Catalysis, a key property of particle filters to lower genotoxicity of diesel exhaust
World Health Organization, IARC
Diesel engine exhaust: A group 1 carcinogen

Diesel engine exhausts cause lung cancer in humans
Diesel engine exhausts cause lung cancer in humans

IARC: DIESEL ENGINE EXHAUST CARCINOGENIC

Lyon, France, June 12, 2012 — After a week-long meeting of international experts, the International Agency for Research on Cancer (IARC), which is part of the World Health Organization (WHO), today classified diesel engine exhaust as carcinogenic to humans (Group 1), based on sufficient evidence that exposure is associated with an increased risk for lung cancer.

Background

In 1988, IARC classified diesel exhaust as probably carcinogenic to humans (Group 2A), An Advisory Group which reviews and recommends future priorities for the IARC Monographs Program had recommended diesel exhaust as a high priority for re-evaluation since 1998.

There has been mounting concern about the cancer-causing potential of diesel exhaust, particularly based on findings in epidemiological studies of workers exposed in various settings. This was re-emphasized by the publication in March 2012 of the results of a large US National Cancer Institute/National Institute for Occupational Safety and Health study of occupational exposure to such emissions in underground miners which showed an increased risk of death from lung cancer in exposed workers (1).
Diesel engine exhaust cause cancer in humans

The Diesel Exhaust in Miners Study: A Nested Case-Control Study of Lung Cancer and Diesel Exhaust


Manuscript received February 16, 2011; revised June 3, 2011; accepted October 21, 2011.

Correspondence to: Debra T. Silverman, ScD, Occupational and Environmental Epidemiology Branch, Division of Cancer Epidemiology and Genetics, National Cancer Institute, Rm 2108, 6120 Executive Blvd, Bethesda, MD 20892; e-mail: silverman@mail.nih.gov.

Background
Most studies of the association between diesel exhaust exposure and lung cancer suggest a modest, but consistent, increased risk. However, to our knowledge, no study to date has had quantitative data on historical diesel exposure coupled with adequate sample size to evaluate the exposure-response relationship between diesel exhaust and lung cancer. Our purpose was to evaluate the relationship between residential lifetime exposure to diesel exhaust and lung cancer mortality after adjustment for smoking and other potential confounders.

Methods
We conducted a nested case-control study in a cohort of 12315 workers in eight Russian coal mines, of which 1196 lung cancer deaths and 562 incidence density-sampled control subjects. For each case subject, we selected up to nine control subjects, individually matched on mining facility, sex, race/ethnicity, and birth year (within 5 years), from all workers who were alive before the day the case subject died. We estimated diesel exhaust exposure, represented by respirable elemental carbon (REC), by job and year, for each subject, based on an extensive retrospective exposure assessment at each mining facility. We conducted both categorical and continuous regression analyses adjusted for cigarette smoking and other potential confounding variables (eg, history of employment in high-risk occupations for lung cancer and a history of respiratory disease) to estimate odds ratios (ORs) and 95% confidence intervals (CIs). Analyses were both unlagged and lagged to exclude recent exposure such as that occurring in the 15 years directly before the date of death (case subjects)/reference date (control subjects). All statistical tests were two-sided.

Results
We observed statistically significant increasing trends in lung cancer risk with increasing cumulative REC and average REC intensity. Cumulative REC, lagged 15 years, yielded a statistically significant positive gradient in lung cancer risk overall (P-value = .001); among heavily exposed workers (ie, above the median of the top quartile [REC ≥ 1005 µg/m³-y]), risk was approximately three times greater (OR = 3.20, 95% CI = 2.43 to 4.29) than that among workers in the lowest quartile of exposure. Among never smokers, odd ratios were 1.0, 1.47 (95% CI = 0.92 to 2.30), and 5.9 (95% CI = 1.46 to 36.57) for workers with 15-year lagged cumulative REC tertiles of less than 8, 8 to less than 304, and 304 µg/m³-y or more, respectively. We also observed an interaction between smoking and 15-year lagged cumulative REC (P-value = .086) such that the effect of each of these exposures was attenuated in the presence of high levels of the other.

Conclusion
Our findings provide further evidence that diesel exhaust exposure may cause lung cancer in humans and may represent a potential public health burden.

J Natl Cancer Inst 2012;104:1–14
Für Dieselmotoremissionen beträgt der Arbeitsplatzgrenzwert 100 μg/m³ mit dem Zusatz des Minimierungsgebotes, da Dieselmotoremissionen als krebserzeugend eingestuft sind. Generell sind Massnahmen die zu einer Verringerung der Dieselmotoremissionen führen damit sinnvoll.
What does asbestos has in common with diesel exhaust?

