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Airport emission particles: exposure characterization and toxicity

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Nanosafety at the National Research Centre for the Working Environment, Copenhagen, Denmark



- Government research institute under the Ministry of Employment
- Nanosafety as strategic research area since 2005

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- At present ca. 45 persons in chemical working environment research
- Advisors for the Danish Working Environment Authorities, EPA, EU, OECD, WHO
- Past and present partners in +30 EU projects on (nano)particle safety

Concern: Association between particulate air pollution and mortality

6 cities with 8000 people

151 urban areas with 500.000 people



Direct correlation between mortality and particle concentration ($PM_{2.5}$):

7 deaths/100 000 persons/year/ ug/m³ PM 2.5

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





Evaluation of cancer risk by IARC (WHO's research institute for carcinogens)

- Almost all current aviation fuel/jet fuels are extracted from the middle distillates of crude oil (kerosene fraction), which is in between the fractions used for gasoline and diesel
- Diesel engine exhaust is classified as carcinogenic to humans (class 1) by IARC
- Gasoline engine exhaust is classified as possibly carcinogenic to humans (class 2B) by IARC
- Carbon black (pure carbon particles) is classified as possibly carcinogenic to humans (class 2B)



Cumulative dose-response relationship between diesel exhaust exposure and lung cancer risk in three epidemiologial studies



EC (µg/m³-year)

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, 2014, EHP



Risk estimate and OELs for DEP based on epidemiological evidence

Table 1. Exposure-response estimates (InRR for a 1-µg/m³ 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)

^aLog-linear risk model (InRR = intercept + $\beta \times$ exposure). Exposure defined as EC in µg/m³-years.

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

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

The new EU OEL for DEP is 50 ug/m³

Based on linear risk function, InRR = 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.

Vermeulen et al, EHP, 2014

Based on this evidence, NL and DK have adapted an occupational expoure limit for diesel engine exhaust at 10 μ m³ REC or EC, respectively

NRCWE studies on airport emissions

- Particle collection and characterisation of particles from a large commercial airport and a non-commercial airfield (PMID: 31182125)
- Animal study where mice were exposed in the lungs to collected particles alongside standard NIST diesel exhaust particles and carbon nanoparticles (PMID: 31182125)
- Work place exposure assessment and biomonitoring at a non-commercial airfield with jet fighters (PMID: 34504215)
- Review of the litterature on airport emissions and health effects (PMID: 33549096)

Bendtsen et al. Particle and Fibre Toxicology	(2019) 16:23
https://doi.org/10.1186/s12989-019-0305-5	

Particle and Fibre Toxicology

RESEARCH

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Airport emission particles: exposure characterization and toxicity following intratracheal instillation in mice

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scientific reports



OPEN Occupational exposure and markers of genetic damage, systemic inflammation and lung function: a Danish cross-sectional study among air force personnel

Maria Helena Guerra Andersen¹, Anne Thoustrup Saber¹, Marie Frederiksen¹, Per Axel Clausen¹, Camilla Sandal Sejbaek¹, Caroline Hallas Hemmingsen², Niels E. Ebbehøj², Julia Catian¹, Kukla Aimonen¹, Joonas Koivisto^{1,6}, Steffen Loft⁶, Peter Møller⁶ & Ulla Vogel^{1,7}

Bendtsen et al. Environmental Health (2021) 20:10 https://doi.org/10.1186/s12940-020-00690-y

Environmental Health

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A review of health effects associated with exposure to jet engine emissions in and around airports



