

Nordic Air Pollution Workshop 2006

March 22 and 23, 2006

Nordic Organising Committee

Steffen Loft (chairman)
Finn Palmgren
Per Schwartze
Raimo Salonen
Bertil Forsberg

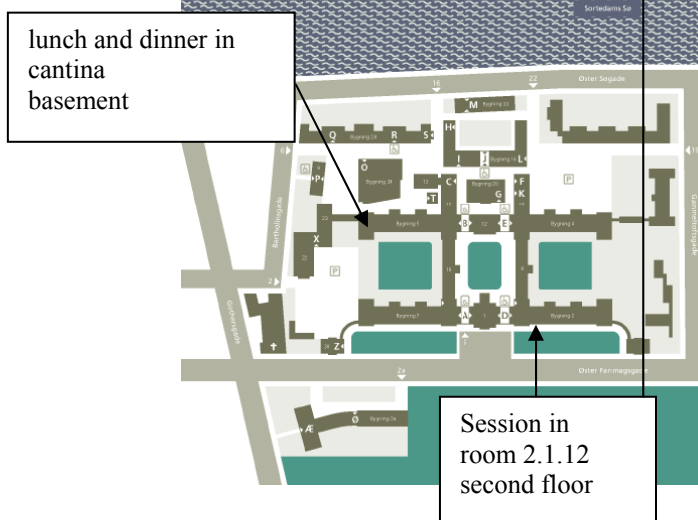
Local Organising Committee

Steffen Loft
Ole Raaschou-Nielsen
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Venue

Centre of Health and Society
University of Copenhagen
Øster Farimagsgade 5
1014 Copenhagen K
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Program for Nordic Air Pollution Workshop 2006.

	Wednesday March 22 room 2.1.12
9.00-9.30	Registration
9.30-9.35	Welcome address
	9.35-12.05 Exposure to air pollution – chairman Finn Palmgren
9.35-10.05	High resolution modelling of exposure to air pollution based on detailed traffic data <i>Martin Hvidberg (NERI, Denmark)</i>
10.05-10.35	Impact of long range transported air pollution on exposure in Norwegian towns (Steinar Larssen, Norwegian Institute of Air Research, Norway)
10.35-11.00	<i>Coffee</i>
11.00-11.30	Contributions from road traffic emissions to urban PM concentrations and exposure <i>Christer Johansson (ITM, Stockholm University, Sweden)</i>
11.30-12.00	Traffic microenvironment in probabilistic modelling of population exposure to fine particles <i>Otto Hänninen (National Public Health Institute, Kuopio, Finland)</i>
12.00-13.00	<i>Lunch</i>
	13.00-15.30 Experimental study of health effects of air pollution – chairman Per Schwarze and Steffen Loft
13.00-13.30	Chemical, toxicological and source characteristics of coarse, fine and ultrafine particles in contrasting air pollution situations in Europe <i>Raimo Salonen (KTL, Finland)</i>
13.30-14.00	Experimental models for health effects of air pollution <i>Håkan Wallin (NIOH, Denmark)</i>
14.00-14.30	Particle properties and effects of particles: what did we learn from comparisons of particles from different European cities <i>Per Schwarze (Norway)</i>
14.30-15.00	Experimental human exposure to diesel and other emissions <i>Thomas Sandström (Sweden)</i>
15.00-15.30	<i>Coffee</i>
	15.30-18.30 Epidemiological study of health effects of air pollution – chairman Ole Raaschou-Nielsen and Bertil Forsberg
15.30-16.00	Source-specific human exposures to and health effects of fine particulate matter in Helsinki <i>Timo Lanki (Finland)</i>
16.00-16.30	Effects of road dust and exhaust particles on daily mortality, hospital admissions and asthma emergency visits in Stockholm <i>Bertil Forsberg (Sweden)</i>
16.30-17.00	Discussion of time-series approaches – appetizer and <i>Bertil Forsberg, Zorana Andersen, Timo Lanki, Steffen Loft</i>
17.00-19.00	<i>Tour and story of the old hospital and visit at the Department of Environmental and Occupational Health with wine bar</i>
19.00-	<i>Dinner in the Cantina</i>



	Thursday March 23 - room 2.1.12
9.00-9.30	Source apportionment of PM10 and hospital admissions and airway symptoms in Copenhagen <i>Zorana Andersen (UC, Denmark)</i>
9.30-10.00	Short- and long-term health effects of air pollution for Norway <i>Per Nafstad (Norway)</i>
10.00-10.30	Air pollution is associated with onset of airway disease in pre-school children <i>Emma Nordling (Stockholm County Council, Sweden)</i>
10.30-11.00	<i>coffee</i>
	11.00-14.00 Specific emissions and effects – Raimo Salonen and Poul Bo Larsen
11.00-11.30	Emission and characterisation of particles from traffic <i>Peter Wåhlin (NERI, Denmark)</i>
11.30-12.00	Air pollution particles as adjuvant in allergic responses <i>Unni Cecilie Nygaard (Norway)</i>
12.00-13.00	Lunch
13.00-13.30	Emission and characterisation of particles from wood stoves <i>Marianne Glasius (NERI, Denmark)</i>
13.30-14.00	Experimental exposure to wood smoke particles in healthy humans: effects on markers of inflammation, coagulation and lipid peroxidation <i>Lars Barregård (Sweden)</i>
14.00-14.30	Intrauterine exposure and genotoxic effects of air pollution in children <i>Lisbeth Knudsen (UC, Denmark)</i>
14.30-15.00	<i>coffee</i>
15.00-16.30	Discussion of time-series approaches and source apportionment in relation to health effects – joint Nordic studies ? <i>Zorana Andersen, Bertil Forsberg, Timo Lanki, Steffen Loft</i>
16.30	End of workshop

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High resolution modeling of exposure to air pollution based on detailed traffic data

Martin Hvidberg, Researcher, mhv@dmu.dk, National Environmental Research Institute (NERI)
Department of Atmospheric Environment

During the last decade techniques and computer programs to support air pollution modeling for exposure assessment on an individual level have become increasingly available and usable.

