phase 1

Overseen by HEI and funded by EMA, EPA, CARB, and API.
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ACES Phase 1 and 2
Background
## US Diesel Emission Standards from 1998 to 2010 (g/bhp-hr)

<table>
<thead>
<tr>
<th>Regulated pollutants</th>
<th>Model Year of Implementation (HDDE) ~540 hp</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1998</td>
</tr>
<tr>
<td>PM</td>
<td>0.1</td>
</tr>
<tr>
<td>CO</td>
<td>15.5</td>
</tr>
<tr>
<td>NOx</td>
<td>4.0</td>
</tr>
<tr>
<td>NMHC*</td>
<td>1.3**</td>
</tr>
</tbody>
</table>

### Model Year of Implementation (Off-Road DE)

<table>
<thead>
<tr>
<th>Regulated pollutant</th>
<th>Tier 2/3</th>
<th>Tier 4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2001-2008</td>
<td>2008</td>
</tr>
<tr>
<td>PM</td>
<td>&lt;11hp ≥750</td>
<td>&lt;11 hp &lt;25</td>
</tr>
<tr>
<td>CO</td>
<td>6-2.6</td>
<td>6-4.9</td>
</tr>
<tr>
<td>NOx</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MMHC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NMHC+NOx</td>
<td>5.6-4.9</td>
<td>5.6</td>
</tr>
</tbody>
</table>

*Nonmethane hydrocarbons

**Included methane
Schematic Representation of HDDE Emission Controls

2007-technology engines

Exhaust Gas Recirculation

Figure adapted from http://www.factsaboutscr.com/scr/default.aspx
Schematic Representation of HDDE Emission Controls

Urea solution

Ammonia (NH₃) oxidative catalyst

NO → NO₂

exhaust

EGR

CO(NH₂)₂ → NH₃

NH₃ + NO + NO₂ → N₂ + H₂O (+NH₃ slip)

↓NOx ↓PM ↓NO₂
(↑NH₃) (↑N₂O)

NH₃ → NO + N₂O + N₂

2010-technology engines

Figure adapted from http://www.factsaboutscr.com/scr/default.aspx
Phase 1 and 2
Set up and Design
Engines Tested

• Phase 1
  – Four model year 2007 HDDE provided by Caterpillar, Cummins, Detroit Diesel Corporation, and Volvo

• Phase 2
  – Three 2010 model-year HDDE provided by Cummins, Detroit Diesel Corporation, and Mack (Volvo Powertrain.)
Engine Test Cycles

• Federal Test Procedure (FTP), also referred to as the composite FTP, used to certify that engines comply with the emission standards—includes a cold start and hot start

• FTP with hot start only, used for characterizing both regulated and unregulated emissions

• 16-Hour Cycle. Developed by researchers at West Virginia University for ACES to represent more closely the real-world operations of modern engines, used for characterizing both regulated and unregulated emissions and for exposing the animals
Overall Experimental Setup

PM composition

OC, elements speciation

NOx, CO, HC
PM mass (regulatory)

Gaseous speciation

PM composition

2007 Heavy Duty Diesel Engine with DPF
Background bag sample of dilution air for CO, CO2, NOx, NO, THC, CH4, and C2-C12 speciation
Regulated PM following CFR Part 1065 using 47 mm Teflon filter
Imagories for carbonyls, alcohols, amines, and cyanide
Sorbent traps for nitroamines, and Sunan matter for SVOC
Auxiliary PM samples on 47 mm filters for inorganic ions (Fluoropore Filter), XRF (Teflon Filter), and ICP-MS (Fluoropore Filter), DFI GC (TX-40 Filter)
XAD traps for gas phase semi-volatile compounds: PAH, oxy-PAH, nitro-PAH, hopanes, steranes, carpanes, polar organics, high molecular weight alkanes and cycloalkanes, dioxins, furans
H Filter (0.4 microns) for particulate-phase semi-volatile compounds: PAH, oxy-PAH, nitro-PAH, dioxins, Furans, hopanes, steranes, carpanes, polar organics, high molecular weight alkanes and cycloalkanes, dioxins, furans
Animal Exposure Chamber (no animals present) PM mass using Teflon filter, size and number using EEP, real time total PM using DNM-230, real time total PM using MSS, OC/EC collection using a pair of quartz filters, and semi-continuous OC/EC (very limited use)
J Proportional bag sample for hydrocarbon speciation of C2 through C12 compounds
K Horiba MEXA 7260 for THC, CO, CO2, NOx, NO analyzers, and CH4 analyser
L FTIR for nitrogen compounds
Phase 1 and 2

