National Research Centre for the Working Environment

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Dieseludstødningspartikler i arbejdsmiljøet

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Particle research at the National Research Centre for the Working Environment, Copenhagen

- Government research institute under the Ministry of Employment
- Nanosafety/particles as strategic research area since 2005
- At present 35-40 persons in chemical occupational health
- Advisors for the Danish Working Environment Authorities, EPA, EU, OECD, WHO
- Past and present partners in >30 EU projects on (nano)particle safety

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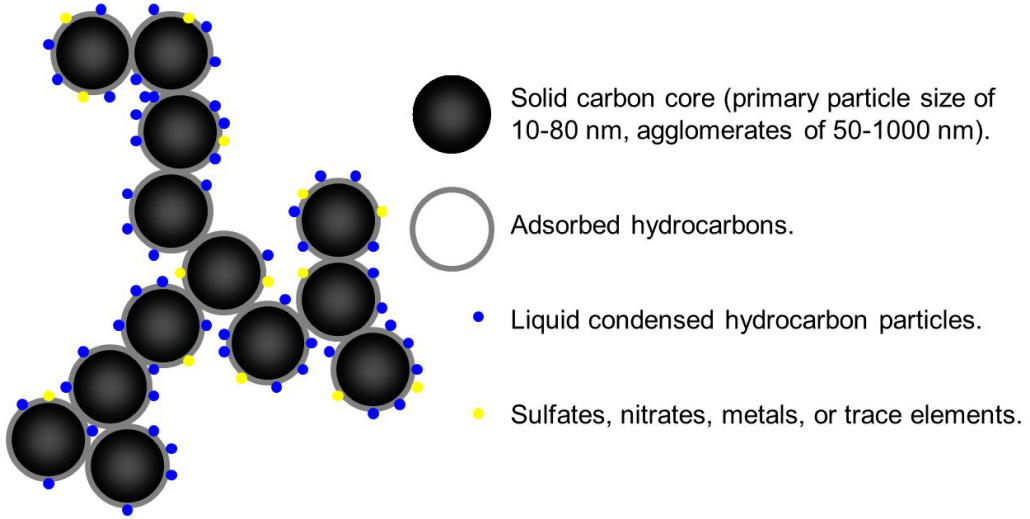
Diesel exhaust emission (DEE)

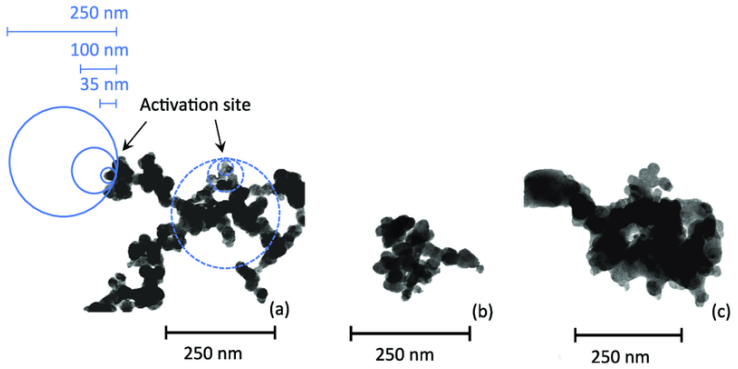
- DEE consists of gases (VOC, CO_x, NO_x) and particles

- Diesel exhaust particles (DEP):
  - Solid carbon core, often aggregated
  - Adsorbed PAH and organic carbon
  - Adsorbed metals
  - Specific surface area ca. 100 m^2/g
  - Can be regarded as a process-generated nanomaterial
  - Elemental carbon, black carbon or PM are often used as measures of DEP

- Classified as carcinogenic to humans



Adapted from Marano, et al. (2002). *Cell Biol Toxicol*. 18(5): 315-320.



95% of the global burden of occupational cancer is caused by 4 agents including diesel engine exhaust

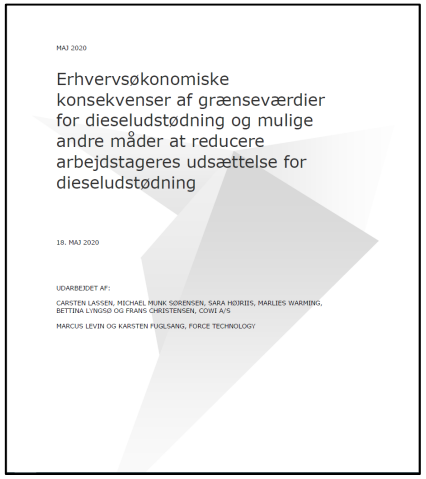
Table 1 Global occupation-attributable cancer deaths and DALYs by carcinogen and cancer type, 2016—number and per cent

Carcinogen	Deaths*	% of deaths	DALYs	% of DALYs
Arsenic†	8073 (2053–14 628)	2.3 (0.6–4.2)	219 218 (57 757–395 480)	3.0 (0.8–5.5)
Asbestos	218 827 (165 455–274 682)	62.7 (47.4–78.8)	3 556 876 (2 657 069–4 514 222)	49.4 (36.9–62.7)
Larynx cancer	3743 (2024–5528)		65 506 (35 042–99 124)	
Lung cancer	181 450 (128 287–236 621)		2 844 282 (1 957 872–3 803 219)	
Ovary cancer	6022 (2984–9404)		93 120 (45 796–149 948)	
Mesothelioma	27 612 (25 559–29 341)		553 967 (507 287–597 783)	
Benzene‡	1899 (596–3123)	0.5 (0.2–0.9)	83 867 (25 512–138 493)	1.2 (0.4–1.9)
Beryllium†	259 (213–312)	0.1 (0.1–0.1)	7223 (5886–8594)	0.1 (0.1–0.1)
Cadmium†	605 (504–709)	0.2 (0.1–0.2)	16 832 (14 142–19 639)	0.2 (0.2–0.3)
Chromium†	1276 (1126–1443)	0.4 (0.3–0.4)	35 452 (31 397–40 172)	0.5 (0.4–0.6)
Diesel engine exhaust†	17 500 (15 195–20 057)	5.0 (4.4–5.8)	485 693 (426 181–553 926)	6.7 (5.9–7.7)
Formaldehyde	1086 (900–1324)	0.3 (0.3–0.4)	46 932 (38 805–56 986)	0.7 (0.5–0.8)
Leukaemia	*608* (505–722)		27 914 (22 861–33 605)	
Nasopharynx cancer	*478* (330–685)		19 018 (12 994–27 091)	
Nickel†	8101 (1243–20 812)	2.3 (0.4–6.0)	221 352 (34 934–563 339)	3.1 (0.5–7.8)
Polycyclic aromatic hydrocarbons†	4526 (3826–5291)	1.3 (1.1–1.5)	125 779 (105 369–145 866)	1.7 (1.5–2.0)
Secondhand smoke	49 246 (25 336–80 957)	14.1 (7.3–22.2)	1 345 915 (703 984–2 186 305)	18.7 (9.8–30.4)
Breast cancer	4864 (1195–8401)		160 494 (39 883–276 832)	
Lung cancer	44 382 (20 655–75 463)		1 185 422 (551 749–2 013 661)	
Silica†	47 999 (21 235–75 452)	13.8 (6.1–21.6)	1 303 949 (576 291–2 042 004)	18.1 (8.0–28.4)
Strong inorganic-acid mists§	3535 (1520–6491)	1.0 (0.4–1.9)	105 226 (45 836–192 418)	1.5 (0.6–2.7)
Trichloroethylene¶	58 (13 –108)	0.0 (0.0–0.0)	1722 (379–3228)	0.0 (0.0–0.0)
Total**	348 741 (269 406–427 386)	100.0	7 199 850 (5 813 091–8 641 244)	100.0