NERI has developed a number of programs and data sets to support air pollution modeling, including estimates of personal exposure to traffic air pollution. The overall name of these tools is AirGIS. AirGIS today supports modeling of outdoor air pollution with high spatial (meters) and temporal (hours) resolution.

Modeling air pollution basically requires data about: emissions, meteorology, background concentrations and local topography.

What happens when air pollution is modeled for a give point in space and time?

Epidemiological studies often requires air pollution estimates for a number of addresses, each for a given time interval.

When such an address is sent into AirGIS a number of processes handle various aspects of describing the local emissions and the local landscape. This information is passed to other processes that handle dispersion and chemical processes to produce the final air quality estimate.

This presentation will go into the details of modeling local air pollution emissions and modeling local area landscape for the purpose of modeling local area air pollution.

The presentation will go through the individual steps from the starting point of an input address to the final air pollution estimate. Dilemmas and pitfalls on the way will be described including a description of the available traffic and building data.

Impact of long range transported air pollution on exposure in Norwegian cities

Steinar Larssen, Leiv Håvard Slørdal, Herdis Laupsa, Harold McInnes and Wenche Aas, Norwegian Institute for Air Research, NILU

Data from air pollution measurements all over Europe (source: AirBase

<http://air-climate.eionet.eu.int/databases/airbase/>) show that the general situation in Europe is that the regional contribution to PM dominates the PM10 and PM2.5 concentrations in urban areas, while this regional contribution to NO2 is much less dominant. The regional scale concentrations are of course highest in the parts of central and eastern Europe, and diminishes towards the Northern parts of Europe. In central parts of Europe the regional scale concentrations are partly due to the country's own contribution and the contribution from 'long range transported' air pollution (LRTAP), while in Northern parts of Europe, the regional scale concentrations are to a large extent caused by LRTAP.

The regional/LRT contribution to cities in Norway has been studied, mainly for Oslo, using measurements and modelling, a combination of the EMEP Unified Model and NILU's urban scale model EPISODE, run within the AirQUIS AQMS software system. The main local sources to PM in air in Norwegian cities, for PM2.5: small scale wood burning and vehicle exhaust; to PM10: here the resuspension of road dust, caused by studded tyres, comes in as a large additional source. For both PM fractions, the LRT contribution comes in addition.

The results of these modelling efforts show that for NO2, the regional contribution to the population exposure in Oslo is small, about 10% to annual average exposure and negligible to the exposure to high-level short-term concentrations above limit values. For PM the LRT contribution is much more important: 40% of the PM10 exposure on an annual basis, and 50% to annual PM2.5 exposure. Looking at the high end of the exposure, only about 12% of the exposure situations above the PM10 short-term limit value (50ug/m3 as daily average) can be attributed to the LRTAP. These figures relate to the 2003 situation. Population exposure is calculated based upon a static population (at residents), and a population weighted average exposure concentration.

The regional contribution to PM concentrations in urban areas in more central regions of Europe would in general be higher both in absolute and relative terms.

Contributions from road traffic emissions to urban PM concentrations and exposure

Christer Johansson, Department of Applied Environmental Sciences (ITM), Section for Atmospheric Science
Stockholm University

Substantial differences in the temporal and spatial distribution of PM10 and particle number concentrations (referred to as PNC) are observed in the urban area of Stockholm. This is due to different impacts of emissions, background concentrations and meteorology. High concentrations of PNC are found at kerb side locations due to vehicle exhaust



emissions verified by high correlation with NO_x concentrations. In Stockholm vehicle exhaust emissions contribute only marginally to the total mass concentration measured as PM₁₀. Instead wear of the road surface is the most important factor for the highest PM₁₀ concentrations observed. The wear is increased drastically due to the use of studded tires and traction sand on streets during winter. Almost 90% of the locally emitted PM₁₀ are due to road abrasion. Particle emissions from the roads are controlled by the wetness of the road surface. The highest concentrations of coarse particles are recorded during dry conditions in spring. Daily mean levels of PM₁₀ frequently exceed the EU-limit value of 50 µg m⁻³ on such occasions. The annual mean urban background PM₁₀ levels are relatively uniformly distributed over the city, due to the importance of long range transport. Non-local sources contribute 60%-70% to the annual mean PM₁₀ level. For PNC, in contrast, local sources dominate the concentrations resulting in large temporal and spatial gradients in the concentrations. Non-local sources of PNC contribute with only 10%-30% to annual levels. Despite these differences in the origin of PM₁₀ and PNC, the spatial distributions of annual mean exposure concentrations are similar due to the common source, namely traffic. This means that people in different areas in Stockholm experiencing a factor of 2 different PM₁₀ exposure due to local sources (as an annual mean value) will also experience a factor of 2 different exposure in terms of PNC (mainly ultrafine particles). At the same time essentially no contrasts in exposure of secondary (long-range transported, LRT) accumulation mode particles will be seen. These results are important for interpretation of epidemiological studies. Health impact studies based solely on spatial differences in annual exposure to PM₁₀ may not separate potential differences in health effects due to ultrafine and coarse particles. On the other hand, epidemiological studies of health effects based on time series analysis of PM₁₀ and PNC, should be able to observe differences in health effects of ultrafine particles versus coarse particles. Likewise, health effects of LRT-particles should be possible to separate from effects due to locally primary emitted particles in time series analysis. LRT-particles (measured at rural sites outside the cities) would not give rise to any spatial contrast in exposure of the population in a metropolitan area like Stockholm.

