The IPIECA Urban Air Quality Management approach

The preparation and application of pollutant emission inventories: exploring the capacity of the IPIECA Toolkit

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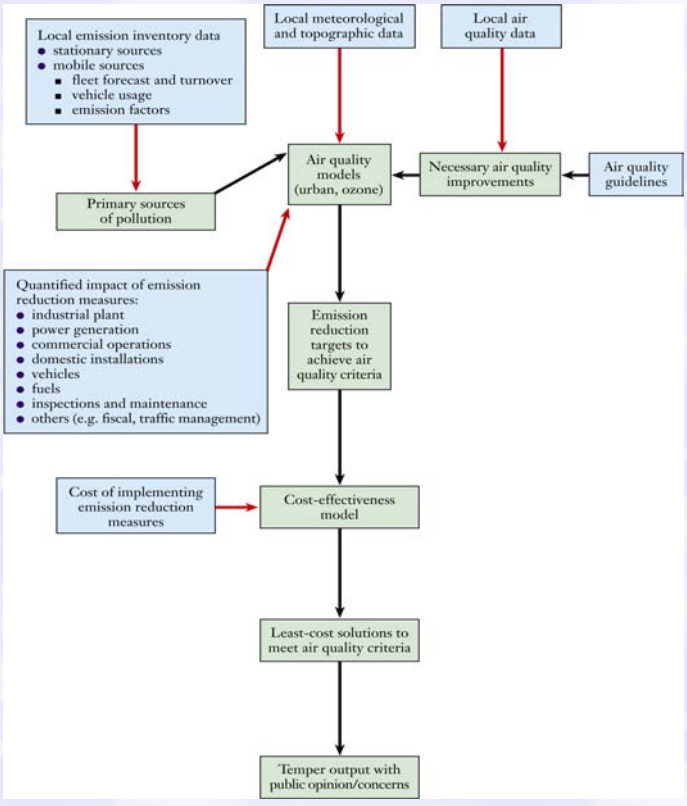
Background to the IPIECA approach

The management of urban air quality is a problem facing cities worldwide, with rapid population growth, and fast economic, industrial and commercial expansion. Adoption of an urban air quality management approach which is based on the principles of sound air quality objectives, cost-effectiveness analysis, use of good science, recognition of the role of all major contributing emission sources, and involvement of all stakeholders, is crucial.

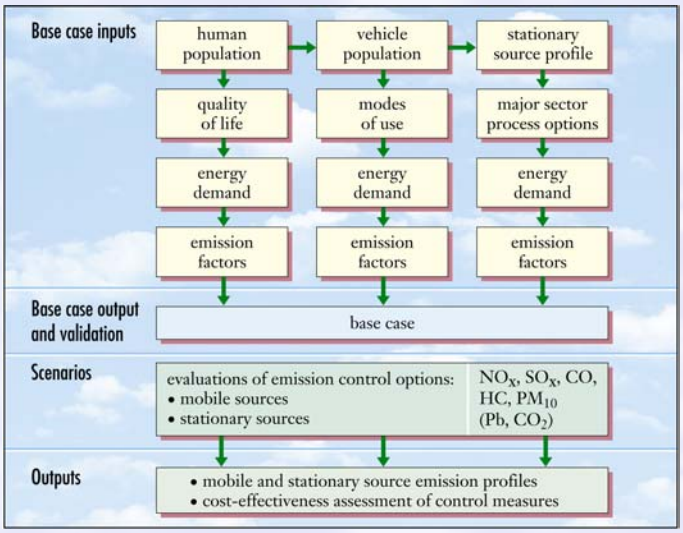
The IPIECA Air Quality Management process below sets out a standardized approach to air quality management which integrates local emission inventory data with the desired air quality improvements to present least-cost solutions for achieving local air quality goals.

IPIECA developed a flexible emission inventory model or "*Toolkit*" to address the development of local emission inventories, which are an essential component of any air quality study. The Toolkit embraces an integrated air quality management approach, facilitates the development and evaluation of local emission databases, and offers the opportunity for rapid screening of a wide spectrum of emission control strategy options by decision makers. The Toolkit also presents outputs, not only in terms of emission reductions, but also in terms of relative cost-effectiveness.



How the Toolkit works

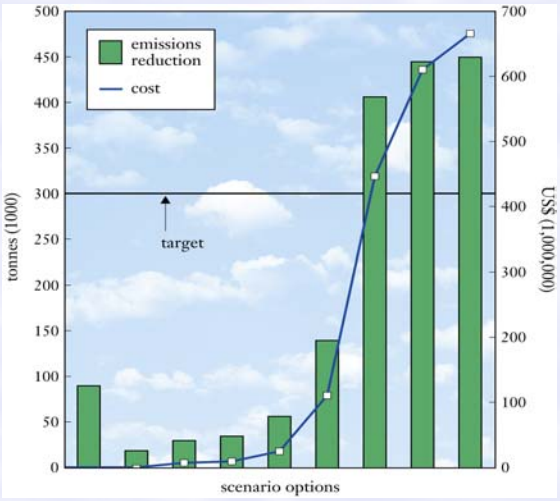
A schematic representation of the Toolkit process is given opposite. The Toolkit is a spatially adaptable model that can characterize mobile, stationary, and natural sources of historical emissions, and can forecast future emissions based on socio-economic growth factors. The Toolkit can be used to perform 'what if' analyses to determine the effectiveness and cost of emission control strategies applied to mobile or stationary sources (or both). The Toolkit therefore places a rapid screening tool in the hands of urban air quality management policymakers.



Data Input Requirements

- Time: The Toolkit can model up to 100 units of time (Years, Days)
- Human Population: The model uses population data characterize growth patterns for the base case and to forecast future growth
- Vehicle Population: The Toolkit classifies vehicles by size, by fuel type, by duty, as well as mode of use (driving modes, average speed and mileage driven within modes, and fuel consumption)
- Other vehicle-related inputs: These include the major fuel quality parameters for the fuel options selected, as well as emission factors drawn from the toolkit database
- Stationary sources: The Toolkit makes provision for type and quantity of residential, commercial and industrial fuels, including power generation.
- Natural Sources: The impact of natural or non-fuel sources of emissions can also be characterised.

Cost Effectiveness calculations



The Toolkit also provides a valuation of the cost-effectiveness of introducing emission control measures based on the costs associated with each measure. The cost data files within the Toolkit give the incremental costs of additional emission control measures, (e.g. introduction of catalysts). The data on vehicle technology is drawn from the European Auto/Oil studies.

Through an ongoing dialogue with the World Bank, the IPIECA Toolkit has been made accessible to cities taking part in the World Bank Clean Air Initiative (CAI). As part of the CAI program, IPIECA and Enstrat International have used the model in support of urban air quality management programmes in Latin American cities, and it has also been used by Shell Global Solutions in New Zealand. The Toolkit is available to non-profit making organizations, on application to the IPIECA Secretariat (http://www.ipieca.org), who will advise on necessary training requirements.

IPIECA

BenMAP, The Environmental Benefits Mapping and Analysis Program: A Tool for Estimating Health Benefits from Air Quality Improvements.

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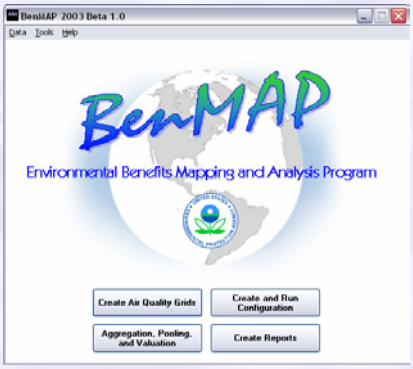
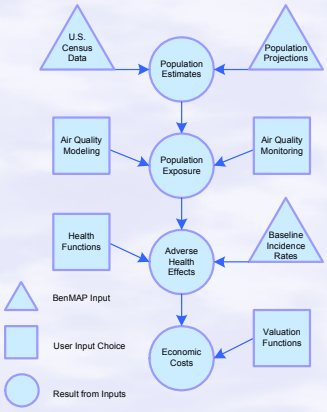
What is BenMAP?

Abt Associates Inc. and the United States Environmental Protection Agency's (US EPA) Office of Air and Radiation have developed a state-of-the-art air benefits model called BenMAP, the Environmental Benefits Mapping and Analysis Program. BenMAP is the US EPA's premier tool for estimating benefits associated with air pollution reduction strategies. BenMAP runs on a Windows© based computer, and is planned for public released by the US EPA after a successful peer review. BenMAP is a GIS-based modeling system that can:

- Create population-level air pollutant exposure surfaces;
- Estimate changes in incidence of a wide variety of health outcomes associated with changes in ambient air pollution;
- Value changes in incidence of health outcomes;
- Provide characterizations of uncertainty in incidence and valuation estimates; and,
- Document key modeling assumptions and analyst judgments through an exportable "audit trail."

BenMAP is designed for many types of users to examine the benefits of policy options for air quality management, including policy analysts, academics, and decisionmakers.

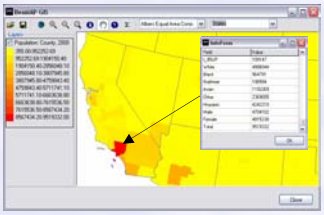




Key Features of BenMAP

BenMAP can handle data at many different levels of spatial aggregation. By overlaying grid-based data files over a geographical area of interest (e.g., the U.S.), BenMAP fits data into each user-specified grid cell. A "grid" can be regularly shaped or based on GIS shapefiles.

Population Estimates are built off of U.S. Census block data and aggregated to a user-specified grid. BenMAP can be easily adapted to accommodate population grids from other countries.



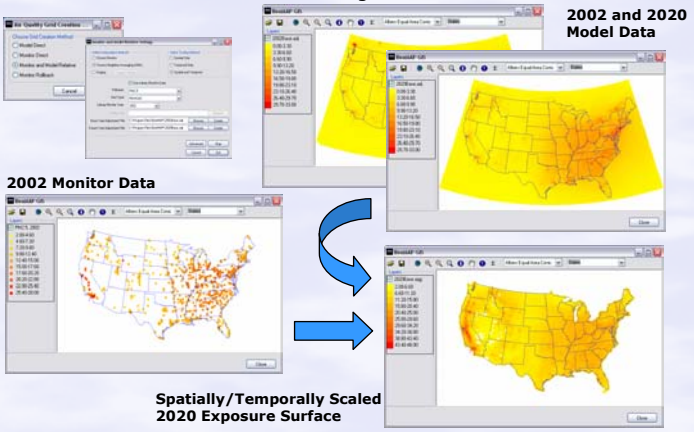
Air Quality Data – BenMAP is able to use a wide variety of air quality data, both monitored and modeled.

- *Model based data* is widely available in standard grid formats (REMSAD, CAMx, UAM-V). Other model formats can be imported by the user.
- *Monitor based data* – BenMAP contains an internal library of U.S. monitor data for multiple years and pollutants.

Key Features (Cont.)

Population Exposure Surfaces (grids) can be calculated in multiple ways, including direct use of monitor or model data, or use of model data with monitor data in a relative sense. Interpolation methods include *Closest Monitor, Voronoi Neighbor Averaging, and Kriging*.

- Users can also quickly assess **"What If?"** scenarios based on user-defined reductions in monitored air quality levels.
- BenMAP includes a powerful **Monitor Filtering** tool that can be used to restrict the monitors used in grid creation.

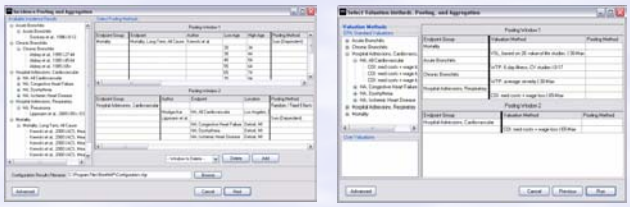


Adverse Health Effects – BenMAP is preloaded with hundreds of C-R functions to estimate adverse health effects. Users can easily add more with the full featured equation editor.

Pooling - BenMAP can also combine the results of multiple C-R functions that estimate the same pollutant-health endpoint combination in order to produce better estimates of the adverse health effect. BenMAP supports three pooling methods: *Subjective Weights, Fixed Effects, and Random/Fixed Effects*.

Valuation - BenMAP can estimate the economic value of avoided incidence based on hundreds of health effect-specific unit values.

Uncertainty - BenMAP characterizes uncertainty surrounding C-R relationships and uncertainty surrounding unit dollar values and total monetized benefits using Monte Carlo methods.



Future Directions for BenMAP

- **International BenMAP** – We are designing BenMAP to allow international researchers to enter their own population, health and air quality data.
- **Advanced Uncertainty Analysis** – BenMAP will be able to evaluate multiple uncertain elements within a given policy analysis, provide a more sophisticated treatment of correlation structures, and provide influence analyses.
- **Addition of Non-Air Impact Assessment** – BenMAP's grid-based modeling structure makes it transferable to assessments related to visibility, mercury deposition and water pollution.
- **Website** with all databases and configurations used in EPA analyses. Users may completely replicate analyses and develop own sensitivity analyses.

Notes

1. Abt Associates Inc., 4800 Montgomery Lane, Suite 600, Bethesda, MD 20814 USA
2. US EPA, Research Triangle Park, NC 27711 USA

Abt Associates Inc.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

Estimating The Health-Based Economic Impacts Of Ground Level Ozone Attributed VOC Emissions: An Alternate Science-Based Approach

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Background

BeTa Version E1.02a

- Created for European Commission DG Environment by Netcen
- Provides marginal external cost of emission of individual pollutants
  - SO_2, SO_2-SO_4, NO_x-NO_3, NO_x-ozone, $PM_{2.5}$, **VOC-ozone**
- Used to support cross media BREF, SLFD, Decorative Coatings Directive

BeTa Version E1.02a - Conceptual Framework

Emission Data ("late 1900's") → GIS Data e.g. Population Dist. → Frequency of Events[1]

Exposure Response Functions[1] (ecological epi studies) → Frequency of Events[1]

Unit cost of event (Willingness to pay) → **Marginal External Costs of Emissions of Individual Pollutants (€/ton)[1]**

[1]Components examined in the current study

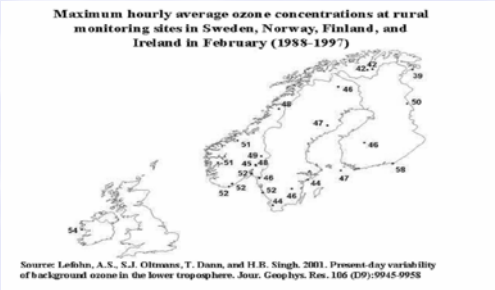
Study Objective: Evaluate Marginal External Costs Of VOC Emissions-Ozone

- Focused on health impacts: ~75% of total VOC impact
- Focused mostly on acute mortality: >99% of health impact
- Evaluated key assumptions with focus on all available data
  - Epidemiology, **human clinical, toxicology, mechanistic data**
- Develop alternative assumptions where supported by latest data
- Re-run BeTa using alternate assumptions: quasi sensitivity analysis

Summary Of Evidence For Acute Ozone Mortality Hypothesis (1a)

Data Category	Summary
Epidemiology	Partial support but serious methodology questions exist
Human clinical and toxicology	No or limited support
Mechanistic	No or limited support
Overall	Limited support

Alternative approach for impact analysis: assume no mortality occurs



Summary Of Major Assumptions in BeTa and Alternate Approaches

Assumption	Alternate Assumption / Approach
Current ambient ozone levels produce acute mortality	Current levels do no produce mortality
There is no threshold for acute ozone mortality	Mortality threshold is evident at current background levels of 0.04-0.05 ppm
RRs of 0.59 per ug/m³ in Barcelona are representative for Europe	Use central value of 0.02-0.3 ug/m³ from recent WHO report
High and low ozone produce equal risk	Use risk ratios from 1-hour averaging times without upward adjustment
Life years lost from acute ozone mortality is 6 months	No reliable value available / suggested
Percent of population affected is 0.099%	No reliable value available / suggested

Summary Of Marginal External Health-Based Cost of VOC Emissions Using BeTa And Alternate Approaches

Approach	Health Effects	Assumptions	Cost/ton (€)
BeTa VE1.02a Base case	Respiratory hospital admissions Asthma provocation Minor restricted activity days Acute ozone mortality	No threshold for all effects High end ER function for mortality	1623
Alternative 1	Respiratory hospital admissions Asthma provocation Minor restricted activity days Acute ozone mortality	No threshold for all effects Central ER function for mortality	566-837
Alternative 2	Respiratory hospital admissions Asthma provocation Minor restricted activity days Acute ozone mortality	No threshold for morbidity Mortality Threshold at 0.05 ppm High end ER function for mortality	88[1]
Alternative 3	Respiratory hospital admissions Asthma provocation Minor restricted activity days	No threshold for morbidity effects	23

[1]Estimated from analysis by Holland et al. 1998

Conclusions And Future Considerations

- Acute ozone mortality and no threshold assumptions have dramatic influence on cost estimates
  - No mortality: reduction of nearly 99% in health-related marginal external costs of VOC emissions
  - Mortality with threshold: reduction of nearly 95%
- Use of more central ER function for mortality has significant impact
  - Reduction of 50-75% in health-related marginal external costs of VOC emissions

Suggestions for Future Work:

- Evaluate effects of NO_x on ozone levels
- Incorporate photochemical reactivity concept to more accurately differentiate impact of individual VOCs
- Perform review of the marginal external costs of environmental impacts

L. Gephart and M. Amoruso, ExxonMobil Biomedical Sciences, Inc.

Impact Assessment System for Urban Air Pollution and Population Exposure

Jensen, S.S., Berkowicz, R., Hertel, O., Hvidberg, M.,Kousgaard, U. and Hansen; H.S.
National Environmental Research Institute (NERI), Denmark

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Introduction

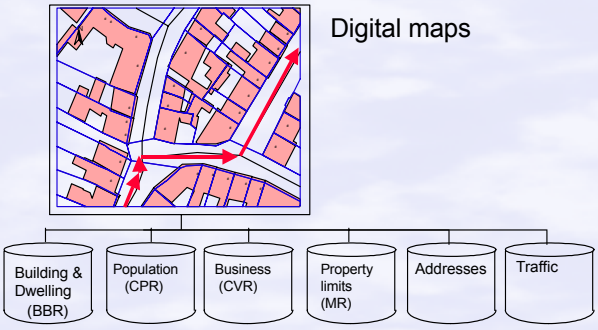
Numerous studies have shown associations between air pollution exposures and morbidity and mortality and the related social costs. AirGIS[1] is a human exposure model system that estimates ambient air pollution levels at very high temporal resolution (hourly) and spatial resolutions (address level). The system is continuously under development to support impact assessment of air pollution on human exposures and health. Pollutants include: NOx, NO_2, O_3, CO, benzene and PM10. Particle numbers and PM2.5 will also be included in the future.

AirGIS - Human Exposure Model System

The concentrations at street levels are estimated with the Danish OSPM model[2]. Emission estimation at individual road links is based on information about traffic intensity from external traffic models and an emission model integrated into the OSPM model. The physical dimensions of the street is estimated e.g. building heights in various wind sectors.

The OSPM model requires urban background air quality and meteorological data. The Danish UBM model estimates urban background concentrations based on a 1 x 1 km^2 emission grid. The emission grid is generated based on a geographical distribution of national emissions based on various geographical weight themes. The regional contribution to the urban background is based on the Danish large-scale model DEHM that relies on the European EMEP emission inventory. The model system has been validated against air quality monitoring data.

Furthermore, AirGIS comprises detailed technical and cadastral digital maps and Danish national administrative databases on buildings, cadastres and populations. It applies a Geographic Information System (GIS).

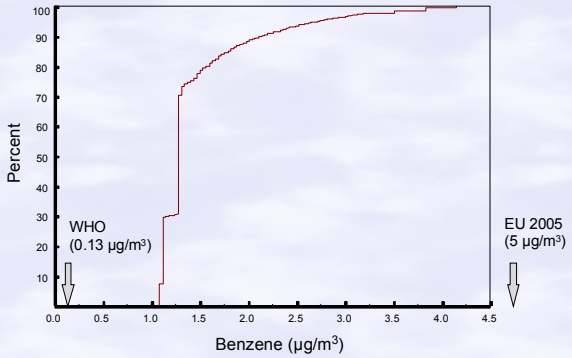


AirGIS is an exposure model system for estimation of exposure at address level or for personal exposure provided that route information is available describing the transport behaviour of an individual.

Urban Air Quality Assessment

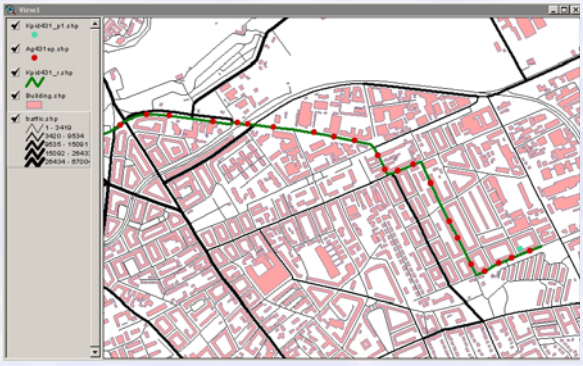
The model system is under implementation for the Greater Copenhagen region as a case study that includes 1.8 million inhabitants out of 5.3 millions in Denmark.

The system can be used in urban air quality management. Mapping and scenario results can be compared with air quality limits and impact assessment of traffic air pollution abatement measures can be carried out like evaluation of environmental zones in cities, particle filters on heavy vehicles and road pricing.



Percentage of people exposed to ambient benzene levels (µg/m³) with the residence address as exposure indicator. Example from the Municipality of Middelfart, Denmark with 10,000 addresses (1996). Similar results will be obtained for the Greater Copenhagen Area.

Air Pollution Epidemiology



Air pollution epidemiology examines the relationship between air pollution and health effects. AirGIS can provide exposure data at address level or personal exposure provided that route information is available. Example of route is illustrated.

Health Impact Assessment

The link between exposures and health impacts is under development based on existing exposure-response relationships. The development of this linkage will enable crude health impact assessment and social cost estimates.

Acknowledgement

Funded by the Centre for Transport Research on Environment and Health Impacts and Policy under the Danish Strategic Environmental Research Programme 2000-2004. Further details on http://www.akf.dk/trip/projekter/32proj.PDF and http://www.akf.dk/trip/projekter/33proj.PDF

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1) Jensen, S.S., Berkowicz, R., Hansen, H. Sten., Hertel, O. (2001) A Danish decision-support GIS tool for management of urban air quality and human exposures. Transportation Research Part D: Transport and Environment, Volume 6, Issue 4, 2001, pp. 229-241.

2) Berkowicz, R. (2000) OSPM – A parameterised street pollution model. Environmental Monitoring and Assessment, Volume 65, Issue 1/2, pp. 323-331.

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Health Effects Indicator Decision Index (HEIDI): A risk-based tool for ranking abatement of air pollution release inventories by expected regional health effects

R.S. McColl, J. Hicks, J.S. Shortreed, and L. Craig

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Policy Background

The Canadian Council of Ministers of the Environment (CCME) is responsible for devising Canada wide standards for major air pollutants such as PM2.5 and ozone, developing control programs for vehicular air emissions, and defining emission reduction strategies for various industrial sectors. The CCME under the *National Framework for Petroleum Refinery Emissions Reductions* (NFPRER) is currently developing an emissions reduction strategy for the petroleum refinery sector *(ref.1)*.

Environment Canada currently collects annual emissions reports on over 200 chemical substances, mixtures, and precursors from all 23 refineries in Canada as part of the *National Pollutant Release Inventory* (NPRI). In setting priorities for abatement. air emissions must be ranked according to their toxicity-weighted health effects, not merely by their emissions mass. Risk-specific environmental indicators (RSEI) consist of toxicity weightings (TW), plus other parameters such as exposed population (EP) and environmental persistence. Various analysis groups are defined according to how these parameters are organized and used to calculate health risk.

Ranking of Emission Reductions

The *Health Effects Indicator Decision Index* (HEIDI) is a risk analysis tool developed by NERAM as part of the NFPRER initiative. It performs a spreadsheet analysis in MS EXCEL to determine the relative weighted health effects of a given set of NPRI substances emitted within a specified refinery site. This tool allows decision-makers to view the priority rankings for air emissions reduction by their toxicity weightings and other RSEI parameters. The priority rankings are displayed for Analysis Groups 1 to 4, including several types of subanalysis within each Group (*ref. 2)*. The HEIDI ranking method has several useful capabilities:

- determines the priority rankings in each refinery for reducing NPRI emissions, according to analysis subgroup

- estimates the effects of regional population distribution profiles and varying atmospheric mixing heights

- provides a 'what-if' scenario analysis in a given refinery to determine the degree of emission reduction required to shift downward the ranking of a specified air toxic

- examines the effect of differences in TW values for air toxics for various jurisdictions (USEPA vs Health Canada), which can produce significant shifts in rankings

HEIDI uses continuous linearized dose-response functions in analysis subgroups 4c and 4d to characterize the TW for threshold-acting agents. This eliminates the computational artifacts of step-function TW parameters such as Reference Concentration (RfC) or Tolerable Concentration (TC).

Findings and Conclusions

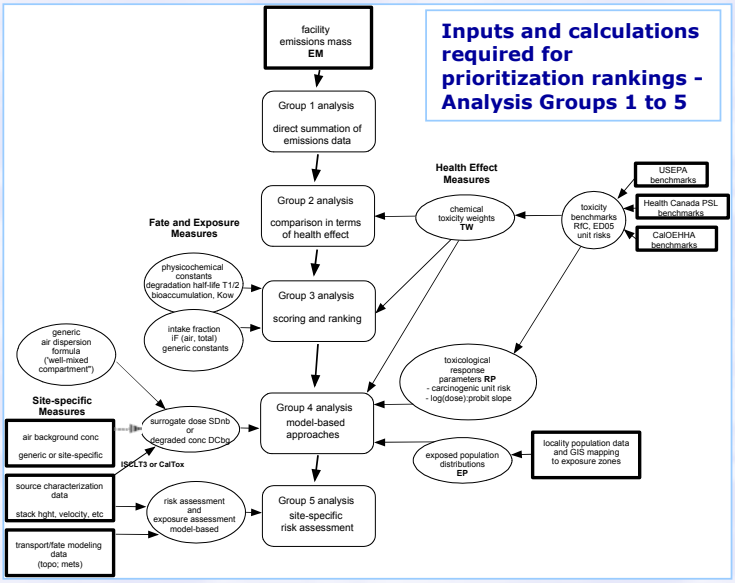
Rankings for air emission reductions in oil refineries are sensitive to assigned TW values and other RSEI parameters.

Differences in TW values in separate jurisdictions (USEPA vs Health Canada) can produce significant shifts in rankings.

Priority outcomes are greatly affected by selective inclusion of RSEI input parameters and risk model assumptions.

Analysis subgroup 4c is the recommended ranking method.

The observed pattern of ranking shifts between various analysis subgroups supports the conclusion that careful consideration of parameter inputs and ranking formulas is important enough to engage in further methodology studies.