*The numbers in brackets are 95% uncertainty intervals.
†Causes lung cancer.
‡Causes leukaemia.
§Causes laryngeal cancer.
¶ Causes kidney cancer.
**Numbers percentages add to more than 100 due to overlapping causes.
DALY, disability-adjusted life year.

Exposure

- Close to sources, ie diesel engine outlet
- Higher in confined areas/inside buildings
- Lower outdoors
- Limited knowledge on Danish exposure levels



MAJ 2020

Erhvervsøkonomiske konsekvenser af grænseværdier for dieseludstødning og mulige andre måder at reducere arbejdstageres udsættelse for dieseludstødning

18. MAJ 2020

UDARBEJDET AF:

CARSTEN LASSEN, MICHAEL MUNK SØRENSEN, SARA HØRIIS, MARLIES WARMING, BETTINA LYNGSØ OG FRANS CHRISTENSEN, COWI A/S

MARCUS LEVIN OG KARSTEN FUGLSANG, FORCE TECHNOLOGY

https://at.dk/media/6236/graensevaerdier-dieselpartikler-2020.pdf

5.4 Færgepersonale, der leder biler og lastbiler på plads

Der er rettet henvendelse til færgeselskaberne Molslinjen, Scandlines og ForSea. Disse selskaber repræsenterer ca. 50% af det samlede antal personbiler med inden- og udenrigsruter [28]. En stor del af de mindre ruter betjener sig af færger med åbne dæk, hvor eksponeringen af personalet her vil antages at være mindre end på de større færger med lukkede dæk.

For personale der arbejder på skibe gælder BEK nr. 9104 af 01/01/2006 "Meddelelser fra Søfartsstyrelsen A, arbejdsmiljø i skibe, kapitel A II C, fysiske arbejdsmiljøpåvirkninger som i "Afsnit C Kræftfremkaldende stoffer og materialer herunder asbest samt mutagener" gennemfører direktiv 2004/37/EF (CMD) og har krav, der på mange måder svarer til Kræftbekendtgørelsens. Bekendtgørelsen angiver, at grænseværdier fastsat af Arbejdstilsynet skal indgå i arbejdspladsvurderingen, men præciserer kun i relation til grænseværdien for asbest, at grænseværdier skal efterleves (modsat Kræftbekendtgørelsens krav). Det vil her antages, at fastsatte danske grænseværdier skal efterleves om bord på skibe.

	Færgepersonale, der leder biler og lastbiler på plads
Hvordan sker udsættelsen?	Udsættelsen sker, når personalet leder køretøjer ud og ind af færgerne i relativt lukkede rum, eller når personalet kører med trucks på området.
Typer af forskellige arbejdspladser med eksponering inden for den enkelte virksomhed	Personale der leder biler ud og ind af færgerne (delvist om bord på færgerne) Personale der kører trucks ind på færgerne. Der vil også kunne være en eksponering af personale, der arbejder på havneområdet, men denne eksponering vil ikke formodes at være større end ved anden brug af trucks udendørs.
Eksponeringskoncentrationer og eksponeringstid	Der er ikke fundet danske målinger, som dokumenterer udsættelsen af færgepersonale. Det er ved dataindhentningen oplyst af et selskab, at der er foretaget målinger af eksponering af personalet, men det har af fortrolighedsgrunde ikke være muligt at videregive resultaterne. Den eneste undersøgelse, der er fundet, er fra år 2000, hvor der ved 20 målinger i Storbritannien blev fundet en middelværdi på 39 µg EC/m³ (se Tabel 3.6). 95% fraktilen er ikke angivet, men må regnes at være væsentligt højere og kan meget vel være over 100 µg EC/m³. Der er desuden fundet en svensk undersøgelse fra 1987 af eksponering af personale der leder køretøjer ind og ud af færger, men denne undersøgelser har ikke målt for ultrafine partikler, BC eller EC (Ulvarson et al., 1987). Medarbejdere, der leder køretøjer ud og ind, vil typisk være eksponeret i 25-30% af arbejdstiden. Dvs. at 95% fraktilen af eksponeringskoncentrationerne i denne tid kan være op til ca. 3 gange grænseværdien. I følge oplysninger fra et af de interviewede selskaber kan der være dage, hvor der grundet vejrforhold, er højere koncentrationer på vogndækket end andre dage. Den britiske undersøgelse er 20 år gammel. Udslip fra dieselkøretøjer er faldet med en faktor 10-20 siden da, og der er installeret forbedret ventilation på færgerne, kan det ikke afvises, at der for enkelte selskaber vil kunne være behov for yderligere foranstaltninger for at være i overensstemmelse med en grænseværdi på 5 µg EC/m³. Den vil dog næppe være over 10 µg EC/m³.