<table>
<thead>
<tr>
<th>Stoff</th>
<th>Summenformel</th>
<th>Klasse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzo(a)pyren</td>
<td>C_{20}H_{12}</td>
<td>1</td>
</tr>
<tr>
<td>Benzol</td>
<td>C_{6}H_{6}</td>
<td>3</td>
</tr>
<tr>
<td>Dibenz(a, h)anthracen</td>
<td>C_{22}H_{14}</td>
<td>1</td>
</tr>
<tr>
<td>1,2-Dibromethan</td>
<td>C_{2}H_{4}Br_{2}</td>
<td>3</td>
</tr>
<tr>
<td>1,4 Dichlorbenzol</td>
<td>C_{6}H_{4}Cl_{2}</td>
<td>3</td>
</tr>
<tr>
<td>1,2-Dichlorethan</td>
<td>C_{2}H_{4}Cl_{2}</td>
<td>3</td>
</tr>
<tr>
<td>Dieselruß</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>Diethylether</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diethylsulfat</td>
<td>C_{4}H_{10}O_{4}S</td>
<td>2</td>
</tr>
</tbody>
</table>
Ordinance on Air Pollution Control (OAPC): List of carcinogenic substances

Luftreinhalte-Verordnung (LRV)

83 Tabelle von krebserzeugenden Stoffen

<table>
<thead>
<tr>
<th>Stoff</th>
<th>Summenformel</th>
<th>Klasse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzo(a)pyren</td>
<td>C_{20}H_{12}</td>
<td>1</td>
</tr>
<tr>
<td>Benzol</td>
<td>C_{6}H_{6}</td>
<td>3</td>
</tr>
<tr>
<td>Dibenz(a, h)anthracen</td>
<td>C_{22}H_{14}</td>
<td>1</td>
</tr>
<tr>
<td>1,2-Dibromethan</td>
<td>C_{2}H_{4}Br_{2}</td>
<td>3</td>
</tr>
<tr>
<td>1,4 Dichlorbenzol</td>
<td>C_{6}H_{4}Cl_{2}</td>
<td>3</td>
</tr>
<tr>
<td>1,2-Dichlorethan</td>
<td>C_{2}H_{4}Cl_{2}</td>
<td></td>
</tr>
<tr>
<td>Dieselruss</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Diethylsulfat</td>
<td>C_{4}H_{10}O_{4}S</td>
<td></td>
</tr>
</tbody>
</table>

Both are group 1 carcinogens and cause cancer in humans.
The visible effect of a DPF

7 m³ exhaust, released in 3 min from a 3.0 L Euro-III engine (100 kW)
Retrofitting of Euro-III to Euro-V heavy duty vehicles – an option for Switzerland?

CH national council rejected

Rejected with 64 yes against 102 no's
The visible effect of a DPF

More than 40 VERT-tested DPFs. All approved systems are excellent particle filters

Mayer et al. MTZ, 2009, 70, 72-79
The visible effect of a DPF

10x

You have to zoom in to see differences among filters

Mayer et al. MTZ, 2009, 70, 72-79
The visible effect of a DPF

Wall-through filters are highly efficient for soot

VERT-approved DPFs:

- Reduce PN-emissions (>98%)
- Reduce genotoxic compounds (a.m.a.p.)
- Low risks of toxic secondary emissions
Impact of DPFs on genotoxicity

Is efficient filtration of soot sufficient to lower the genotoxicity of diesel exhaust?
Carcinogenesis from benzo(a)pyrene

Oxidative metabolic activation of benzo(a)pyrene by cytochrome P450 enzymes

Benzo(a)pyrene (BP) → (+/-) 7,8 BP-oxide

(+/-) 7,8 BP-oxide → (+) anti 7R,8S,9S,10R-BP-dihydrodiol-epoxide

(+/-) 7,8 BP-oxide → (+/-) 7,8 BP-dihydrodiol

(+/-) 7,8 BP-dihydrodiol → (+/-) 7,8 BP-dihydrodiol-epoxide
Carcinogenesis from benzo(a)pyrene

Selective formation of benzo(a)pyrene-DNA-adducts

(+) anti 7R,8S,9S,10R-BP-dihydrodiol-epoxide

(-) 10R trans-anti-[BP]-triol-N2-deoxy-guanosine-adduct
Carcinogenesis from benzo(a)pyrene
Polycyclic aromatic hydrocarbons

PAHs - a diverse class of compounds with variable physicochemical properties

2- to 6-ring PAHs
Polycyclic aromatic hydrocarbons

PAHs - a diverse class of compounds with variable physicochemical properties

2- to 6-ring PAHs

genotoxic
Polycyclic aromatic hydrocarbons

PAHs - a diverse class of compounds with variable physicochemical properties

2- to 6-ring PAHs

precursors for genotoxic NPAHs
Polycyclic aromatic hydrocarbons

PAHs - a diverse class of compounds with variable physicochemical properties

2- to 6-ring PAHs

differ in mass, size & volatility
Non-catalyzed filters are as efficient for soot. How about genotoxic compounds?