CCME Prioritization project
Analysis Guide

Analysis Group	Sub group	calculation of Indicator Element (IE)	emission		estimated dose			measure of toxicity				persons at risk	
			emission mass EM = tonnes*1000 [kg]	emission concentration EC = EM*1/V $V_{di} = pi*r^2*h$	degraded concentration DC = EC *f(1/$T_{1/2}$)	fate concentration FC=EC*iF intake fraction iF (air, total)	surrogate dose SD_{nb} no background	degraded concentration plus background $=DC_{bg}+bg$	toxicity benchmark RfC	toxicity weight TW	response parameter RP	slope-modifying parameter SP	exposed population EP
1		EM	+	-	-	-	-	-	-	-	-	-	-
2	a	EC x RfC	-	+	-	-	-	-	+	-	-	-	-
	b	EC x TW	-	+	-	-	-	-	-	+	-	-	-
3	a	DC x TW	-	-	+	-	-	-	-	+	-	-	-
	b	FC x TW	-	-	-	+	-	-	-	+	-	-	-
4	a#	SDnb x TW x EP	-	-	-	-	+*	-	-	+	-	-	+
	b¤	DCbg x TW x EP	-	-	-	-	-	+**	-	+	-	-	+
	c¦	DCbg x f(RP) x EP	-	-	-	-	-	+**	-	-	+	-	+
	d	DCbg x f(RP, SP) x EP	-	-	-	-	-	+**	-	-	+	+	+
5		full risk assessment model	+	+	+	+	+/-	+*	+	-	+	+/-	+

USEPA REIS formula for IE
¤ Health Canada PSL formula for IE
¦ Response Parameter formula for IE (dose-response function with default slope = 1)
Response Parameter formula for IE modified by Slope Parameter (for dose-response function where slope - 1)
*site-specific ISCLT model (or similar transport and fate air model)
** generic well-mixed compartment formula, or site-specific ISCLT model

HEIDI Health Effects Indicator Decision Index version 1.3
Stephen McColl and John Hicks are the authors of this work

NERAM Refinery Prioritization project for CCME (air toxics)
front end interface sheet

4 **ENTER FACILITY SELECTION**

ENTER MIXING HEIGHT (KM) 1.5 default mixing height = 1.5 km

1= AB Scotford
2= NB St John
3=BC Burnaby
4= hypothetical worst-case refinery

100% = no reduction in current emission levels
10% = reduction to 10% of current emission levels

RESIDUAL EMISSIONS TARGET indicates what is the target level of continuing emissions after abatement

hypothetical worst-case refinery **ENTER RESIDUAL EMISSIONS TARGET (1% to 100% of current level)**

			100	100	100	100	100	100
formula	**Analysis Group**	***subgroup***	**mercury**	**MTBE**	**ethylbenzene**	**benzene**	**toluene**	**n-hexane**
EM	**1**	***1***	6	1	5	3	2	4
EC x 1/RfC	**2**	***2a***	5	2	4	1	6	3
EC x TW		***2b***	6	4	5	1	3	2
DC x TW	**3**	***3a***	4	6	3	1	5	2
FC x TW		***3b***	2	6	4	1	5	3
SDnb x TW x EP	**4**	***4a***	4	6	3	1	5	2
DCbg x TW x EP		***4b***	4	6	3	1	5	2
DCbg x f(RP) x EP		***4c***	4	2	6	1	5	3
DCbg x f(RP, SP) x EP		***4d***	4	2	6	1	5	3

NOTE: the decision index is currently configured for an exposed population distribution (EP) that approximates the Greater Toronto area for this reason, the scenario reflects only what would occur hypothetically for the 3 refinery facilities if they were located in the western GTA

valid index results will only be produced when accurate site-specific **population distribution values are provided by a GIS population databa**
valid index results will only be produced when accurate site-specific **background air toxics concentrations are provided by a monitoring dat**

(1) Assessment of comparative human health risk-based prioritization schemes for petroleum refinery emissions reduction. NERAM Report to CCME. May 26, 2003.

(2) Pennington, DW & Bare, JC. (2001). Comparison of chemical screening and ranking approaches: The waste minimization prioritization tool versus toxic equivalency potentials. Risk Analysis, vol. 21, No. 5, 897-912.

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Proposed Framework for an Effectiveness Analysis for PM Reduction

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The relationships between generated air pollution and human adverse effects are complicated. The sources of air pollution are many and varied, their relation to human exposure is complex, and the effects of exposure on individuals are varied. Therefore, the relationship between various reduction initiatives and their corresponding benefits are challenging. In order to promote a benefit/cost discussion we propose to develop, with interested parties, a straw-man framework to visualize the important components of the relationships. The framework, based on the principals of decision analysis, is suggested as a start toward developing a method for performing an effectiveness analysis to identify the most important remedial actions. The basic idea is to lay out the individual components, or nodes, in a pathway from pollution generation to human effect. All interested parties would contribute to the identification of nodes, to the mathematical model form (without numeric values on parameters) of how each node operates, and to the inter-relationships among the nodes.

The benefit of building the model would be to achieve agreement among interested parties as to what model sections are important to consider, how the sections might work, how the sections fit together, and where any anomalies might exist in the logic flow. Developing the model is seen as a major step in the process, possibly taking the bulk of the effort. Using the model, different parameter values would be suggested, in the form of point estimates or distributions.

After the model is fully described it is then a relatively straightforward mathematical exercise to determine which nodes are critical in the benefit/cost relationship and/or where more effort is needed to develop more precise parameter estimates or a better structure of the model node.

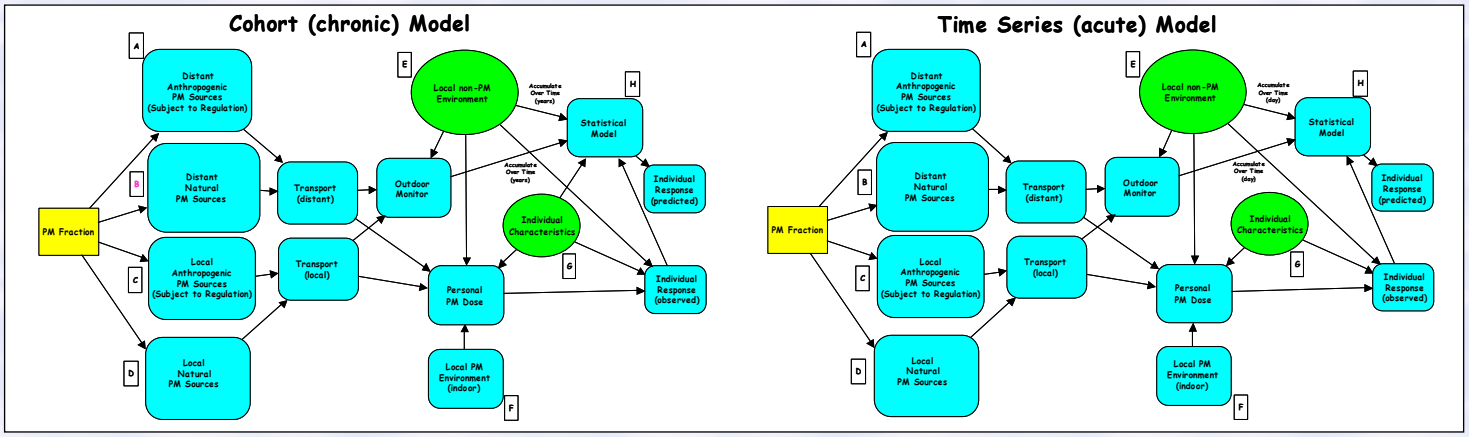
Developing a model is based on a series of questions, such as but not limited to:

- What is the overall goal or research question?
- What are the major decision points?
- What is the flow of information from exposure to health risk?
- What are the most significant uncertainties, and is it possible to obtain additional information to reduce the uncertainty of a decision?

The diagram is a first attempt to identify the main nodes in the flow diagram from source to health endpoint. Each node has a code that relates to a suggested starting list of items to be considered.

As with any method this approach has strengths and limitations. A key strength is the ability to frame the problem and allow stakeholders to express their concerns; this will stimulate explicit analysis of the effects of social values, estimates of uncertainty, projected costs, and efficiencies of control options on decisions. Also, it will facilitate the important public policy development process of open examination of the tradeoffs involved in deciding whether to perform additional research. A comprehensive, transparent description of methods and assumptions of the analyses are critical to realizing the above strengths.

On the other hand, to date there has been limited use of this method in real world environmental health research priority setting. This lack of a body of examples of real world applications likely reflects the complexity in modeling and solving this type of question and the difficulties in valuing outcomes and characterizing the uncertain and variable model inputs (Yokota, et al, 2003a, 2003b). A major difficulty is the valuing health and other non-monetary outcomes, although this problem may be reduced in the future with the increased activity in health outcome valuation research. Also, we note that in some instances additional research may actually increase the perceived uncertainty if the new research reveals more complexity and uncertainty than previously recognized. However, this information is valuable if the goal is to



A & C - Distant & Local Anthropomorphic PM Sources

- Power plant, coal, gas, oil
- Internal combustion engine (auto/stationary)
- Barbeque grill
- Manufacturing, smelters, refiners, paper mill
- Mining, Water treatment, Etc.

B & D - Distant & Local Natural Sources

- VOC
- Pollen, Animal, Insect, Sea Salt
- Fires, Crustal Material, Etc

E- Local non-PM Sources

- Hydrogen ions
- SOx, H2SO4, Nox, HNO3
- O3, CO, VOCs,
- Temperature, Humidity (and variation)
- Altitude, Water hardness
- Annual average heating/cooling degree-days
- Etc.

F- Local PM Environment

- Cigarette smoking
- Space heaters: kerosene, gas
- NOx
- Wood burning stoves, candles
- Re-suspension/personal cloud, Cleaning,
- Cooking, washing, showering
- Biological sources (pollen, mites, moulds, insects, micro-organisms, pet allergens)
- Emissions from indoor materials/substances (VOCs, lead, radon, asbestos, various synthetic chemicals)
- Etc.

G- Individual Characteristics

- Age, Gender, Martial status, Height
- Race group, Body mass index
- Pet ownership, Socioeconomic factors
- Smoking & alcohol history
- Diet history
- Daily stress, Blood sugar, COPD

G- Individual Characteristics (cont)

- Exercise patterns (including how much time spent outdoors)
- Occupation and dust exposure
- History of medical conditions
- Blood pressure
- Health status (diabetes control, cardiovascular system, lung function)
- Etc.

H- Statistical Model

- (terms directed to the Statistical Model node are not always included)
- Poisson response model
- Cox proportional hazards
- Data mining methods
- Etc.

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A TOOL TO EVALUATE IN REAL-TIME AND FORECASTING MODE THE AIR QUALITY IMPACT OF INDUSTRIAL EMISSIONS

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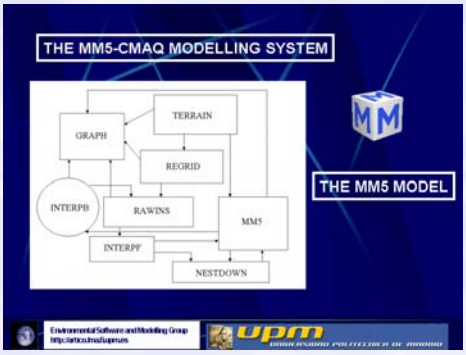

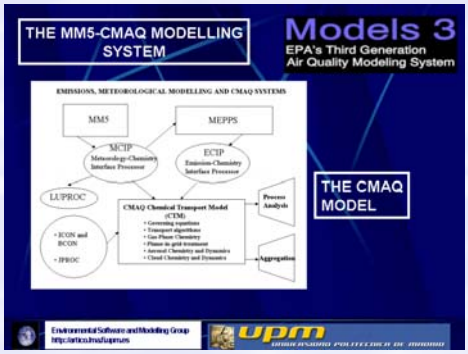

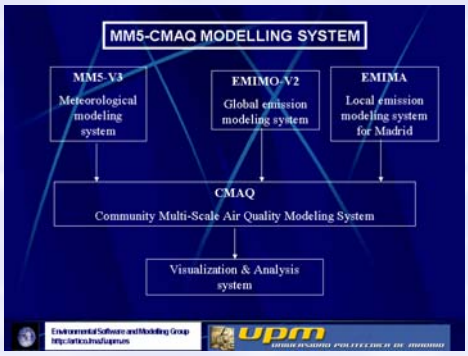

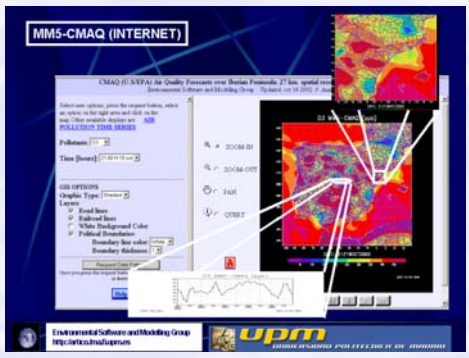

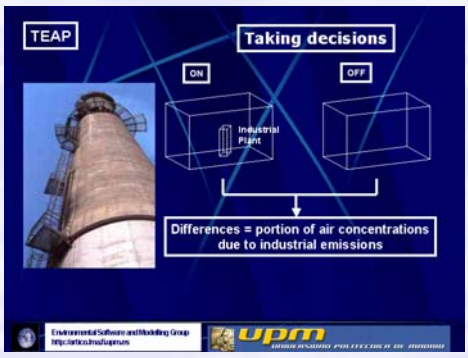

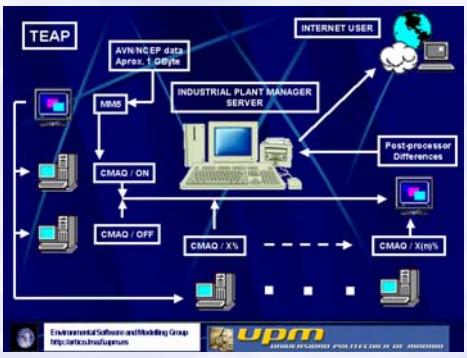

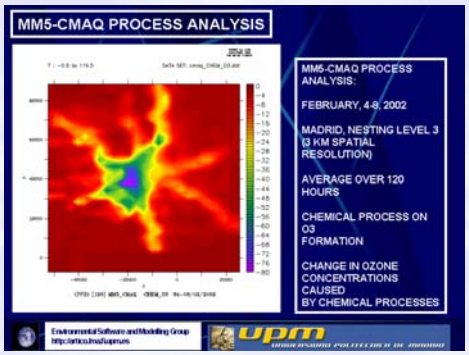

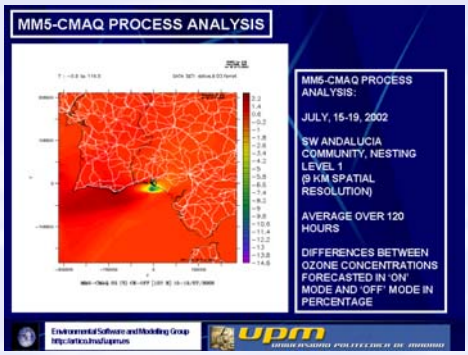

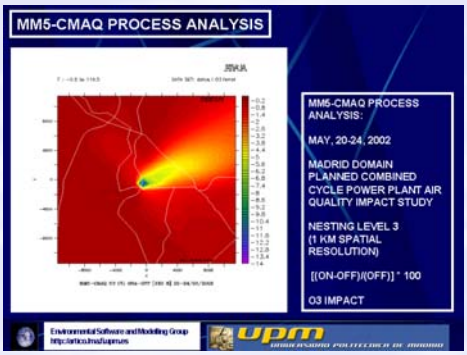

This tool uses themost advanced meteorological and dispersion models (with aerosol chemistry). It provides a full analysis of the impact of the industrial plant emissions in time and space. It can be run in real time and forecasting mode and it can also be used for Air Quality Impact Analysis for pre-operational analysos for power plants and general industrial plants. The tool is developed under the EUREKA project TEAP.

- **MM5 is a non-hydrostatic mesoscale mode developed by PSU/NCAR (USA).**
- **CMAQ is the Community Multiscale Air Quality Modelling System developed by EPA (USA)**
- **EMIMO is an emission inventory model developed by UPM (Spain).**

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No-regret policy measures for PM from traffic sources.

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Objective

To evaluate the cost and benefit of emission reduction scenarios for traffic in Flanders Belgium. Based on a review of the scientific information and the uncertainties and gaps in the scientific knowledge, it is felt that proximity to traffic is one of the most important and most certain factors that contributes to the health impact of particulate matter. When taking into account the uncertain information both on costs and benefits, is it possible to inform policy makers on the efficiency of reduction measures?

Methods

We use an updated emission inventory for primary PM2.5 and PM10 emissions in Flanders.

We develop emission scenarios for traffic for 2010 and calculate the cost of these scenarios.

On the other side benefits of reduced health impacts are valued according to the ExternE methodology, i.e. through a monetary valuation of externalities following an impact pathway approach.

Given different hypotheses for the health effects of particulate matter of traffic, a range of benefits can be derived for the emission scenarios.

Traffic emission scenarios

- S1: BAU scenario, including 10 ppm sulphur in fuel and public buses with CRT
- S2: CRT on 25% of heavy-duty vehicles and buses of type Euro 1, Euro 2 and Euro 3
- S3: Introducing 5% of biodiesel
- S4: Reducing the share of diesels to the level of 2002
- S5: Introducing a 30% share of hybrid diesels
- S6: Sum of S2 to S5

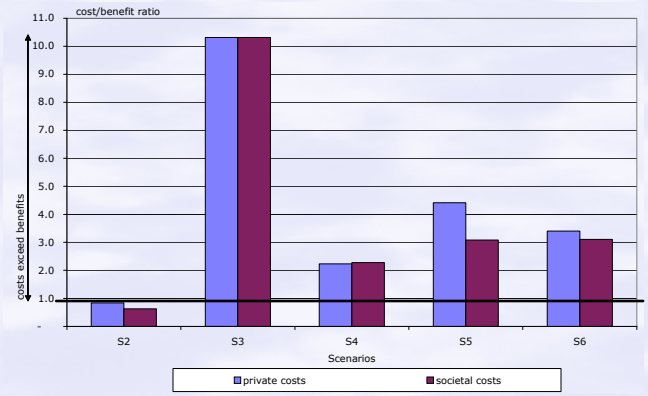

Figure 1: Cost benefit ratios for all scenarios, based on private or societal costs and external benefits

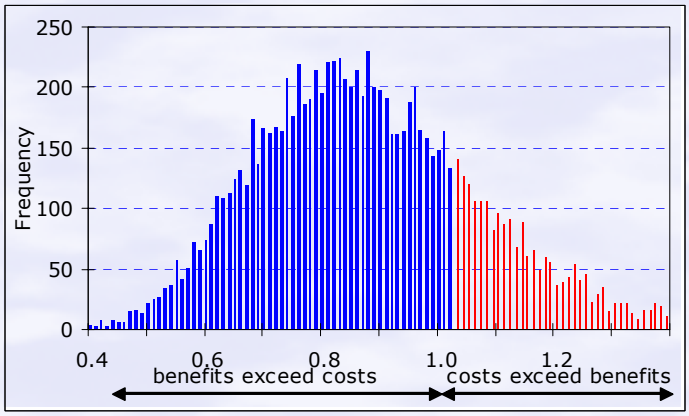

Figure 2: S2 Cost benefit ratio, based on private costs and external benefits, taking into account uncertain benefits

Results

Speeding up extra measures to reduce PM is only beneficial in case of CRT for trucks (S2) (figure 1). Other scenarios cost more than the public health benefit.

Taking into account uncertainty of external costs for health effects of PM, benefits exceed costs with 78% certainty (figure 2).

When trying to achieve a cost/benefit ratio of 1 for S6, the external cost per tonne of PM2.5 emitted has to increase by a factor of 3.4. This is an unrealistic high external cost for PM.

Secondary benefits?	S2	S3	S4	S5	S6
through CO_2	-	-	-	+	0/-
through NO_x	0	-	0/+	+	0/+
through SO_2	0	0	0	0	0
through VOC	+	0	-	0	-

- = negative; + = positive; 0 = no effect.

Secondary benefits are either negative, reducing benefits or positive but too small to change the cost-benefit ratio

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Flemish environmental administration, Tritel and the JOULE program of the European Commission

VITO Flemish Institute for Technological Research

Air Pollution Modelling for Support to Policy on Health and Environmental Risks in Europe (APMoSPHERE).

David Briggs[1], Danielle Vienneau[1], Kees de Hoogh[1], Asbjorn Aaheim[2], Gerard Hoek[3], Chris Dore[4], Mike Petrakis[5], Gavin Shaddick[6]

AIRNET
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Rationale

The policy need:

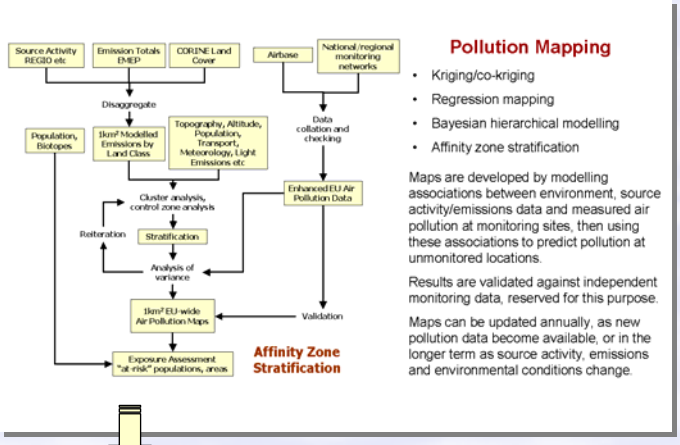
- Proper implementation and monitoring of policies to combat air pollution requires reliable, consistent and detailed information on emissions and air quality. Information is also needed to guide and monitor the effects of the many sectoral policies (e.g. transport, energy, tourism) that affect air quality.

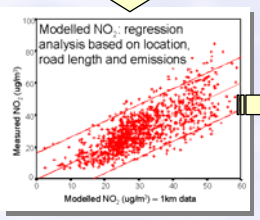
The science need:

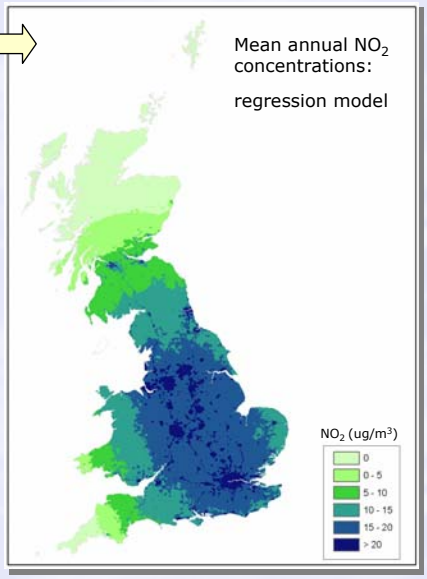
- Many of the impacts of air pollution on human health are complex and, at the local or individual level, small: what makes them important is the large populations at risk. In order to understand and assess these risks it is vital to monitor conditions over the whole EU and to analyse data from different areas in a consistent form.

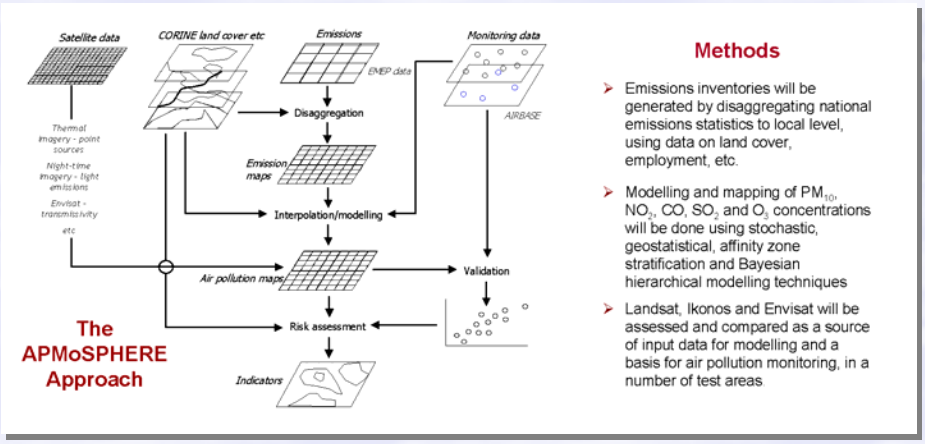
The information need:

- Current air pollution monitoring cannot provide all the data needed to support policy and science. New monitoring technologies, including Earth Observing satellites, offer great potential, but information is needed to help design and use these systems effectively, and methods are needed to extrapolate the monitoring data to areas which cannot be directly monitored.









Methods

- Emissions inventories will be generated by disaggregating national emissions statistics to local level, using data on land cover, employment, etc.
- Modelling and mapping of PM_{10}, NO_2, CO, SO_2 and O_3 concentrations will be done using stochastic, geostatistical, affinity zone stratification and Bayesian hierarchical modelling techniques
- Landsat, Ikonos and Envisat will be assessed and compared as a source of input data for modelling and a basis for air pollution monitoring, in a number of test areas

Air Pollution Information: the Problem

- Ground-level air pollution is highly variable, over both time (hours-days) and space (length-scales of 100 m +)
- Ground-based air pollution monitoring is expensive, so can never resolve these variations adequately, and cannot therefore meet the needs for information in full
- Commitment of member states to provide best available information has not yet been assured – so EU data sources are not optimal
- Existing sources of data on emissions are too coarse to provide reliable information for source attribution or air quality modelling
- EO data are little used – and technologies are not yet able to provide measurements of pollutants of interest

Objectives

- to produce a detailed (1km) inventory of atmospheric emissions (PM_{10}, SO_2, NO_x, CO) by major sector for the EU
- to develop and test a range of different GIS-based methods for mapping air pollution on the basis of these emissions estimates, in combination with other routinely available data sets (including air pollution monitoring data)
- to assess the capability of new generation satellites as a basis for air pollution modelling and monitoring at regional and local scales
- using these various methods and data sets to generate detailed (1km) and updatable maps of air pollution
- Based on the results, to develop a set of policy-related indicators on potential ecological, population exposures and health risks by intersecting pollution maps with population and land cover data
- to provide an assessment of the air pollution situation in the EU, and implications for future air quality monitoring and policy

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Imperial College London

APMoSPHERE
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ASSESSING AND ADAPTING TO THE HEALTH EFFECTS OF CLIMATE CHANGE IN CANADA

P. Berry, Health Canada

Aerosol number concentration measurements in five European cities during HEAPSS

P. Aalto, P. Paatero, M. Kulmala, K. Hämeri, F. Forastiere, G. Cattani, A. Marconi, J. Cyrys, S. Von Klot, K. Zetzsche, A. Peters, L. Bouso, G. Castaño, J. A. Palacio, J. Sunyer, T. Lanki, J. Pekkanen, B. Sjöval, N. Berglind, T. Bellander, F. Nyberg

AIRNET
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Objectives

Epidemiological research during the last decade has indicated that exposure to air pollution at the levels presently measured in European urban environments is associated with an increase in mortality and with a variety of health conditions, including emergency room visits and hospital admissions for respiratory and cardiovascular diseases. Particulate matter (PM) appears to be the air pollutant most consistently associated with adverse health outcomes. Although the toxicological mechanism has not been established, the small size fraction of ambient aerosols, measured as PM10 (particles with aerodynamic diameter less than 10 μm) or PM2.5 (less than 2.5 μm), rather than the larger particles, is considered to be responsible for most of the health effects. The number of concentrations of ultrafine particles (0.01 to 0.1 μm) are hypothesized to be of particular concern (1) .