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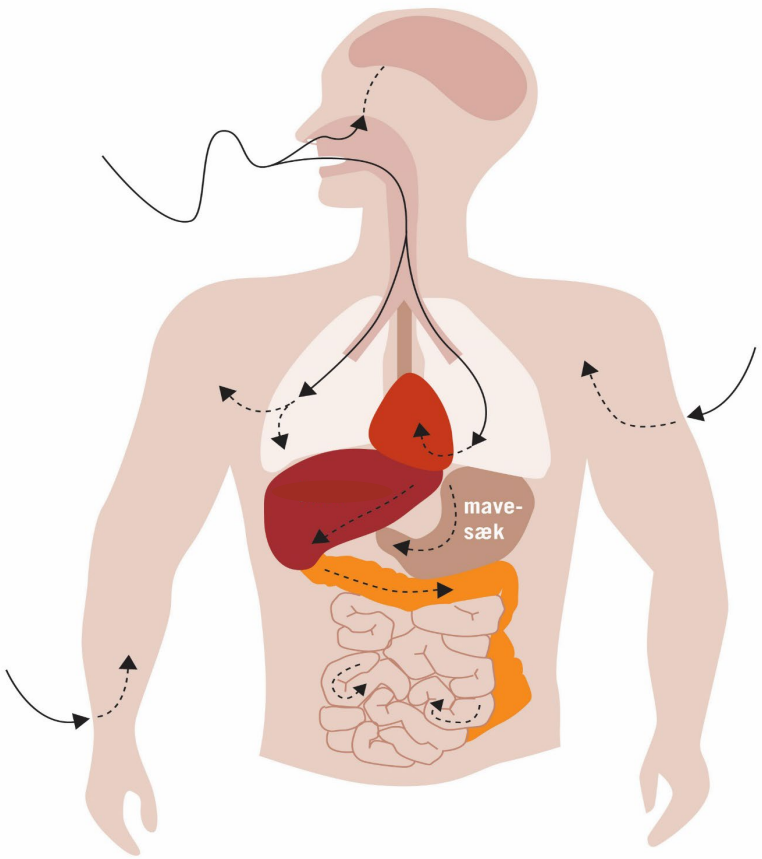
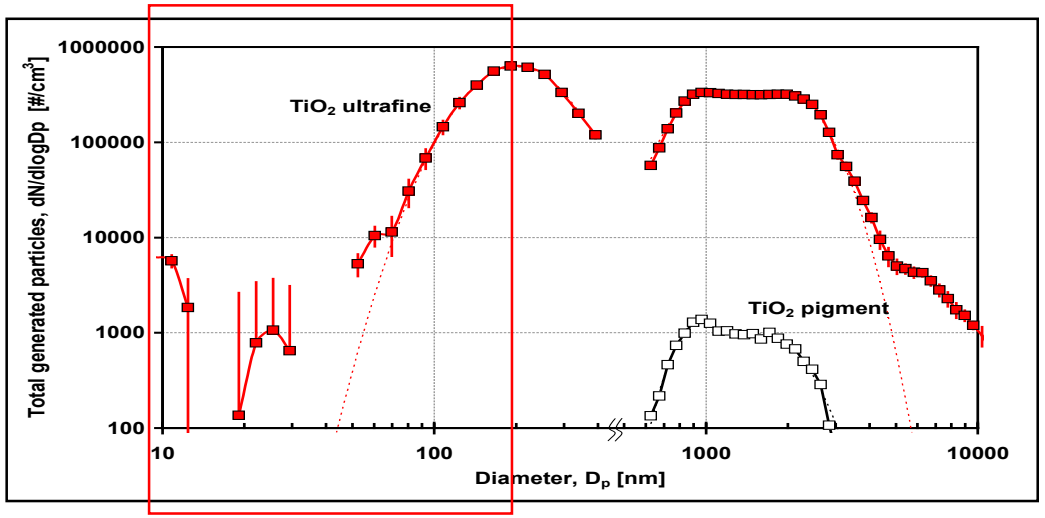
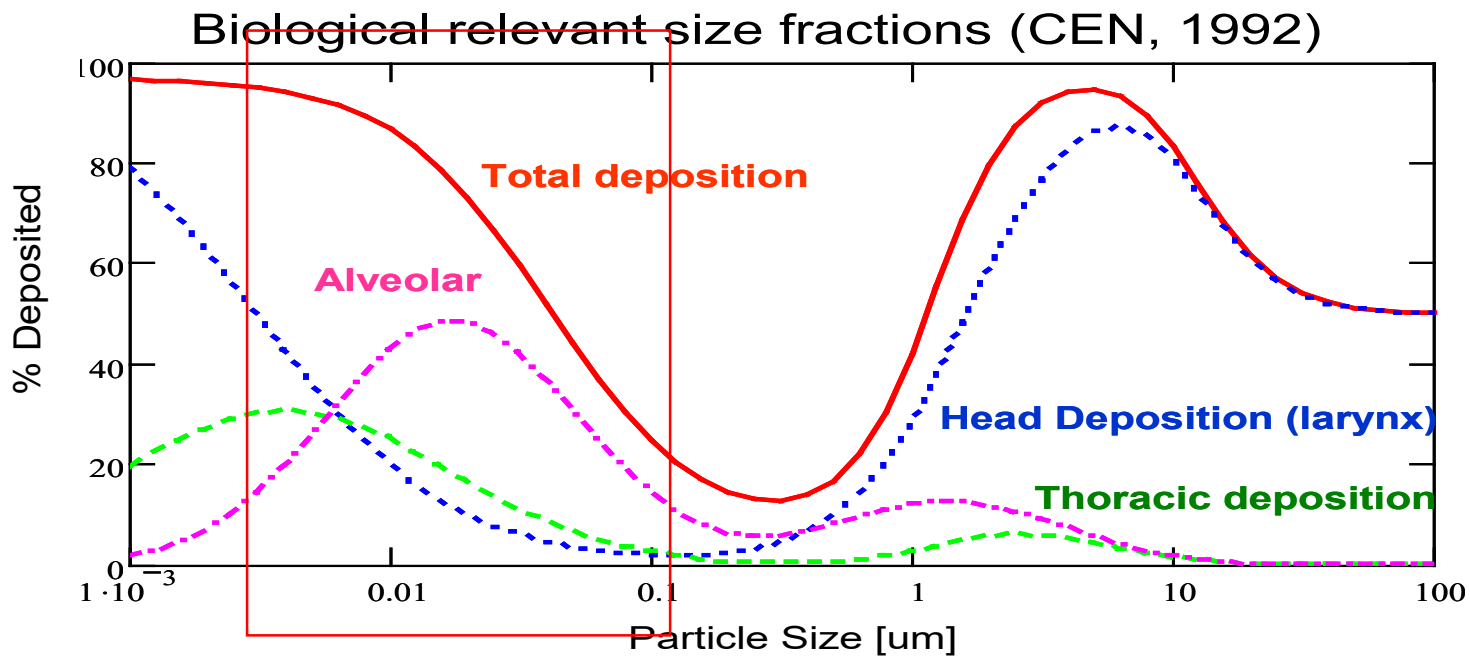
Why are they hazardous?

