Advanced Collaborative Emission Study (ACES)

New-technology diesel engine emissions characterization and chronic rodent inhalation bioassay

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Combustion Generated Nanoparticles

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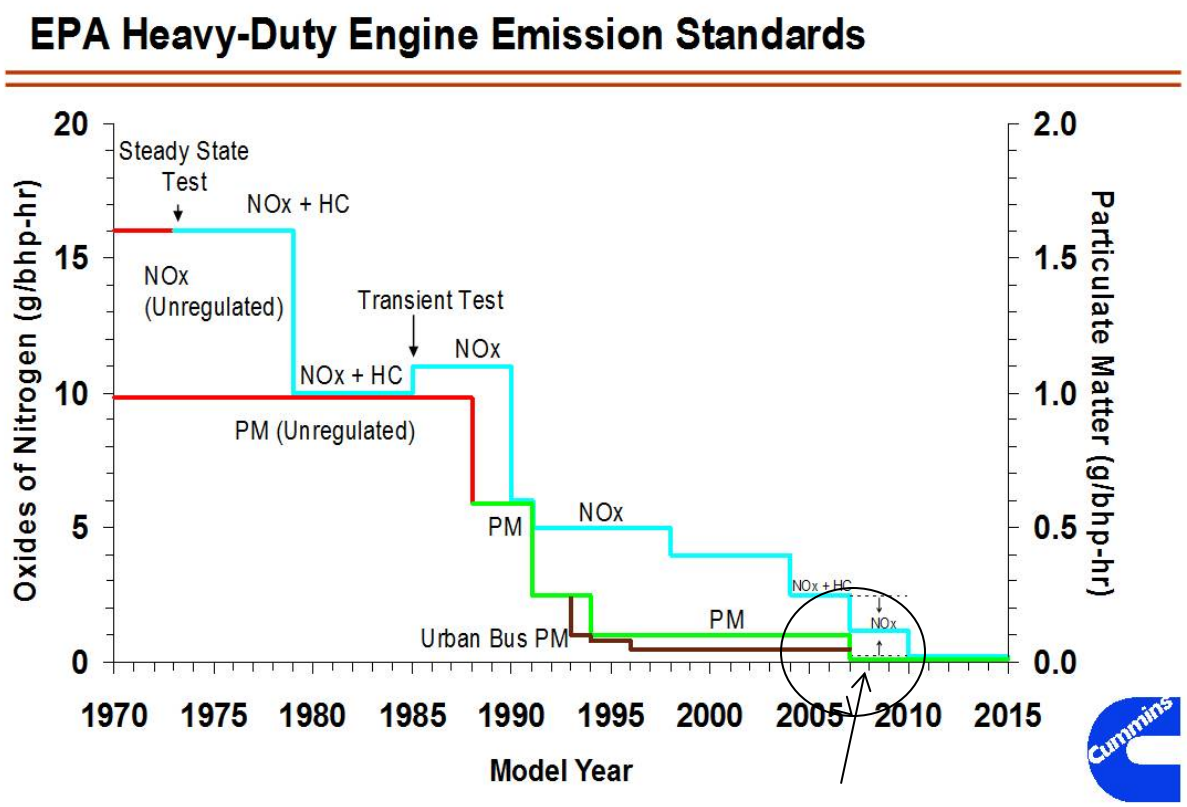
Maria Costantini, Imad Khalek, Jacob MacDonald, and Christopher Tennant

Regulation of Diesel Engine Emissions



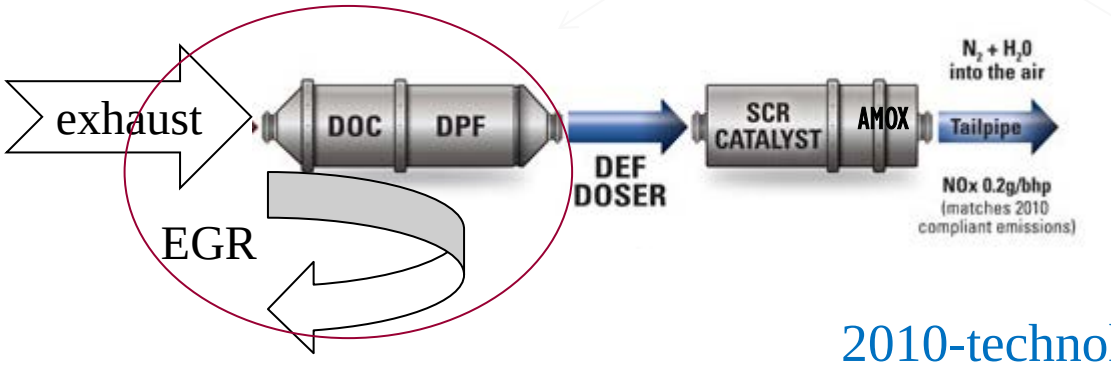
ACES engines

Similar patterns in Europe

Rationale for ACES Study

- A combination of advanced-technology diesel engines, after-treatment systems, reformulated fuels and reformulated oils were needed to meet the new standards