The main objective of the HEAPSS-project (Health Effects of Air Pollution on Susceptible Subpopulations) is to quantify the risk of hospitalisation and of death due to air pollution, in particular airborne ultrafine particles, in individuals with coronary heart disease. This comprises the collection of a database of cross-European data on ultrafine particles.

Methods

The study is performed in five European cities - Augsburg (Germany; 0.45 million inhabitants), Barcelona (Spain; 1.5 million inhabitants), Helsinki (Finland; 0.5 million inhabitants), Rome (Italy; 2.7 million inhabitants), Stockholm (Sweden; 1 million inhabitants) - chosen so as to include a variety of geographical conditions and air pollution characteristics.

The measurement sites inside the cities are selected so that the concentrations measured represent an city-wide background concentrations (urban background). Three of the sites, Barcelona, Stockholm and Helsinki are elevated sites, Augsburg site is located inside a park. The site in Rome is located by a fairly busy road and could be classified as a kerbside site. Stockholm and Rome have also secondary sites. In Stockholm the secondary site is located inside a street canyon and in Rome inside a park (urban background).

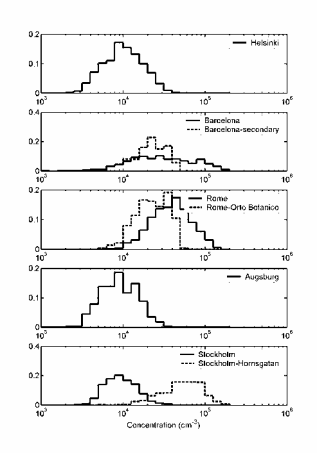
Ultrafine particle number concentrations are measured with a condensation particle counter TSI-3022 (TSI-Incorporated) (2). The sample is drawn to the instrument with a flow rate around 1.5 LPM through a Stainless Steel tube with an inner diameter of four millimeters. The tube length was tried to be kept as short as possible, less than four meters. Later on at some sites sample flow drier was added to the sample line to prevent water accumulation inside the instrument.

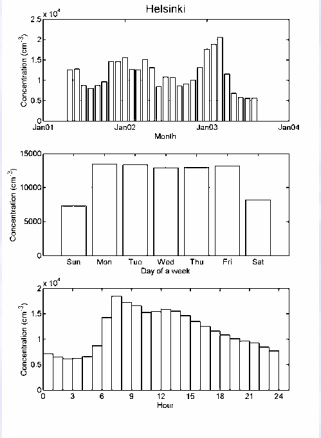
Results

Measurements at all five sites started during May 2001 and have continued since then.

The top table on the right column present the yearly averages of the particle number concentration in 1/cm^3 from all the five sites. Data is not yet fully analyzed and some poor quality data might appear. Average concentrations are highest in the southern cities especially during the winter. The three northest cities have equally low concentration. Fairly strong annual variation in the concentration is evident especially in Rome and Barcelona. Daily variation is strongest in Barcelona and Helsinki which both are influenced by the sea with cleaner marine air.

Year	Helsinki	Stockholm	Augsburg	Barcelona	Rome
2001	11530± 8921	10194± 6008	11535± 6694	65674± 62511	41682± 32765
2002	11680± 8921	10164± 6406	9402± 7439	46431± 51159	49994± 39297
2003	11530± 11780	10614± 7983		22091± 24731	39389± 30377





The northern cities, Stockholm and Helsinki are also effected by the strong meteorological inversions especially during the spring, which prevent the particles produced inside the city to be mixed to cleaner free tropospheric air.

The secondary urban background site of Rome is cleaner than the kerbside site. The Stockholm street canyon site has almost ten times higher daily mean concentration than the urban background site. Secondary site of lower concentrations was in operation also in Barcelona.

The lower left graph presents the histograms of particle concentrations. Barcelona has the highest variation in concentration.

The lower right graph shows as an example variation of monthly mean, weekday dependency and hourly dependency of particle concentration in Helsinki. Highest concentrations are observed during the winter months. All the working days have almost the same average concentration. Lowest concentrations are observed on Sundays. During one day highest concentrations are observed during the morning rush hours before the break-up of nocturnal boundary layer.

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SAPALDIA 2

Swiss Cohort Study on Air Pollution and Lung Diseases in Adults

Methods, Participation and Air Pollution Development

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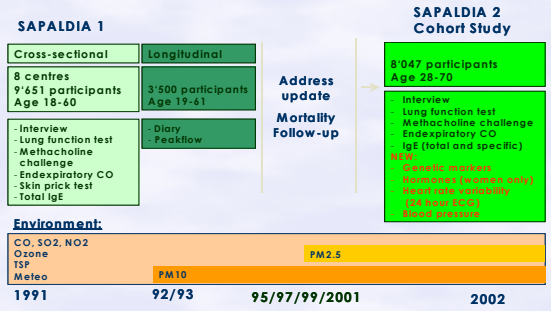
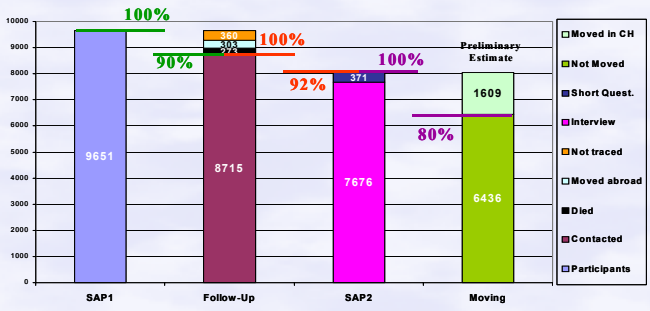
Major Aims of SAPALDIA 2

- To investigate the long term health effects (pulmonary, cardiovascular) of ambient air pollution
- To assess the 11-year course of respiratory and atopic symptoms and diseases
- To assess cardiovascular parameters (heart rate variability) in a subsample of particpants age >50
- To investigate associations between individual, long-term air pollution exposure estimates and i) decline of pulmonary function; ii) course of respiratory and atopic diseases/status and iii) heart rate variability

Study Design

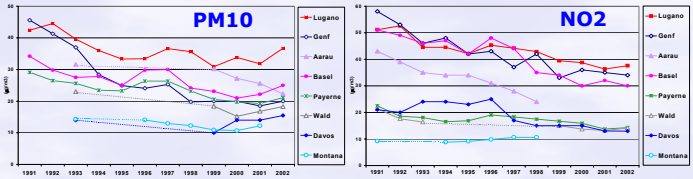


Participation and Moving Patterns



Air Pollution Exposure Assessment

11-Year Course of Air Pollutants

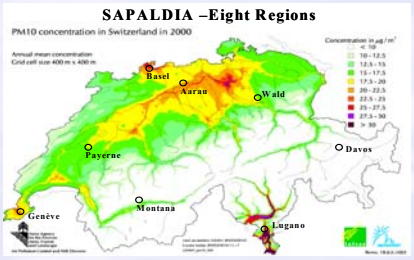


Traffic-Related Exposures: Black Smoke

- On PM10 and PM2.5-Filters
- 11 Monitoring stations (1-3 per region)
- Annual averages 2000-2002

Individual Exposure Assignment

- Annual GIS-Maps 1991-2002 for NO2 and PM10
- Based on emissions (household, traffic, off-road, industry)
- Gauss' dispersion models
- Validation with measured data



- Linkage with geo-coded addresses 1991-2002
- Calculation of individual 11-year AP-exposures

Conclusions

Moving and Participation Patterns

- Relatively stable study population
  - ✓ 90% could be contacted again
  - ✓ 20% moved within Switzerland
- High participation rate
  - ✓ 92% of those contacted and 83% of original cohort participated again

Individual Exposure Assignment

- Individual long term exposure estimates possible
  - ✓ Taking into account individual moving histories and course of air pollution levels 1991-2002
- Estimates for traffic exposures possible
  - ✓ Black Smoke and NO2

➔ Valid study basis for detecting long-term health effects of low-level air pollution and of traffic exposures

Prepared for AIRNET/NERAM 2003, November 5-7 2003, Rome (It)

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Acknowledgement:
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Urban air pollution and respiratory emergency visits at Paediatric Unit, Reggio Emilia, Italy.- Preliminary results

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Introduction and object

A large body of epidemiological research has focused on urban air pollution exposure and its consequences on respiratory function. Fewer studies have had the possibility to analyse less severe end points such as emergency room visits focusing on respiratory symptoms. Recurrence of respiratory symptoms are events certainly more numerous than admissions to hospital, they increase the demand for medical attention and they can be taken as indicators of people's life-quality worsening.

Reggio Emilia is a town of northern Italy with almost 150.000 inhabitants, with only one Hospital with a Paediatric Unit where all children emergency visits (E.V.) are fully registered; moreover an air pollution monitoring station network is working since 1989, and in winter 2001-2002 the alarm threshold has often been exceeded .

The aim of this study is to evaluate short term effects of urban air pollution levels on children E.V. due to respiratory symptoms.

Fig. 1: Reggio Emilia

Methods

Data of emergency visits

Yearly, the number of E.V. for all causes of Reggio Emilia inhabitants under 15 years of age is about 13.000.

The study has collected E.V. for respiratory symtoms during the period 03/01/2001 - 03/31/2002; children with more than 5 E.V. were not enrolled.

The E.V. were classified into 2 groups:

- allergenic –obstructive pathology (group ASTHMA)
- Other respiratory patologies (group OTHER).

Air pollution and weather variables

Reggio Emilia Department of the Agenzia Regionale Prevenzione Ambiente (ARPA) has collected air pollution monitoring data during the study period (Fig. 2).

The pollutants considered were: NO_2, SO_2, CO, TSP, PM 10, O_3, allergenic pollens.

Regional Metereological Service has collected weather variables data (temperature, humidity, precipitations).

Fig. 2: Reggio Emilia air pollution monitoring network

Statistical methods

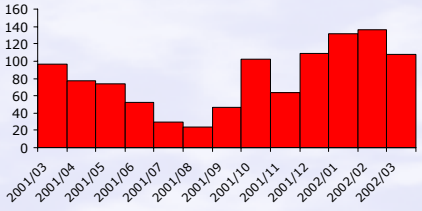
To analyse the correlation among E.V. and air pollution and weather variables, the statistical model GAM (generalized additive model) was performed. Non parametric smoother function, long temporaly trend and seasonality were considered into the model.

Results

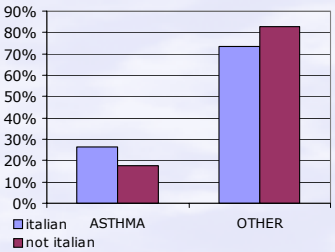
857 children
- 357 females → 444 E.V.
- 497 males → 611 E.V.

Children median age is 2 years.

797 E.V. (76%) were due to respiratory patologies of group OTHER and 254 to group ASTHMA (24%); only 26 E.V. (2,5%) were followed by admission to hospital.



Graf. 1: Number of E.V. for month



Graf. 2: Percentual of E.V. for nationality and group

Most of E.V. happen during the week-end and in the period from december to march (47,6%) (Graf. 1).

The 24,6% of E.V. is relative to "non italian children" (with non italian father).

The E.V. in the ASTHMA group are more frequent in italian children (26,5%) than in non italian (17,3%) (Graf. 2).

E.V. characteristics (time, day, month) and mean (number of E.V. per subject) are not different beetween the 2 groups.

The E.V. rate [1], stratified by nationality, is 43,2‰ among italian children and 144,8‰ among non italian.

	PM10 µg/m³	TSP µg/m³	SO2 µg/m³	NO2 µg/m³	CO mg/m³	O3 µg/m³	O3 summer µg/m³
Mean	51,2	60,4	9,3	49,0	1,4	68,6	107,5
D. S.	30,6	34,3	2,3	13,8	0,7	46,7	32,2
Min	5,0	3,7	4,6	21,6	0,4	4,5	30,9
Median	44,7	52,4	8,9	47,5	1,1	66,2	105,4
Max	196,8	272,9	20,9	107,5	4,6	200,5	200,5

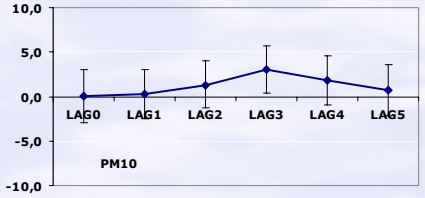
Tab. 1: Pollutant daily concentration

Mean PM10 concentration during the study period was 51,2 µg/m³ (air quality standard: 40 µg/m³ annual mean) (Tab. 1).

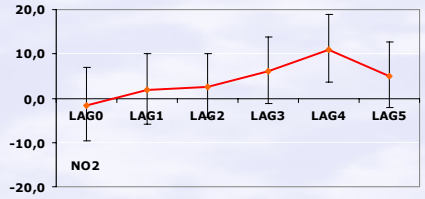
A significant association is observed beetween E.V. and some pollutant variables.

Particularly, the increase in the daily E.V. is associated with PM10 and NO_2 measurements (Graf. 3-4).

The percent increase in E.V relative risk, for 10 µg/m³ increase in PM10, is 3 (CI 95%: 0.4-5.7) (lag 3), while is 11 (IC 95%: 3.6-18.8) for an analogous change in NO_2 (lag4).



Graf. 3-4: Percentual change in E.V. relative risk associated with 10 µg/m³ increase of PM10 e NO_2 measurements.



Discussion and conclusion

The study shows a small but significant association beetween air pollution (PM10 e NO_2) and paediatric E.V. due to respiratory symtoms. This association is indipendent from the effect of temperature, humidity and pollen trend.

In contrast, no significant association is found for ozone and other pollution measurements.

Several studies have reported similar positive association.

[1] E.V. rate: E.V. number per 1.000 inhabitants in Reggio Emilia municipality on 12/31/2001 (non italian children: 1.871, italian children: 18.069)

Health impacts of ozone levels in Sweden -a national assessment

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[3] The Swedish National Institute for Public Health

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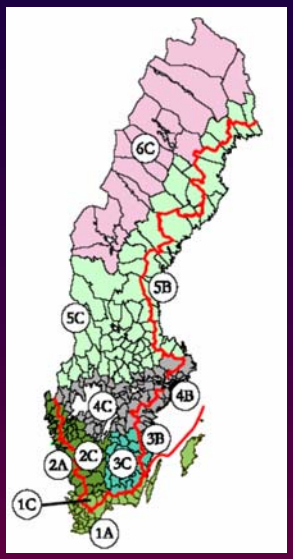
Aim

The aim of this study is to quantify the health impacts of ground level ozone in Sweden, and at the same time to indicate a possible method for a national estimate of the health impacts of certain types of air pollution.

Method

The Swedish population

(8.9 million) was divided into 29 sub populations for which time series of daily maximum 8-hour ozone averages during the year 2000 have been estimated. For seven large cities we obtained data from urban background measurements. For the rest of the country 22 series of ozone levels (11 for urban areas) were estimated from 6 rural EMEP stations.



Population data

were collected from Statistics Sweden. **Baseline data (2000) on all cause mortality and respiratory hospital admissions at county level were obtained from registers kept by The National Board of Health and Welfare.**

Calculations

were done using the AirQ tool developed by WHO/ECEH. As exposure-response coefficients we used an increase of 0.4% and 0.7% in mortality and admissions, respectively, per 10 µg/m3 increase in ozone.

Results

In total (Table 1) it is calculated that more than 1000 deaths per year are brought forward due to ozone levels above 60 µg/m³. Approximately 2000 respiratory hospital admissions per year are estimated due to ozone levels above 60 µg/m³. The cumulative number of excess cases can be quantified from any level for every sub population (Figure 1)

Tabel 1. Estimated yearly reduction in number of deaths and admissions by avoiding levels above 60 and 80 µg/m³, respectively.

	>60 µg/m³	>80 µg/m³
Fatalities	1121	434
Resp hosp adm	1808	700

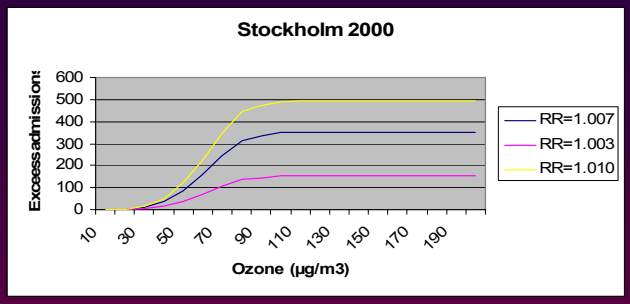
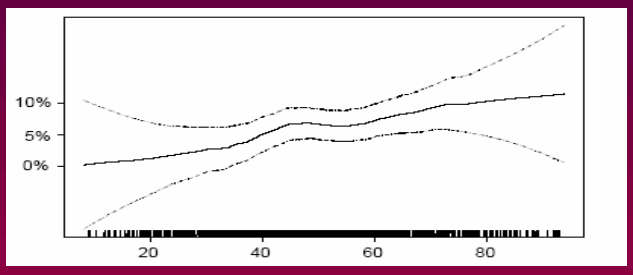


Figure 1. The cumulative number of respiratory admissions related to ozone exposure in Stockholm (1.2 million people) according to AirQ calculations.



Figur 2. The ozone induced increase (%) in respiratory hospital admissions in Stockholm (1997-1999) as a smooth function of the 8-hour maximum lag 01.

Conclusions and uncertainties

- **Ground level ozone is a major air pollution problem in Sweden, resulting in increased mortality and morbidity**
- **Swedish studies tend to show stronger ozone effects than typical, why the magnitude of the impact may be underestimated (Figure 2).**
- **The impact may be overestimated if a threshold exists, but the literature and Swedish data do not support the existence (Figure 2)**

Long-term Effects of Air Pollution & Mortality

The **Ge**rman **Co**hort on **Mo**rtality & Air Pollution in women (**GECOMO - Air**)

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Background

- Three American and one Dutch cohort study showed an association of long-term exposure to air pollution with mortality, in particular with cardiopulmonary mortality.
- In some studies, this association was stronger in men compared to women.

Objective

- To study the effect of ambient air pollution on mortality in a cohort of women.

Methods

Study design

- Cohort study

Study population

- Participants of several cross-sectional studies performed in North Rhine Westphalia from 1985 through 1994.
- N = 4752 Women aged 55 years (at recruitment) with German nationality.

Study region

- 7 cities in North Rhine-Westphalia:

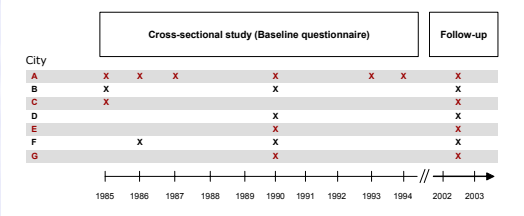
Germany

North Rhine-Westphalia

Study methods

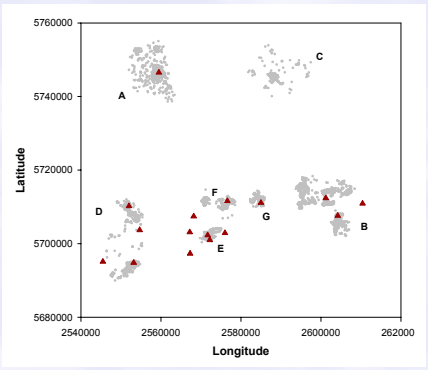
- Questionnaire data (cross-sectional studies = baseline)
- Follow-up (Figure 1)
  - Vital status & Residential history

Figure 1. Chronology of cross-sectional studies and follow–up.



- Exposure assessment using data from
  - air pollution monitoring sites (Figure 2)
  - Geographic information systems (GIS)

Figure 2. Location (Gauss-Krueger-Coordinates) of participants' addresses (grey dots) and air pollution monitoring sites (red triangles).



Statistical analysis

- Survival analysis using Cox's proportional hazards models with adjustment for potential confounding variables

Application of Results

- To provide information about the health impact of exposure to ambient air pollution in order to facilitate decision-making with regard to public health measures.

Collaborators

- GSF – National Research Center for Environment and Health, Institute of Epidemiolgy, Neuherberg, Germany: Ulrike Gehring, Joachim Heinrich, H.-Erich Wichmann
- Ludwig-Maximilians-University of Munich, Institute of Medical Data Management, Biometrics and Epidemiology, Chair of Epidemiology, Munich, Germany: Ulrike Gehring, H.-Erich Wichmann
- Environmental Health Research Institute (IUF) at the Heinrich-Heine-Universität Düsseldorf, Düsseldorf, Germany: Ursula Krämer, Dorothee Sugiri

The Study is funded by the Landesumweltamt North Rhine-Westphalia

Health impact assessment of PM_{10} exposures in the city of Caen, France.

Glorennec P., Monroux F. Ecole Nationale de la Santé Publique (National School of Public Health), Rennes, France.

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Context and objectives

The city of Caen is located in north-western France. Air pollution is mainly due to traffic sources. The aim of this work is to assess the public health impact of both short (with short term effects) and chronic (with long term effects) exposures to PM_{10}.

Methods

The standard World Health Organisation (WHO) methodology (1) for an Health Impact Assessment (HIA) has been used to calculate the attributable deaths and hospital admissions. Population exposure is estimated from PM_{10} (Particulate Matter <10 µm) concentrations collected by the local air quality measurement network : Aircom. The relative risks have been modelled by the exposure-risk functions established in epidemiologic studies carried out in the general population. The Aphea-2 (2-4) program, wich combines european time-series studies, have been used for short exposures effects, and a meta risk calculated (5) from cohort studies for long exposures effects. Health impact have been calculated with the EIS-PA software (6) to assess :

• for the short exposures : total impact with reference to the baseline level of 10 µg/m3, health gain for a daily 10 % reduction of pollution, health gain for a daily compliance to 40 µg/m3 (EU regulatory level for annual mean in 2005)

• for the chronic exposures : health gain for a 5 µg/m3 annual reduction.

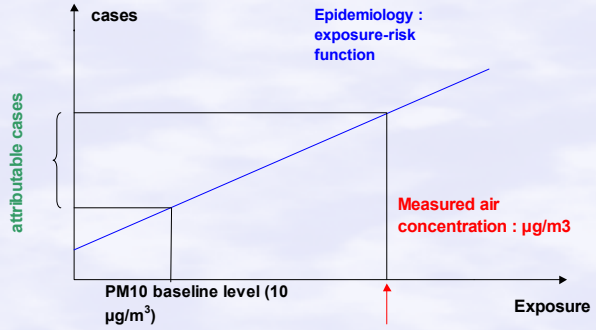


Fig 1. HIA principle

	PM10 (µg/m3)
Min	2
P 5	7
P 25	12
P 50	17
P 75	25
P 95	42
Max	87
Daily mean	19
Standard deviation	11,4
% missing values	1%

Table 1. PM 10 outdoor exposures, Caen 1998-2002.

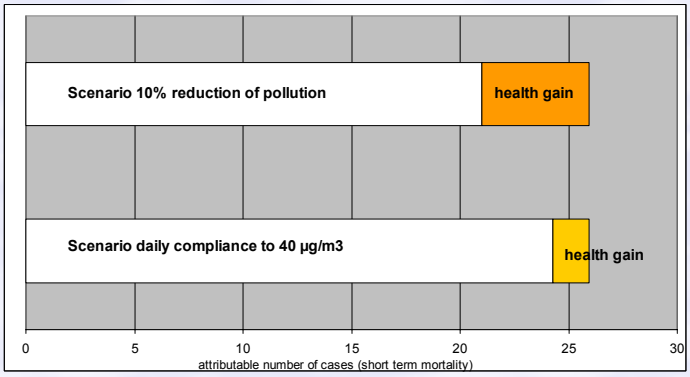
Findings

Table 2 : health impact of outdoor particulate air pollution. Caen 1998-2002.

	Health effect	Attributable cases (CI 95 %)
	Anticipated death	26 (17-35)
Short term	Respiratory hospital admissions	16 (11-23)
	Cardiovascular hospital admissions	27 (11-44)
Long term	Mortality	90 (54-126) *

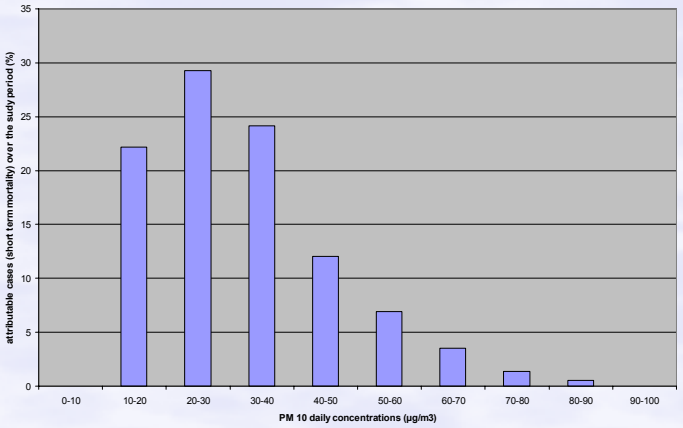
* for a 5 µg/m^3 reduction of the annual mean.

Fig 2. Health efficiency of particulate air pollution policy scenarios, Caen 1998-2002.



Reducing daily levels of pollution is more efficient than avoiding peaks. Health impact is mainly due to moderate, but frequent, levels of pollution.

Fig 3, Health impact vs exposure levels. Caen 1998-2000.



Discussion

Some major uncertainties and ways of reducing it :

• long term effects : validity in Europe, and accuracy of relative risks : to go ahead with European cohort studies

• hazard identification/health end points : major effects such as bronchitis and asthma attacks could not be assessed : to know the local baseline risks in France

Variability and its uses :

• geographic : when necessary (town planning), needs to be assessed with population time activity patterns inquiries and city wide PM10 measurements

• temporal : it has been used here to describe the respective contribution of polluted and 'normal' days.