Aerodynamic size in air is the important predictor of pulmonary deposition during inhalation exposure

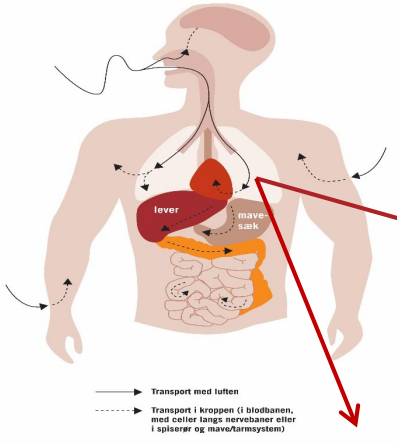
Nanomaterials aggregate in air







Low clearence of nanoparticles from the lung



Fine particles

Nanoparticles

Ultrafine particles

Airways

Particle deposition and removal by mucociliar clearance

Particle phagocytosis by macrophage

INFLAMMATION

release of mediators

ROS

Inflammatory cells and inflammatory mediators

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Marianne Dybdahl

Inhaled TiO_2 nanoparticles in the lung are removed very slowly

Mice inhaled 40 mg/m^3 nanosized TiO_2 1 hour daily for 11 days.

TiO_2 content in lung tissue was measured by ICP-MS.

Exposure	Days after exposure	N	TiO_2 in lung (mg/kg) (mean ± sd)	Procent of deposited dose
TiO_2	5	3	63 ± 10	24%
Air	5	3	< 8	
TiO_2	25	3	55 ± 30	21%
Air	25	3	< 1	

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Hougaard et al, PF&T, 2010

From IARC classification as human carcinogen to occupational exposure limit: 9 years, short or long time..

2012: IARC classified diesel exhaust as carcinogenic to humans

2016: The Danish Working Environment Authority asked NRCWE to provide documentation for health-based occupational exposure limit for diesel exhaust particles

2018: NRCWE submitted the documentation ultimo 2018. The social partners negotiated an occupational exposure limit based on the documentation for health-based risk estimates and a Socioeconomic assessment report

2019-2020: Additional scientific data was published and communicated

2021: The EU occupational exposure limit of 0.05 mg/m^3 effective from Feb 20th, 2021 was replaced by a Danish occupational exposure limit at 0.01 mg/m^3 on July 1st, 2021

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The available evidence

The documentation report is avaiable at https://nfa.dk/da/Forskning/Strategiske-forskningsomraader/Kemisk-arbejdsmiljo/Graensevaerdier

Critical effects:

- Focus on inhalation exposure

- IARC (2014):
  - sufficient evidence of carcinogenicity of diesel engine exhaust, diesel exhaust particles, diesel partilcle extracts in experimental animals
  - sufficient evidence that diesel engine exhaust is carcinogenic to humans and causes lung cancer

- DECOS/NEG (2016): critical effects of diesel engine exhaust inhalation are lung cancer and inflammation

Proposed key characteristics of carcinogens

Table 1. Key characteristics of carcinogens.

Characteristic	Examples of relevant evidence
1. Is electrophilic or can be metabolically activated	Parent compound or metabolite with an electrophilic structure (e.g., epoxide, quinone), formation of DNA and protein adducts
2. Is genotoxic	DNA damage (DNA strand breaks, DNA–protein cross-links, unscheduled DNA synthesis), intercalation, gene mutations, cytogenetic changes (e.g., chromosome aberrations, micronuclei)
3. Alters DNA repair or causes genomic instability	Alterations of DNA replication or repair (e.g., topoisomerase II, base-excision or double-strand break repair)
4. Induces epigenetic alterations	DNA methylation, histone modification, microRNA expression
5. Induces oxidative stress	Oxygen radicals, oxidative stress, oxidative damage to macromolecules (e.g., DNA, lipids)
6. Induces chronic inflammation	Elevated white blood cells, myeloperoxidase activity, altered cytokine and/or chemokine production
7. Is immunosuppressive	Decreased immunosurveillance, immune system dysfunction
8. Modulates receptor-mediated effects	Receptor in/activation (e.g., ER, PPAR, AhR) or modulation of endogenous ligands (including hormones)
9. Causes immortalization	Inhibition of senescence, cell transformation
10. Alters cell proliferation, cell death or nutrient supply	Increased proliferation, decreased apoptosis, changes in growth factors, energetics and signaling pathways related to cellular replication or cell cycle control, angiogenesis

Abbreviations: AhR, aryl hydrocarbon receptor; ER, estrogen receptor; PPAR, peroxisome proliferator–activated receptor. Any of the 10 characteristics in this table could interact with any other (e.g., oxidative stress, DNA damage, and chronic inflammation), which when combined provides stronger evidence for a cancer mechanism than would oxidative stress alone.

Release of toxic substances; fx PAH, metals

Surface-dependent ROS generation

Deposited total surface area

Shape (HARN)

Smith MT EHP, 2016, PMID: 26600562

Mechanism of action

- Both DEP and NO_x induces inflammation. **So inflammation cannot be used for risk assessment of DEP**
- 2 year cancer studies in rats: Diesel Engine Exhaust (DEE) induces lung cancer, but not filtered diesel engine exhaust. Thus, **the particulate fraction is the carcinogenic component (Brightwell 1989)**.
- Both inhalation of diesel engine exhaust and instillation of diesel exhaust particles and diesel exhaust particle extracts induced mutations in lungs of mice (Hashimoto 2007)
- Evidence that both carbon core and diesel exhaust particle extracts (PAH, OC) contribute to carcinogenicity (Hashimoto, 2007, Heinrich 1995)
- PAH adduct formation and particle **surface-induced ROS: primary genotoxicity and non-threshold effects**

Five different two-year inhalation studies in rats

Table 3. Diesel engine exhaust inhalation studies in rats with observed dose carcinogenicity response