Traffic microenvironment in probabilistic modelling of population exposure to fine particles

Otto Hänninen^{1,2,*}, Raimo O. Salonen¹, and Matti Jantunen¹

¹ KTL, Environmental Health, Centre of Excellence for Environmental Health Risk Assessment, POB. 95, FI-70701 Kuopio, Finland; ² WHO Regional Office for Europe, European Centre for Environment and Health, Görrestrasse 15, D-53113 Bonn, Germany.

* Email: oth@ecehbonn.euro.who.int

Modelling of exposures to air pollution provides a necessary tool for comparison policy options that attempt to reduce the adverse health effects associated with traffic emissions. Selection of modelling techniques must balance between detailness of the outputs and requirements set for the input data needed to run the models. Probabilistic modelling techniques allow for efficient representation of the variability of concentrations, durations of time spent in various microenvironments, and exposures without requiring personal time-activity data or location-specific street canyon modelling.

Particulate matter exposures to local traffic generated particles occur throughout the day everywhere including indoor microenvironments, but the rather limited time, typically 5%, spent in traffic has a very significant contribution to the total exposure of urban populations.

Models need to be validated against experimental data. Measurement of traffic-specific PM exposure data is complicated by difficulties in identifying carbon rich tailpipe particles with the typical elemental analysis techniques. Chemical mass-reconstruction and exposure/concentration closure have been applied data from simultaneous measurements of personal exposures and concentrations in the main microenvironments (work, residence) collected in the *EXPOLIS* study in Helsinki and six other European cities (Jantunen *et al.*, 2005). This demonstrates a preliminary approach to solve the source identification problem for traffic particles.

The results from the source apportionment study have been applied to further studies to model traffic-related PM_{2.5} exposures in the Helsinki metropolitan area with the business as usual scenario from 2002 to 2025, indicating a clear drop in tailpipe particulate exposure and total PM_{2.5} exposure, but an increase in resuspension particulate exposure, during selected episodic days. Exposure to local traffic particles was high on a temperature inversion day, but the highest PM_{2.5} total exposure was experienced during an episode of long-range transported particulate pollution.

References

Jantunen M, Hänninen O, Ilacqua V, Karppinen A, Kukkonen J, and Aarnio P. Exposure levels to exhaust-generated and total PM_{2.5} in traffic. Proceedings of the 5th International Conference on Urban Air Quality, Valencia, Spain, March 29-31., 2005.



CHEMICAL, TOXICOLOGICAL AND SOURCE CHARACTERISTICS OF COARSE, FINE AND ULTRAFINE PARTICLES IN CONTRASTING AIR POLLUTION SITUATIONS IN EUROPE (PAMCHAR)

Acknowledgements: Field campaign work by the local teams and funding by the EU-FP5 (QLK4-CT-2001-00423), Academy of Finland (Contract 53307) and Tekes (Contract 40715/01). Experimental models for health effects of air pollution

Raimo O. Salonen¹, Maija-Riitta Hirvonen¹, Pasi Jalava¹, Mikko Happonen¹, Arja Hälinen¹, Arto Pennanen¹, Markus Sillanpää², Sanna Saarikoski², Karri Saarnio², Anna Frey² and Risto Hillamo²

¹National Public Health Institute (KTL), Department of Environmental Health, Kuopio, Finland ²Finnish Meteorological Institute (FMI), Air Quality Research, Helsinki, Finland

Urban air fine particles (PM_{2.5}; diameter < 2.5 µm) have been associated with extensive premature mortality and morbidity among subjects with chronic cardio-respiratory disease, but the responsible sources and constituents as well as toxicity mechanisms for the particulate effects are not well known. We conducted 7-week sampling campaigns in six European cities during selected seasons of public health interest: Duisburg (autumn), Prague (winter), Amsterdam (winter), Helsinki (spring), Barcelona (spring) and Athens (summer). Urban air coarse (PM_{10-2.5}), fine (PM_{2.5-0.2}) and ultrafine (PM_{0.2}) particulate samples were collected with a modified Harvard high volume cascade impactor for toxicological studies, while PM_{10-2.5} and PM_{2.5} samples were collected in parallel with three virtual impactors for in-depth chemical and source characterisation. There were major differences in inorganic and organic compositions and sources of the complex urban air particulate mixture between the sampling campaigns. These differences were consistently reflected in the inflammatory activity of the particulate samples in the mouse RAW264.7 macrophages and the mouse lung. The inflammatory activity of the PM_{10-2.5} samples was higher than that of the PM_{2.5-0.2} samples, but the latter exhibited larger differences between the sampling campaigns. The PM_{0.2} samples had negligible inflammatory activity, but some of them were highly cytotoxic. In addition to emission sources, atmospheric photochemical activity is suggested to be a major factor determining the toxicity of urban air particulate matter.