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OXIDATIVE POTENTIAL OF ENVIRONMENTAL PARTICULATE MATTER FROM SITES WITH VARYING TRAFFIC DENSITY: CORRELATION BETWEEN ANTIOXIDANTS DEPLETION AND ARACHIDONIC ACID RELEASE

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INTRODUCTION and STUDY DESIGN

World-wide, epidemiological studies have consistently demonstrated an association between airborne concentrations of particulate matter (PM) and cardiovascular and pulmonary morbidity and mortality. The specific contribution of traffic PM to this effect is largely unknown. A European Union funded project (Health effects of particles from motor engine exhaust and ambient air pollution; HEPMEAP) was established to assess the toxicological potential of PM collected from various sites across Europe with established contrasts in traffic density. The project sought to test the hypothesis that the toxicity of ambient PM samples was related to their chemical composition. Thirty-two coarse (PM2.5-10) and fine (PM0.1-2.5) samples were collected from a variety of European metropolitan and rural areas using a high volume impactor and their oxidative activity assessed in a synthetic model of the respiratory tract lining (RTLF) fluid by determining their capacity to deplete ascorbate, urate and reduced glutathione (200μM of each) following a 4h incubation (37ºC, pH7.4) at a PM concentration of 50μg/ml.The PM2.5-10 and PM0.1-2.5 inflammatory response was evaluated by measuring the release of arachidonic acid (AA), tumour necrosis factor alpha and interleukin-6 from a monocytic/macrophagic cell line (RAW 264.7) following exposure to 20 and 60mg/cm^2. The AA release induced by the thirty two coarse samples at 60mg/cm^2 in RAW 264.7 cells supernatants was also shown to be strongly associated ($p<0.001$) with the loss of GSH ($r^2=0.61$) and ascorbate ($r^2=0.36$) from the synthetic RTLF. As the loss of these antioxidants is largely driven by the concentration of water leachable transition metals the impact of metal chelation on the macrophage AA response was investigated. Transition metals have been implicated in determining toxicity mainly through their ability to generate reactive oxygen species (ROS) (1). ROS generated through Fenton-like chemistry may activate mitogen-activated protein kinases (MAPK) cell signalling cascades (2), which in turn may phosphorylate and activate cytosolic phospholipase (3). Four samples eliciting the greatest AA release, according to our screening data and collected from four different sites (table 1) were studied. Increased availability of AA may increase prostaglandins and leukotrienes synthesis which may play a role in the changes in airway tone associated with the allergic response. In order to evaluate the role of PM iron content in inducing AA release samples were pre-treated with the membrane-impermeable iron-cupric metal chelator diethylenetriaminepentaacetic acid (DTPA). This chelator does not cause any cytotoxicity at the concentrations of 0.1 mM and 1mM used in the present study. Ferrous ion content was determined using the chromogenic chelator bathophenantroline disulphonate which forms a ferrous complex that absorbs strongly at 535nm. Total iron was determined after a pre-incubation of PM with ascorbate. Coarse particles at 60 μg/cm^2 but not at 20 μg/cm^2 showed a marked AA release which was significantly reduced with DTPA pre-treatment as shown in Fig.1. The effect of DTPA on fine PM-induced AA release was less clear. DTPA also induced a significant reduction in AA release induced by iron-rich residual oil fly ash (ROFA) particles suggesting a clear involvement of iron in the induction of A2 phospholipases in this model.

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RESULTS

Table 1. Iron Content

Samples Codes	Location	Time of the year	Iron Content μM/mg PM
10 & 13 (NL)	Sassenheim	December	19.1 +/- 0.89
18 & 19 (NL)	Amsterdam	March	58.8 +/- 0.89
24 & 25 (DL)	Ostbanhof	April	190.2 +/- 4.73
HIA (NL)	Dutch Tunnel		102.2 +/- 3.22

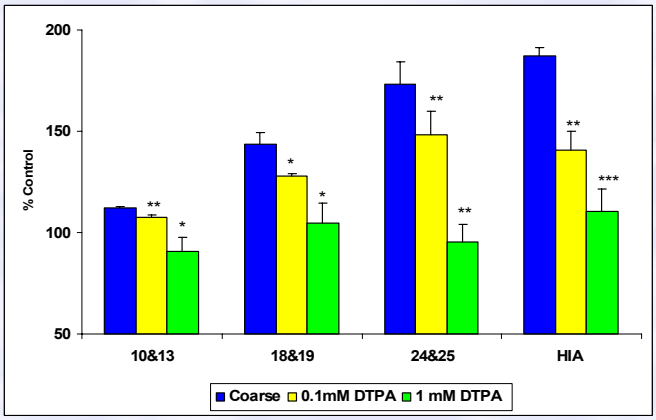

Fig. 1. Effect of coarse particles on [^{3}H]AA release in RAW 264.7 cells. Cells were pre-labelled with [^{3}H]AA and then incubated for 5h with the particles at 60 μg/cm^2 with and without DTPA. The radioactivity released by untreated cells was taken as 100%. Values are means of three experiments assayed in triplicate. The iron-chelator DTPA significantly (*p<0.05; **p<0.01; ***p<0.005) inhibited the particles-induced AA release.

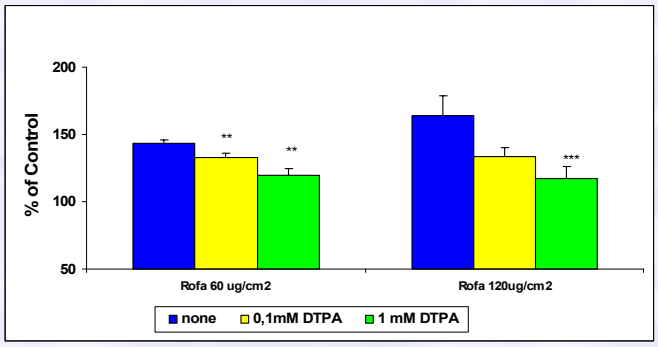

Fig. 2. Effect of DTPA on Rofa-induced [^{3}H]AA release in RAW 264.7 cells. Cells were prelabelled with [^{3}H]AA and then incubated for 5h with Rofa particles at 60 and 120 μg/cm^2 with and without DTPA. The radioactivity released by untreated cells was taken as 100%. Values are means of six experiments assayed in triplicate. DTPA significantly (** p <0.01; ***p<0.005) inhibited the Rofa-induced AA release.

Conclusions

These data indicate that (a) the oxidative potential of PM is strongly related to their capacity in induce AA release from macrophages and (b) that this relationship can be explained in terms of the bioavailable pool of iron in these PM samples. These relationships were only apparent in the coarse fraction. The absence of a clear cut association with PM0.1-2.5 may reflect differences in cellular uptake into the macrophages compared with coarse PM.

BITTERFELD Study

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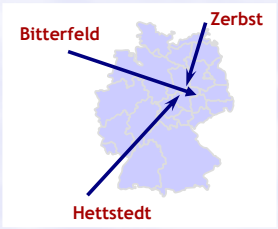
Background

- After German reunification in 1990, the East-German population rapidly adopted a modern "Western lifestyle".
- Some of these lifestyle factors may have affected the development of allergies.
- If environmental and lifestyle factors contributed to the higher prevalence of allergies among West Germans, an increase in prevalence of allergies among East Germans after German reunification would be expected.

Study area



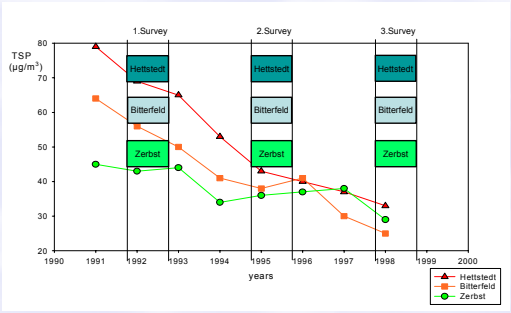
Objectives

Objectives

- To determine the impact of ambient air pollution on children's health.
- To study temporal changes in the prevalence of allergic and non-allergic diseases in East German children.
- To investigate determinants of asthma and allergic diseases in children.

Study design

- Three cross-sectional surveys: 1992-93, 1995-96, 1998-99



Study population & methods

Study population

- 2400-2800 children in each survey resulting in 7611 questionnaires from 5360 different children
- In each survey the study population consisted of 5 to 7 year-old school entrants, 8 to 10 year-old third graders and 11 to 14 year old sixth graders.

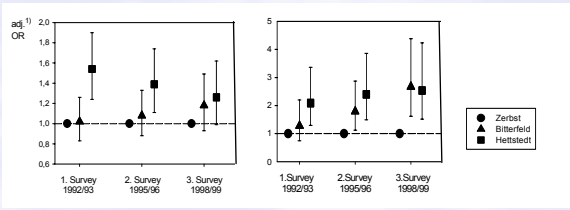
Study methods

- Questionnaire
- Serum specific IgE
- Heavy metal analyses in blood & urine
- Lung function testing

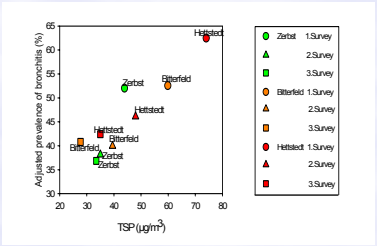
Results

- Children from the most polluted area (Hettstedt) showed poorer respiratory health (Heinrich et al. EHP 1999, Heinrich et al. AJRCCM 2002).

Bronchitis **Hay fever**



- Non-allergic respiratory morbidity improved along with improved air hygiene (Heinrich et al. Epidemiology 2003).



- Hettstedt ambient particles were associated with increased risk of allergic sensitization in children (Heinrich et al. EHP 1999) and influence the severity of allergic respiratory disease in mice (Gavett et al. EHP 2003).
- Numerous publications on determinants of asthma and allergies and temporal changes of several health outcomes (http://www.gsf.de/epi/en/index_ag_unm_epi_en.htm).

Collaborators

- GSF – National Research Institute of Environment and Health, Institute of Epidemiology, Neuherberg, Germany: Joachim Heinrich and H.-Erich Wichmann for the Bitterfeld study group
- Gesundheitsamt Bitterfeld, Kreiskrankenhaus Wolfen, Gesundheitsamt Hettstedt, Klinikum Mansfelder Land, Gesundheitsamt Zerbst
- Umweltbundesamt, Institut für Wasser-, Boden-, Lufthygiene.
- US-EPA-NHEERL, Chapel Hill, NC U.S.A.

The study is funded by the Umweltbundesamt (UBA)

Air pollution and hospitalisations for first myocardial infarction (MI) in the HEAPSS* cohort

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AIRNET
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* **HEAPSS: Health Effects of Air Pollution on Susceptible Subpopulations**

Aims

HEAPSS study aims to determine whether air pollution increases the risk of first acute myocardial infarction (MI) in general population, or the subsequent risk of cardiac events among patients who have survived their first MI. Of particular interest in the study are effects of ultrafine particles (particles <0.01 μm).

The associations between 1. MI and air pollution will be reported here.

Methods

HEAPSS cohort consists of patients hopitalised for their first acute MI during city-specific enrolment periods within the years 1992-2000 in 5 European cities. Patients were recruited either from hospital admission registries (Helsinki, Stockholm, Rome) or from MI registries (Augsburg, Barcelona). The two types of registries have different upper age limits (table 1).

Air pollution data was collected from existing networks of fixed outdoor monitors. Ultrafine particles were measured for almost two years in all cities using condensation particle counters (CPC). Daily concentration of ultrafine particles was estimated for the entire study period using measurements of gaseous air pollutants, particles, and meteorology.

Statistical analyses were accomplished using Poisson regression adjusted for the effects of meteorology on MI.

Results

There were almost 27 000 cases of acute myocardial infarction during the patient enrolment period (table 1). The bulk of the MI cases were found in the study centres where hospital admission records were utilized. Thus, results of these centers drive the pooled results.

Daily air pollution levels were higher in southern European cities than in the northern cities. Highest CO levels were found in Rome, and highest SO2 levels in Barcelona (table 1).

City-specific correlation coefficients between measured and estimated ultrafine particles concentrations ranged from 0.88 to 0.91.

Correlation coefficients (Barcelona not yet available) between ultrafine particles and CO ranged from 0.48 to 0.83, and between ultrafine particles and PM10 from 0.06 to 0.53 (PM10, particles <10 μm; in Augsburg PM10 estimated using other variables).

Increase of 10 000 ultrafine particles/cm^3 was associated with 1.1 % increase in the risk of 1. MI (table 2). Increase of 1 mg/m^3 carbon monoxide was associated with 2.8% increased risk of MI. PM10 or SO2 were not consistently associated with the incidence of MI, nor were O_3 or NO_2 (data not shown).

Table 1. Number of hospitalised first myocardial infarction cases, and daily air pollution levels (median)

	Augsburg	Barcelona	Helsinki	Rome	Stockholm
Hospitalized cases	1694	1467	5148	8799	9555
% men	75	78	52	67	58
Age limits	35-74	35-79	35-	35-	35-
Air pollution					
Ultrafine particles [1]	12400	-	13600	46000	11800
PM_{10} (μg/m^3)	43.5 [1]	57.4 [1]	21.0	48.5	12.5
CO (mg/m^3)	0.9	0.9	0.4	2.3	0.4
SO_2 (μg/m^3)	4.6	11	3.3	4.6	2.9

[1] *Estimated using other variables*

Table 2. Associations between daily air pollution levels and incidence of first myocardial infarction. Pooled results of 5 cities (4 in ultrafine particles)

	Exposure	Rate ratio	Confidence limits
Ultrafine particles	**Current day**	**1.013**	**1.000-1.026**
	Previous day	1.001[1]	0.959-1.045
CO	**Current day**	**1.028**	**1.002-1.055**
	Previous day	1.010	0.984-1.038
PM10	Current day	1.003	0.995-1.011
	Previous day	1.002	0.994-1.010
SO2	Current day	1.000	0.996-1.004
	Previous day	1.003	0.999-1.006

[1] *city-specific estimates heterogenous*
-rate ratios calculated for a change of 10000/ 1 cm3 in UF, 1 mg/m3 in CO, 10 μg/m3 in PM10, 1 μg/ m3 in SO2
-in bold results that are significant (p< 0.05)

Discussion

Although air pollution effects differed between individual cities, according to our pooled results acute exposure to CO and ultrafine particles increases the risk of hospitalisation for a new myocardial infarction. Both of these pollutants are linked to local traffic emissions. Thus, the study suggests that by regulating vehicle exhaust emissions detrimental effects associated with air pollution could be diminished. As no associations were observed with PM10, routine monitoring of finer PM fractions (ultrafine particles/PM2.5) should be considered.

Current study provides a database of cross-European ultrafine particle measurements. There's still a lack of long measurement series of both ultrafine particles and PM2.5. In this study, retrospective estimation of ultrafine particles was tried for the first time - successfully. However, as other pollutants were used to estimate ultrafine particles, the possibilities to separate health effects between ultrafine particles and other pollutants are limited. Estimation of PM2.5 using the collected airport visibility data is under consideration.

Hospital admissions represent only a fraction of all MIs in the population since acute fatal events are often not hospitalized. Associations between air pollution and incidence of all MIs will be analysed in 3 study centres where appropriate mortality data is available. In all 5 centres, personal characteristics possibly modifying the effects of air pollution are being evaluated.

HEAPSS study was funded by European Union (QLK4-CT-2000-0708)

Studies of Air Pollution and Health in Denmark

Steffen Loft, Institute of Public Health, University of Copenhagen, DenmarK; s.loft@pubhealth.ku.dk

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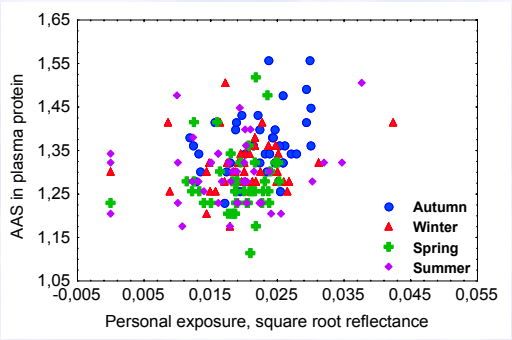
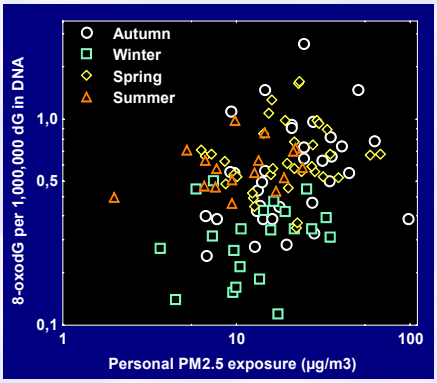
Biomarkers and Air samplers for Assessment of Exposure and Effects of Urban Air Pollution – BIOAIRPEX

In this project determinants and biomarkers of internal and biologically effective dose and susceptibility of personal exposure to particulate matter (PM2.5) and NOx are assessed in 50 healthy subjects in each of the 4 seasons over one year. Individual samplers and samplers inside and outside the subjects' residence as well as in urban background and busy streets are compared. Biomarkers include a battery of genotoxicity markers, including oxidative DNA damage by comet assay and 8-oxodG and PAH DNA adducts as well as markers of oxidative stress and hematology. Susceptibility markers include a battery of metabolism enzymes. So far, data under publication include significant correlations between biomarkers of oxidative stress and personal exposure to PM2.5 black smoke.

The study is continuing with measurement of personal exposure to ultrafine particles and relations to biomarkers of exposure in specified traffic scenarios. The generated data are also used for validation of modeling of exposure based on geographic information systems.



Relationship between personal exposure to black smoke measured as reflectance of PM2.5 filter and oxidative stress in plasma assessed as oxidation of amino acids (AAS: aminoadipic semialdehyde)



Relationship between personal exposure to PM2.5 by mass on filter and oxidative damage to DNA in lymphocytes in terms of guanine oxidation ((-oxodG: 8-oxodeoxyguanosine per mill. Deoxyguanosine)

Hertel O, Solvang Jensen S, Andersen HV, Palmgren F, Wåhlin P, Skov H, Nielsen IV, Sørensen M, Loft S, Raaschou-Nielsen O. Human exposure to traffic pollution. Experience from Danish studies. Pure Appl Chem 73 137-145, 2001

Sørensen M, Dragsted LO, Hertel O, Knudsen LE, Loft S. Personal $PM_{2.5}$ exposure and markers of oxidative stress in blood, Environ Health Perspectives, in press

Ongoing epidemiological studies of health effects of air pollution in Denmark

The studies take advantage of a number of large ongoing population studies to address the most pertinent aspects of the health effects related to exposure to traffic generated air pollution, especially particles. Modelling is the main tool for exposure assessment. Overall coordination is by Steffen Loft.

1. *Self reported symptoms*

Annoyance and symptoms in relation to exposure to air pollution from road traffic will be addressed in 6.000 people studied in the National Health Survey 2000. Principal investigator Lis Keiding, lk@si-folkesundhed.dk

2. *Acute cardiopulmonary mortality*

The risk of acute morbidity and mortality of cardiovascular and pulmonary diseases requiring admission to hospital during 2 years will be addressed in relation to traffic generated air pollution at the residence, workplace and commuter route. Case-cross-over, nested case-control and time-series designs will be applied in four large population studies in Copenhagen providing a population at risk of approximately 25.000. Principal investigator: Steffen Loft, S.Loft@pubhealth.ku.dk

3. *Birth weigth*

Intrauterine growth retardation and perinatal/infant mortality will be addressed in relation to modelled air pollution concentrations at the residence of 15.000 pregnant women from the Danish National Birth Cohort. Principal investigator: Ole Raaschou-Nielsen, ole@cancer.dk

4. *Asthma in children*

Exposure to fine particles (PM2.5) as a causal factor in asthma development will be addressed by 3 one-week measurements in the bedrooms of each of 400 infants at high risk of asthma development (Copenhagen Prospective Study of Atopy in Childhood). Principal investigator: Ole Raaschou-Nielsen, ole@cancer.dk

Daily recorded sympotoms over the first three years of life will be correlated to daily air pollution levels including sizefractionated particles measured in urban background and estimated by modelling at the residence. Principal investigator: Steffen Loft, S.Loft@pubhealth.ku.dk

5. *Asthma and COPD in adults*

The development of self-reported asthma and bronchitis in relation to exposure to traffic generated air pollution at the small area level will be studied in a health survey conducted on 3000 persons from the Glostrup Study. Principal investigator: Torben Sigsgaard, ts@mil.aau.dk.

6. *Lung cancer.*

In a case-cohort design 800 lung cancer cases are identified among participants in a number of Danish population cohorts. A sub-sample of another 800 participants is selected from the cohorts. Residential history (from 1960) is traced by use of population registries and interviews with participants or next-of-kin. Information on work place addresses and transportation between home and work are also collected by interviews. Cumulative exposure to air pollution is estimated by modelling. Relative risks for lung cancer in association with air pollution will be estimated by regression techniques and adjusted for smoking habits and occupational exposures. For part of the study population (250 cases, 250 controls) PAH-DNA adducts will be measured in white blood cells stored in a biobank as a marker of biological relevant exposure andgenetic polymorphisms in metabolism and DNA repair enzymes will be analysed as possible effect modificators Principal investigators: Steffen Loft, S.Loft@pubhealth.ku.dk and Ole Raaschou-Nielsen, ole@cancer.dk.

PHEWE

Assessment and Prevention of acute Health Effects of Weather conditions in Europe

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BACKGROUND

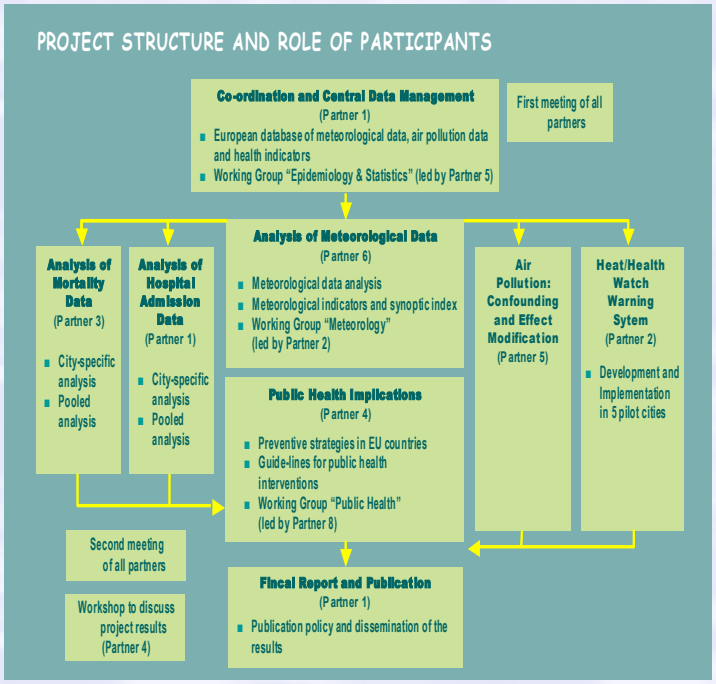
Epidemiological studies indicate that exposure to extreme weather conditions (e.g. warm and cold temperatures) are associated with an increase in mortality for cardiovascular and respiratory diseases. A large scale study in areas with different meteorological conditions applying a standardised methodology will produce estimates of the effects of environmental variables on health for Europe and contribute to policy development and public health decision making.

AIMS

The objective of this study is to evaluate the association between weather conditions and acute health effects (mortality and hospital admissions) in 17 European cities, both during hot and cold season. The study will experiment the use of Heat/Health Watch-Warning Systems (HHWWS) in European cities, and provide information for public health policies on prevention and implementation of adaptive actions.

SPECIFIC OBJECTIVES

- ✓ Create a European database of meteorological variables, health indicators, and air pollution data.
- ✓ Compare and classify specific weather conditions in different European cities according to selected meteorological indicators.
- ✓ Perform city-specific and pooled analysis on the effects of weather on daily mortality and daily hospital admissions, using a time-series approach (seasonal analysis).
- ✓ Analyse the role of air pollution as potential effect-modifier on weather related health effects.
- ✓ Implement and evaluate Heat/Health Watch Warning systems (HHWWS) to predict and to alert the population of oppressive weather conditions related to adverse health effects in 5 pilot cities (Rome, Paris, Barcelona, London, Budapest).
- ✓ Develop a framework of public health intervention strategies to minimise adverse health effects in the participating cities and in Europe.
- ✓ Disseminate the results to various groups: policy makers, health and environmental scientists, national, regional, and local governments, and the general public.



PROGRESS DURING THE FIRST YEAR

(Project duration: August 1st, 2002 – July 31st, 2005)

- ✓ The European database was completed. Health indicators collected were: mortality and hospital admission data for cardiovascular, cerebrovascular, and respiratory causes (Table 1). Meteorological data (air temperature, dew point temperature, wind speed, wind direction, sea level pressure, total cloud cover, solar radiation, precipitation, visibility) and air pollution data (SO_2, TSP or BS, PM_{10}, NO_2, O_3, CO) were collected for the same periods.
- ✓ The first all-participants meeting was held in Rome in March 2003.
- ✓ Three working groups "Epidemiology & Statistics", "Meteorology", and "Public Health" were established. Each of these WGs will discuss the specific methodological aspects and will follow the progress of the different work packages.
- ✓ The analysis of the meteorological variables for each city was performed.
- ✓ The protocol for the analysis of mortality and hospital admission data was defined and the city-specific analysis is in progress.
- ✓ In three of the five HHWWS pilot cities a collaborative network was created at a local level.
- ✓ Up-dated information on the progress of the project can be found on the website www.epiroma.it

Table 1. Database

city	mortality data	hospital admission data
Athens	1992-1996	
Barcelona	1991-2000	1994-1997
Bucharest	1992-1996	
Budapest	1992-2001	1997-2000
Cracow	1990-1996	
Dublin	1990-2000	
Helsinki	1988-2000	
Ljubljana	1997-1999	1997-2001
London	1992-2000	1992-2000

city	mortality data	hospital admission data
Milan	1990-2001	1990-2001
Paris	1991-1998	1991-2000
Prague	1992-2001	
Rome	1992-2000	1995-2000
Stockholm	1990-2000	1990-2000
Turin	1990-1999	1995-2000
Valencia	1995-2000	1996-2000
Zurich	1990-1996	1990-1996

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Dept. of Astrogeophysics, University of Joannina, Greece, P. Kassomenos.
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Dipartimento di Epidemiologia ASL RME

Quality of life and management of living resources
Key action n. 4: Environment and health
EU contract QLK4-CT-2001-00152
EC contact: Callum Searle - callum.searle@cec.eu.int

APHEIS: Influence of traffic-related PM_{10} emissions on public health in 1999

Hans-Guido Mücke and Sylvia Medina on behalf of the Apheis network

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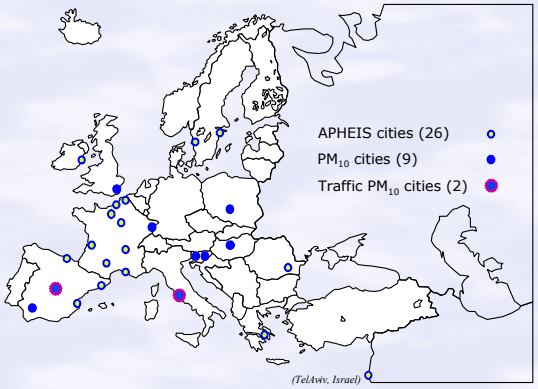
Background and objective

Apheis (Air Pollution and Health: A European Information System) has been designed to provide decision makers, environmental professionals and the public of Europe with a comprehensive, up-to-date and easy-to-use information source on air pollution and health.