Reference	Strain (sex) Group size	Exposure	DEP mg/m³	NO_2	NO ppm	NOx	Lung tumor incidence M	Lung tumor incidence F
Mauderly et al., 1986	F344 (M/F)	Clean air and DEE (1980 5.7-L V8)	0				1.4%	
	N = 221-230	7 h/d, 5 d/w for up to 30 months	0.35	0.01			0.7%	
			3.5	0.3			4.6%*	
			7.0	0.7			16.1%*	
Brightwell, 1989	F344 (M/F)	Cond. air and DEE (VW Rabbit 1.5-L)	0				1.5%	0.8%
	N = 72 or 144	16 h/d, 5d/w for 24 + 6 months	0.7				1,.4%	0.0%
			2.2	0.9-2.8			4.2%	15.3%*
			6.6				22.5%*	54.2%*
Mauderly et al., 1994	F344 (M/F)	Cond. air and DEE (Two ´88 LH6 GM 6.2L V8)	0				3.0%	0.0%
	N = 100	16 h/d, 5d/w for 24 + 6 months	2.5	0.7		8.8	5.0%	8.0%
			6.5	3.8		24	9.0%	29.0%*
Heinrich et al., 1995	Wistar (F)	Clean air and DEE (Two VW 40-kW 1.6-L)	0					0.5%
	N = 100-220	18 h/d, 5d/w for 24 + 6 months	0.84	0.3		4.7		0.0%
			2.5	1.2		14		5.5%*
			7.0	3.8		33		22.0%*
Stinn et al., 2005	Wistar (M/F)	Clean air and DEE (VW 1.6-L)	0				4.0%	0.0%
	N = 99	6 h/d, 7 d/w for 24 + 6 months	3		7	9	18.0%*	28.0%*
			10		23	28	34.7%*	56.9%*

The table is adapted from IARC Table 3.2 (IARC 2014). DEE, DEPs (measured particulate matter in mg/m³). Cond.: Conditioned. Brightwell also included a filtered exhaust exposure with 99.7% of the mass removed. No increased tumor incidence was observed.

F344; inbred
Wistar: outbred

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Dose-response relationship for carcinogenicity in male and female rats

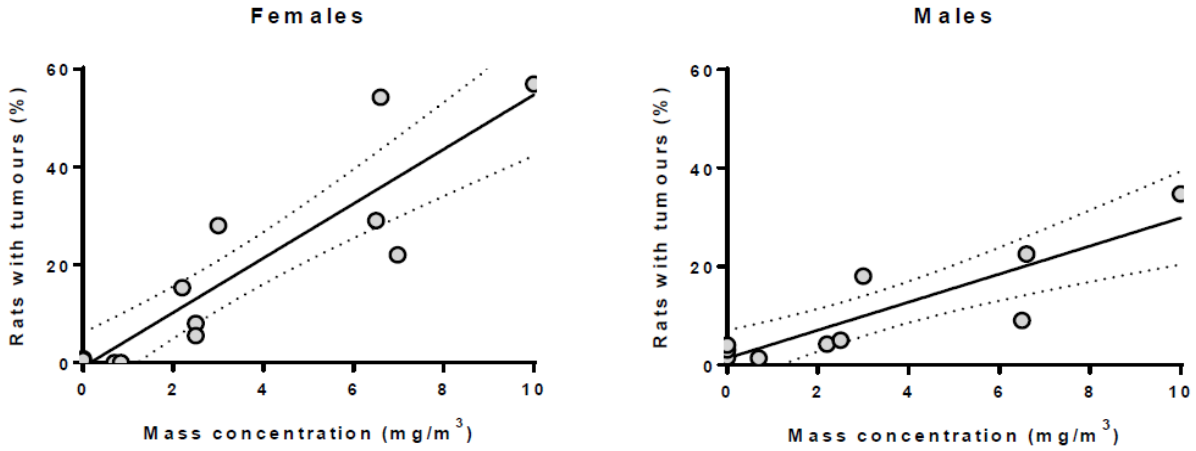

Figure 2. **Frequency of female and male rats with tumors as a function of DEP mass concentrations in the chronic inhalation studies by (Brightwell et al. 1989;Heinrich et al. 1995;Mauderly et al. 1994;Stinn et al. 2005). Dotted lines represent 95% confidence interval for the regression lines. Females: y = 5.6x – 0.088; Males: y = 2.8x +1.4**

Traditional technology diesel engine exhaust (DEE) vs new technology DEE

- New technology engines emit:
  - Lower amount of PM
  - DEP with lower EC content (13% of PM instead of 75%)
  - Relatively higher NO_x emissions

- All epidemiological studies and the majority of chronic inhalation studies were performed using tradtional DEE
- One chronic inhalation study in rats using new technology DEE used 12 ug/m^3 DEP as the highest dose. No increased cancer incidence compared to controls were found with group sizes of 100 males and 100 females.
- The NFA working group regarded the DEP concentrations as being too low to allow detection of DEP-induced cancer (considering that 1:1000 has required 5-50 ug/m^3 in other chronic inhalation studies)
- A comparison of 5 diesel exhaust particles including 2 biodiesels showed that DNA damage levels correlated with EC content (Bendtsen et al 2020, PFT; PMID: 32771016)

Systematic meta-analysis of epidemiological studies

Research

All *EHP* content is accessible to individuals with disabilities. A fully accessible (Section 508–compliant) HTML version of this article is available at http://dx.doi.org/10.1289/ehp.1306880.

Exposure-Response Estimates for Diesel Engine Exhaust and Lung Cancer Mortality Based on Data from Three Occupational Cohorts

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[1]Division of Environmental Epidemiology, Institute for Risk Assessment Sciences, Utrecht University, Utrecht, the Netherlands;

- Inclusion criteria:
- 1) DEE exposure was given as cumulative expoure in the exposure response analysis
- 2) an appropriate no/low exposure group was used as control
- 3) no major methological shortcomings were identified.

Three epidemiological studies in the meta-analysis

	Steensland (1998)	Garshick (2012)	Silverman (2012)
Design	Nested case-control study All cases and control died 1982-83 and were long-term Teamster members (N=10 699) (1949-1990)	Cohort study of male workers in the US trucking industry (1985-2006)	Nested case-control of 8 non-metal mining facilities including 12,315 workers (1947-1997)
N	994 lung cancer cases, 1,085 controls	31 135 male workers, 779 lung cancers Mechanics excluded (unreliable JEM)	198 lung cancer and 562 controls
Exposure	JEM based on 242 samples quantified 1988-89	JEM based on 4000 measurements 2001-6	JEM based on 700 measurements 1998-2001
Adjustments	Age, race, smoking, diet, self-raported asbestos	Not adjusted for smoking	Adjusted for smoking

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Cumulative dose-response relationship based on 3 studies