Experimental models for health effects of air pollution

Håkan Wallin, National Institute of Occupational Health, Denmark; hwa@ami.dk

Particle properties and effects of particles: what did we learn from comparisons of particles from different European cities

Per E. Schwarze, Norwegian institute of Public Health

Multicentre epidemiological studies show some heterogeneity with respect to risk estimates of particle effects. This could be due to different particle properties. Three EU-funded projects have sampled different size fractions of PM in different European cities and investigated their toxicological potential. In the RAIAP project the fractions were investigated with regard to inflammatory and adjuvant effects in vivo and in vitro after thorough chemical and biological characterisation. The coarse fraction had more inflammatory potential than the fine fraction, measured as increased cytokine release from lung cell lines or primary lung cells. A similar pattern was found in BAL after tracheal instillation in rats. There were also seasonal differences between particle samples, with the spring and summer samples showing more pro-inflammatory potential than the winter samples. The differences between fine and coarse samples followed the pattern of low and high endotoxin levels, but endotoxin levels could not explain the variations between different coarse samples. Neither the metal nor the PAH composition alone could explain these differences, indicating that several factors were involved in eliciting the effects. The mouse foot pad method indicated that fine particles were more potent than coarse particles in eliciting an adjuvant effect measured as specific IgE. However, no systematic differences were found between fine and coarse particles on other ovalbumin-induced effects (number of lymph node cells, expression of cell surface receptors and *ex vivo* cytokine production). Moreover, no significant difference between these fine and coarse particles could be detected in a respiratory allergy mouse model, with the exception that coarse particles were more potent to elicit histopathological lesions.



Experimental human exposure to diesel and other emissions

Thomas Sandström, Dept of Respiratory Medicine and Allergy, University Hospital, Umeå, Sweden
e-mail: thomas.sandstrom@lung.umu.se

The associations between air pollutants and respiratory and cardiovascular morbidity and mortality have been well described by epidemiological studies over the last decades. In contrast, the biomedical counterparts to symptoms and disease aggravation have been poorly understood. This has mainly been investigated by a range of toxicological methods in-vitro and in animal experimental models aimed to determine the mechanistic aspects involved.

During the last years special attention has been directed towards human experimental studies in order further address the hypothesis that particle air pollution induces oxidative stress with acute inflammatory and reactive effects in the cardiovascular and respiratory systems leading to adverse health effects present in healthy subjects as well as compromised people with diseases like asthma, COPD and cardiovascular disease.

Well validated experimental exposure models with diluted diesel engine exhaust as well as ozone and nitrogen dioxide have been used for controlled experiments in humans. Measurement of lung function, bronchial hyperresponsiveness, nasal lavages, induced sputum, bronchoscopy with bronchoalveolar lavages and biopsy sampling have been demonstrated to be valuable techniques for determining biomedical responses linked to adverse health effects in different groups of subjects.

Heart rate variability analyses and bilateral underarm plethysmography techniques with arterial and venous canulations have recently been proven to be valuable complementary techniques for studies of cardiovascular effects linked to particulate matter exposure in-vivo in healthy and disease compromised subjects.

Recent publications

Behndig AF, Mudway IS, Brown JL, Stenfors N, Helleday R, Duggan ST, Wilson SJ, Boman C, Cassee FR, Frew AJ, Kelly FJ, Sandström T, Blomberg A. Airway antioxidant and inflammatory responses to diesel exhaust exposure in healthy humans. *Eur Respir J.* 2006;27:359-65.

Mills NL, Törnqvist H, Robinson SD, Gonzalez M, Darnley K, MacNee W, Boon NA, Donaldson K, Blomberg A, Sandström T, Newby DE. Diesel exhaust inhalation causes vascular dysfunction and impaired endogenous fibrinolysis. *Circulation.* 2005 Dec 20;112(25):3930-6.

Pourazar J, Mudway IS, Samet JM, Helleday R, Blomberg A, Wilson SJ, Frew AJ, Kelly FJ, Sandström T. Diesel exhaust activates redox-sensitive transcription factors and kinases in human airways. *Am J Physiol Lung Cell Mol Physiol.* 2005 Nov;289(5):L724-30.



Source-specific human exposures to and health effects of fine particulate matter in Helsinki

Timo Lanki

Unit of Environmental Epidemiology, National Public Health Institute, Kuopio, Finland. timo.lanki@ktl.fi

Within the European ULTRA study, we have apportioned mass of fine particles ($PM_{2.5}$) between different sources in Helsinki, and linked the obtained source-specific $PM_{2.5}$ concentrations to indicators of cardiorespiratory health. The ULTRA study consisted of two parts: ULTRA 1 was carried out 96-97 (Nov-Apr) and ULTRA 2 98-99 (Nov-May).

The study panel in ULTRA 1 consisted of 57 adult asthmatics whose respiratory health was monitored. In ULTRA 2, 47 elderly persons with coronary heart disease visited biweekly a clinic for evaluation of cardiorespiratory health. Particulate and gaseous ambient air pollution was monitored at a central site. In addition, personal and indoor $PM_{2.5}$ were measured during the 24-h preceding the clinic visits in ULTRA 2. Outdoor $PM_{2.5}$ was apportioned between sources using principal component analyses and multivariate regression. Indoor $PM_{2.5}$ was apportioned using a 2-way model in the Multilinear Engine.