For health impact assessment air quality measurements of urban background monitoring sites should be considered (defined by the Apheis guideline on exposure assessment).

Because the location of PM_{10} monitoring sites of Madrid and Rome were characterised as traffic-related the opportunity is given to analyse the influence of PM_{10} emissions of vehicular traffic, and consequently their impact on public health, in comparison to urban background PM_{10} measurements of seven other Apheis cities.



PM_{10} concentration findings

In 1999, the PM_{10} mean concentration ranged from 20 to 45 µg/m³. The specific traffic-related analyses show first, that PM_{10} of traffic related-sites of Madrid and Rome is in the upper concentration level (37 and 43 µg/m³) such as the PM_{10} urban background data of cities in eastern Europe (Celje, Cracow and Ljubljana), and they have approximately the same number of days per year with exceeded 24-hour limit values. Second, a comparison within western European cities show that PM_{10} urban background levels of e.g. London and Strasbourg (~22 µg/m³) are about two times lower than those of Madrid and Rome.

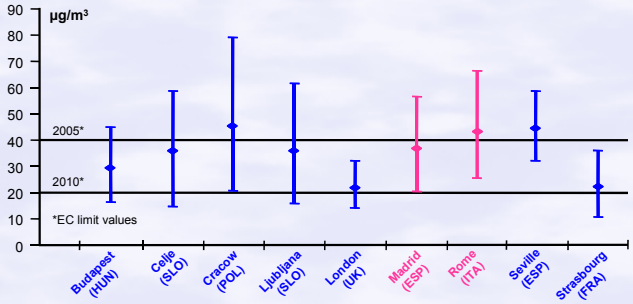


Fig. 1: Annual mean, 10th and 90th percentiles of PM_{10} concentration

Furthermore, the PM_{10} concentration in Seville is 44 µg/m³, as high as in Rome. Although PM_{10} was measured at urban background monitoring sites, the city of Seville identified vehicular transport as the main cause for PM_{10} air pollution.

Health impact assessment findings

In terms of health impact assessment, the result with the biggest potential health benefit was found for a reduction of the annual PM_{10} mean value to a level of 20 µg/m³ (PM_{10} EC limit value for 2010). It shows that in Madrid and Rome, the corresponding reductions in the number of deaths per 100,000 inhabitants per year attributable to chronic effects of PM_{10} would range between 50 and 70, comparable to Budapest and Ljubljana, and are more than ten times higher than in other western European cities (e.g., London and Strasbourg: 5 deaths/100,000 inhabitants).

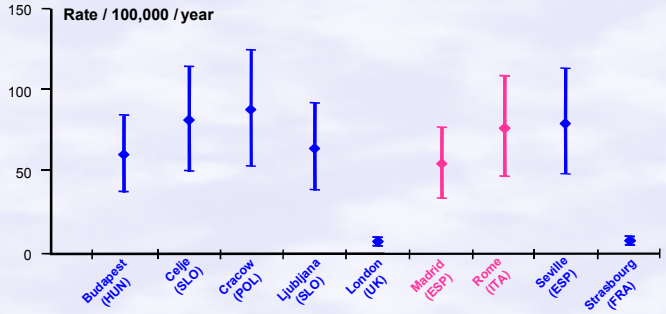


Fig. 2: Potential benefits of reducing annual mean values of PM_{10} to a level of 20 µg/m³ (2010 limit values for PM_{10}) - Number of deaths per 100,000 inhabitants (95 % confidence limits) attributable to the chronic effects of PM_{10}

Conclusions

In 1999, western European cities with a significant influence of vehicular traffic show higher PM_{10} concentrations and an increased mortality rate due to acute and chronic effects of particles compared to people in eastern Europe who are exposed to urban background level.

By chance, a discrepancy within the characterization of air monitoring stations was identified, which could result in data misinterpretations. Within Apheis data compilation air monitoring meta data of Madrid and Rome was reported as traffic-related. The PM_{10} concentration in Seville was comparable to Rome, but PM_{10} monitoring sites were characterised as urban background. The city of Seville identified vehicular traffic as the main cause of PM_{10} air pollution.

As this study shows, to avoid misinterpretations in the comparison of air quality data and in the assessment of their health impact a harmonised and standardised air monitoring siting criteria should be used within Europe, as fixed at the EC Council Decision (97/101/EC of 27 January 1997) to establish a reciprocal exchange of information and data from networks and individual stations measuring ambient air pollution within Member States (revised by Commission Decision 2001/752/EC of 17 October 2001).

[Acknowledgement: Apheis is supported by European Commission DG Health and Consumer Protection programme on pollution-related diseases and participating institutions in 26 cities of 12 European countries.]

For further information visit www.apheis.org

Umwelt Bundes Amt
für Mensch und Umwelt
Federal Environmental Agency

INSTITUT DE VEILLE SANITAIRE

Acute effects of particulate matter on respiratory diseases in Austria.

Manfred NEUBERGER, MD, Michael G. SCHIMEK, DPhil PhD, Hanns Moshammer, MD, Helger HAUCK, PhD

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Air Quality in Austria

Improvements with gaseous pollutants – problematic fine particles

During the 1980s Austria achieved the highest SO_2 reduction among the signatory states of the Helsinki Protocol[1], however, other sources of fine particulate matter like diesel traffic increased. At present annual averages of 20-30µg/m³ are measured for PM_{10} and 15-20µg/m³ for $PM_{2.5}$.

The Austrian Project on Health Effects of Particulates-AUPHEP in 1999-2001 investigated short term effects of particulate matter (PM) on lung function, morbidity and mortality in Vienna, Linz, Graz and a rural control area.

site/ period	PM_1 µg m^{-3}		$PM_{2.5}$ µg m^{-3}		PM_{10} µg m^{-3}		TSP µg m^{-3}		CPC cm^{-3}	
	mean	max	mean	max	mean	max	mean	max	mean	max
VIENNA										
year	**14.9**	**75.1**	**18.6**	**96.4**	**26.5**	**104.6**	**36.1**	**153.5**	**26234**	**62835**
winter	15.5	75.1	19.5	96.4	26.9	104.6	37.5	153.5	31119	62835
summer	14.2	32.6	17.5	42.7	26.1	58.3	34.6	75.6	20555	41749
LINZ										
year	**14.7**	**48.3**	**18.8**	**76.4**	**29.9**	**127.4**	**42.9**	**193.8**	**23387**	**82520**
winter	17.6	48.3	22.3	76.4	35.3	127.4	50.8	193.8	30650	82520
summer	11.9	34.4	15.2	46.4	24.5	60.6	35.4	99.8	16154	47517
GRAZ										
year	**17.5**	**70.4**	**21.1**	**81.2**	**31.0**	**114.1**	**38.4**	**142.1**	**22540**	**54075**
winter	20.9	70.4	26.7	81.2	38.3	114.1	45.7	142.1	29300	54075
summer	14.1	40.9	15.5	43.4	24.3	59.1	31.1	74.6	16374	39289

Hospital admissions

Table 1: *Percent increase in respiratory hospital admissions per 10 µg PM/m³*

Vienna	PM_1		$PM_{2.5}$		PM_{10}	
age	male	female	male	female	male	female
1-6		13.23		8.01		9.31
7-14				6.27		5.53
65+			5.47	5.60	4.22 x	
rural						
1-6						
7-14						
65+			9.89	10.53		4.22

x) estimate for lag 2 (for lag 10 it was 4.23)

Increased admissions for respiratory diseases in Vienna and the rural control area

Applying semi-parametric generalised additive models daily hospital admissions in Vienna for respiratory diseases (asthma and COPD) were found related to high concentrations of PM. For $PM_{2.5}$ this relationship became significant in elderly Viennese (age 65 years and older) after a lag of 2 days in males and a lag of 3 days in females. A second peak of respiratory morbidity at lag 10 reached significance for PM_{10} in elderly men only. In the rural area only the second peak was found, reaching significance for $PM_{2.5}$ at lag 10 for males and at lag 11 for females. Besides this increase of hospital admissions in the elderly which has also been observed in other studies at higher concentrations of PM, we detected in Vienna an increase of respiratory admissions in children which was mainly due to asthma and significant for girls only. In pre-school children this increase reached significance after a lag of 4 days and in school children after a lag of 2 days. **Table 1** shows significant estimates for the increase in hospital admissions encoded ICD 490-496 at hospital discharge.

Independent of the PM effects we found an immediate increase of respiratory admissions in children of both sexes after an increase in NO_2, which is related to motor traffic.

Mortality

Increased cardiovascular and respiratory mortality in Viennese elderly

Based on several severe air pollution episodes, a temporal correlation between high concentrations of particulate matter (PM) and SO_2 pollution and acute increases in respiratory and cardiopulmonary mortality had been established in Vienna for the 1970's. After air pollution had decreased in Austria in the 1980's - as documented by data on SO_2, and total suspended particles (TSP) - no such associations between day-to-day changes of SO_2 and TSP with mortality have been documented any more.

But ongoing analysis of the AUPHEP data show that at least in Vienna there is still a significant association of cardiovascular mortality with particulate matter (PM_{10}, $PM_{2,5}$, PM_1).

Lung function of children

Acute effects in school children

At the elementary school nearest to the monitoring station in Linz 164 children participated in medical examinations for signs of acute infection and in lung function tests performed every second day on about 20 of them, so that each of the 69 girls and 95 boys had spirometry about every fortnight from October 2000 to May 2001. Forced oscillatory resistance as well as FVC, FEV1, MEF_{25}, MEF_{50}, MEF_{75}, and PEF were obtained [2]. In all analyses, each child served as its own control, because we used the difference of the individual lung function parameter to the arithmetic mean of this parameter of all tests during the school year as the outcome variable.

The mixed model showed significant results for the carbonaceous fraction of $PM_{2.5}$, indicating bronchial obstruction on days when particulate total carbon (TC) was increased. The lung function impairment was not significantly related to elemental carbon (EC), but to organic carbon in $PM_{2.5}$, which had a higher variable concentration across the period of observation. Total mass of fine particulate matter (PM_1 as well as $PM_{2.5}$) showed a tendency to increase bronchial obstruction. The coefficients were also negative for PM_{10} (-1.07), particle number concentration measured by condensation particle counter (-0.89), and the gases NO_2 (-0.56) and SO_2 (-0.13).

Key messages

In spite of improvements in Austrian air quality both particulate matter and NO_2 still pose a threat to the health of children and elderly people.

Sources for these air pollutants such as road traffic and in particular diesel powered vehicles (which today make up more than 50% of Austrian cars due to low prices for diesel fuels) have to be reduced.

Acknowledgements:

This study was supported by the Clean Air Commission of the Austrian Academy of Science and conducted within the AUPHEP project. We thank the Municipality of Linz (Departments of Health and Environment) and the AUPHEP team for their support.

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2. **Moshammer H, Neuberger M (2003). The active surface of suspended particles as a predictor of lung function and pulmonary symptoms in Austrian school children. Atmospheric Environment 37: 1737-1744**

Lung inflammation in children with short-term exposure to ambient ozone: evidence of a threshold

Marc Nickmilder[1], Sylviane Carbonnelle[1], Claire de Burbure[1] and Alfred Bernard[1]

AIRNET
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Introduction

Ozone is a major pollutant produced by sunlight-driven reactions involving mainly nitrogen oxides and volatile organic compounds (Fig. 1). During summertime, ground levels of ozone may peak at values exceeding 200 µg/m^3 in central or southern regions of Europe and in many other areas of the world. This gas can produce a variety of pulmonary effects, including a decrement in lung function, inflammatory reactions, an increase in epithelial permeability and airway resistance, and asthma exacerbation.

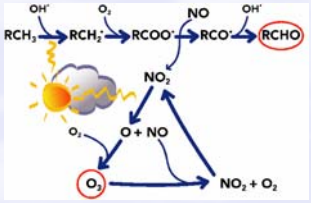

Figure 1

During the last few years, one specific test has emerged and been developed to evaluate the extent of inflammation or damage in the lung: the measurement of exhaled NO, a very sensitive marker of airways inflammation and oxidative stress.

The aim of this study was to assess the inflammatory effect of ambient O_3 in healthy children using nitric oxide in exhaled air (eNO) as a non invasive test.

Methods

• Participation of six groups of children (n = 11-15; age: 6.5- 15 years) attending summer camps in rural southern Belgium in 2002 (Fig. 2).

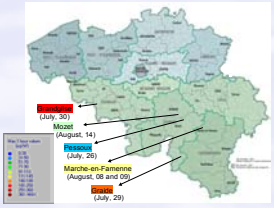

Figure 2

• Ambient O_3 concentrations were continuously monitored and ranged from 48 to 221 µg/m³, 1 hour maximal mean (Fig. 3).

• Other air pollutants were monitored hourly.

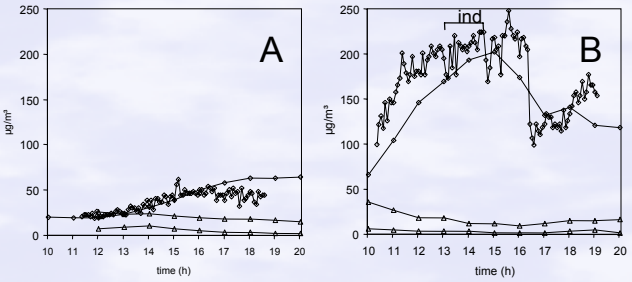

Figure 3: Diurnal variations of O_3 (dark diamonds for in-house measurements and white diamonds for station measurements), NO (dark triangles) and NO_2 (white triangles).
Graph A: day with the lowest ozone levels
Graph B: day with the highest levels

• Children remained outdoors doing various recreational activities.

• Lung function tests and eNO (NIOX®, Aerocrine, Sweden) were measured twice, in the morning (10:00-12:00 am) and evening (18:00-20:00 pm).

• Age, sex, height and weight of each subject were recorded during the morning test.

Results

• Whilst lung function tests didn't show any particularly consistent decrease, a highly significant increase in eNO was found from an ambient 1-hour O_3 level of 167 µg/m³ (Fig. 4 & 5).

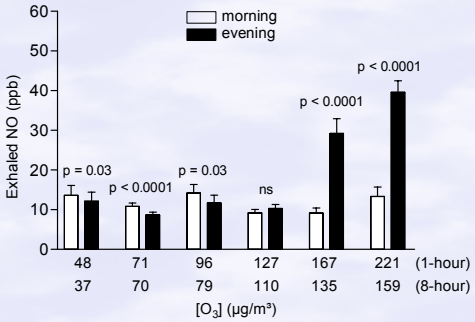

Figure 4

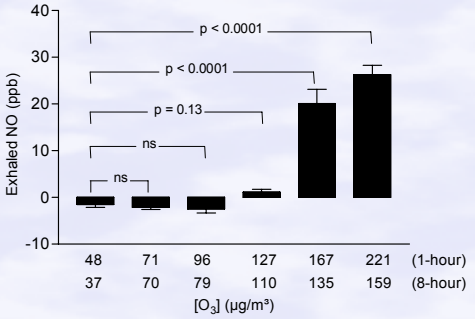

Figure 5

• A multivariate analysis did not reveal any influence of the age, sex and BMI of the children.

Conclusion

•The exact threshold for this O_3-induced increase in eNO might lie around 130 µg/m³ since from this level onwards the significant diurnal decrease of eNO observed in control camps was abolished.

•The observations suggest that ambient ozone produces early inflammatory changes in the airways of children from levels slightly below current air quality standards.

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HELI-O-S

An Epidemiological Assessment of Individuals Biological Uptake of Particulate Air Pollution and Related Health Effects

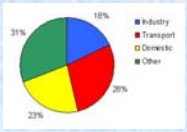
Susan O' Connell & Ian P Matthews

INTRODUCTION

The effects of air pollution on health have long been of interest to scientists, doctors and policy makers. Most studies however concentrate on the results exposure to air pollution has on those whose health is already compromised in some way so as to make them more susceptible to the effects i.e. those with cardiovascular or respiratory illnesses. While there is a lot of evidence supporting the view that exposure to air pollution and in particular to the particulate fraction does indeed have an effect on health of those more susceptible sections of the population there is very little in the way of effects at an individual level or of effects on the healthy section of the population.

This study aims to show that residential exposure to air particulates has an effect at an individual level and that even healthy individuals are at risk from the effects.

»Vehicular emissions are the most common source of particulates in urban areas.



»~25% of particulate pollution in the UK being the result of road transport.

»This figure has been shown to rise to 75-80% on high pollution days.

Criteria for inclusion in the study

»Participants must be living in one or other of exposed or unexposed areas

»Participants to be male of ages 50 70

»Non smokers for at least three years

»No history of chest or heart disease, diabetes and/or arthritis or other prolonged illnesses

»To have been in good health for at least six weeks prior to taking part in the study

Objectives

»It is hypothesised that on completion of the analysis a significant difference will be seen between the levels of ultrafines in the exposed and unexposed areas and possibly between indoor and outdoor levels.

»It is also hypothesised that there will be an effect on various blood parameters in participants from the exposed areas with a lesser or possibly no effect on those in unexposed areas and that these effects may be attributable to the prolonged residential exposure to particulate air pollution.

Home Visits are arranged with all eligible participants

Blood and urine samples are taken from partcipants and tested for various factors that may indicate a reaction as a result of prolonged exposure to high levels of particulates

Environmental Measurements are taken both inside and outside the home using a TSI Ultrafine Particle Counter called a P-Trak™

The P-Trak™ is a small handheld device with the ability to measure particles in the range of 0.02 to greater than 1 micrometer and has a concentration range of 0 to $5x10^5$ particles/cm^3.

Materials & Methods

An exposure comparison study has been designed to show whether healthy individuals residing in proximity to high volumes of traffic have biological uptake of pollutants and suffer effects such as inflammatory responses, increased oxidative stress and changes in blood properties such as coagulation and viscosity.

CARDIFF

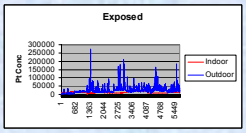
Exposed: Households located on a busy roadside with high traffic flows *eg households on main access roads near the city centre*

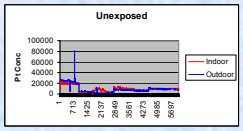
Unexposed: Households located in quiet areas away from busy roads and with little passing traffic ***eg households in small housing estates on the outskirts of the city***

Exposed

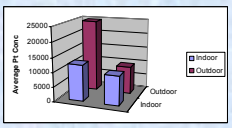
Unexposed

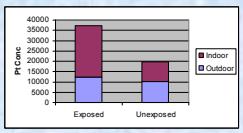
Results





Indoor and Outdoor comparisons of the levels of ultrafines





Comparisons of the average levels of ultrafines in exposed and unexposed

Conclusions

Data Collection is still in it's early stages and as such, although some preliminary investigation appears to suggest a clear difference in levels of ultrafines between exposed and unexposed areas, it is not possible to make draw any significant or meaningful conclusions from the data at this point

Dept Epidemiology Statistics and Public Health
University of Wales College of Medicine

References

Sources of uncertainty in calculating mortality and morbidity attributable to air pollution

Talar Sahsuvaroglu and Michael Jerrett
McMaster University, Hamilton, Canada

AIRNET
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NERAM
Network for Environmental Risk Assessment and Management

Introduction

This work evolved from a request from Clean Air Hamilton, a multi-stakeholder group tasked with advising Hamilton City Council on air pollution policy. We were asked to update the estimated mortality and morbidity attributable to air pollution in the City.

By conducting an in-depth study in one location, our aims were:

- to illustrate the sensitivity of health effect estimates to a wide range of possible underlying assumptions.
- to give policymakers a better sense of the assumptions and estimations that underlie estimates of mortality and morbidity attributable to ambient air pollution exposure.
- to highlight some of the differences in expectations between policymakers and scientific research.

Methods

- Dose-response relationships were derived based on pooled and averages estimates published in the scientific literature from 1997 to 2001. Estimates were applied to local air pollution and hospital admissions for the years 1995-1999, and mortality for 1995-1997.
- A programming limitation in the generalized additive models (GAM) revealed that risk estimates may have been overestimated by as much as 42%. Adjustments were applied to study data to account for this overestimation.
- Health effect estimates are normally compared to a zero pollution level, considered by many to be practically unattainable. Estimates were thus calculated using a baseline of the lowest quintile of measured pollution values, as mean-min 20%.
- Local estimates derived from Hamilton-specific models were also conducted. Additional sensitivity analyses were based on pooled random effects models and from chronic studies from other jurisdictions.

Figure 1. Location map of Hamilton, Canada

Equations used for calculations

$$H = B * \Delta H\% * P$$

where:
H = annual health outcome
B = base number of outcomes per year
ΔH% = percent change in health outcome per unit increase of pollutant
P = annual pollution average

Sample Calculation

$$HO = 3730 \frac{\text{deaths}}{\text{year}} \times 0.076 \frac{\text{deaths}}{\mu g/m3} \text{ per } 100 \text{ deaths} \times 27.9 \ \mu g/m3 = 79.09 \frac{\text{deaths}}{\text{year}}$$

Results and Discussion

- Sensitivity of estimates to choice of model can be seen as mortality and morbidity estimates can range by 278 annual deaths, 563 respiratory admissions and over 1600 cardiovascular admissions in one year.
- Mortality increased from 298 to 374 annual deaths, respiratory admissions increased from 144 to 607, while cardiovascular admissions increased from 257 to 2000, using more recent literature findings and local health and pollution averages.
- Combining both the 42% GAM adjustment and 20% baseline adjustment decreased the mortality estimate to **96** deaths, and morbidity estimates to **139** respiratory deaths and **479** cardiovascular admissions (Figure 2).
- Estimates based solely on Hamilton-specific data resulted in higher mortality counts than pooled data (**96** vs. **90**). Chronic exposure calculations resulted in **232** annual deaths due to fine particulate pollution (Table 1).

Figure 2. Differences in mortality and morbidity estimates according to model utilized

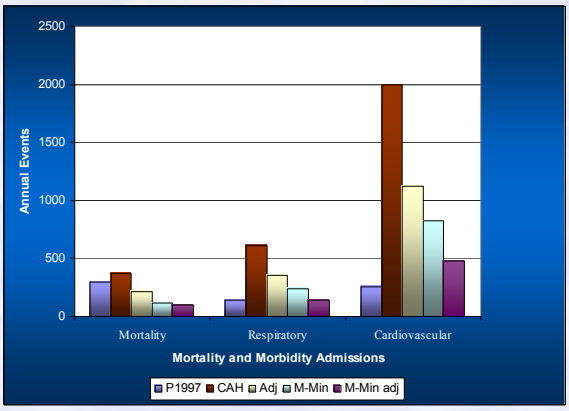

Table 1. Summary and Comparison of Mortality Counts Estimated for Different Models, Based on 1997 Hamilton Pollution Values

Pollutant	NT mortality (average incidences/year) average of estimates							
	P1997	CAH	Adj	M-min	M-min adj	Pooled	Hamilton	Chronic
PM_{10}	97	73	43	24	14	31		
$PM_{2.5}$		110	64					232
CoH							256	
SO_2	16	53	31	27	16	22	73	
NO_2	81	134	78	46	27	14	108	
CO	3	10	6	6	3	0	5	
O_3	102	105	61	62	36	23	122	
Total	298	374	217	119	96	90		232
Total **		411	238				564	

P1997 = HAQI report, Pengelly 1997
CAH = Current reanalysis for City of Hamilton
Adj = Adjusted value of CAH, for overestimate of 42%
M-min = Estimate calculated for pollution values of mean – min (lower quintile) for 1997
Total ** = Totals calculated with CoH as particulate measure instead of PM_{10}

Conclusions

- The wide range of estimates resulting from the different models substantiates our aim of highlighting the impact of scientific uncertainties and model assumptions.
- Contrary to advice from the academic literature, policymakers were interested in total numbers of deaths and admissions from all pollutants.
- While we compensated for potential overestimates by using only multi-pollutant models, we still believe that the summation of all air pollutants inflates mortality and morbidity admissions.
- Conversely, the total burden of illness due to air pollution includes other important health effects that were not into account in these analyses, such as asthma exacerbation, that may lead to underestimates of morbidity.

Future directions

Based on our analysis and experience with advising policymakers, we conclude with three suggestions for future research.
1. An effort should be made to reach a consensus on standardized reporting methods
2. An immediate need exists for widely accepted health accounting conventions.
3. Many areas of air pollution research require considerable work before complete estimates can be ascribed. Priority areas include studies on the chronic health effects of air pollution , multi-pollutant studies, and on health outcomes such as asthma

Acknowledgments:
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Health data was made available by the Central West Health Planning Information Network. Pollution data was supplied by the Ontario Ministry of Environment.

Urban air pollution and emergency room visits for respiratory complaints

M.A.Vigotti, F.Chiaverini, P.Biagiola, G.Rossi

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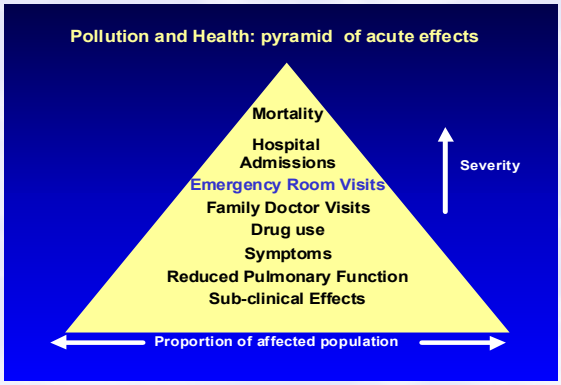
Background

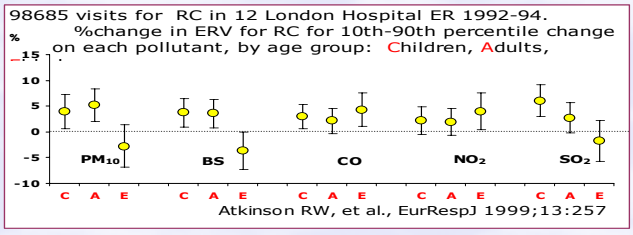
Several studies indicate that exposure to NO_2 and PM, homes proximity to roadsides/motorways, exposure to high rates of road traffic particularly to truck traffic increases the risk of respiratory symptoms.
High levels of air pollution may start and/or aggravate respiratory symptoms and may result in an urgent visit to an Emergency Room (ER)

ER visits are more sensitive indicator of acute respiratory effects of short-term elevation in air pollution since only the more serious cases seen in the ER are likely to be hospitalized. ER are not restrictedby bed availability.
Therefore among patients seen in an ER the clinical spectrum of respiratory illnesses is probably greater.