Results and Conclusions
Regulated Emissions of PM, NOx, and CO (g/bhp-hr)

Regulatory FTP cycle

<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Standard</td>
<td>Standard</td>
<td>Standard</td>
<td>Standard</td>
</tr>
<tr>
<td>PM</td>
<td>0.1</td>
<td>0.1</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>CO</td>
<td>15.5</td>
<td>15.5</td>
<td>15.5</td>
<td>15.5</td>
</tr>
<tr>
<td>NOx</td>
<td>4.0</td>
<td>2.0</td>
<td>1.2</td>
<td>0.2</td>
</tr>
</tbody>
</table>
# Comparison of Emission Rates of Selected Gases

Table 2. Emissions rates (g/bhp-hr) of selected gases in exhaust from 2007- and 2010-technology engines (16-hour cycle)

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>2007 Engines</th>
<th>2010 Engines</th>
<th>% Reduction Relative to 2007 Technology Engines</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO\textsubscript{2}</td>
<td>590.2±22.7</td>
<td>571.3±41.4</td>
<td>a</td>
</tr>
<tr>
<td>N\textsubscript{2}O*</td>
<td>0.010±0.003</td>
<td>0.073±0.030</td>
<td>(+730)</td>
</tr>
<tr>
<td>NO\textsubscript{2}</td>
<td>0.73±0.20</td>
<td>0.046±0.029</td>
<td>94</td>
</tr>
<tr>
<td>SO\textsubscript{2}</td>
<td>0.00112±0.00025</td>
<td>0.00033±0.00016</td>
<td>71</td>
</tr>
<tr>
<td>NH\textsubscript{3}**</td>
<td>&lt;0.0001</td>
<td>0.0025±0.0014</td>
<td>(+2400)</td>
</tr>
</tbody>
</table>

a: No discernible change within the measurement uncertainties

* 2014 N\textsubscript{2}O emission standard =0.1 g/bhp-hr

**Corresponding to a concentration of 0.22 ppm. Proposed European standard is 10-15 ppm. No US standard
## Changes in PM Mass and Composition

### Emission Rate

<table>
<thead>
<tr>
<th>Year</th>
<th>EC, mg/hr</th>
<th>OC, mg/hr</th>
<th>EC/OC</th>
<th>Composition</th>
</tr>
</thead>
<tbody>
<tr>
<td>1998 (Hot FTP)</td>
<td>3445.0±1110</td>
<td>1180.0±70.7</td>
<td>2.9</td>
<td>EC 70% OC 27% Sulfate 3%</td>
</tr>
<tr>
<td>2004 (CARB cycle)</td>
<td>22.6±4.7</td>
<td>52.8±47.1</td>
<td>0.4</td>
<td>EC 13% OC 32% Sulfate 52%</td>
</tr>
<tr>
<td>2007 (16-hr cycle)</td>
<td>12.2±6.2</td>
<td>39.2±33.6</td>
<td>0.3</td>
<td>EC 15% OC 65% Sulfate 1% Nitrate 13%</td>
</tr>
<tr>
<td>2010 (16-hr cycle)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Composition