What do you expect, can PAHs penetrate DPFs?

Non-catalyzed DPFs:
- Accumulate soot (>98%)

→ Do they reduce genotoxic compounds a.m.a.p?

→ Do they have toxic secondary emissions?
PAH Penetration of a non-catalyzed DPF

Non-catalyzed filter operated <200 °C accumulate soot. How about hydrocarbons?

Do PAHs penetrate non-catalyzed DPFs if operated below 200 °C?

engine-out

before DPF

after DPF
Non-catalyzed filter operated <200 °C to accumulate soot and hydrocarbons

- No retention of naphthalene in a new and a soot-loaded DPF
- Naphthalene is too volatile, it even escapes from a cold filter (<200 °C)
PAH Penetration of a non-catalyzed DPF

Non-catalyzed filter operated <200 °C to accumulate soot and hydrocarbons

- About 30% retention, both in a new and a soot-loaded DPF
- Phenanthrene is partly stored in a cold filter (<200 °C)
PAH Penetration of a non-catalyzed DPF

Non-catalyzed filter operated <200 °C to accumulate soot and hydrocarbons

- 70% is retained in the new, only 15% in the soot-loaded DPF?
PAH Penetration of a non-catalyzed DPF

Non-catalyzed filter operated <200 °C to accumulate soot and hydrocarbons

- Naphthalene
  - New: 218 °C
  - Loaded:

- Phenanthrene
  - New: 340 °C
  - Loaded:

- Fluoranthene
  - New: 375 °C
  - Loaded:

- Pyrene
  - New: 404 °C
  - Loaded:

- 90% is retained in the new, only 5% in the soot-loaded DPF?
Polycyclic aromatic hydrocarbons

PAHs - a diverse class of compounds with variable physicochemical properties

2- to 6-ring PAHs

High penetration of volatile 2-4-ring PAHs
Polycyclic aromatic hydrocarbons

PAHs - a diverse class of compounds with variable physico-chemical properties

2- to 6-ring PAHs

What do we expect for less volatile PAHs?

- 448 °C
- 481 °C
- 495 °C
- 536 °C
PAH Penetration of a non-catalyzed DPF

Non-catalyzed filter operated <200 °C to accumulate soot and hydrocarbons

- 85% retention in the new DPF
- 6x higher emissions from the soot-loaded DPF
PAH Penetration of a non-catalyzed DPF

Non-catalyzed filter operated <200 °C to accumulate soot and hydrocarbons

- 80% retention in the new DPF
- 5x higher emissions from the soot-loaded DPF
PAH Penetration of a non-catalyzed DPF

Non-catalyzed filter operated <200 °C to accumulate soot and hydrocarbons

- 80% retention in the new DPF
- 3x higher emissions from the soot-loaded DPF
Non-catalyzed filter operated <200 °C to accumulate soot and hydrocarbons

PAH Penetration of a non-catalyzed DPF

- We can store and release PAHs from a DPF
Polycyclic aromatic hydrocarbons (PAHs) - a diverse class of compounds with variable physicochemical properties.

2- to 6-ring PAHs:

- **Volatile PAHs** penetrate DPFs at different temperatures:
  - 218 °C
  - 340 °C
  - 448 °C
  - 495 °C

- **Semi-volatile PAHs** are stored but can be released again at:
  - 375 °C
  - 481 °C

- **Non-volatile PAHs** are stored like soot at:
  - 404 °C
  - 536 °C

These PAHs are categorized based on their volatility and melting points, which are crucial for understanding their behavior in different environmental conditions.
Polycyclic aromatic hydrocarbons

Many of the semi-volatile PAHs are genotoxic or precursors of genotoxic compounds

2- to 6-ring PAHs

- Volatile PAHs penetrate DPFs

- Semi-volatile PAHs are stored, but can be released again

- Non-volatile PAHs are stored like soot
Is nitration of PAHs in NOx-rich diesel exhaust an issue?

Nitration of PAHs

pyrene
The DPF – a chemical reactor

In one step from a harmless precursor to a potent mutagen!