Some previous evidences

In 741 children 7-11 years old living in non-urban towns (Netherlands 1987-90) respiratory symptoms reported by either children or parents were correlated with a reduced respiratory function ($FVC, FEV_{1,0}$, PEF, FEF_{25-75}) but were not associated with air-pollution
(Hoek G, et al. Int. J. Epidemiol. 1999 28: 293-299)

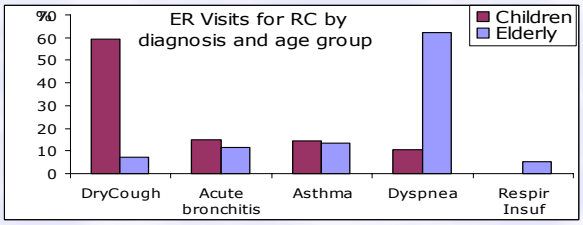


In Sao Paulo do Brazil in 1986 prevalence of respiratory symptoms among children of low-income families was much higher in areas with medium-high pollution levels, but was similar to all other children in the low pollution area (Ribeiro H. Soc Sci Med. 1989;29(8):959-64)

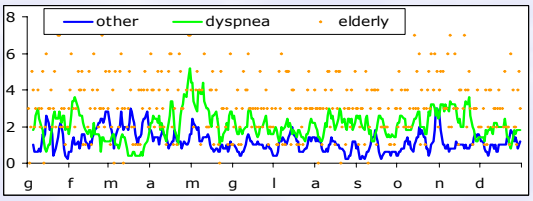
Twelve years later in 1998 in the area where both PM and SO_2 levels decreased, there was a reduction in the prevalence of respiratory symptoms
(Ribeiro H et al.Soc Sci Med. 2003;57(11):2013-22)

Urban air Pollution and ER visits for respiratory complaints, Pisa, Italy, 2000

Pollutants and meteo: PM_{10}, NO_2 , CO, temperature,relative humidity, total rain
Health data: 1724 visits (533 for children,1191 for elderly) collected and coded by medical personnel.
758 visits for dyspnea in elderly were excluded
Methods: Poisson autoregressive models allowing for overdispersion were used to model the outcomes. Generalised Additive Models for studying non linear relationships between confounders and morbidity.Up to 5 days lags were investigated



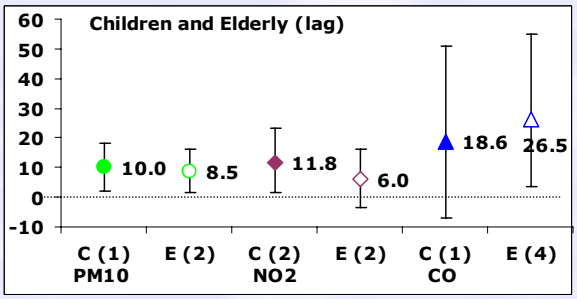
Time trend of daily ER visits for elderly: 5 days moving average and absolute values



Distributions of daily values, Pisa 2000

Pollutant		unit	mean	s.d.	Min	Median	Max
PM_{10}	24h	$\mu g/m^3$	35.4	15.8	9.5	31.6	100.1
NO_2	24h	$\mu g/m^3$	45.6	11.0	21.3	44.8	74.0
CO	24h	mg/m^3	1.5	0.7	0.3	1.4	3.5
Meteo							
Temp	24h	°C	15.8	6.1	1.1	15.6	27.0
Rel.Hum	24h	mm/Hg	82.7	11.1	42.8	83.6	99.6
Max Rain	24h	mm	2.2	7.1	0.0	0.0	90.6
E.R.visit							
Children	24h	num.	1.4	1.3	0.0	1.0	6.0
Elderly	24h	num.	1.2	1.1	0.0	1.0	6.0

% change of ER visits for RC for an increase of 10 $\mu g/m^3$ of PM_{10} or NO_2 and 1mg/ m^3 of CO, Pisa 2000



Results confirm also in Pisa that an increase in daily levels of urban air pollution is associated, in children and in elderly, with a decrease in quality of respiratory health that require a medical attention

Is there a threshold for associations between ozone concentrations and health outcomes?? A review.

Dr Heather Walton, Air Pollution Unit, UK Department of Health

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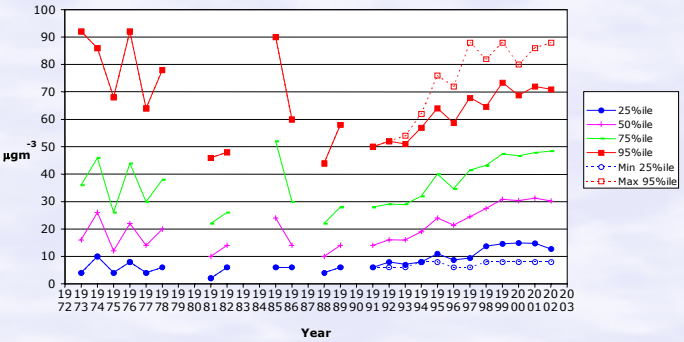
Introduction

Background mean concentrations of ozone are rising in urban areas of the UK (Figure 1), although peaks are declining. This trend is predicted to continue as a result of global warming and reductions in nitric oxide emissions from vehicles. To what degree is this a matter for public health concern? If levels are increasing but are always below a threshold for health effects, then no health impact would be expected. Is there sufficient evidence for a threshold to depart from a precautionary assumption of linearity? Thresholds could be indicated by a lack of an association in seasons or places with low ozone concentrations or on days with low ozone concentrations within individual studies. The literature was assessed for information on this, looking in particular for good contrasts in ozone levels and clear separation of effects from other pollutants.

Figure 1 Running 8-hour mean ozone concentrations in London



Methods

Studies on the St George's Air Pollution Epidemiology Database for 8 hour, 1 hour and 24 hour average ozone were obtained and searched for information on seasonal differences, maximum ozone concentrations and descriptions of the shape of the dose-response relationships. The use of multi-pollutant models when addressing these aspects was also checked. Outcomes covered were all cause, respiratory and cardiovascular mortality and respiratory and cardiovascular admissions, concentrating mainly on all ages associations. Panel studies on lung function and respiratory symptoms were also covered except for seasonal differences as most were summer only.

Results

Season Many studies show greater associations in the summer but only 12 studies gave coefficients by season and specified seasonal ozone concentrations. Several of these divided the year into just two 6 month periods which limited the contrast in ozone concentrations. In the example below (Table 1), there is extensive overlap in the ozone concentrations yet there is still a marked difference in coefficient by season. This might be due to different patterns of confounding by other pollutants in summer and winter. Only 3 studies[1-3] adjusted for other pollutants and only one of these[1] included seasonal maximum ozone levels. This study found no association in winter when levels were below 33 ppb 24 hour average.

Table 1 Seasonal differences in Brisbane, Australia

Season	Mean ozone (8 hour) (range)	Outcome	Estimate % per 5ppb (95% CI)
Summer Oct - Mar	20 ppb (3-63)	All-cause mortality	1.48 (0.49-2.48)
		Respiratory mortality	1.14 (-2.65-5.07)
		Cardiovascular mortality	1.19 (-0.58-2.99)
Winter Apr-Sep	16 ppb (2-57)	All-cause mortality	0.65 (-0.72-2.04)
		Respiratory mortality	0.51 (-2.41-3.51)
		Cardiovascular mortality	0.73 (-1.81-3.33)

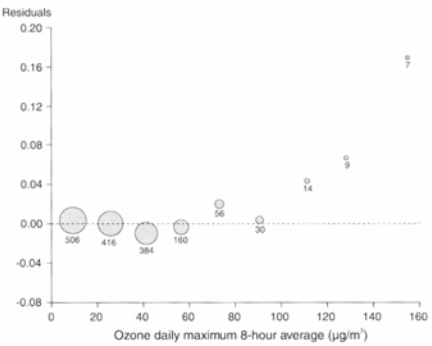
Source: Simpson *et al*; 1997 Arch. Env. Health 52:442-454

Geographical differences There are several studies[4-9] which have found positive and statistically significant associations stable to adjustment for other pollutants in places where maximum ozone levels were only 60 to 80 ppb 8 hour average. Positive significant adjusted associations with asthma symptoms have been found in Paris[10] with a maximum ozone level of 40 ppb 8 hour average. Ozone was not associated with respiratory symptoms in non-asthmatics in studies with Northern European ozone levels[11-14] except during vigorous exercise[15]. Plotting of study coefficients against maximum ozone concentration in the study was not pursued. Interpretation is difficult as linear coefficients may be imposed on data with thresholds in the individual studies.

Results (cont.)

Shapes of relationships There were apparent thresholds in 13 studies of mortality or hospital admissions but only 1 of these studies[16] adjusted for other pollutants. 7 studies suggested linear associations of which two[1-2] adjusted for other pollutants. Adjustment for other pollutants is important as it may affect the shape of the relationship. For example in the figure below[17], days to the left are more likely to be winter days and days to the right are more likely to be summer days. Ozone may be correlated with particles negatively in winter and positively in summer. This makes interpretation of the shape difficult. There were also very few panel studies[18-20] that examined the shape of relationships with adjustment for other pollutants.

Figure 6.1 **Relationship between peak 8 hour average ozone concentrations and hospital admissions plotted as residuals[1]**



[1] In this figure the area of the circles is proportional to the data available at the given concentrations. The number of 8 hour periods of given concentration is shown by the figures adjacent to the circles

Messages for policy makers

- Seasonal differences and shapes of relationships may appear to suggest thresholds but may be due to different patterns of confounding by other pollutants.
- Only a small number of studies have adjusted for other pollutants when addressing these aspects and some of these suggest linear relationships.
- Positive significant associations have been found below 40 - 80 ppb 8 hour average
- There is insufficient evidence at present to depart from an assumption of linearity
- Linear associations can be used to estimate the health impact of policies to alleviate the rise in ozone

The opinions expressed represent those of the author and not the Department of Health. References 1. Moolgavkar et al (1995) 2. Hoek et al (1997) 3. Wong et al (1999) 4. Simpson et al (1997) 5. Anderson et al (1996) 6. Wong et al (2002) 7. Bremner et al (1999) 8. Petroeschevsky et al (2001) 9. Ponce de Leon et al (1996) 10. Desqueyroux et al (2002) 11. Hoek et al (1999) 12. Declercq et al (2000) 13. Hoek et al (1995) 14. Ward et al (2002) 15. Brunekreef et al (1994) 16. Fairley et al (2003) 17. COMEAP (1998) 18. Higgins et al (1990) 19. Higgins et al (1995) 20. Schwartz et al (1994) Full references available on request. Further details www.doh.gov.uk/comeap/index.htm under Current Issues. Acknowledgements Tony Bush NETCEN (Fig 1); Professor Ross Anderson's team; COMEAP.

HEALTH EFFECTS OF LONG-TERM EXPOSURE TO AIR POLLUTANTS IN SCOTLAND

Christina Yap[1], Iain J Beverland[1], Raymond M Agius[2], David Hole[3], Chris Robertson[1], Geoff Cohen, Deborah Henderson[1], George Morris[4], Mathew R Heal[5], Gerry Fowkes[5], Robert Elton[5]

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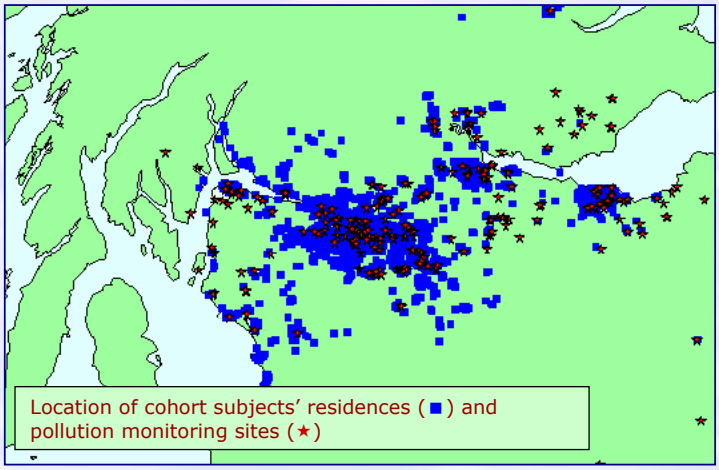
1. Introduction

- It is increasingly recognised that health impacts of air pollution may depend largely on the effects of long-term exposures
- Interacting effects of long and short-term exposures are poorly understood
- Current epidemiological methods are limited by poor exposure classification
- Scotland has very high nationally reported mortality rates for both coronary heart disease & lung cancer. Correspondingly epidemiologists have already established cohorts in urban areas with detailed baseline data on risk factors
- Many subjects reside in areas with detailed long-term records for black smoke and SO_2

Historical air pollution (smog from coal fires) in Edinburgh



Location of cohort subjects' residences (■) and pollution monitoring sites (★)

2. Objectives

To investigate and quantify:

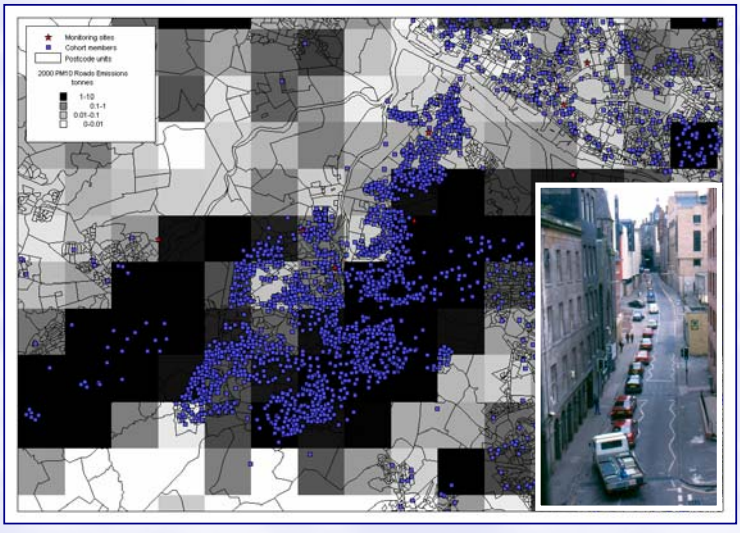
- cause-specific health effects of long-term exposure to air pollutants;
- extent of cause-specific shortening of life from short-term exposures to air pollutants;
- effects of pollutant exposure on possible susceptible population sub-groups;
- coherence in medical outcomes, confounding effects, and biologically relevant exposure/latency periods;
- use of different exposure metrics.

3. Some aspects of study design

- Outcomes in 26,360 subjects in 3 cohorts will be studied between 1975 and 2002.
- Detailed baseline risk data & unique advantages of the Scottish Health Record Linkage system (including algorithmic linking of individual hospital admission & mortality records) will enable novel ways of quantifying effect magnitudes in susceptible population sub-groups & coherence in medical outcomes.
- The potential for confounding & effect modification by both individual & aggregate level factors (including smoking, deprivation, occupation, prior ill health, physiological factors, and gaseous co-pollutants) will be examined.
- Extensive (>50 year duration) pollution exposure databases will enable investigation of exposure & latency durations & temporal changes in pollution concentrations that are most relevant to health outcomes.
- Alternative exposure metrics based on inventoried, source-specific, pollutant emissions will be investigated.

4. Anticipated benefits to end-users

- Quantification of the health impacts of long-term exposure to air pollutants
- Research will inform policy on future air quality standards in relation to exposure metrics, magnitudes & durations
- Identification of susceptible population sub-groups that may require additional protection



Residence location of cohort subjects in Paisley & Renfrew superimposed on the National Atmospheric Emissions Inventory grid for traffic related particles (PM_{10}).

[1]University of Strathclyde, [2]University of Manchester , [3]University of Glasgow, [4]Scottish Centre for Infection & Environmental Health, [5]University of Edinburgh

FACTORS INFLUENCING INDIVIDUAL NITROGEN DIOXIDE EXPOSURE IN STOCKHOLM

Tom Bellander and Martin Kruså

National Institute of Environmental Medicine, Karolinska Institutet, Stockholm and
Department of Environmental Health, Stockholm County Council, Stockholm, Sweden

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AIM OF THE STUDY

Individual exposure to NO_2

Personal exposure - Ambient air level

Residence - Workplace

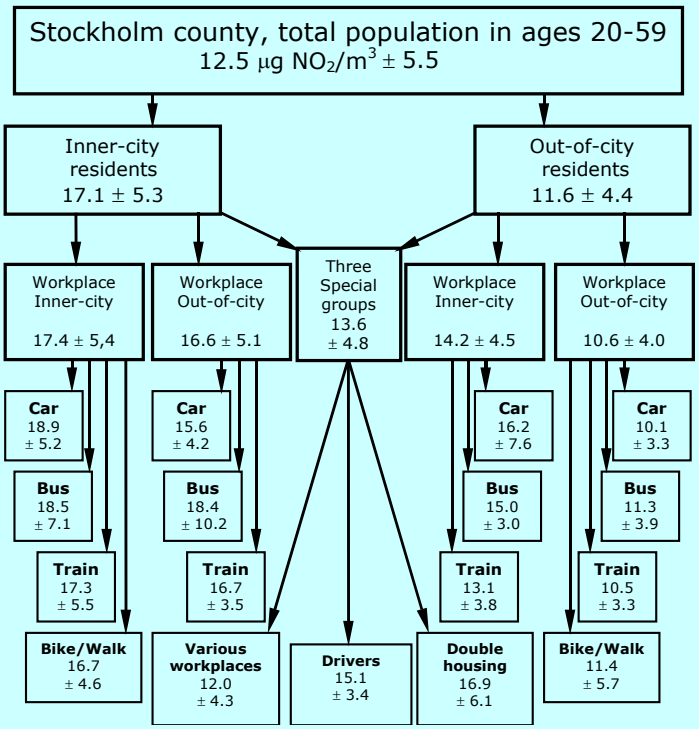
Mode of transportation

MATERIALS & METHODS

- Random population sample of 240 persons
  - aged 20 - 59 years
  - non-smokers
  - permanent work within Stockholm County
  - no gas appliances at home
- Stratification by location of home, work and mode of transport to work
- Individual NO_2 exposure by small diffusion monitors carried for a week at two occasions, in the period May 1999 - May 2000. Daily activity patterns recorded in diary
- Air levels of NO_2 at workplace and home from dispersion modeling
- Urban street and background levels from at three fixed monitoring stations

RESULTS

Stockholm County inhabitants' exposure to NO_2 ($\mu g/m^3 \pm$ stdev)



- Inner-city residents were more exposed than out-of-city residents.
- For out-of-city residents, place of work was important.
- Mode of transport did not seem important.

Factors influencing personal NO_2 exposure

Multiple regression model, r^2=0.46

Factor	IQR	Coef. µg/m³	95 % C.I.	
NO_2 at residential address	12.3	0.26	0.19	0.33
NO_2 at workplace	11.9	0.22	0.16	0.28
NO_2 at street level	8.2	0.27	0.17	0.35
NO_2 at roof level	4.6	0.15	0.03	0.27
Bedroom facing large street (prop.)	7.3%	2.6	1.4	3.8
Hours in room with gas. appl.	0.0*	0.44	0.34	0.55
Hours in smoky room/free flames	5	0.26	0.18	0.35
Hours in traffic/garage	6.5	0.15	0.05	0.25

- Individual one week exposure levels were strongly correlated to the estimated annual average levels at home and work
- Bedroom facing a large street increased the exposure level
- Individual exposure levels were correlated to the current street (more) and urban background (less) levels
- Time spent in certain environments influenced the individual exposure level:
  - in a room with gas appliance
  - in a room with free flames or smoke
  - in traffic or a garage

*Only 33 out of 383 weeks of measurement had times greater than 0, up to 24 hours

CONCLUSIONS

• Individual NO_2 exposure varied mainly due to estimated NO_2 levels at home address and work

• Variation in NO_2 at street level was correlated to individual NO_2 exposure. Roof level variation was less influential.

• Time spent in traffic was only weakly related to individual NO_2 exposure

• Mode of transport had no significant influence on individual NO_2 exposure.

• Indoor sources may give significant contributions

Impact of iron and steel industry and waste incinerators on human exposure to dioxins, PCBs and heavy metals: results of a cross-sectional study in Belgium

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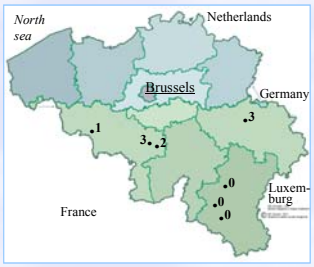
Introduction

An epidemiological study was carried out in 2000/2001 in Wallonia, Belgium, to quantify the dioxins (PCDD/Fs), polychlorinated biphenyls (PCBs) and heavy metals in subjects living in the vicinity of a municipal solid waste incinerator (MSWI) or a sinter plant and compare them to levels in a rural unpolluted area.

Materials and methods

A total of 142 volunteers living around these facilities were recruited. Fifty-one subjects aged 21 to 80 years were living within a distance of 2 km from a MSWI in a rural area (Thumaide). Thirty-three subjects aged 33 to 65 years were recruited within a distance of 2 km from a MSWI in an industrial area (Pont-de-Loup). Fifty-eight subjects aged 25 to 67 years were living within a distance of 4 km from two iron and steel plants in the suburbs of two industrial cities (Liège, n=12 and Charleroi, n=46). These subjects were compared with 63 controls from an unpolluted area in the Ardenne (Belgium).

Figure 1: Localisation of the facilities



- 1: MSWI 1 (*Thumaide*, rural area)
- 2: MSWI 2 (*Pont-de-Loup*, industrial area)
- 3: Sinter plants (*Charleroi and Liège*)
- 0: Referents (*Ardenne*)

The volunteers provided approximately 200 ml of fasting blood and a urine sample. We quantified serum concentrations of dioxins (17 PCDD/Fs congeners), coplanar PCBs (IUPAC n° 77, 81, 126 and 169) and 12 PCB markers (IUPAC n° 3, 8, 28, 52, 101, 118, 138, 153, 180, 194, 206 and 209). We also measured three heavy metals: urinary cadmium (Cd), urinary mercury (Hg) and blood lead (Pb).

Results

The mean concentrations of pollutants in blood or urine in the different groups are given in Table 1. Results are similar after adjustment for confounding factors (age, sex, body mass index, fat consumption, tobacco consumption or alcohol consumption).

The mean Cd, Hg and Pb concentrations of exposed subjects were not significantly increased in comparison with referents.

By contrast, whilst no increase was found in residents around the MSWI in the industrial area or the sinter plants, subjects living around the MSWI in the rural area had on average significantly higher serum levels of dioxins and coplanar PCBs than referents.

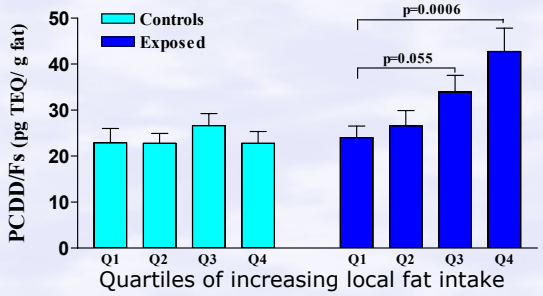
Table 1: Concentrations of pollutants in blood or urine

	Referents n=63	MSWI 1 n=51	MSWI 2 n=33	Sinter plants n=58	Total n=205
Dioxins and polychlorinated biphenyls					
PCDD/Fs (pg TEQ/g lipids)	23.9 [5.0-71]	37.9* [9.2-101]	24.1 [11-113]	23.8 [5.2-57]	26.8 [5.0-113]
Coplanar PCBs (pg TEQ/g lipids)	7.0 [1.5-29]	10.3* [0.2-44]	5.7 [0.2-20]	6.3 [1.2-37]	7.2 [0.2-44]
PCDD/Fs+cPCBs (pg TEQ/g lipids)	31.3 [6.7-100]	48.7* [9.4-145]	30.1 [13-133]	30.7 [7.6-84]	34.7 [6.7-145]
Σ12 PCB markers (ng/g lipids)	418.3 [141.5-764.1]	450.4 [122.9-1275]	374.8 [194.3-818.3]	404.5 [124.8-909.4]	416.3 [122.9-1275]
Heavy metals					
Cd urine (µg/g creatinine)	0.49 [0.13-2.17]	0.62 [0.20-1.95]	0.43 [0.07-2.78]	0.49 [0.06-4.46]	0.51 [0.06-4.46]
Hg urine (µg/g creatinine)	1.95 [0.52-6.17]	1.80 [0.79-18.8]	2.11 [0.95-10.35]	1.79 [0.5-6.12]	1.89 [0.5-18.8]
Pb blood (µg/L)	45.8 [5.00-164]	43.3 [11.0-149]	39.4 [17.0-123]	42.2 [12.0-190]	43.1 [5.00-190]

Geometric mean [min-max]. *p value < 0.05.

A two-way ANOVA on age-adjusted dioxin levels revealed a significant interaction between residence around incinerators and the consumption of fat from local origin, especially from bovine and poultry products. Whereas dioxin levels in referents did not vary with local animal fat consumption, dioxin levels in subjects living around the incinerators increased proportionally to their intake of local animal fat, almost doubling in subjects with the highest fat intake (figure 2).

Figure 2: Increase in dioxin body burdens with local fat consumption



Conclusions

- Human exposure to emissions from MSWIs can increase dioxin and coplanar PCB body burden of residents.
- This increase is dependent of the consumption of animal products from the local food chain.
- This increase is likely to occur only when dioxin emissions exceed 5 ng TEQ/Nm³. This threshold is largely above emission standards currently in force in most countries (between 0.1 and 1 ng TEQ/Nm³).

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Supported by the Ministry of Environment of the Walloon Region

UCL

ECRHS II

Correlations between Surrogates of Exposure to Ambient Air Pollution ($PM_{2.5}$ mass, Sulphur, NO_2, black Smoke, Silicon) across 21 European Centres

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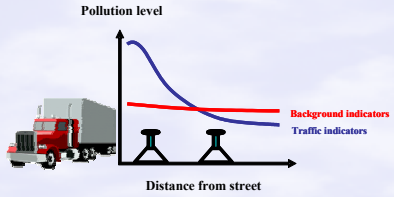
Introduction

The European Community Respiratory Health Survey II (ECRHS II) is a cohort study aimed at assessing the **long-term effects** of air pollution.

In addition to **$PM_{2.5}$**, the core indicator of air pollution exposure assessment in ECRHS II, **NO_2**, **Black Smoke**, and **28 chemical elements** on $PM_{2.5}$ were measured.

Correlations between annual means of pollutants indicate to what degree their effects may be distinguishable in long-term health analyses.

However, different **spatial variability** of these indicators may affect their **comparability**. Of specific concern is the influence of the sampling location on measured levels of pollutants heterogeneously distributed in space, such as traffic emissions.