<table>
<thead>
<tr>
<th>Year</th>
<th>EC</th>
<th>OC</th>
<th>Sulfate</th>
<th>Nitrate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1998</td>
<td>70%</td>
<td>27%</td>
<td>3%</td>
<td></td>
</tr>
<tr>
<td>2004</td>
<td>13%</td>
<td>32%</td>
<td>52%</td>
<td></td>
</tr>
<tr>
<td>2007</td>
<td>15%</td>
<td>65%</td>
<td>1%</td>
<td>13%</td>
</tr>
</tbody>
</table>
Particle Number (PN/bhp-hr) Comparison with 2004 engine

With 2007 and 2010 engines PN was two orders of magnitude lower than 2004 engines
Particle number was 72% lower in 2010 engines relative to 2007 during the 16-hour cycle.
Major Conclusions of Phase 1
2007 Engines

• All regulated emissions were lower than the 2007 standards
  – PM emissions were 90% lower than the standard and 99% lower than 2004 engine emissions

• NO$_2$ emissions were higher than 2004 engine emissions due to the use of catalyzed particulate filters

• Emissions of unregulated pollutants (such as PAHs and metals) were substantially lower than 2004 emissions

• Overall emissions of the four 2007 engines were very similar and no engine had higher or lower emissions of all the major components or classes of components
  – The engine to use in Phase 3 was chosen randomly
Major Conclusions of Phase 2

• Emissions of regulated and regulated pollutants from 2010-engines were lower than in Phase 1
  – The major reduction was for NOx which decreased by 94%
  – NO\textsubscript{2} was also decreased by the same %
  – PM emissions also went down

• Emissions of N\textsubscript{2}O and NH\textsubscript{3} increased but very slightly
Summary of Emission Reductions Between Phase 1 and Phase 2

BSFC – Brake-specific fuel consumption
GWP – global warming potential
Main Difference Between Phase 1 and 2

- Trap regeneration occurred 1-3 times during the 16-hr cycle in 2007 engines, but did not occur in 2010 engines
  - emissions of PM were higher during regeneration
- Lack of regeneration was thought to be associated with
  - Reduction of SO2 and sulfate and overall reduction of PM mass and number because desorption of sulfur from the DPF can only occur at the high temperature that occur only during regeneration. Sulfate can serve a precursor for nanoparticle formation and may be another reason for the lower particle number emission rate
  - Reduction in EC, PM number, and metals due to higher filtration efficiency of the loaded DPF
Acknowledgments

- Imad Khalek, Southwest Research Institute, ACES Phase 1 and 2 Principal Investigator
- Christopher Tennant, CRC, ACES Phase 1 and 2 Project Officer
- CRC Oversight Panel for ACES Phase 1 and 2
- Nigel Clark, West Virginia University, developer of the 16-hour cycle

All figures presented are adapted from CRC ACES Phase 1 (2009 and Phase 2 (2013) reports, Khalek et al (AWMA 2011 and 2015-in press)

The CRC reports can be obtained at www.crcao.org
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- Overall Conclusions
  - Rashid Shaikh
ACES Phase 3B Goals

• Health effects of lifetime exposure of rats to emissions from 2007-compliant diesel engines = New Technology Diesel Exhaust (NTDE)

• Hypothesis: Emissions will not cause an increase in tumor formation or substantial toxic health effects… although some biological effects may occur.

• Characterize exposure atmospheres throughout the exposure period
Phase 3B - Rat Exposures to NTDE

• Expose male and female rats (Wistar Han strain, 140 of each sex/exposure group).