- Although substantial public health benefits were expected from these reductions, there was interest in evaluating the new technologies to assess any unforeseen changes in the emissions and effects as a result of the technologies



2007-technology engines

2010-technology engines

Figure adapted from http://www.factsaboutscr.com/scr/default.aspx

ACES Consisted of 3 Phases

Phase 1 and 2 – Emission characterization

Conducted at Southwest Research Institute

Emissions Characterization

- Phase 1: characterization of emissions from 4 new HDDEs that met the 2007 PM standards
- Phase 2: Characterization of emissions from 3 new HDDE that met the 2010 NOx standards

- Engines were tested over different test cycles (FTP and a 16-hour cycle)
- Both regulated and unregulated pollutants (more than 700 species) were measured

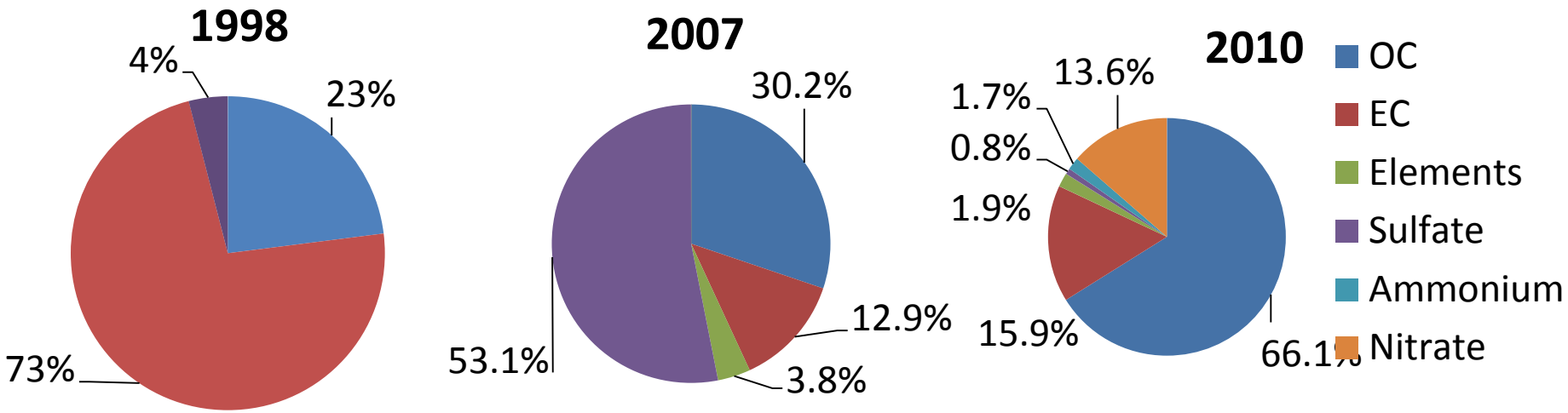
Regulated Emissions of PM, NOx, and CO* (g/bhp-hr)

Regulatory FTP cycle

	MY 1998	MY 2004	MY 2007			MY 2010		
	Standard	Standard	Standard	Measured Phase 1	% below standard	Standard	Measured Phase 2	% below standard
PM	0.1	0.1	**0.01**	0.0014	86	0.01	0.0008	92
CO	15.5	15.5	15.5	0.48	96	15.5	0.5	97
NOx	4.0	2.0	**1.2**	1.09	9	**0.2**	0.08	60

*Hydrocarbons are also regulated, but were very low at uncertainty level in 2007 and 2010 engine exhaust