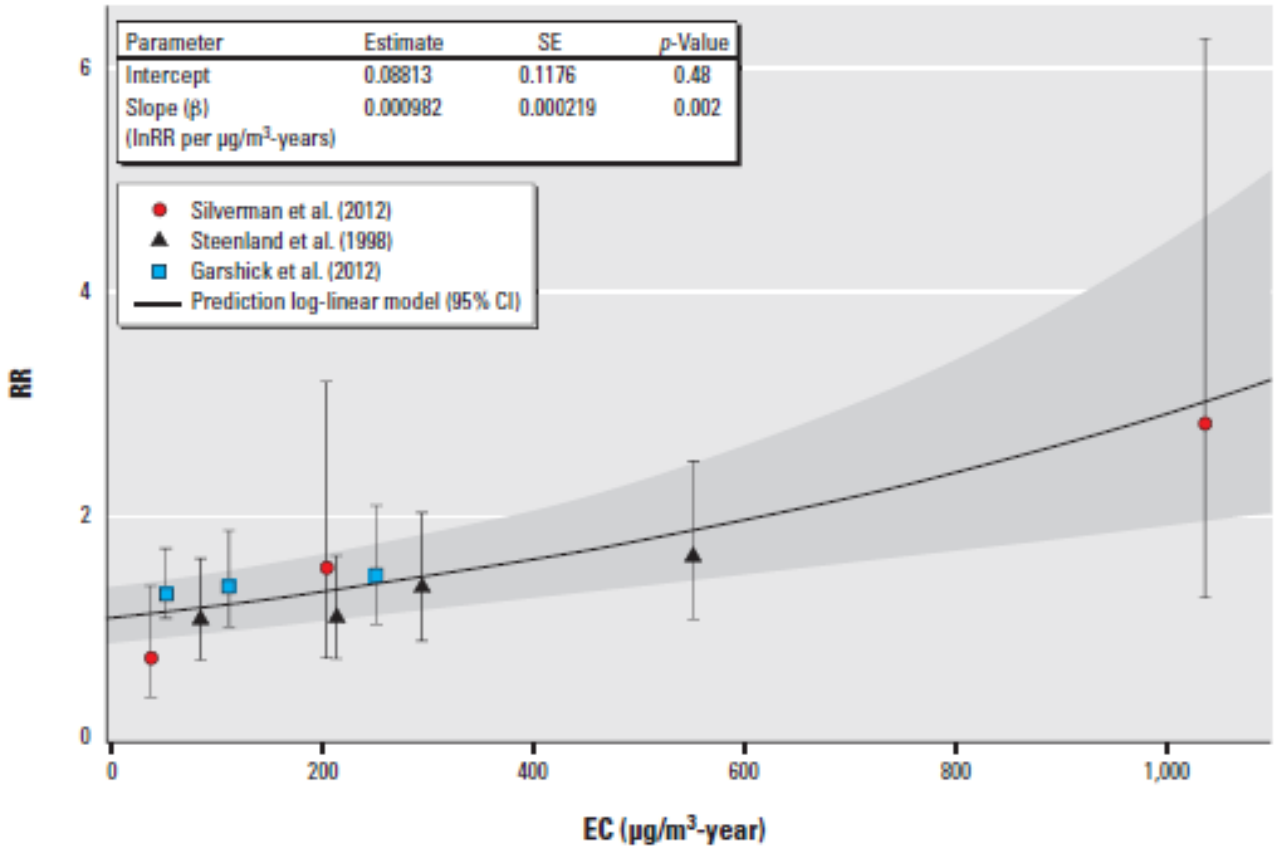


Figure 1. Predicted exposure–response curve based on a log-linear regression model using RR estimates from three cohort studies of DEE and lung cancer mortality. Individual RR estimates [based on HRs reported by Garshick et al. (2012) or ORs reported by Silverman et al. (2012) and Steenland et al. (1998)] are plotted with their 95% CI bounds indicated by the whiskers. The shaded area indicates the 95% CI estimated based on the log-linear model. The insert presents the estimates of the intercept and beta slope factor, the SE of these estimates, and the associated *p*-values.

Vermeulen et al, EHP

Risk estimate for DEP based on epidemiological evidence

Table 1. Exposure–response estimates (lnRR for a 1-μg/m^3 increase in EC) from individual studies and the primary combined estimate based on a log-linear model.

Model[a]	Intercept	β (95%CI)
All studies combined	0.088	0.00098 (0.00055, 0.00141)
Silverman et al. (2012) only	–0.18	0.0012 (0.00053, 0.00187)
Steenland et al. (1998) only	–0.032	0.00096 (0.00033, 0.00159)
Garshick et al. (2012) only	0.24	0.00061 (–0.00088, 0.00210)

[a]Log-linear risk model (lnRR = intercept + β × exposure). Exposure defined as EC in μg/m^3-years.

Table 2. Excess lifetime risk per 10,000 for several exposure levels and settings, United States in 2009.

Exposure setting	Average EC exposure (μg/m^3)	Excess lifetime risk through age 80 years (per 10,000)
Worker exposed, age 20–65 years	25	689
Worker exposed, age 20–65 years	10	200
Worker exposed, age 20–65 years	1	17
General public, age 5–80 years	0.8	21

Based on linear risk function, lnRR = 0.00098 × exposure, assuming a 5-year lag, using age-specific (5-year categories) all cause and lung cancer mortality rates from the United States in 2009 as referent.