• Duration - Lifetime = 28 months for males, 30 months for females, 16 hr/day, 5 days/wk.
  10 rats/exposure group evaluated at 1, 3, 12, 24 months. Remainder (=100+) evaluated at terminal sacrifice

• **NTDE:** Three dilutions of whole emissions + clean air controls
  – 4.2 ppm NO$_2$ = High
  – 0.8 ppm NO$_2$ = Medium
  – 0.1 ppm NO$_2$ = Low

  **NO$_2$, rather than PM, chosen as target pollutant**
**ACES Phase 3B - Core and Ancillary Studies**

- **Core study PI** Jake McDonald, Lovelace Respiratory Research Institute (LRRI), Albuquerque, NM
  - Exposures conducted at LRRI; atmospheres characterized with animals in chambers
  - > 100 health endpoints measured = histopathology (including tumor incidence), hematology, serum chemistry, pulmonary function

- **Ancillary studies** – samples sent from LRRI at 1, 3, 12, and 24 months

- **Goal** – evaluate genotoxic and other endpoints
  1. Jeffrey Bemis – Litron Labs, Rochester, NY
     Micronucleus formation in red blood cells
  2. Lance Hallberg – U Texas-Galveston
     Genotoxic and oxidative stress endpoints
  3. Daniel Conklin – University of Louisville
     Vascular markers of inflammation and thrombosis, cardiac and aortic remodeling
HEI Review of Reports

• Biologic findings – core and ancillary studies
  – ACES Special Review Panel = experts in pathology, vascular and inflammatory responses, statistics, and genotoxicity
  – Pathology Working Group = pathologists, reviewing all histopathology findings

• Characterization of components of exposure with animals in chambers
  – Exposure Characterization Panel = expertise in exposure assessment
Key findings and conclusions

• Exposure concentrations targets of NO₂ met.
• Most abundant pollutants - CO₂, CO, NO and NO₂.
• Engine-derived particle concentrations very low = < 10μg/m³
• Ultrafine particles detectable (2-8x10⁵/cm³), associated with filter regeneration events (1-2 per 16-hour exposure cycle).
• Very low levels of volatile (VOC) and semi-volatile organic (SVOC) compounds and SO₂.

• Animals inside exposure chambers contributed to chamber atmosphere, in particular levels of SVOC and fine PM.
Core study - Biologic Findings
ACES Review Panel findings and conclusions

• The investigators successfully completed a complex, lifetime exposure study of rats to 2007-compliant diesel emissions (= NTDE).

• Overall agreement with authors’ findings
Core Study - Histopathology (I)

• Key findings:
  – **No increase in tumor formation** over background in the lung or any other organ of rats
  
  – **Major difference compared to long-term exposures to “traditional” diesel exhaust** containing PM, which showed:
    • Lung tumors, associated with PM exposure at $\approx 1 \text{ mg/m}^3$
    • Pre-cancerous changes in lung, including inflammatory response and presence of soot particles

  *See Mauderly et al [1994] and Heinrich et al [1995]*)
Effects of 30-Month Exposure to TDE in rat lung

TDE, original magnif. 100x

Large area of lung involved:
1) Black diesel soot particulates present free in alveoli,
2) Numerous pulmonary alveolar macrophages, and in interstitial tissues.
3) Chronic inflammation, characterized by a mixed inflammatory cell infiltrate and fibrosis.
4) Centriacinar (gas exchange region) epithelial hyperplasia and bronchiolization
The Gas Exchange Region of the Lung

Blood supply of alveoli

Capillary network of one alveolus
Some subtle changes in the centriacinar region of the lung:
1) ↑ airway epithelial cell number;
2) bronchiolization;
3) some ↑ in macrophages;
4) interstitial fibrosis
Histopathologic changes in the centriacinar (gas-exchanging) region of the lung in ACES after long-term exposure to NTDE were similar to changes after long-term exposure to oxidizing pollutant gases, in particular NO₂.

ACES study, NTDE

24-month rat exposure to NO₂
Core Study - Respiratory Endpoints

- NTDE exposure associated with a few small decreases in respiratory endpoints

- Some measures of expiratory flow -
  - Predominantly at the highest exposure level and more in females than males.