Five main source categories were identified: crustal source, long-range transported particles (LRTP), local combustion including traffic, oil combustion, and salt source. In ULTRA 1, decreased peak expiratory flow was associated especially with local combustion, but also with LRTP. In ULTRA II, urinary concentration of lung Clara cell protein (indicator of damaged lung epithelial barrier) was associated with LRTP. Further, increased occurrence of ST segment depressions (indicator for ischemia) was associated with local combustion, but also with LRTP. Decreased heart rate variability (measure of autonomic nervous control of heart) was associated with LRTP. Central outdoor concentrations estimated well daily variation in exposures to LRTP and combustion particles. Three identified indoor sources (resuspension, cooking, Cu source) accounted for 27% of indoor $PM_{2.5}$.

Effects of road dust and exhaust particles on daily mortality, hospital admissions and asthma emergency visits in Stockholm

Bertil Forsberg, Kadri Meister, Bo Segerstedt

Occupational and Environmental Medicine, Dept of Public Health and Clinical Medicine, Umeå University, SE 901 87 Umeå, Sweden

Traffic produces several types of particles, road dust and ultrafine exhaust particles being two extremes. It is not very likely that exhaust particles and road dust have the same effects on health, but still today they are lumped together and considered equally harmful in health impact assessments as in the recent report from CAFÉ. This lack of specific exposure-response data seriously limits the scientific support provided for pollution management and control strategies.

We studied the associations between daily ($n=824$) urban background levels of PNC and NO_x , respectively, in Stockholm and daily respiratory and cardiovascular hospital admissions as well as daily number of deaths. The mean PNC was 10350 particles/cm³ and the mean NO_x level was 23 $\mu\text{g}/\text{m}^3$.

We also studied PM_{10} effects in Stockholm based on 217 days with high concentrations of road dust (mean roof level PM_{10} was 23 $\mu\text{g}/\text{m}^3$), identified using the 85th percentile for local non-exhaust PM_{10} time-series adjusted for rural background PM_{10} and urban levels of NO_x , representing local exhaust emissions.

Poisson regression by using penalized spline models in `mgcv` package of R is used for the statistical analysis. The mean concentration of the same day and the day before (lag 01) is considered as exposure. Smooth functions of time and different weather variables are used in the models.

For an increase corresponding to the IQR, we found PNC and NO_x to result in about the increase in hospital admissions. However, only the association between NO_x and respiratory admissions was statistically significant ($p<0.05$). Only PNC had a tendency to correlate with daily mortality. When compared to PM_{10} from other sources, PM_{10} from road dust seems to have a stronger effect on emergency visits for asthma and as strong effect on respiratory admissions, but no effect on CVD admissions or on daily mortality.



Source Apportionment and Size Distribution of PM in relation to Hospital Admissions in Children and Elderly in Copenhagen

Zorana J. Andersen, Peter Wåhlin, Ole Raaschou-Nielsen, Thomas Scheike, Steffen Loft
University of Copenhagen, National Environmental Research Institute, Danish Cancer Society

Background: An association between particulate air pollution and morbidity and mortality is well established. However, little is known about which sources and size fractions of particulate matter contribute most to the adverse health effects, which would provide important insights into biological mechanisms as well as enable more efficient air pollution control.

Methods: For a 6 year period (01.01.1999-31.12.2004) we examined associations between urban background PM₁₀ in the presence of gaseous pollutants (CO, NO₂) and hospital admissions due to cardiovascular and respiratory disease in the elderly (age ≥ 65), and asthma in children (age 5-18) in Copenhagen, Denmark. We studied further hospital admissions associations between fractions of PM₁₀ assigned to six sources (biomass, secondary, oil, crustal, sea salt, and vehicle) from a 1½ year campaign (03.05.2002-31.12.2003). Finally, we used the data on the size distribution of the number concentration of PM (15.05.2001-31.12.2004) to assess the association with hospital admissions, for total number concentration as well as for the four discernible size modes (12nm, 23nm, 57nm and 212nm). We used Poisson generalized additive model adjusted for overdispersion, season, day of the week, public holidays, influenza epidemics, grass pollen, and meteorology, with up to 5 days lagged exposure.

Results and Conclusions: We found positive associations between PM₁₀ and the three health outcomes, robust in the presence of CO and NO₂. Different PM₁₀ sources were important for the three health outcomes: crustal and secondary sources showed strongest associations with cardiovascular, biomass with respiratory, and vehicle and biomass with asthma admissions. At present, no single PM₁₀ source can be attributed to all morbidity. We found positive associations with ultrafine particles for all three outcomes, which appeared weaker than the associations with PM₁₀. The number concentrations of the larger size modes (212 nm and 57 nm) showed strongest associations with CVD and RD in elderly, while the smaller size mode (23 nm) showed strongest association with asthma in children. All results point at higher susceptibility of children.

Epidemiological studies of short and long term effects of air pollution in Norway.