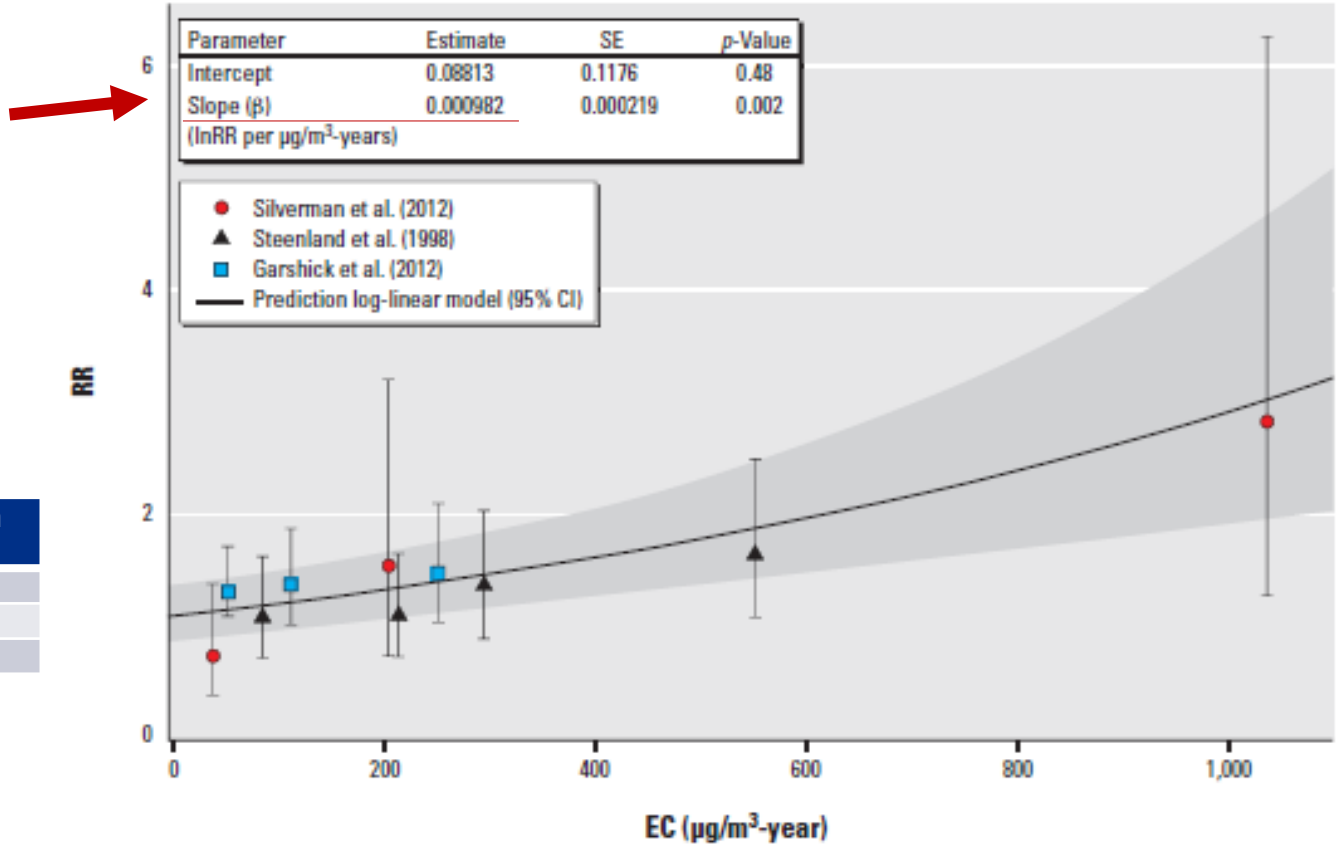
The EU OEL for DEP is 50 ug/m^3

Vermeulen et al, 2014, EHP

Derived health-based OEL based on epidemiological data

Risk estimates were calculated based on Danish lung cancer incidence (4.9%) and the slope of the dose-response relationship

Excess lung cancer risk (45 years of work)	DEP air concentration (measured as EC)
1: 1 000	0.45 µg/m³
1: 10 000	0.045 µg/m³
1: 100 000	0.0045 µg/m³



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Vermeulen et al, EHP, 2014

Lung cancer risk based on 2 year inhalation studies in rats based on lung burden

Table 7. Observed cancer incidence following DEP exposure in (Heinrich et al. 1995)

DEP concentration	0	2.5 mg/m³	7.0 mg/m³
Cancer Incidence	1/217	11/200	22/100
Lung burden (mg/lung)		23.7	63.9

Observed cancer incidence at 2.5 mg/m³:
(11/200 – 1/217)/(1-1/217) = 0.05 = 5%

Lung deposited dose in rats at 2.5 mg/m³: 23.7 mg/lung.

The human equivalent dose is:

(Rat deposited dose) x (human alveolar surface area)/(rat alveolar surface area) =
23.7 mg x 102 m²/0.4 m²= 6 043.5 mg DEP per human lung.

Assuming 16.8% deposition as previously reported for humans by (NEG/DECOS)(Taxell and Santonen 2016).

Using the values above, a lung burden of 6 043.5 mg in humans would require that workers are exposed to:

Air concentration =
6 043.5 mg/(8h/day x 5 days/week x 45 weeks/year x 45 years x 1.2 m³/h x 0.168) =
0.37 mg/m³

Thus, at an air concentration of 0.37 mg/m³ during a 45-year work life, an excess lung cancer incidence of 5% is expected. Assuming a linear dose-response relationship, then 1% excess lung cancer is expected at:

(0.37 mg/m³)/5 = 0.074 mg/m³

Table 8. Excess cancer risk

Excess lung cancer risk	DEP air concentration
1: 1 000	7.4 µg/m³
1: 10 000	0.74 µg/m³
1: 100 000	0.074 µg/m³

Lung cancer risk based on air concentrations in animal studies

Method II

Risk estimates were calculated as recommended by ECHA (ECHA 2012a; SCHER/SCCP/SCENIHR 2009), based on the 2 year DEE inhalation study in rats by (Heinrich et al. 1995) (Table 4):

Excess cancer risk:

Observed excess cancer incidence at 2.5 mg/m³:

(5/200- 1/217)/(1-1/217)= 0.0506 = 5 %

Correction of dose metric for humans during occupational exposure (8h/d):

2.5 mg/m³ x (18 h/day)/(8 h/day) x (6.7 m²/10 m²) = 3.769 mg/m³

Calculation of unit risk for cancer:

Risk level = exposure level x unit risk
0.0506 = 3 769 µg/m³ x unit risk
Unit risk = 1.34 x 10^{-5} per µg/m³

Table 9. Calculated excess lung cancer incidence at DEP mass concentrations based on method II

Excess lung cancer incidence	DEP Air concentration (µg/m³)
1: 1 000	74
1: 10 000	7.4
1: 100 000	0.74

Summary of risk estimates for epidemiological studies, low- and high responders in rat studies

Table 13. Overview of exposure levels in terms of EC, resulting in extra cancer risk levels at 1:1000, 1:10 000 and 1: 100 000 based on a non-threshold based mechanism of action using different approaches

	Suggestion of an OEL for DEP calculated as EC			
Excess lung cancer incidence	Human studies	Method I, µg/m³ Rat inhalation study of DEE*	Method II, µg/m³ Rat inhalation study of DEE*	
	Vermeulen	Heinrich	Heinrich	Brightwell
1: 1 000	0.45 µg/m³	5.6 µg/m³	56	15
1: 10 000	0.045 µg/m³	0.56 µg/m³	5.6	1.5
1: 100 000	0.0045 µg/m³	0.056 µg/m³	0.56	0.15

Method I is based on lung deposition. Method II is based on air concentrations and following ECHA guidelines.*For traditional DEPs, it is assumed that 75% of the mass is EC (Taxell and Santonen 2016).