Katja M. Bendtsen^{1*}, Elizabeth Bengtsen¹, Anne T. Saber¹ and Ulla Vogel^{1,2}



Review: Expoure assessments by others

Table 1. Reported levels of occupational exp	osures of BC and particles in airports		
Black carbon			
Mean black carbon concentrations measured at different micro-environments of airports and in commercial flights	3.78 μg/m ³ (during boarding/disembarking) 3.16 μg/m ³ (airport concourse)	NA	Targino et al. (2017)
	2.78 μ g/m ³ (inside aircraft with open doors). 0.81 μ g/m ³ (inside aircraft on the ground with closed doors)		
Particles			
Total mean concentration of 10 daily UFP samples with personal monitors placed with crew chief and hangar operator	6.5×10^3 particles/cm ³ (downwind site)	 2.5 x 10⁴ particles/cm³ (crew chief) 1.7 x 10⁴ particles/cm³ (hangar operator) Median number concentrations for 2 months measurement period 	Buonanno et al. (2012)
Geometric means of personal exposure to particle number concentration carried out in five different occupational groups	NA	37×10 ³ UFP/cm ³ (baggage handlers) 5×10 ³ UFP/cm ³ (landside security) 12-20×10 ³ UFP/cm ³ (catering drivers, cleaning staff and airside security)	Møller et al. (2014)
Particle and metal exposure in ambient air and in airport workers using exhaled breath condensates	 1.0 x 10⁴ - 2.1 x 10⁷ particles/cm³ (apron workers) 10³-10⁴ (office staff) Airport workers were exposed to significantly smaller particles (mean geometric size: 17.7 nm) compared to office workers (mean geometric size: 23.7 nm). 	Particulate content was found in exhaled breath condensates, but no difference was found between the two study groups	Marie-Desvergne et al. (2016)
Maximal UFP number concentration of UFP exposures investigated for 33 male employees working in an airport taxiway	9.59 x 10^6 (during support tasks in taxiing and taking off of the aircraft)	2.44 x 10 ³ particles/cm ³ Median UFP number concentration	Marcias et al. (2019)

Bendtsen et al, PMID: 33549096

Review: Health effects related to airport emissions

- Biomonitoring studies:
- <u>Cavallo et al. (2006)</u>: PAH exposure correlated with biomarkers of DNA damage in 41 airport employees in jobs with high or medium exposure risks in Leonardo da Vinci airport in Rome
- <u>Erdem et al. (2012)</u>: Urinary metabolites 1- and 2-naphthol as biomarkers of jet fuel correlated with biomarkers of DNA damage among 43 aircraft fuel maintenance staff, fuel specialists, and mechanics
- Lammers (2020): 21 healthy non-smoking volunteers were repeatedly exposed for 5 h to ambient air near Schiphol Airport, while performing intermittent moderate exercise. Exposure was associated with decreased lung function (mainly FVC) and a prolonged QTc interval (a biomarker of cardiovascular function).
- Andersen et al. (2021): Cross-sectional study of 79 employees at a Danish air force military base. Exposure to particles, PAH, organophospate esters (OPE) and biomarkers of DNA damage and inflammation was assessed. Jobs with potentially high exposures were compared to jobs with potentially low exposure. Crew chiefs had increased exposure to 1 specific PAH and one OPE. No effect on biomarkers were observed.

- Epidemiological study:
- Møller et al. (2017 and 2019): A prospective, occupational cohort study in CPH, encompassing 69175 men in unskilled positions as baggage handlers or in other outdoor work. No associations between cumulative apron-years and cardiovascular disease outcomes were found. On the other hand, since the exposed group had a mean age of 24-35 years, a 22-year follow-up may have been too short to detect cardiovascular effects

Study: Jet engine emission particles

- Aircraft engines emit large amounts of nanosized carbon-based particles
- Primary particle size ca. 15 nm (smaller than DEP)
- Aggregate in air

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- PAH content similar to standard NIST DEPs
- Metal content similar to standard NIST DEPs
- Airport emissions from a commercial airport were much more complex and contained salt crystals, organic particles ect in addition to the small soot particles
- Overall conclusion: Jet engine emission particles are similar to diesel exhaust particles (but somewhat smaller)







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EM picture of jet fighter emissions during taxi (from a chasing car)

Animal study of health effects following lung exposure to airport emission particles

- Animal studies are used to establish causal relationships, which is difficult in epidemiological studies
- Mice were exposed by pulmonary expoure to JEP (jet emission particles) and CAP (Commercial airport particles) by pulmonary instillation at 3 dose levels and followed for 1, 28 and 90 days
- Pulmonary expousre (intratracheal instillation) can be used for hazard comparision and ranking
- Endpoints:
 - Lung histology (Biopersistence and histological changes)
 - Lung inflammation (biomarker of toxicity)
 - Acute phase response (biomarker for risk of cardiovascular disease)
 - DNA damage in lung, lung fluid cells, liver (biomarker for risk of cancer)