Per Nafstad, Department of General Practice and Community Medicine, Medical Faculty, University of Oslo and Division of Epidemiology, Norwegian Institute of Public Health, Oslo

A major challenge for air pollution researchers is to contribute to the understanding of how air pollution affects human health. Available epidemiological methods are better tools for studying short term than long term health effects. Accordingly, health effect assessments rely heavily on time series studies while possibilities to address long term effects of air pollution are limited still. A large number of time series studies have been published. Weather and topographic conditions in many Norwegian cities create conditions favouring within city variation in air pollution exposure, which may open for other approaches for studying health effects of air pollution. During the last years several cohorts and cross sectional studies have been initiated in which the assessment of participants' air pollution exposure is based on modelling of air pollution at different addresses within a city. These initiations include studies of mortality, development of chronic diseases as well as potential intermediate markers of disease development like lung function, blood pressure and markers of inflammation. This presentation aims to cover recent and ongoing Norwegian epidemiological studies addressing health effects of ambient air pollution and methodological considerations concerning long term vs short term health effects of air pollution exposure.



Air pollution is associated with onset of airway disease in pre-school children

Emma Nordling¹, Niklas Berglind^{1,2}, Erik Melén^{1,3}, Gunnel Emenius¹, Jenny Hallberg⁴, Fredrik Nyberg^{2,5}, Göran Pershagen², Magnus Svartengren^{1,4}, Magnus Wickman^{1,2}, and Tom Bellander^{1,2}

1. Department of Occupational and Environmental Health, Stockholm, Sweden
2. Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden
3. Centre for Allergy research, Karolinska Institutet, Stockholm,
4. Department of Public Health Sciences, Karolinska Institutet, Stockholm.
5. AstraZeneca R&D Mölndal, Mölndal, Sweden Sweden

Introduction: Urban air pollution can trigger asthma symptoms but there is conflicting evidence on effects of long-term exposure on lung function, as well as on the onset of airway disease and allergy in preschool children. The aim of this study was to assess the impact of exposure to air pollution from road traffic and from house heating early in life, on lung function, wheezing and sensitization in children up to the age of four.

Methods: The spatial distribution of air pollution from traffic was assessed using source-specific nitrogen oxides (traffic-NO_x) and inhalable particulate matter (traffic-PM₁₀) as indicators, based on emission databases and dispersion modelling in a 25 to 500 meter gridnet. Air pollution from residential heating was assessed as sulphur dioxide (heating-SO₂). Estimated levels were used to assign monthly residential outdoor levels for the first year of life of 4,081 children in a prospective birth cohort, by linking to geocoded home addresses. The average source-specific contribution from road traffic to residential NO_x and PM₁₀ levels were 23.2 and 3.9 µg/m³, respectively, during the children's first year of life. Parents provided questionnaire data on symptoms and exposures when the children were approximately 2 months, 1, 2 and 4 years old. At 4 years, 73% of the children participated in clinical examination including specific IgE in blood and peak expiratory flow (PEF).

Results: Higher residential levels of air pollution from traffic during the first year of life were associated with lower lung function at four years of age: 5.4 l/min lower PEF (95% CI: 0.05, 10.7) for a 6 µg/m³ (5th-95th percentile) difference in residential traffic-PM₁₀. These levels tended to be associated with persistent wheezing, odds ratio (OR) 1.64 (95% CI: 0.90-3.00), and with sensitisation to inhalant allergens, especially to pollen, OR 2.30 (95% CI: 1.23-4.29). Results were similar using traffic-NO_x as indicator. No associations to heating-SO₂ (average source-specific contribution 2.5 µg/m³) were seen.

Conclusions: In a Swedish birth cohort we found associations between residential outdoor levels of air pollution from traffic during the first year of life, and three different indicators of airway disease in 4-year old children: lower peak expiratory flow, persistent wheezing and sensitisation to pollen. This indicates that modest exposure to air pollution from traffic early in life may have a negative effect on the respiratory health of pre-school children.

Emission and characterisation of particles from traffic

Peter Wåhlin, National Environmental Research Institute, Denmark

Traffic is an important source of airborne particles in the whole size range from ultrafine particles (< 0.1 µm) to coarse particles (> 2.5 µm). The particles are emitted due to many different processes of which the most important are turbulent raising of soil dust and road dust, wear of asphalt concrete, wear of rubber tyres, wear of brake disks and brake linings, soot formation in diesel engines, condensation of volatiles in the exhaust, and secondary particle formation by chemical conversion of exhaust gases. In the presentation will be given a schematic overview of the typical particles sizes and the chemical composition of particles from the different processes, their relative contributions, and the contributions compared with the contributions from non-traffic sources.

Traffic particles, especially the particles in the ultrafine size fraction, are often suspected of contributing more seriously to adverse health effects than other particles, but this is difficult to confirm on a non-speculative basis. In time series analysis of hospital admissions in urban areas, it is a problem that the day-to-day variation of the concentration of traffic particles is small compared with the variation of large contributions from other particle sources. Even in short term exposure studies of people in busy streets, it is not possible to combine measured health effects with specific particle types, because the different types of traffic particles in the fine/ultrafine size range are emitted in nearly constant mutual proportions.



Air pollution particles as adjuvants in allergic responses

Unni C. Nygaard, Norwegian Institute of Public Health, Oslo

Particulate air pollution may have contributed to the increased prevalence of asthma and allergic diseases in industrialized countries during the last decades. Numerous epidemiological studies indicate that exposure to elevated levels of PM₁₀, PM_{2.5} and PM_{0.1} worsen asthmatic symptoms. The long-term effects of PM exposure on development of asthma and allergy are less clear, but some studies have reported an association between allergic sensitisation and road traffic pollution. A wide range of particles, like diesel exhaust particles, ambient city air particles and various inert, insoluble particles have been shown to increase the production of allergic (IgE) antibodies in various animal models. Also in humans, intranasal co-exposure of diesel exhaust particles with allergen has been shown to increase the allergic response towards the (neo-)allergen. Overall, these studies suggest that various air pollution particles may have an adjuvant effect on allergic responses.