- Diffusion capacity of carbon monoxide = $DL_{CO}$
  - Measure of gas exchange in gas exchange region of lung
  - Trend across all exposure groups in males and females at 3, 12, and 24 months
NTDE → small ↓DL_{CO} = ↓ gas diffusion from the alveolus to the capillary bed

Suggests that the histopathology changes in the centriacinar region associated with NTDE exposure may result in small physiological changes

Gas exchange (O_2 and CO_2) between the alveolus and the capillary bed
Core Study - Biochemical Effects

- **Few changes** in inflammatory endpoints in lung tissue, bronchoalveolar lavage fluid or blood.

- Small ↑ in heme oxygenase (HO) 1, interleukin 6, keratinocyte-derived chemokine (lung tissue)
- Small ↑ in μtotal protein, total white blood cells (lung lavage)
  - Both sexes

- Conclusion - NTDE exposure results in very mild oxidative stress and lung inflammatory responses
Changes in Lung Tissue Heme Oxygenase (HO)-1 Levels Over 24 Month Rat Exposures

C = control; L, M and H = low, medium and high level NTDE
Ancillary Studies – ACES Review Panel Conclusions

- **Genotoxic endpoints - No exposure-related changes**
  - no increase in *micronucleus formation* in reticulocytes (early red blood cells)
  - no change in *lung DNA damage* (Comet assay), or *oxidative stress* (= levels of TBARS in hippocampus and of 8-OH deoxyguanaine in plasma)

- **Vascular endpoints – most unchanged**, a few exposure-related changes (of these: mostly high dose and in females).
  - Review Panel – no coherence among changes, so biological significance unclear

- **Overall conclusions – NTDE did not cause genotoxic changes and had little or no effect on > 20 vascular markers of inflammation or thrombosis**
ACES 3B – Health Study
Overall Conclusions

• Lifetime exposure to NTDE in rats did not induce tumors in rats, in contrast to lifetime exposure to TDE

• Exposure to NTDE had few biological effects
  – Study hypothesis supported

• The few histological changes after NTDE exposure consistent with exposure to NO$_2$. 
ACES - HEI Phase 3B Reports and Reviews

• All Phase 3B core and ancillary studies reports + ACES Review Panel Commentary - downloadable as HEI Report #184
• ACES Program Summary – to be issued in summer 2015.
• All available at: http://pubs.healtheffects.org
Overall Conclusions

Rashid Shaikh
Conclusions of ACES
ACES Phases 1 and 2

• Goal: Characterization of emissions from MY 2007 and 2010 HDDE engines
  – Equipped with EGR, other engine control systems and after-treatment technologies (DOC, DPF, SCR [+AMOX])
  – Highly effective (90 to 99%) in reducing the tailpipe emissions of PM, PN, NOx, PAHs, and more than 100 other compounds. Meet, and exceed, current US EPA standards
Conclusions of ACES
Phase 3

• Goal: Assessment of health effects in rats
  – Life-time inhalation exposure in rats to emissions from a MY 2007 engine
  – Produced no increase in tumors or any precancerous changes in the lung
  – A few effects were observed, most likely associated with NO₂ present in 2007 engine exhaust
  – No consistent changes seen in 100+ other markers of toxicity
  – Stark contrast with studies with traditional diesel exhaust ⇒ carcinogenic
Limitations of ACES Studies

- **Emissions Characterization:**
  - Efficacy of SCR under certain driving conditions – not investigated
  - Regeneration of DPF in MY 2010 engines – not observed in Phase 2

- **Health Effects Study:**
  - Rat study – caution in extrapolation to humans
  - Was there sufficient statistical ‘power’ to detect effects?
    - But, PM levels are highly reduced in NTDE
Impact

• Diesel emissions – still an issue from older engines
  – Diesel fleet turnover currently at ~35% but highly variable geographically

• Ambient levels of PM, EC and NOx going down – e.g. in MATES IV study

• Promise for developing countries
  – India, China and others taking steps to reduce fuel sulfur levels
Thank you

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Geoffrey Sunshine: gsunshine@healtheffects.org

ACES Reports
Reports can be downloaded from:
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Coordinating Research Council
http://crcao.org/publication/index.html