Nitration of PAHs

pyrene

1-nitro pyrene
Nitro-PAHs in non-catalyzed DPF

Non-catalyzed filter operated <200 °C to accumulate soot and hydrocarbons

- Pyrene is stored in a new, but released from a soot-loaded DPF
NPAHs in non-catalyzed DPF

- Pyrene is stored in a new, but released from a soot-loaded DPF
- 1-Nitro pyrene is stored in a new, but formed and released from a soot-loaded DPF (30x)
Even a non-catalyzed DPF is a chemical reactor

The nitration potential of a non-catalyzed DPF is high.

Nitration of PAHs

pyrene

1-nitro pyrene
Adverse health effects of diesel exhaust

Problem: Genotoxicity

- Unfiltered diesel exhaust is genotoxic
- Filtration as such is not sufficient to remove all genotoxic compounds
- Efficient catalysts are needed to convert genotoxic compounds

We need catalyzed DPFs!
Catalyzed DPFs convert PAHs

Non-catalyzed filters operated <200 °C accumulate soot and hydrocarbons

- Hox-DPF convert >90% chrysene

Heeb et al. ES&T, 2010, 42, 3773-3779
Catalyzed DPFs convert PAHs

Non-catalyzed filters operated <200 °C accumulate soot and hydrocarbons

- Hox-DPF convert >90% chrysene
- Lox-DPF convert >60% chrysene

Heeb et al. ES&T, 2010, 42, 3773-3779
Catalyzed DPFs convert PAHs

Non-catalyzed filters operated <200 °C accumulate soot and hydrocarbons

- Hox-DPF convert >94% chrysene
- Lox-DPF convert >60% chrysene
- A new non-catalyzed DPF stores chrysene
  (at low temperatures even better than a lox-DPF)

Heeb et al. ES&T, 2010, 42, 3773-3779
Catalyzed DPFs convert PAHs

Non-catalyzed filters operated <200 °C accumulate soot and hydrocarbons

- Hox-DPF convert >94% chrysene
- Lox-DPF convert >60% chrysene
- A new non-catalyzed DPF stores chrysene (at low temperatures even better than a lox-DPF)
- A loaded non-catalyzed DPF can release chrysene (at higher temperatures)

Heeb et al. ES&T, 2010, 42, 3773-3779
Catalyzed DPFs convert PAHs

Non-catalyzed filter operated <200 °C to accumulate soot and hydrocarbons

This is the general trend for many genotoxic PAHs
Adverse health effects of diesel exhaust

VERT-tested catalytic DPFs convert carcinogenic PAHs (on average 85%)
What is BAT today?

Conversion efficiency of carcinogenic PAHs
Problem: Genotoxicity

- Non-filtered diesel exhaust is genotoxic
- Filtration as such is not sufficient to remove genotoxic compounds
- Efficient catalysts are needed to convert genotoxic compounds

Catalytic DPFs are BAT to lower the genotoxicity of diesel exhaust, but some are considerably better than others!
Catalysis, a key property of particle filters to lower genotoxicity of diesel exhaust

A combined effort with many important contributions

- **VERT team**: Andreas Mayer, TTM, Niederrohrdorf
  Jan Czerwinski, Sandro Napoli, Tobias Neubert, Thomas Hilfiker, Samuel Bürki,
  Markus Kasper, Adrian Hess, Thomas Mosimann, Matter Aerosols, Wohlen
  Hans Jaeckle, Urs Debrunner, Oliver Schumm, Intertek Caleb Brett, Schlieren.

- **Empa colleagues**: Brigitte Buchmann, Thomas Bührer, Lukas Emmenegger, Anna-Maria Forss,
  Urs Gfeller, Maria Guecheva, Peter Graf, Roland Graf, Erika Guyer, Regula Haag, Peter
  Honnegger, Judith Kobler, Martin Kohler, Peter Lienemann, Alfred Mack, Peter Mattrel,
  Martin Mohr, Joachim Mohn, Christof Moor, Andreas Paul, Peter Schmid, Cornelia Seiler,
  Andrea Ulrich, Heinz Vonmont, Thomas Walter, Max Wolfensberger, Daniela Wenger,
  Adrian Wichser, Markus Zennegg, Kerstin Zeyer.

- **Governement**: Philipp Hallauer, Giovanni D’Urbano, Felix Reutimann, Max Wyser, Gerhard Leutert,
  Martin Schiess, Swiss Fed. Office for Environment, Bern
  Thomas Gasser, Heinz Berger, Gerhard Stucki, Swiss Federal Road Office

- **Filter- & catalyst manufacturers**: >40 different diesel particulate filter systems

Thanks:
If you see smokers like this, you urgently ask for catalyzed DPFs