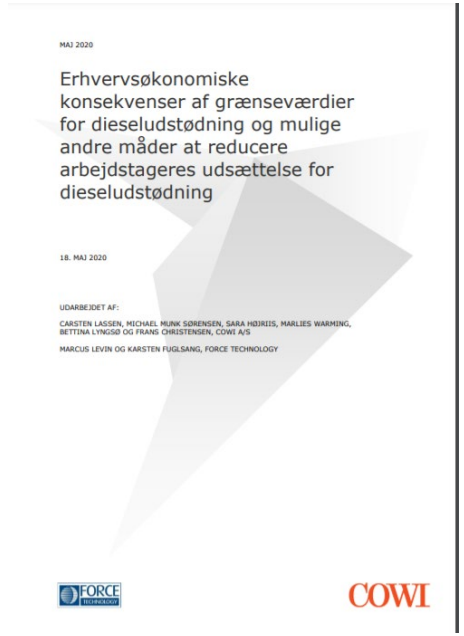
Dutch Committee on Occupational Safety (DECOS) in 2019:

- The DECOS estimates that the exposure concentrations of respirable elemental carbon (REC) in the air, which serve as parameter for exposure to diesel engine exhaust powered by petroleum-diesel fuels, and which corresponds to:
- 4 extra death cases of lung cancer per 100,000 (target risk level), for 40 years of occupational exposure, equals to 0.011 µg REC/m^3,
- 4 extra death cases of lung cancer per 1,000 (prohibition risk level), for 40 years of occupational exposure, equals to 1.03 µg REC/m^3.
- The exposure levels are 8-hour time-weighted average concentrations.

https://www.healthcouncil.nl/documents/advisory-reports/2019/03/13/diesel-engine-exhaust.

Assessment of socioeconomic consequences

- Assessment of the socioeconomic consequences of an occupational exposure limit at 50 and 5 ug EC/m^3
- Associated health effects were not assessed
- Conclusions:
- Urban background levels are now 0.5-2 ug EC/m^3
- An OEL at 50 ug EC/m^3 would not infer any additional expenses for industry
- An OEL at 5 ug EC/m^3 would infer additional expenses for industry to lower diesel exhaust exposure
- An OEL at 1 ug EC/m^3 would infer considerable additional expenses for industry to lower diesel exhaust exposure



https://at.dk/media/6236/graensevaerdier-dieselpartikler-2020.pdf

Additional data: pooled analysis of diesel case-control studies published in 2020

- New pooled analysis of 14 case-control studies with almost 17,000 lung cancer cases and 21,000 controls,
- Exposure was estimated using job-exposure matrices.

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ORIGINAL ARTICLE

Diesel Engine Exhaust Exposure, Smoking, and Lung Cancer Subtype Risks

A Pooled Exposure–Response Analysis of 14 Case–Control Studies

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Additional pooled analysis of diesel case-control studies

The two studies were not directly comparable, and consequently we averaged the slopes of the dose-response curve

Risk-estimates for lung cancer (EC)	based on Meta-analysis (Vermeulen)	based on pooled study (Ge et al.)	average of two studies
1:100	4.5 µg/m^3	12 µg/m^3	6 µg/m^3
1:1000	0.45 µg/m^3	1.3 µg/m^3	0.67 µg/m^3
1: 10 000	0.045 µg/m^3	0.13 µg/m^3	0.067 µg/m^3

EC= Elemental carbon, ie the carbon core

The EU OEL of 50 µg/m^3 respirable EC would correspond to a risk estimate of 4-10% risk of lung cancer

Biomonitering study on volunteers shows effect of expoure to 10 µg/m³ EC

- 29 voluneers took the train for 6h/day for three conseqtive days, either diesel-driven trains or electric trians.
- In the dieseldriven trains, the average expure was 10.3 µg/m³ EC as compared to 0.8 µg/m³ EC in electric trains.
- After 3 days of exposure for diesel engine exhaust, the volunteers had:
  - Increased levels of DNA damage in blood cells as indication of carcinogenic exposure
  - Slightly reduced lung function

	Diesel	Electric	P-value
#observations	54	29	
Black Carbon (EC) (µg/m³)	10.3 ± 2	0.8 ± 0.5	< 0.001
#ultrafine particles/cm³	189 200 ± 91 900	8100 ± 2400	< 0.001
DNA-damage	0.18 ± 0,13	0.12 ± 0.13	0.025
Lung function (FEV_1, L)	3.24 ± 0.96	3.32 ± 0.96	0.0003

Andersen et al. 2019, Part Fibre Tox

New Danish occupational exposure limit for diesel exhaust at 0.01 mg/m³ July 1st, 2021 (similar to the Dutch OEL)

Bekendtgørelser

Bilag 2 - Grænseværdier for luftforureninger m.v.

Bilag 2 til Arbejdstilsynets bekendtgørelse nr. 1426 af 28. juni 2021 om grænseværdier for stoffer og materialer

77-73-6	Dicyclopentadien (1996)	0,5	2,7	
60-57-1	Dieldrin	-	0,25	HK
	Emissioner fra dieseludstødning(2021)	-	0,01[8)]	EK
111-42-2	Diethanolamin (1996)	0,46	2	H
109-89-	Diethylamin (1996)	5	15	EH

The Danish social partners will re-negotiate in 2024 whether the OEL can be lowered to 0.005 mg/m³

How do you measure diesel engine exhaust exposure

- Diesel engine exhaust particles are measured as Elemental carbon (EC)
- Exposure should be measured in the inhalation zone of the workers
- NIOSH 5040 method:
- Total dust is collected on a quartz filter
- The content of elemental carbon is measured using thermic or optical methods
- Several companies offer occupational exposure measurements for diesel engine exhaust exposure

Conclusions

- Denmark now has (together with the Netherlands) the lowest occupational exposure limit for diesel exhaust particles in EU and in the world
- The OEL will be revisited, and possibly further lowered in 2024

- Diesel engine exhaust is classified as carcinogenic to humans by IARC
- There is a considerable body of evidence for the dose-response relationship in humans and in rodents
- The European occupational exposure limit of 50 ug/m^3 corresponds to an excess life time risk level of 4-10% for lung cancer
- Volunteers exposed to diesel engine exhaust at 10 ug/m^3, the Danish and Dutch occupational exposure limit, have increased levels of DNA damage in blood cells, suggesting genotoxic/carcinogenic exposure

- Exposure should be as low as reasonably achievable (AT)

National Research Centre for the Working Environment

14-04-2023

Tak for opmærksomheden