Both types of airport emission particles induced dose dependent inflammation and acute phase response, similar to carbon nanoparticles and standard diesel exhaust particles





Day 1 mRN Lung Saa3 6 18 54 6 18 54 CAP 18 54 162 NIST2975 le control = 1). Mean ± S Ε Fig. 6 mRNA levels of Sag3 in lung. Sag1 liver, and SAA3 plasma protein on day 1 (scatter plots, mean + SEM). Sag3 mRNA in lung tissue and Sag1 mRNA hiver tissue were used as biomarkers of pulmonary and hepatic acute phase response, following exposure to particles collected at the apron of a mercial airport and in a jet shelter at a non-commercial airfield. SAA3 protein was measured in plasma. Sog in lung and liver was m 8 and 90 post-exposure, and SAA3 on day 1 and on day 28 for highest particle doses

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Two different airport emission particles cause the same health effects in mice as standard NIST diesel exhaust particles and carbon nanoparticles

- The two airport emission particles have similar physico-chemical properties as diesel exhaust particles, although they are slightly smaller
- The two airport emission particles induce the same health effects in mice as two NIST standard diesel exhaust particles including:
 - inflammation (general toxicity marker),
 - acute phase response (biomarker of cardiovascular risk)
 - weak indications of DNA damage (biomarker of cancer risk)

Summary

- Airport emission levels reported in many studies are relatively high, but below current occupational exposure levels
- Aircraft emission particles have similar physico-chemcial properties as diesel exhaust particles and carbon nanoparticles
- Aircraft emission particles have similar health effects as diesel exhaust particles and carbon nanoparticles in mice including increased inflammation and biomarkers of risk of cardiovascular disease and cancer
- Biomonitoring studies find associations between exposure to airport emissions and biomarkers of DNA damage, lung function and cardiovascular function.
- Taken together, the evidence suggests that aircraft engine emission particles and airport emissions have similar health effects as diesel exhaust particles and other traffic-related emissions





Thank you for your attention

Metal and polyaromatic hydrocarbons (PAH) in collected particles

 \succ The two particle types had very similar content of PAH and metals

Ra

83

103

4/ND

> Levels were similar to that of standard NIST diesel exhaust particles

Extrac	ted elements fr	om analysis of	4 mg JEP and CAP		Table 5. Content of	ТОГАП
	JEP	CAP	NIST 2975	СВ	Ref. NIST2975ª	Ref. Cl
Li	3	17	1/ND	3/ND	-	-
Mg	950	8,655	291/281	ND /ND	-	-
AI	3,057	9,735	ND	203/0		• -
V	6	11	5/1	ND	0.0±0.0	<1
Cr	17	146	90/102	ND		<1
Mn	134	125	11/11	1 /ND	- <u> </u>	· -
Fe	2,788	5,386	814/743	498/-	0.0±13	11
Co	9	15	7/8	0/-	0.1±0.1 ne	<1
Ni	200	249	55/65	0/-	0.5±0.7	<2
Cu	1,147	14,884	24/5	13/3	0.9±0.6 her	<1
Zn	7,433	31,897	13,926/17,003	ND	10±4	-2
Ga	1	3	ND	ND	act	•
As	4	5	1/2	-/1		<2
Se	5	14	ND /2	ND		<10
Rb	7	8	ND	ND	·	
۰.	A A	107	Q/1	0/1	red	
Ag	62	35	ND	ND	-	-
Cd	6	3	ND	ND	-	<0.4
In	ND	1	ND	ND	-	-
Cs	1	1	ND	ND	-	-