Changes in PM Composition

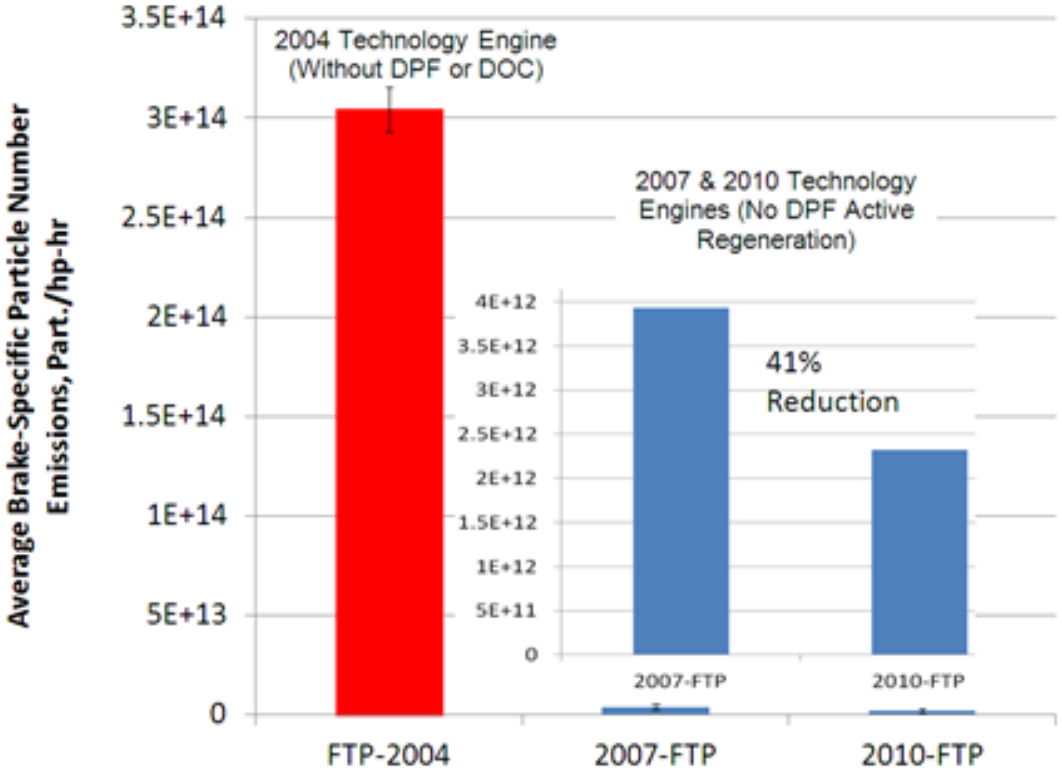


	1998 (Hot FTP)	**2007 (16-hr)*** (16-hr cycle)	**2010 *** (16-hr cycle)
Composition	EC 73% OC 23% Sulfate 4%	EC 13% OC 30% Sulfate 53%	EC 16% OC 66% Sulfate 1% Nitrate 13%

*Trap regeneration occurred 1-3 times during the 16-hr cycle in 2007 engines, but did not occur in 2010 engines. PM reduction and compositional changes between 2007 and 2010 engines could be due to lack of trap regeneration

Particle Number (PN/bhp-hr)

Comparison of 2007 and 2010 engines with 2004 engine



With 2007 and 2010 engines PN was two orders of magnitude lower than 2004 engines

Major Conclusions of Phases 1and 2

- All regulated emissions were lower than the 2007 and 2010 standards
- NO_2 emissions from 2007 engines were higher than 2004 engine emissions due to the use of catalyzed particulate filters, but were reduced by 94% in 2010 engines
- Emissions of NH_3 and N_2O increased in 2010 engines, but were below proposed standards (NH_3) and 2014 standard (N_2O)
- Emissions of unregulated pollutants (such as PAHs and metals) were substantially lower than 2004 emissions

ACES Consisted of 3 Phases

Phase 3- Health Effects Study in Rats

Conducted at Lovelace Respiratory Research Institute

ACES Phase 3 Goals

- Assess health effects of lifetime exposure of rats to emissions from a 2007-compliant diesel engine = 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 chamber exposure atmospheres throughout the exposure period

Phase 3B - Rat Exposures to NTDE

- Expose male and female rats (Wistar Han strain) for a lifetime = 28 months for males, 30 months for females, 16 hr/day, 5 days/wk.