The characteristics of the particles affect their adjuvant potency. Studies indicate that the insoluble particle core play an important role in the adjuvant effect, while adsorbed chemicals and allergens may further increase or modulate the immune response. At the same weight dose, ultrafine particles seem to be more potent adjuvants than larger particles. The currently used PM weight is a poor measure of the amount of the smaller and possibly more potent particles.

Measures of particle surface area or particle number, rather than particle mass, should therefore be included in further studies of particles and allergic diseases.

EMISSION AND CHARACTERISATION OF PARTICLES FROM WOOD STOVES

M. GLASIUS, P. WÄHLIN, M. KETZEL, J. MØNSTER, R. BOSSI, R. BERKOWICZ
and F. PALMGREN

¹ National Environmental Research Institute (NERI), Department of Atmospheric Research, Frederiksborgvej 399, DK-4000 Roskilde, Denmark

Residential wood combustion is a source to ambient particles and e.g. polycyclic aromatic compounds (PAH) and volatile organic compounds. Since this type of emission occurs in residential areas it is important to study and assess the population exposure and possible health effects. There are about 300,000 wood combustion appliances used regularly in Denmark and the number is increasing.

We have investigated the impact on local air quality in two residential areas in Denmark. The study areas differ since the first is an area with row houses heated by electrical heating and wood combustion, while the second area is an area with detached houses heated by combustion of natural gas and wood.

Measurement results from the first area showed increased average levels of PM_{2.5} of about 4 µg m⁻³ during a six-week winter period (Glasius et al., 2006), while the impact in the second area was lower, as expected. The diurnal variation in the residential areas showed elevated particle levels (PM_{2.5}) in the evening and night as expected from local heating sources, while PM_{2.5} at the nearby rural background site shows a diurnal pattern with minor, broad peaks. Elevated levels of PAH were also observed.

The results will be compared with emission measurements of particles and PAH.

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Experimental exposure to wood smoke particles in healthy humans: effects on markers of inflammation, coagulation and lipid peroxidation

Lars Barregard¹, Gerd Sällsten¹, Pernilla Gustafson¹, Linda Johansson², Samar Basu³, Lena Andersson¹, Lennart Stigendal⁴

¹Department of Occupational and Environmental Medicine, Sahlgrenska University Hospital and Academy, Goteborg University, Sweden ²Department of Energy Technology, Swedish National Testing and Research Institute (SP)

³Department of Public Health and Caring Sciences, Geriatrics and Clinical Nutrition, Uppsala University, Sweden.

⁴Department of Haematology and Coagulation, Sahlgrenska University Hospital, Goteborg, Sweden.

Background: Particulate air pollution is known to increase cardiovascular morbidity and mortality. Proposed mechanisms underlying this increase include effects on inflammation, coagulation factors, and oxidative stress, which could increase the risk of coronary events and atherosclerosis. We recently performed a study to examine whether short-term exposure to wood smoke affects markers of inflammation, blood hemostasis and lipid peroxidation in healthy humans.

Methods and Results: Thirteen subjects were exposed to wood smoke and clean air in a chamber during two 4-hour sessions, 1 week apart. The mass concentrations of fine particles at wood smoke exposure were 240–280 $\mu\text{g}/\text{m}^3$, and number concentrations were 95,000–180,000/ cm^3 . About half of the particles were ultrafine (<100 nm). Blood, urine and breath samples were taken before and after the experiment. Exposure to wood smoke increased the levels of serum Amyloid A, a cardiovascular risk factor, as well as factor VIII in plasma and the factor VIII/von Willebrand factor ratio, indicating a slight effect on the balance of coagulation factors. Moreover, there was an increased urinary excretion of free 8-iso-Prostaglandin_{2 α} , a major F₂-isoprostane, though this was based on nine subjects only, indicating a temporary increase in free radical-mediated lipid peroxidation. Effects were shown also on breath markers.

Conclusion: Wood smoke particles at levels that can be found in smoky indoor environments seem to affect inflammation, coagulation and possibly lipid peroxidation. These factors may be involved in the mechanisms whereby particulate air pollution affects cardiovascular morbidity and mortality. The exposure setup could be used to establish which particle characteristics are critical for the effects.

Intrauterine exposure and genotoxic effects of air pollution in children

Lisbeth E. Knudsen, Tina Dam Mikkelsen, Marie Pedersen (UC, Denmark)

Fetal growth and development is extremely dependent on placenta functionality as the major role of the placenta is to exchange oxygen, nutrients, hormones, and waste product between the pregnant mother and the fetus. Exposures to environmental toxicants and drugs during pregnancy are associated with a number of adverse effects in the child ranging from minor growth retardations to malformations. Exposure to tobacco smoke and environmental pollution is associated with lower birth weight, retarded growth and neurodevelopment. Knowledge of fetal exposure to environmental toxicants is sparse and necessary for risk assessment within reproductive health and environment. Substances able to cross the placenta have the potential to cause adverse effects directly on fetal development or the function of the placenta, while substances not able to cross the placenta can harm the fetus through an effect mediated in the mother. The barrier function of the placental membrane depends on molecular size, polarity, lipid solubility and protein binding of the substances. Dual recirculating perfusion of a single cotyledon in the human placenta can contribute to a better understanding of the transplacental transfer (including toxicokinetics of the mother, fetus and the placenta) and the toxicity of different substances.