3/3

Table 3. Content of 16 PAH in airport-collected particles

ef. NIST2975	f. NIST2975ª Ref.		g particles	JEP mg/g particles	NIST1650B ^{\$} (mg/g)	NIST2975 ^s (mg/g)	
-		-	ND	ND	0.007(0.0004)	0.004(0.0001)	
-		-	(0.0009)	0.01(0.002)	0.001(0.00004)		
		· _	ND	ND	0.0002(0.00002)	0.0005(0.00003)	
00+00		<1	0.00007)	0.001(0.0002)	0.001(0.00004)	0.003(0.0002)	
		1	(0.0005)	0.001(0.00008)	0.07(0.004)	0.02(0,0003)	
			ND	0.001	0.008(0.0004)	0.00005(0.000002)	
		- 0.00		0.001(0.00008)	0.05(0.001)	0.03(0.0005)	
0.0±13		11	,0.0007)	0.007(0.00007)	0.04(0.001)	0.002(0.0002)	
0.1±0.1	ne	<1	ND	ND	0.006(0.0004)	0.001(0.00004)	
0.5±0.7		<2	ND	ND	0.01(0.0006)	0.006(0.0001)	
0.9±0.6	her	<1	(0.0009)	0.02	0.009(0.0009)	0.01(0.003)	
1014	n	. ~	<i>.</i> (0.0004)	0.009(0.0004)	0.001(0.0001)	0.0008(0.00004)	
	ace	•	ND	ND	0.0004(0.00008)	0.0005(0.00005)^	
-	, yre	<2	ND	ND	0.004(0.0002)	0.002(0.0001)	
-	en	<10	ND	ND	0.006(0.0003)	0.002(0.00009)	
-		-	081	0.05	0.22	0.086	
- · · · · - 	red with own	 - יוחה מהביסחחה	ed as blank cor	rected mean values (N=2) wit	h standard deviation in pa	renthesis. The PAH	
-		-					

Exposure assessment at the non-commerial airfield

The instruments reached their upper detection limit

Also the particle counter in the breathing zone of the Crew Chief (Klarmelder)

Particle size <500 nm

The majority <10 nm







Exposure levels for the Crew Cheif at the non-commercial airfield

Table	• 2 Average exposures and doses of jetfighter personnel at a non-commercial airfield													
Event	<i>t</i> , [min] <i>n</i> , ×10 ⁶ [cm ⁻³]	<i>m</i> , [μg m ⁻³]	<i>m_{ΡΜ4}</i> , [μg m ⁻³]	$DR_N, \times 10^{10}$ [min ⁻¹]	HA, n[%]	TB, n[%]	AL, n[%]	DR _m , [µg min ⁻¹]	HA, m[%]	TB, m[%]	AL, m[%]	Particles [× 10 ¹²]/ Event	Mass [µg]/ Event
PL	15.1	7.7	1086	537	15	21.2	27.2	51.6	18.7	84.6	4.7	10.7	2.26	280
PA + FT	21.3	2.67	410	228	5.4	21.7	27.7	50.7	7	83.6	4.9	11.5	1.15	150
t _{PM4}	170	1.22	194	89	2.4	21.4	27.4	51.3	3.5	85.8	4.6	9.6	4.12	600

Average exposures and doses during Plane Leaving (PL), Plane Arrival and fueling the plane (PA + FT combined), and over one flight cycle (t_{PM4}). From left to right: average event time (*t*) in minutes, average particle number concentration (*n*), mass concentration (*m*) and mass fraction smaller than 4 µm (m_{PM4}), inhaled number dose per minute (DR_N), predicted fraction of particles deposited in extra-thoracic (*HA*), tracheo-bronchial (*TB*) and alveolar (*AL*) lung regions, inhaled mass dose per minute (DRm), predicted fraction of mass deposited in extra-thoracic (*HA*), tracheo-bronchial (*TB*) and alveolar (*AL*) lung regions, total particles per event and total mass per event

Exposure levels in a jet engine test facility: $2-4 \times 10^6$ particles/cm³ (data in supplementary)

Inhaled TiO₂ nanoparticles in the lung are removed very slowly

Mice inhaled 40 mg/m³ nanosized TiO₂ 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)	Procent of
			(mean ± sd)	deposited dose
TiO ₂	5	3	63 ± 10	24%
Air	5	3	< 8	
TiO ₂	25	3	55 ± 30	21%
Air	25	3	< 1	



Hougaard et al, PF&T, 2010

Inhalation of nano-TiO₂ results in long lasting inflammation

160000 Total number of cells in BAL fluid 140000 □ Control ⊠ TiO2 120000 ** *** 100000 80000 60000 40000 20000 0 Macrophages Neutrophils Lymphocytes

Types and numbers of cells in lung fluid

After 4 weeks



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After 5 days

Hougaard et al, 2010