	Months after start of exposure (males and females)				
Assignment	**1**	**3**	**12**	**24**	**28 and 30**
Chronic bioassay (histopathology)	-	-	-	-	200
Intermediate sacrifice (biologic endpoints)	20	20	20	20	-
Cumulative total	20	20	20	20	280

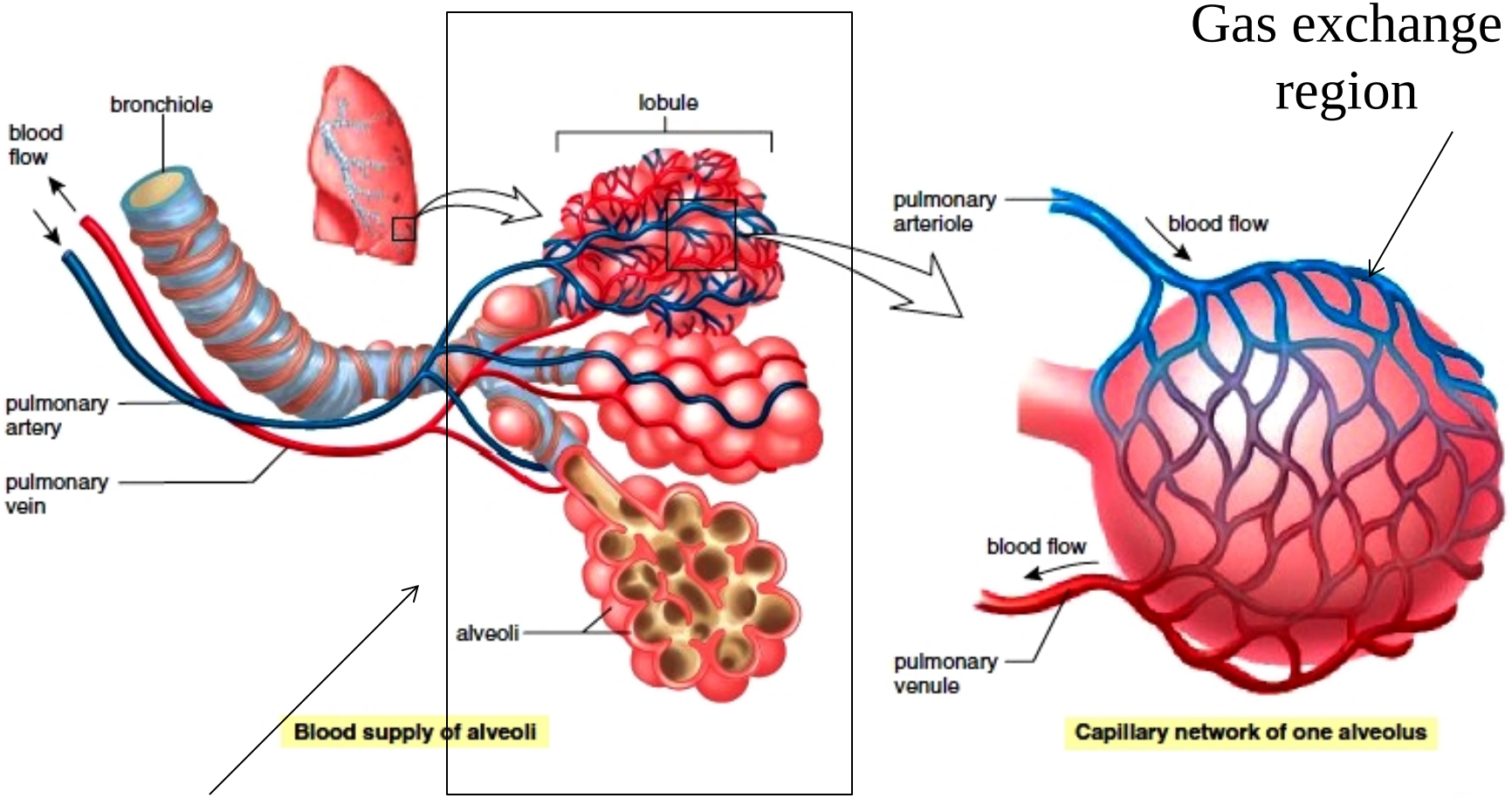
- 2007 engine: Three dilutions of whole emissions + clean air controls
  - 4.2 ppm NO_2 = High (filter PM = 12.3 ug/m^3)
  - 0.8 ppm NO_2 = Medium (filter PM = 1.07 ug/m^3)
  - 0.1 ppm NO_2 = Low (filter PM = 0.3 ug/m^3)

- NO_2, rather than PM, chosen as target pollutant

Core Study - Histopathology (I)