Aims

Derive estimates of long term exposure which reflect …

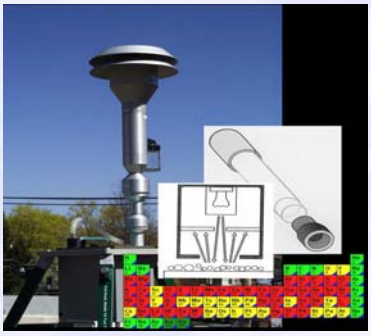
- ***specific*** **aspects of air pollution**

and

- ***representative*** **levels for population**

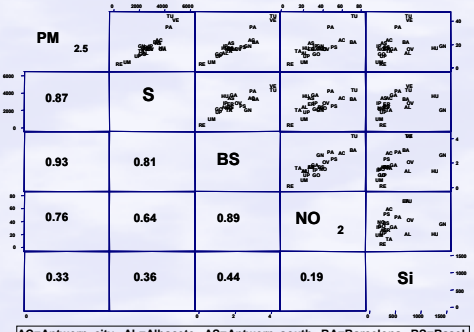
Method

- Parallel exposure measures, same sites:
  - $PM_{2.5}$ (Basel Sampler/BGI, 7 days/month)
  - NO_2 (Palmes Tubes, monthly only)
  - Reflection of $PM_{2.5}$ (Black smoke (BS))
  - Elements on $PM_{2.5}$ (ED-XRF)
    - Sulphur (s), Lead (Pb), 26 others
- Assumptions for indicators:
  - Background: $PM_{2.5}$ (urban), S (long range)
  - Traffic: NO_2, BS (diesel), Pb (gasoline)
  - Crustal material: Si



Results and Discussion

Correlations of $PM_{2.5}$, S, NO_2, BS, and Si annual means



AC=Antwerp city, AL=Albacete, AS=Antwerp south, BA=Barcelona, BS=Basel, ER=Erfurt, GA=Galdakao, GN=Grenoble, GO=Gothenburg, HU=Huelva, IP=Ipswich, NO=Norwich, OV=Oviedo, PA=Pavia, PS=Paris, RE=Reykjavik, TA=Tartu, TU=Turin, UM=Umea, UP=Upsala, VE=Verona

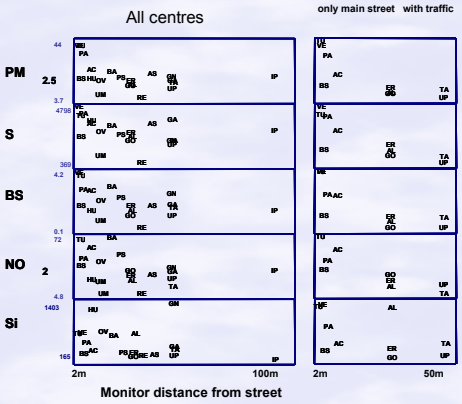
Pearson correlations between annual means of anthropogenic pollutants ($PM_{2.5}$, S, BS and NO_2) range from 0.64 to 0.93. $PM_{2.5}$, S and BS correlated better with each other than with NO_2. Their correlations with Silicon are significantly lower ($0.19<r<0.44$)*.

Very high correlations between pollutants make it difficult to assign health effects to specific indicators or emission sources. Therefore, the health relevance of these pollutants should be investigated separately.

* For epidemiological investigation of long-term effects of air pollution, annual means serve as exposure estimates.

Evaluation of influence of sampler locations

Sampler to street distance vs. pollutant levels

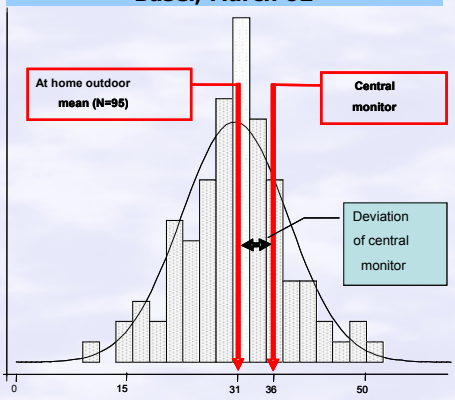


Over all centres pollutant levels are independent of the distance to the nearest street. But after the exclusion of centres where the street closest to the monitor is a "side street" or only has "low" traffic volume, a clear trend for higher levels at stations closer to a street is observable (right column).

The observed pattern may reflect true background levels as distance to street seems to be correlated with expected pollution levels and therefore acts as a confounder. E.g. all stations in North Italy are close to a street (PA, TU, VE) whereas stations in Sweden (GO,UP) and Estonia (TA) are further away.

We created several variables expected to depend on the proximity to traffic but being independent of absolute pollution levels, namely ratios between traffic and background indicators and weekdays vs. weekend ratios. So far we see no evidence for a major influence of local traffic. We will further investigate this using more detailed data on traffic, wind patterns and station characteristics.

Spatial distribution of NO_2 at home, outdoors, simultaneously, Basel, March 02



Spatially distributed NO_2 measurements at participants homes will be used to calculate differences between central monitor values and the average of NO_2-at home levels. This will provide an estimate for the deviation of the central sampler from what can be considered a representative mean.

Conclusions

- **Various potentially source specific indicators of exposure are available**
- **Comparability of indicators may be affected by several factors:**
  - **spatial variability**
  - **differences of sampling locations**
  - **methodological differences**
- **Exposure data will be evaluated on this matter before health effects analyses**

This work was carried out by T Götschi (1), L Bayer-Oglesby (2), M E Hazenkamp-von Arx (2), C Luczynska (3), J Sunyer (3), S Villani (3), J Heinrich (3), D Norbäck (3), B Forsberg (3), D Jarvis (3) N Künzli (1,2) and the ECRHS Working Group Air Pollution (3)

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AMBIENT AIR POLLUTION AND CHILDREN'S EXPOSURE TO LEAD AND CADMIUM IN A SMELTER REGION IN BULGARIA

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BACKGROUND

A smelter plant in the region of South Bulgaria produces lead, cadmium, other non-ferrous metals and hard-alloy products.

Such productions are known to emit into the atmosphere particulates, sulfur dioxide, and airborne lead, cadmium and other heavy metals.

The presence of such a powerful source of hazardous emissions with a potential human health risk required exposure assessment of the population in the area.

OBJECTIVES

- Assessment of external exposure of the general population in the area by monitoring environmental levels of lead and cadmium.
- Assessment of children's internal exposure by measuring blood lead levels
- Assessment of maternal/infant exposure by measuring lead levels in the placenta, maternal and cord blood.

MATERIALS and METHODS

Air pollution monitoring

Period: 1991-99

Sampling sites. Three fixed monitoring stations for ambient air quality in residential areas in the vicinity of the plant: Site 1 ("*Assenovgrad*"); Site 2 ("*Kuklen*'); Site 3 ("*Dolno Voden*")

Sample analysis. Determination of lead and cadmium by atomic absorption spectrometry (Perkin Elmer 3100 with FID)
Detection limit: 0.002 μg/cm^3

Data processing: Lead and cadmium levels calculated for monthly and annual averages, median, minimal, maximal concentrations, and 98 percentile.

Children's lead exposure

Sample 100 children, aged 11-12 , residing at sites 1 & 3
50 children, same age, from a control area

Determination Blood lead
(atomic absorption spectrometry, extraction with methylisobutyl ketone and ammonium pyrolidine dithiocarbamate)

Data analysis Comparisons (T test) :
- Plant area versus control area
- Site 1 versus site 3
- Plant area 1991 versus 1995

Maternal / infant lead exposure

Sample 76 maternal-infant pairs (sites 1 & 3)

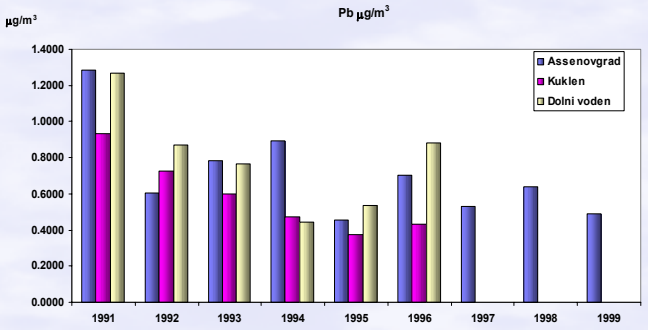
Determination Lead in maternal & cord blood and placenta
(atomic absorption spectrometry, Perkin Elmer 5100 Zeeman)

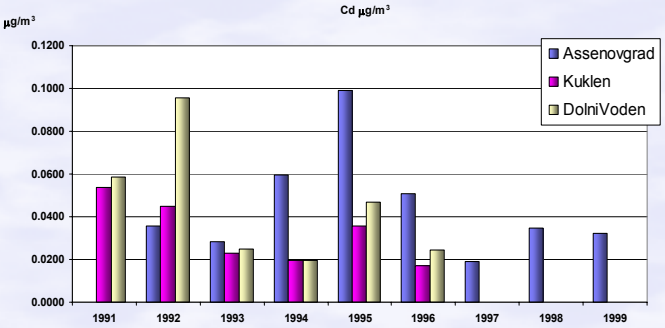
Data analysis Comparisons (T test) :
- Maternal & infant blood lead levels in smokers and non-smokers
- Maternal & infant lead levels in normal and pathological birth outcomes

RESULTS

Children's blood lead
(Distribution of individual values)

Blood lead (μg/L)	*Plant area Site 1*	*Plant area Site 3*
< 150	56%	28%
150 - 200	17%	28%
201 - 250	17%	14%
> 250	10%	20%





CONCLUSIONS

- ***General population exposure to lead and cadmium takes place in the vicinity of a non-ferrous metal smelter in Bulgaria; ambient air in residential areas is contaminated with these metals at levels above the national MAC values.***
- ***Environmental contamination results in significantly increased levels of blood lead and cadmium in local children population aged 11-12 in comparison to children of the same age in a non-industrial area.***
- ***Maternal exposures during pregnancy result in high lead levels in placenta and infant blood at birth; abnormal birth outcomes (particularly pre-term births) are associated with significantly higher maternal/fetal lead exposures in comparison to normal births.***

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Lung hyperpermeability and asthma prevalence in schoolchildren: unexpected associations with the attendance of indoor chlorinated swimming pools

Bernard A.[1], Carbonelle S.[1], Nickmilder M.[1], Michel O.[2], Higuet S.[2], de Burbure C.[1], Buchet J.-P.[1], Hermans C.[1], Dumont X.[1] and Doyle I.[3]

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Introduction

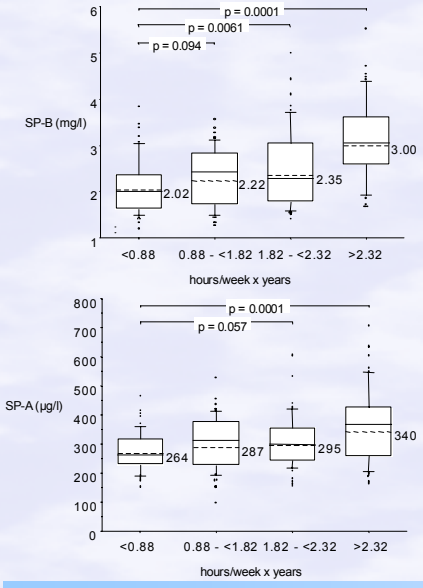
• For several decades, chlorination has been used to destroy microbiological pathogens and organic matter in swimming pools.
• When reacting with organic matter brought by swimmers (e.g. urine, sweat), hypochlorous acid generates a complex mixture of potentially harmful disinfection by-products from which the most volatile and also most concentrated in the air of indoor pools is nitrogen trichloride (NCl_3), a powerful irritant.

The aim of this study was to investigate whether exposure to nitrogen trichloride in indoor chlorinated pools may affect the respiratory epithelium of children and increase the risk of some lung diseases such as asthma.

Observations

1. Chronic effects of air pollution on the respiratory epithelium:

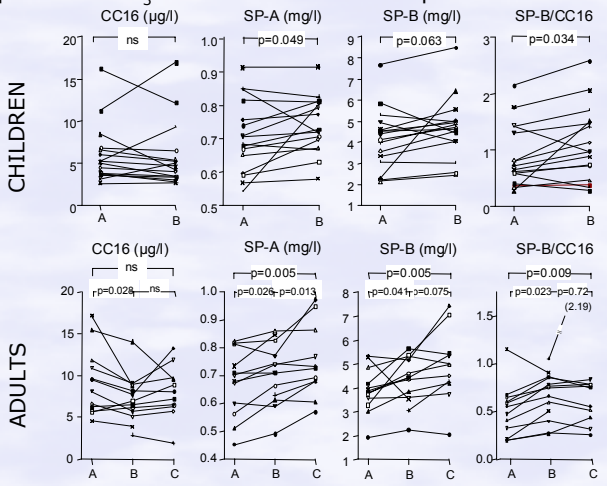
• 226 healthy children aged 8-12 years were recruited from seven primary schools, two in Brussels and five in a rural area (Ardenne).
• The cumulated pool attendance of these children ranged from less than 0.55 to 6.42 hours/week x years.
• The integrity of lung epithelium was assessed by measuring three lung specific proteins in serum: the surfactant-associated proteins A and B (SP-A and SP-B) and the 16 kDa Clara cell protein (CC16).



A positive dose-effect relationship was found with cumulated pool attendance and serum SP-A and SP-B. No clear dose-effect relationship was found for serum CC16 (ANOVA, p = 0.07).

2. Acute effects of air pollution on the respiratory epithelium:

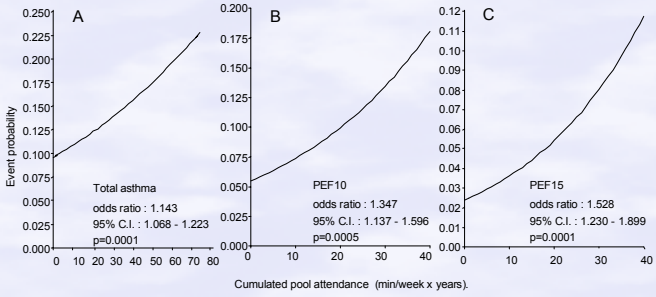
• Lung specific proteins were measured in the serum of 16 children and 13 adults both before and after a 1- to 2-hour exposure to NCl_3 in an indoor chlorinated pool.



These changes in serum levels of lung proteins were reproduced in children and adults attending an indoor pool with a mean NCl_3 concentration of 0.490 mg/m^3. Serum SP-A and SP-B were already significantly increased after one hour by the pool side without swimming.

3. Relationships between pool attendance and asthma prevalence:

• 1,881 children (15 Brussels primary schools) aged 6-14 years: cumulated pool attendance of 0.7 to 2.5 hours/week x years.
• Screening for asthma: a child was considered EIB-positive (exercise-induced bronchoconstriction test) when his PEF (peak expiratory flow) dropped by 10 % or more (PEF10) or 15 % or more (PEF15) at 5 or 10 min after a 6 minute-runabout indoors.
• Total asthma prevalence was calculated as the sum of the prevalence of PEF10 plus the prevalence of EIB-negative children actually treated for asthma.



PEF10, PEF15 and total asthma prevalences were significantly correlated with cumulated pool attendance indices.

Conclusions

Regular attendance of chlorinated pools by young children is associated with an exposure-dependent increase in lung epithelium permeability and an increase in the risk of developing asthma, especially in association with other risks factors such as exposure to pets. These findings lead us to postulate that the increasing exposure of children to chlorination products in indoor pools might be an important cause of the rising incidence of childhood asthma and allergic diseases in industrialized countries. Further epidemiological studies should be undertaken to test this hypothesis.

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HELI-O-S

Clara cell protein (CC16) as a biomarker for ozone exposure in humans

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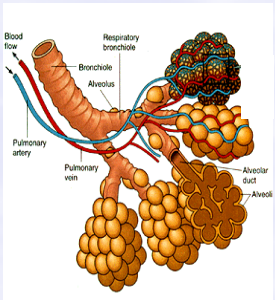
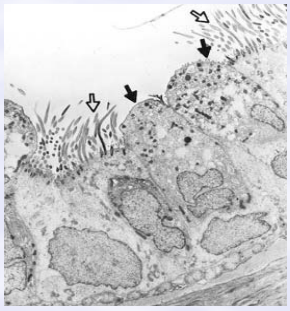
Background

Ozone (O_3) is an important air pollutant known to impair lung function and induce airway inflammation. Epidemiological studies have shown associations between variation in daily ambient levels of ozone and adverse health effects. Clara cell protein (CC16) is a 16-kDalton anti-inflammatory protein produced and secreted by the bronchiolar Clara cells. CC16 diffuses from the respiratory tract into the blood and has been considered a sensitive marker of increased permeability of the lung epithelial barrier (1). An association between serum CC16 and ambient ozone levels has been found in Italian cyclists and CC16 has been suggested to be a promising biomarker for ozone exposure (2).





Clara cell protein (CC16) is the major protein secreted by the bronchiolar Clara cells

Aim

To evaluate the usefulness of CC16 as a biomarker for ozone exposure by addressing its sensitivity under controlled experimental conditions.

Methods

Twenty-two healthy subjects were exposed to 0.2 ppm of ozone and filtered air on two separate occasions, at least three weeks apart. Peripheral blood samples were drawn and lung function assessed at five time-points: 2 hours pre-exposure, immediately before and after exposure as well as 2 and 4 hours post-exposure. CC16 was determined in serum using a latex immunoassay (3).

Results

Exposure to ozone significantly increased the serum concentrations of CC16 at two and four hours post-exposure relative to parallel air exposure values, 12.0±4.5 vs. 8.4±3.1 μg/L [mean±SD] ($p<0.001$) and 11.7±5.0 vs. 7.9±2.6 μg/L ($p<0.001$). Ozone serum CC16 concentrations at 2 h post-exposure were significantly increased compared to the immediate pre-exposure value ($p<0.01$). After air challenge, serum CC16 concentrations were significantly decreased at 2 and 4 hours post-exposure compared with the pre-air exposure concentration, $p<0.01$ and $p<0.001$ respectively. (Figure 1)

Plasma from archived material revealed that ozone exposure resulted in an increase in CC16 concentrations which persisted until 6 hours post-exposure; 9.1±2.6 vs. 7.1±1.7 μg/L ($p<0.01$) but returned to control values at 18 h post-exposure. (Figure 2)

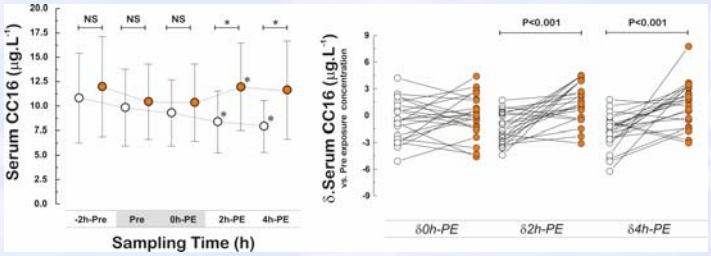


Figure 1

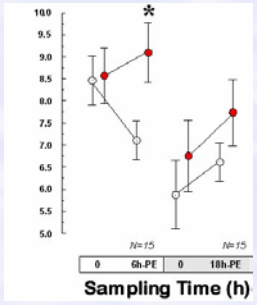


Figure 2

Ozone induced a significant decrease in FEV_1 immediately post exposure ($p=0.002$) (Figure 3). A corresponding fall was also detected in VC ($p=0.001$).

No significant correlations were observed between baseline serum CC16 concentrations and baseline lung function values. Neither was any relationship noted between the CC16 and lung function responses (ozone – air) at any time-point post-exposure.

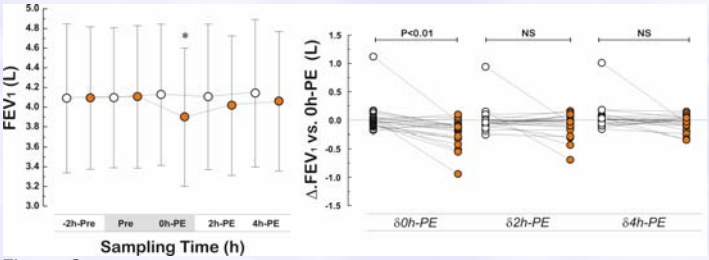


Figure 3

Discussion and Conclusions

Serum levels of CC16 increased after exposure to ozone, peaking around 2-4 hours post-exposure with levels back to baseline at 18 h.

As CC16 is synthesized and secreted almost exclusively by the lung Clara cells, the enhanced serum concentrations can only be explained by a leakage of the protein across the lung epithelial barrier.

Increases in serum CC16 seem to correspond with an evolving ozone-induced airway inflammation.

No association was observed between the magnitude of the ozone-induced lung function decrements and CC16 responses, suggesting that impaired lung function was not related to epithelial injury.

A consecutive decrease in CC16 levels was detected on the air exposure day. This may suggest a diurnal variation of the baseline CC16 levels.

The data suggest serum CC16 to have a potential as a biomarker for ozone exposure.

The question of a potential diurnal variation in baseline serum CC16 concentrations needs further evaluation.

References:

1. Hermans and Bernard. *Am J Respir Crit Care Med* 199;159:646-678
2. Broeckaert *et al. Lancet* 1999;353:900-901
3. Bernard *et al. Eur Respir J* 1992;5:1231-1238

UMEÅ UNIVERSITET

Evaluation of the mutagenicity of PM10 and PM2.5 collected in an industrial and urban area of Antwerp

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Introduction and Aim

Epidemiological studies over the last decades suggest rather consistently that inhalable particulate matter may be responsible for increased rates of lung cancer.
Limited research has been performed to measure the toxicity of particles in Flanders. Particle monitoring is restricted to measurement of PM10 concentrations and sporadical chemical analyses.
The aim of this study is to investigate the toxic activities associated with coarse (PM10) and fine (PM2.5) particles, collected in the industrial environment of the harbour (Petrol Quay) and the city center of Antwerp, Borgerhout.
Human alveolar epithelial cells (A549) are used for this purpose. Cytotoxicity is evaluated with the Alamar Blue assay, mutagenicity with the micronucleus test.

PM10 and PM2.5 samples

Samples were collected by the department of Environmental Measurements (Vito).
Sampling periods were not simultaneous for both locations.

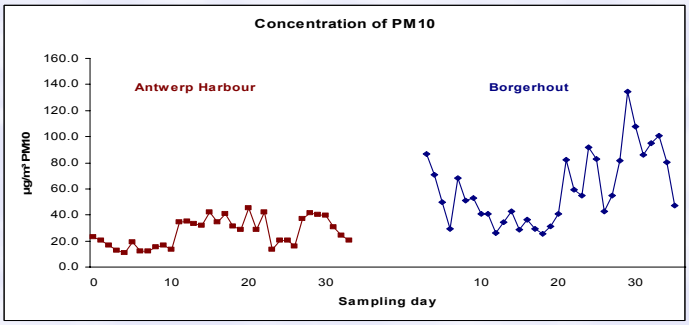
Average PM10 concentration is twice as high in the city as compared to the harbour.
The percentage of fine particles (PM2.5), contributing to the total amount of coarse particles (PM10) counts 54% in the urban location and 82% in the industrial location.


Eberline sampler


Borgerhout Antwerp: urban


Harbour Antwerp: industrial

Location	Average PM_{10} concentration	Average $PM_{2.5}$ concentration	Sampling Period
Borgerhout	57 µg/m³	31 µg/m³	12/02/03 – 31/03/03
Antwerp Harbour	28 µg/m³	23 µg/m³	20/12/02 – 31/01/03

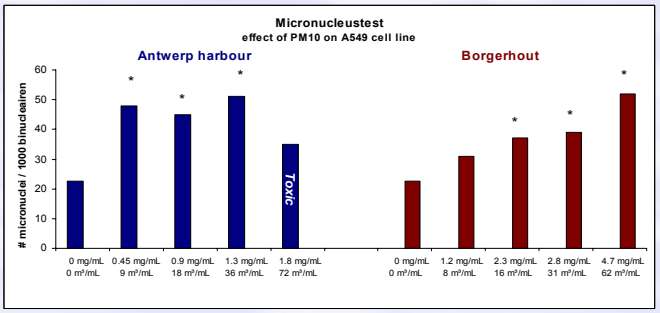


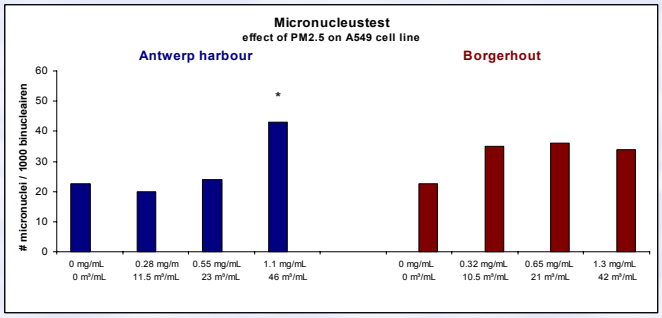
Methods

Particles were suspended in bidistilled sterile water supplemented with 0.1% tween-80. Particle mass is gravimetrically assessed by weighing the filters before and after sampling.
Human alveolar epithelial cells (A549) were exposed for 48 hours to the particles to examine *in vitro* the toxicological potential of both PM10 and PM2.5.
Cytotoxicity was assesed using the AlamarBlue assay.
Mutagenicity was assessed using the cytokinesis-block micronucleus assay on binucleated cells *(Fenech et al., Mut Res, 147:29-36 (1985), with modifications)* .

Results

Particles from both areas and both size-fractions showed no significant cytotoxicity (results not on poster).
PM10 and PM2.5 originating from both areas were able to cause mutagenic effects. The micronucleus frequency in binucleated cells was significantly increased.
Although the m³ equivalents added to the cells are comparable, the mass of particles (mg/mL), reflecting the concentration (µg/m³), is two-fold higher in the urban area. Particles from the industrial area are more potent than the urban particles to evoke a mutagenic response.
PM10 is more mutagenic compared to PM2.5 for both locations.





*: Significant difference between the marked sample and the 0% dose, assessed with Statistical Tables from Kastenbaum et al., *Mut Res, 9 (1970) 549-552.*

Conclusion

Routine concentration measurements of PM10 and PM2.5 are insufficient to predict the genotoxic potential.
The increase in micronucleated cell frequencies in this study is related to both location and particle size.
This research needs to be confirmed by more environmental measurements and precise determination of particle mass, number and composition.

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Influence of the PAH on the DNA Damage Detected in Unexposed and Occupationally Exposed Donors from Košice

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[2] Chair of the Epidemiology and Preventive Medicine, CM UJ, Kraków, [3] Department of Molecular Biology of the P.J.Šafárik University, Košice, Slovakia, [4]Cancer Biomarkers and Prevention Group, Leicester, UK

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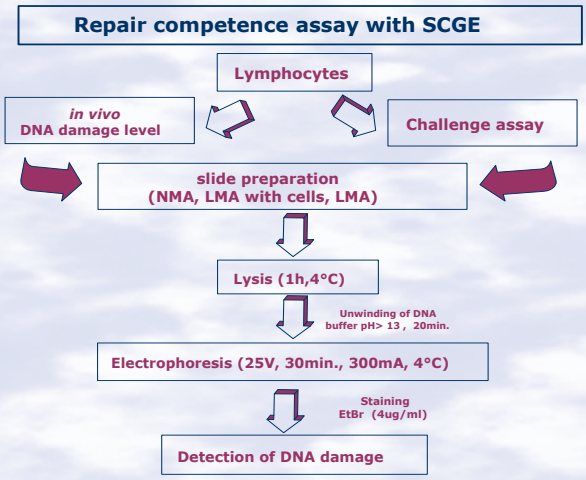
Introduction

The aim of our study was to investigate a cellular response to complex environmental genotoxic mixture and the possible influence of the occupational exposure to PAH on susceptibility to the induction of the oxidative types of the DNA damage. Lymphocytes isolated after samplings in Košice were transported frozen (in dry ice) to the laboratory of the DREB in the INP in Kraków, Poland, where they were stored in -70^{0}C for further treatments. A repair competence assay after a challenging dose of X-rays (oxygen radicals, oxidative and DNA damage inducing agent) was proposed for the study and the alkaline version of single cell gel electrophoresis (SCGE) assay was applied for the analysis of the DNA damage induced by various *in vitro* treatments [1,2]. The DNA damage was estimated by the automatic evaluation of the comet size that was performed with Komet 3.0 software from Kinetic Imaging, Liverpool, UK. For each treatment, 100 cells were analyzed (2 times 50 cells for each of two replicate electrophoresis). From various measures the tDNA - fraction of the DNA in the comet tail and TM- a comet tail moment, which is the fraction of the DNA in the comet tail multiplied by the tail length were used for the evaluation of the efficiency of treatments. Studies were performed in the presence of the standardizing samples of lymphocytes from the same referent pool of lymphocytes of healthy male donor (MS).