A family pilot study was conducted in the Czech Republic to test the hypothesis that exposure to air pollution with particulate matter (PM) in children results in detectable effects indicated by a number of biomarkers of exposure and early effects. Significant higher frequencies of micronuclei were found in the children living in the Teplice area as compared with those living in the Prachatice area. The increased MN frequency may be associated with elevated polycyclic aromatic hydrocarbon (PAH) concentration of the PM_{2.5} measured in the ambient Teplice air, but other factors like genotoxic compounds from the diet or protective effect of micronutrients were not addressed.



List of participants in Nordic Air Pollution Workshop 2006

Name	e-mail
Alireza Afshari	ala@sbi.dk
Alix Rasmussen	ar@dmi.dk
Anna Wallén	anna.wallen@itm.su.se
Anne Mette Boisen	amb@ami.dk
Anne Saber	ats@ami.dk
Anoop Sharma	ash@ami.dk
Bent Hansen	danalytic@post.tele.dk
Berit Grevstad	bgr@ami.dk
Bertil Forsberg	bertil.forsberg@envmed.umu.se
Birthe Busch Thomsen	bibuse@tmf.kk.dk
Birthe Vraa-Dam Sørensen	b.vraa@pubhealth.ku.dk
Christer Johansson	christer.johansson@itm.su.se
Christian Lange Fogh	clf@mst.dk
Christian Stevns Hansen	stevnshansen@gmail.com
Elvira Vaclavik	elva@pubhelath.ku.dk
Emma Nordling	emma.nordling@sll.se
Erik Nilsson	
Erik Swietlicki	Erik.Swietlicki@nuclear.lu.se
Finn Plamgren	fpj@dmu.dk
Gerd Sällsten	gerd.sallsten@amm.gu.se
Hans Orru	orru@ut.ee
Herman Autrup	ha@mil.au.dk
Hilde Balling	hib@sst.dk
Håkan Wallin	hwa@ami.dk
Ingunn Skogstad Riddervold	isri@mil.au.dk
Jaana Kettunen	jaana.kettunen@ktl.fi
Jacob Mønster	jgm@dmu.dk
Jakob Fryd	jafryd@tmf.kk.dk
Jakob Löndahl	jakob.londahl@nuclear.lu.se
Jan Byman	jan.byman@naturvardsverket.se
Jan Sundell	jas@mek.dtu.dk
Janne Berger	janne.berger@nilu.no
Janne Kjærsgaard	jakj@pubhealth.ku.dk
Jes Fenger	jfe@dmu.dk
Jesper Stubkjær	jfsemail@yahoo.com
Jette Bornholdt Lange	jbo@ami.dk
Kadri Meister	kadri.meister@envmed.umu.se
Karin Sørig	ksh@ami.dk
Lars Barregard	lars.barregard@amm.gu.se
Lars Gunnarsen	lbgsbi@sbi.dk
Lisbeth E. Knudsen	l.knudsen@pubhealth.ku.dk
Lykke Forchhammer	lforchhammer@hotmail.com
Maria Ullerstam	Maria.Ullerstam@naturvardsverket.se
Marianne Glasius	mag@dmu.dk
Marianne Paulsen	marianne.paulsen@rh.hosp.dk
Marie Pedersen	m.pedersen@pubhealth.ku.dk
Martin Hvidberg	martin.hvidberg@dmu.dk
Martin Roursgaard	mro@ami.dk
Mathias Ketzel	mke@dmu.dk
Mette Sørensen	mettes@cancer.dk
Mikael Skou Andersen	msa@dmu.dk
Nicklas Raun Jacobsen	nrj@ami.dk
Ole Hertel	oh@dmu.dk
Ole Raaschou-Nielsen	ole@cancer.dk



Oliver Bischof	oliver.bischof@tsi.dk
Otto Hanninen	otto.hanninen@ktl.fi
Per Nafstad	Per.Nafstad@fhi.no
Per Schwarze	Per.Schwarze@fhi.no
Pernilla Gustafson	pernilla.gustafson@amm.gu.se
Pernille Danielsen	p.danielsen@pubhealth.ku.dk
Peter Molnár	peter.molnar@amm.gu.se
Peter Vangsbo Madsen	pvm@dmu.dk
Peter Wåhlin	pew@dmu.dk
Pia Konggaard Nielsen	pia_k@nielsen.mail.dk
Poul Bo Larsen	pbl@mst.dk
Raimo Salonen	raimo.salonen@ktl.fi
Rikke Hansen	rha@ami.dk
Steen Solvang Jensen	ssj@dmu.dk
Steffen Loft	s.loft@pubhealth.ku.dk
Tatjana Ilyina	ilitat@gmail.com
Thomas Sandstrom	thomas.sandstrom@lung.umu.se
Timo Lanki	timo.lanki@ktl.fi
Tina Dam Mikkelsen	t.d.mikkelsen@pubhealth.ku.dk
Ulla Vogel	ubv@ami.dk
Ulrik Korsholm	usn@dmi.dk
Unni Cecilie Nygaard	Unni.Cecilie.Nygaard@fhi.no
Zorana Andersen	z.jovanovic@biostat.ku.dk

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