- **Key findings:**
  - **No increase in tumor formation** over background in the lung or any other organs of rats
  - **Subtle changes in the centriacinar region** of the lung
  - **Major difference compared to long-term exposures to "traditional" diesel exhaust** containing PM, which showed:
    - Lung tumors, associated with PM exposure at ≈ 1 mg/m^3
    - Pre-cancerous changes in lung, including inflammatory response and presence of soot particles

The Gas Exchange Region of the Lung

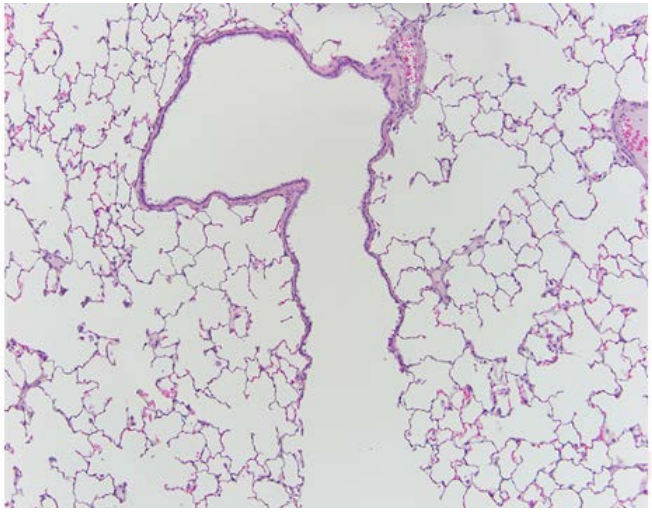


Regions of histopathology study (centriacinar region)

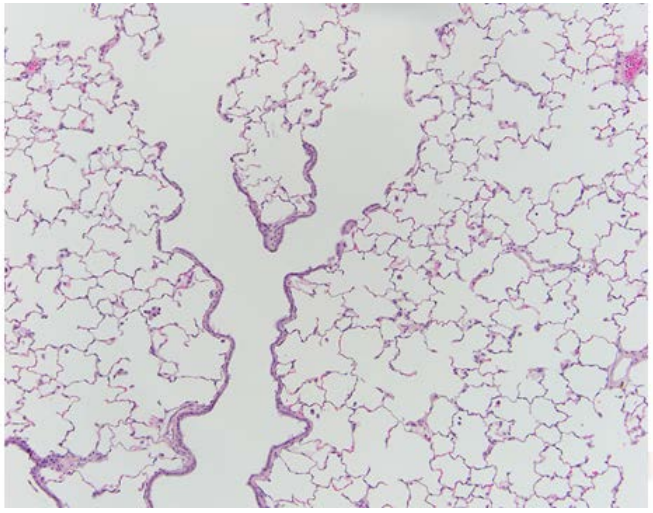
Core Study - Histopathology (II)

(30-month exposure, female rat lung)

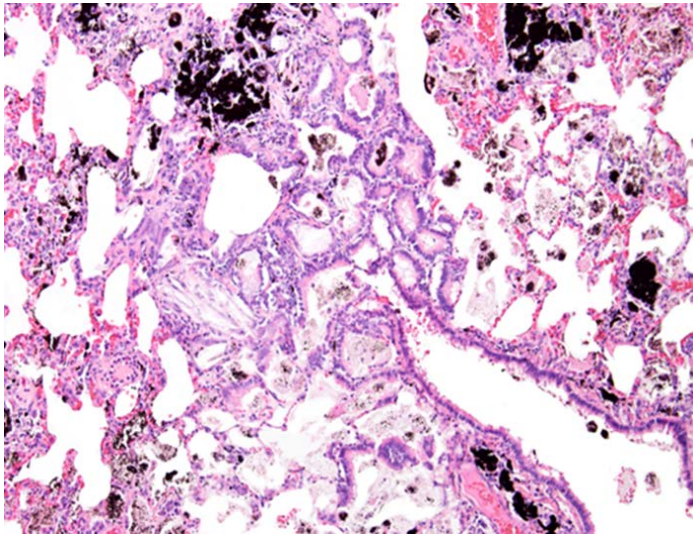
ACES control x100



ACES – high dose NTDE x100



1988's Engine – high dose x100



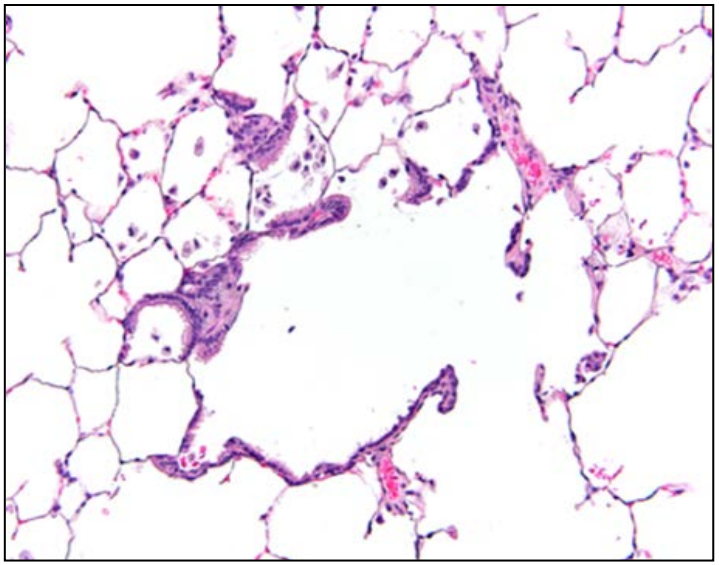
Some subtle changes in the centriacinar region of the lung:

1) ↑ airway epithelial cell number (hyperplasia);
2) bronchiolization;
3) interstitial fibrosis
4) some ↑ in macrophages

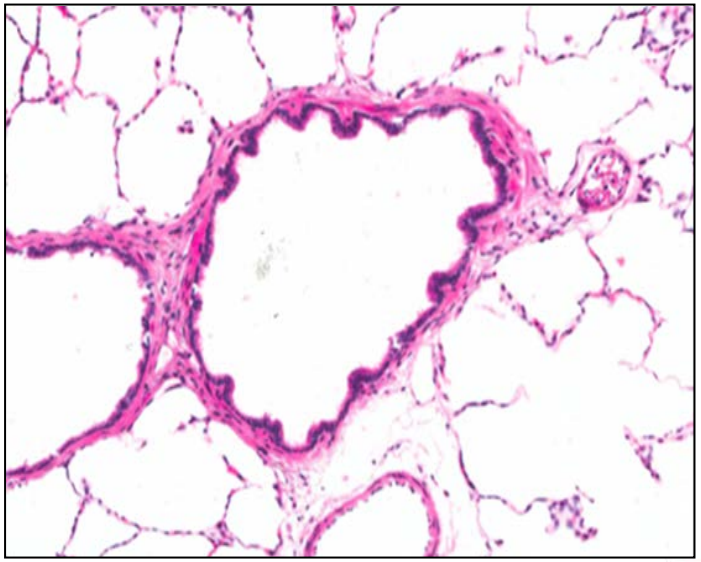
Core Study - Histopathology (III)

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 NO_2.

ACES study, high level NTDE



Mauderly 1987 NO_2 bioassay



Core Study - Other Endpoints

Respiratory endpoints

- **Small decreases** in expiratory flow and reduced diffusion capacity of carbon monoxide = DLCO
- Suggests that the histopathology changes in the centriacinar region associated with NTDE exposure may result in small physiological changes

Biochemical and Genotoxic Endpoints

Few changes in markers of inflammation or thrombosis in lung tissue, bronchoalveolar lavage fluid or blood

No consistent changes in many other biochemical markers

No exposure-related changes in genotoxic endpoints

= no increase in micronuclei in reticulocytes (early red blood cells)

= no change in lung DNA damage (Comet assay), or oxidative stress

ACES Phase 3B – Overall Conclusions

- Lifetime exposure to NTDE did not induce tumors in rats, in contrast to lifetime exposure to TDE at a similar dilution ratio
- The few histological changes after NTDE exposure consistent with exposure to NO_2
  - NO_2 was reduced by more than 90% in 2010 engines
- Exposure to NTDE had few biological effects
  - Study hypothesis supported
- No obvious new toxic species in the 2007 engine exhaust

ACES - Final Considerations

- Emissions Characterization
  - 2010 engines test results did not include the emission contribution of the infrequent active regeneration. Future work should include the contribution of regeneration
- Health Effects Study in rats
  - Caution in extrapolation to humans
- Overall
  - ACES demonstrates the effectiveness of the emission controls in greatly reducing both PM and NOx and suggests a similar reduction in the toxicity of NTDE

Acknowledgments

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- ACES Reports can be downloaded from
  - Health Effects Institute www.pubs.healtheffects.org
  - Coordinating Research Council http://crcao.org/publication/index.html