Materials and Methods

Donors: 55 unexposed (av. age 33.4y)

50 exposed (av. age 32y)



Evaluated parameters;

SVI_T, SVI_{TM} - standardized *in vivo* DNA damage level estimated for TDNA and TM parameters in comet assay

$SUSC_T$, $SUSC_{TM}$ – standardized cells susceptibility to X-rays estimated for TDNA and TM parameters

SRD_T, SRD_{TM} - standardized the percent of no repaired (residual) damage detected in cells after challenging doses assay estimated for TDNA and TM parameters

Results and Conclusions

Table. 1 PAH occupational exposure influence on cellular capacities in the results obtained with SCGE assay.

EXP	SVI_T ±SD	SVI_{TM} ±SD	$SUCS_T$ ±SD	$SUCS_{TM}$ ±SD	SRD_T[%] ±SD	SRD_{TM}[%] ±SD
0	6.56	1.84	11.91	5.82	60.73	41.75
	1.76	0.65	2.89	2.02	21.96	26.32
1	6.91	1.90	12.00	5.34	69.82	49.07
	1.94	0.70	2.85	1.60	25.60	26.10
Sig.	ns	ns	ns	ns	0.001	ns

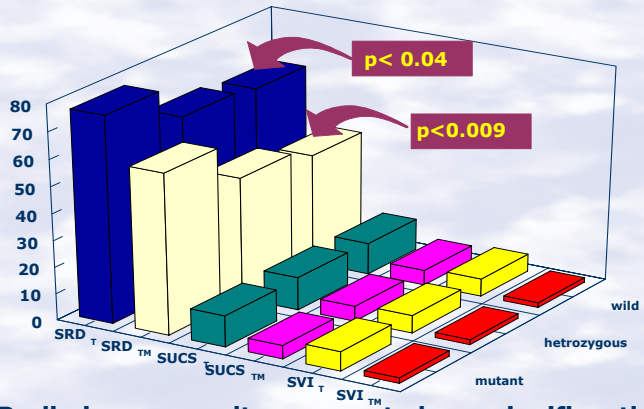
Significantly lower efficiency of the repair process of induced by the challenging dose damage in cells of occupationally exposed to PAH donors was observed.

Table. 2 Influence of smoking history(SH=0 nonsmokers, SH=1 smokers) on cellular capacities in lymphocytes.

SH	SVI_T ±SD	SVI_{TM} ±SD	$SUCS_T$ ±SD	$SUCS_{TM}$ ±SD	SRD_T[%] ±SD	SRD_{TM}[%] ±SD
0	6.73	1.88	12.17	5.78	62.56	41.32
	1.78	.68	2.87	1.94	23.55	24.29
1	6.68	1.86	11.70	5.40	67.53	49.40
	1.94	.66	2.85	1.74	24.49	28.15
Sig.	ns	ns	ns	ns	ns	0.04

Significantly negative influence of the smoking on the efficacy of the repair process was observed.

Figure. 1 Influence of GSTP I/V polymorphism on cellular capacities the results with SCGE assay.



Preliminary results suggested a significantly lower efficiency of repair process of the damage induced in lymphocytes of genetically polymorphic donors with mutation in exon 5 GSTP1 (GSTP1 Ile/Val).

Reference:

1.A.Cebulska-Wasilewska, D.Nowak, W.Niedźwiedź, E.D.Wagner, M.Plewa, Mut. Res. 446 (1999) 57-65.

2. D.W. Fairbairn, P.L.Olive, K.L. O'Neill. Mut. Res. 339 (1995) 37-59.

Acknowledgments:

Research was partially supported by grants: EC-EXPAH QLK4-CT-2000-00091/SPUB-M 620/E-77/SPUB-M/5.PRUE/DZ74/2001-2003. Available assistance by E.Bartel, I. Pawlyk, J. Swakoń and J. Wiltowska, is greatly appreciated.

INFLUENCE OF OCCUPATIONAL EXPOSURE TO PAHs ON LYMPHOCYTES SUSCEPTIBILITY TO THE INDUCTION OF DNA DAMAGE (sampling in Sofia)

A.Cebulska-Wasilewska[1,2], A. Panek[1] I.Pawłyk[1], P.Farmer[3], T.Popov[4],

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Abstract

The aim of the study was to investigate a possible influence of the occupational exposure to PAHs on cellular susceptibility to the induction of the oxidative type of damage in lymphocytes collected from donors in Sofia. Similarly to the studies performed in the previous groups of donors the alkaline version of the SCGE assay was applied to evaluate the DNA damage present in lymphocytes after various treatments: in defrosted lymphocytes, in lymphocytes right after exposure to the challenging dose 4Gy of X-rays (as an oxygen radicals and oxidative damage inducing agent) and in lymphocytes after various periods of incubation following the X-rays exposure, to investigate kinetics and efficiency of the repair process. Lymphocytes were isolated from the whole blood samples of 78 healthy males: 25 donors unexposed (average age 39 years), 53 donors occupationally exposed to PAH (27 drivers of average age 43.3 and 26 policemen of average age 32.5 years). Among donors: 26.9% have never smoked, 20% gave up smoking and 52.6% were recent smokers. Evaluation of the DNA damage (tDNA, TM, TL) as a function of repair (post-irradiation incubation time) was done on the basis of two independent repetitions of the SCGE assay applied to each donor for the studies of kinetics. From the results obtained, susceptibility to the induction of the DNA damage, repair competence from the residual (unrepaired) damage and repair rate from the half life of the DNA damage repair process kinetics were evaluated. No difference between referent and exposed groups was observed in the damage induced either *in vivo* or by the challenging dose. Although, the residual damage on average is higher in exposed donors, and half the time of the repair kinetics longer, suggesting lower repair competence in lymphocytes from exposed donors, though observed difference is statistically insignificant. However, when evaluated repair competence was stratified to three subgroups; referent, policemen and drivers repair efficacy decreased statistically significantly (81% then 89% and 67% respectively $p < .004$). Surprisingly, no significant difference was observed between groups analysed according to the category of smoking habits, however, neither PAH nor B(α)P average concentrations were significantly differentiated between investigated sub groups. Our preliminary results also suggest, possibly due to different lifestyles, lowering of the repair rate with decreasing education level ($r^2 = -0.32$, $p < 0.02$), however, final conclusion should be drawn up when all kinetics data will be available.

Materials and methods

Lymphocytes isolated from the whole blood samples collected from 78 healthy males (26.9% never smoke, 20% were former and 52.6% recent smokers). Among donors:

- ❑ donors unexposed (n=25, average age 39 years)
- ❑ donors occupationally exposed to PAH (drivers;n=26, average age 43.3 years & policemen;n=27, average age 32.5 years)

Challenging dose of 4Gy of X-rays (as an oxygen radicals and oxidative damage inducing agent) and alkaline version of the SCGE assay was applied to evaluate the DNA damage present in:

- ❑ defrosted lymphocytes
- ❑ in lymphocytes right after exposure to the challenging dose
- ❑ in lymphocytes after various periods of incubation following the X-rays exposure (to study kinetics and efficiency of repair process Fig.1).

Results

- ❑ No significant difference between referent and exposed groups was observed in the damage induced either *in vivo* or by the challenging dose. (Table 1.)
- ❑ Residual damage on average is higher in exposed donors, and half time of the repair kinetics longer (Table 1.) though observed difference are statistically insignificant.

Table 1. Influence of occupational exposure

EXPOSURE		$SVIV_{TM}$	$SUCS_{TM}$	SRD_{TM} [%]	$t(1/2)_{TM}$
unexposed	MEAN	1.61	9.10	41.60	3.75
	±SD	0.91	1.62	17.74	2.00
exposed	MEAN	1.51	8.50	44.51	4.35
	±SD	1.00	3.03	19.35	3.57

Abbreviations:

$SVIV_{TM}$ - standaryzed *in vivo* DNA damage level in the defrosted cells estimated for TM parameters,
$SUSC_{TM}$ – standardized sucsceptibility to X-rays estimated for TM parameters,
SRD_{TM} - standardized sucsceptibility to X-rays estimated for TM parameters,
$t(1/2)_{TM}$ – mean half lives time of repair estimated for TM parameters.

Table 2. Comparison of results obtained for three supgroups donors stratified according job.

JOB		$SVIV_{TM}$	$SUCS_{TM}$	SRD_{TM} [%]	$t(1/2)_{TM}$
referent	MEAN	1.61	9.10	41.60	**3.75**
	±SD	0.91	1.62	17.74	**2.00**
drivers	MEAN	1.44	8.36	45.76	**5.46**
	±SD	0.94	3.76	23.27	**4.27**
policemen	MEAN	1.58	8.65	42.98	**3.10**
	±SD	1.07	2.07	13.85	**2.01**
p<		0.79	0.61	0.79	**0.019**

Repair rate statistically significant deffered for various jobs categories (Table 2).

No significant difference was observed between groups analysed according to the category of smoking habits (SUCS – 8.25-9.60-8.57, SRD – 42.67-43.13-43.65 for nonsmokers, former and recent smokers respectively) however, neither PAH nor B(α)P average concentrations were significantly differentiated between investigated subgroups

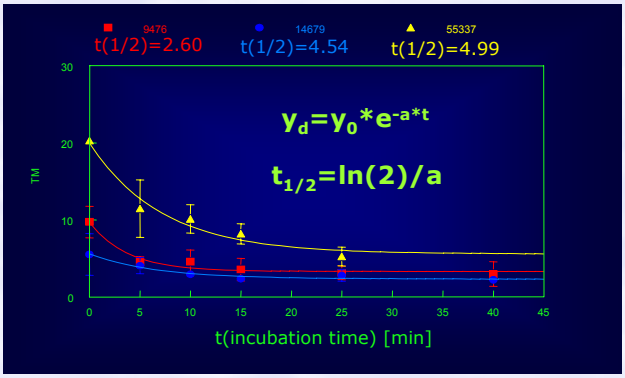

Fig. 1. Kinetics of repair of the DNA damage induced by the challenging dose of X-rays in lymphocytes of various donors

Conclusion

Our preliminary results suggest:

- **lower although insignificantly repair competence in lymphocytes from exposed donors,**
- **lowering of the repair rate with decreasing education level ($r^2 = -0.32$, $p<0.02$), possibly due to different life style,**
- **final conclusion should be drawn up when all kinetics data will be available.**

[1]Department of Radiation and Environmental Biology, the H. Niewodniczanski Institute of Nuclear Physics, Kraków, Poland [2]The Epidemiology and Preventive Medicine Department, CMUJ, Kraków, Poland, [3]Cancer Biomarkers and Prevention Group, Leicester, UK [4]Department of Toxicology, National Center of Hygiene, Sofia, Bulgaria.

Acknowledgments:

1. Research was partially supported by grants: EC-EXPAH QLK4-CT-2000-00091/SPUB-M 620/E-77/SPUB-M/5.PRUE/DZ74/2001-2003. Available assistance by J.Adamczyk, E.Bartel, J. Swakoń and J. Wiltowska, is greatly appreciated.

Reference:

1. A.Cebulska-Wasilewska, D.Nowak, W.Niedźwiedź, E.D.Wagner, M.Plewa, Mut. Res. 446 (1999) 57-65.
2. D.W. Fairbairn, P.L.Olive, K.L. O'Neill. Mut. Res. 339 (1995) 37-59.

Lung permeability, antioxidant status and NO_2 inhalation: a selenium supplementation study in rats.

de Burbure C.Y.[1], Heilier J.-F.[1], Nève J.[2], Becker A.[3], Albrecht C.[3], Borm P.J.A.[3], Gromadzinska J.[4], Wasowicz W.[4], Rydzynski K.[4], Bernard A.M[1].

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Introduction

Little is known about antioxidant status, selenium status in particular, and lung response to NO_2 which acts as a proinflammatory air pollutant. In this study, the effects of a low selenium diet (1.3µg Se/day) with or without selenium supplementation were studied in 128 two-month old male Wistar rats exposed to various concentrations of NO_2.

Materials and methods

Groups of Se-normal and Se-deficient rats were exposed to 1 or 10 ppm NO_2 6 h/day, 5 days/week for 4 weeks (Groups I and II respectively, long-term exposures), 5 ppm for 6 h/day for 5 days (Group III, sub-acute exposure), 50 ppm for 30 minutes (Group IV, acute exposure) or used as controls breathing normal atmospheric air (Group V). Rats were sacrificed immediately after the experiment or 48 hours later in order to estimate recovery (see Table 1).

Table 1: NO_2 exposure conditions

NO_2 Exposure conditions	Groups of rats	Total exposure to NO_2		Se status	Recovery
		ppm	ppm days		
1ppm 28d 6h/d 5d/wk	I (4x8rats)	28	5	Se^+/Se^-	0/48h
10ppm 28d 6h/d 5d/wk	II(4x8rats)	280	50	Se^+/Se^-	0/48h
5ppm 5d 6h/d	III(4x8rats)	25	6.25	Se^+/Se^-	0/48h
50ppm 30min	IV(2x8rats)	1.04	1.04	Se^+/Se^-	0
Controls	V(2x8rats)	0	0	Se^+/Se^-	0

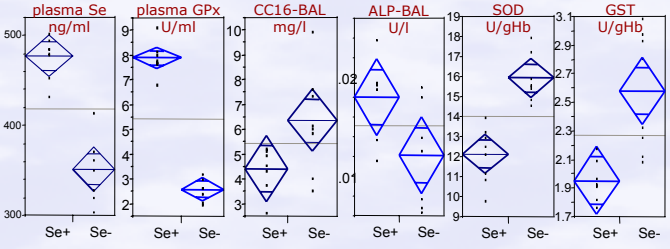
Se^+: diets with normal selenium content
Se^-: diets with low selenium

Along standard measurements of lipid peroxidation (TBARS, chemiluminescence), antioxidative enzymes (GPx, SOD, GST, Cp), lung damage (alkaline and acid phosphatases), lung permeability (total protein, albumin) and inflammation markers (cell populations), new biomarkers such as CC16 (Clara cell protein) were also determined, both in serum and in broncho-alveolar lavage fluid (BAL).

Results

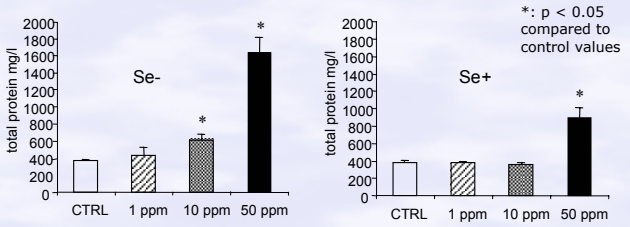
• Selenium status did not only significantly influence GPx, SOD, GST, and even ALP activity prior to any NO_2 inhalation experiments, CC16 also showed unexpected changes, decreasing following selenium supplementation (Fig. 1).

Figure 1: Changes in plasma and BALF composition according to Selenium status before NO_2 inhalation.



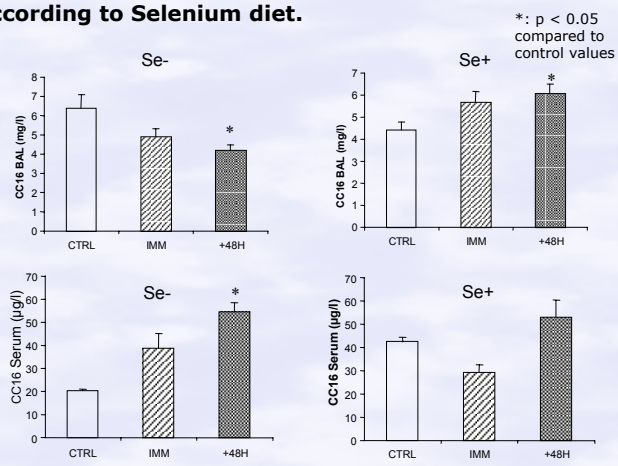
• The protective role of normal selenium status with respect to NO_2 lung toxicity was evident both for long-term and acute exposures (Groups II and IV), as the increase in BALF-total proteins (and corresponding decrease in serum), indicating increased lung permeability, was significantly more pronounced in selenium-deficient animals compared to rats receiving adequate selenium (Figure 2).

Figure 2: Total protein concentrations found in BALF immediately following NO_2 inhalation experiments according to Selenium status.



• Serum CC16 further confirmed its key role as an early marker of increased lung permeability during the various inhalation experiments (Figure 3).

Figure 3: CC16 mirror levels in BAL and serum following exposure to 1ppm NO_2 for 28 days and according to Selenium diet.



Conclusions

• These findings confirm the important role of antioxidant status, and selenium status in particular, in the lung's own defense mechanisms such as the integrity of the alveolo-capillary barrier and the production of anti-oxidants like CC16 when challenged by air pollutants such as NO_2.

• However, in view of the unexpected decreased CC16 secretion observed in selenium-supplemented rats, further corroborated in the human studies of the BIOART project, one should exert caution when advocating selenium supplementation.

• Further studies on the complex interactions of selenium and CC16 in their protective antioxidant properties in the lung are required as these would shed light on the important fields of lung disease, asthma and allergy.

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These studies are supported by the European Union (BIOART project IC15CT980336)

UCL

EXPAH: Risk assessment of the air pollution mixtures - `*in vitro* study´

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AIRNET
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AIMS

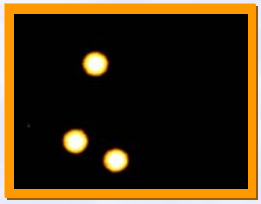
Assessment of genotoxicity of the complex mixture of organic compounds (extractable organic matter, EOM) associated with respirable urban air particles (particulate matter, PM<10 μm) in human cells cultivated *in vitro*.

Air pollution mixture

extractable organic matter (EOM) was prepared by dichloromethane extraction of PM10 collected during winter and summer seasons by high volume samplers (HiVol) in three European countries, Czech Republic (Prague, localities **PRG-SM** and **PRG-LB**), Slovak Republic (**Košice**) and Bulgaria (**Sofia**). For evaluation of biological activities of EOMs *in vitro*, EOMs were re-dissolved in DMSO

AIR POLLUTION

Locality	Season	PM10 (μg/m³)	EOM (μg/m³)
PRG-LB	summer	**26.39**	**3.72**
	winter	**38.97**	**10.86**
PRG-SM	summer	**36.91**	**4.96**
	winter	**62.59**	**14.93**
KOŠICE	summer	**24.30**	**1.67**
	winter	**57.99**	**15.30**
SOFIA	summer	**29.72**	**3.95**
	winter	**89.88**	**24.60**

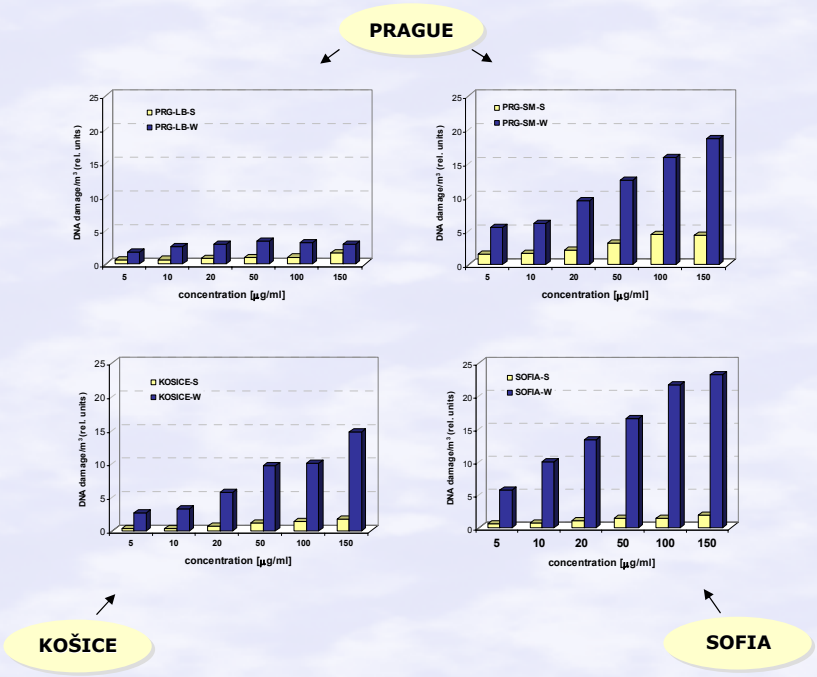


„Nuclei" of control, unexposed cells. DNA is stained with fluorescent dye. and analysed by a fluorescent microscope (magnification 250x)

2 h exposure of human cells to EOM

„Nuclei" of human cells exposed *in vitro* to the complex mixture of organic compounds associated with air pollution. DNA damage induced by EOM resulted in DNA migration ". Damaged cells look like „comets".

GENOTOXICITY OF AIR POLLUTION



Summary

EOM induced dose-dependent increase of DNA damage in human cells *in vitro*.

Highly significant differences in *air pollution genotoxicity* (EOM μg/m³) were detected between seasons (winter *vs.* summer) and localities.

Air pollution genotoxicity rose in order:
summer: PRG-LB~KOŠICE~SOFIA<PRG-SM;
winter: PRG-LB<KOŠICE<PRG-SM<SOFIA;

Oxidative DNA damage does not seem to be a dominant DNA lesion induced by EOM.

Acknowledgement

The authors acknowledge the European Commission 'Quality of life and management of living resources' programme (QLK4-CT-2000-00091) for financial support.

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Pulmonary epithelial integrity in children
– relationship to swimming pool attendance and ambient ozone exposure

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Objective

The aim of the present project was to measure lung function and possible changes in serum levels of Clara cell protein (CC16) in relation to ambient ozone exposure and other environmental factors. CC16 is a new biomarker of early adverse effects on the airways.

Subjects and Methods

Based on a larger study in November 2001, 57 healthy children (33 boys and 24 girls) with a mean age of 10.8 ± 0.4 years, and with no history of respiratory or kidney disease were reexamined in May 2002. Subjects who reported pollen allergy and/or who had a FEV1 <80% of the predicted value in November were not invited. All lung function tests were performed by the same nurse, and blood was sampled before and after 2 hrs light outdoor exercise Ozone exposure was estimated as the total exposure measured between 7 am until the second blood sample was taken. Mean exposure dose was estimated by assuming an exposure level of 50% of the outdoor concentration during time spent indoors. Information on exposure to chlorine in pools was obtained via a questionnaire.

Results

FEV1 was significantly higher after exercise than before both in children who had regularly attended chlorinated swimming pools and in children not swimming (Table 1). The FEV1 in swimmers before and after exercise was lower than in non-swimmers, p<0.05. The mean daytime outdoor level of ozone during the study ranged from 77–116 µg/m^3, and estimated individual exposure varied from 352–914 µg/m^{3}hour. The difference (D) in CC16 was defined as afternoon value - morning value. On average these values were two hours separate, range 1.5–3.0.

Regardless of inclusion of all children or only those not attending chlorinated swimming pools, there were no consistent differences between CC16 levels in serum before (S1) and after exercise (S2) and the difference was not statistically significant. However, among children regularly swimming in chlorinated swimming pools (n=21), there was considerably less difference (S2-S1) in CC16 levels than among non– swimmers (n=33), p<0.001, t- test). The average CC16 level both before and after exercise was lower than in non-swimmers, Wilcoxon W p<0.01 (Table 2). When the relationship between ozone exposure and CC16 levels were examined, no significant correlations were found. However, when S2 was considered, a correlation coefficient of r= 0.18 was found (p=0.20) when all individuals were included. If swimmers were excluded, r was =0.32 (p=0.07).

Conclusion

This study in Swedish school children did not show a statistically significant relationship between CC16 levels in serum and ambient ozone exposure. Lower CC16 levels and less variation in CC16 values before and after exercise among children regularly attending chlorinated swimming pools may be explained by a somewhat decreased pool of CC16 in the Clara cells in the lungs of these children. Such a decreased pool may be a result of repeated release of CC16 and other epithelial protective proteins as a result of toxic insult to the epithelium of the lung. Such an interpretation is supported by the observation (*Carbonnelle S et al, Bio-markers 2002;6:464-78*) that disturbances of the lung epithelial barrier may occur in children exposed to elevated levels of airborne NCl_3 when attending chlorinated swimming pools.

Acknowledgement

Financial support was received from the European Commission (*HELIOS project, QLK4-1308*), the Swedish Environment Protection Agency, and FORMAS.

Table 1. Forced expiratory volume during one second (FEV_1) before (S1) and after (S2) outdoor exercise in swimmers and non-swimmers.

Children	FEV_1 (S1)	FEV_1 (S2)	Paired t-test
Swimmers, n= 22	2.07 ± 0.25	2.11 ± 0.25	p<0.001
Non swimmers, n= 34	2.25 ± 0.32	2.29 ± 0.33	p<0.004
All, n=56	2.18 ± 0.31	2.22 ± 0.32	p<0.001

Table 2. CC16 µg/l in children before (S1) and after (S2) exercise.

Category	CC16 µg/l (S1)	CC16 µg/l (S2)
Swimmers, n= 21	5.7 ± 2.4	5.2 ± 1.8
Non swimmers, n= 33	8.2 ± 2.8	8.0 ± 2.6

Statistically significant difference between swimmers and non swimmers p<0.01

AIRGENE STUDY GROUP

AIR POLLUTION AND INFLAMMATORY RESPONSE IN MYOCARDIAL INFARCTION SURVIVORS: GENE-ENVIRONMENT INTERACTION IN A HIGH RISK GROUP (AIRGENE)

INTRODUCTION

Epidemiological research during the last decade has indicated that exposure to air pollution at the levels presently measured in European urban environments is associated with an increase in mortality and also with a variety of health conditions, including emergency room visits and hospital admissions for respiratory and cardiovascular diseases. Particulate matter (PM) appears to be the air pollutant most consistently associated with adverse health outcomes.

Although the toxicological mechanism has not yet been established, the small size fraction of ambient aerosols, measured as PM10 (particles with an aerodynamic diameter less than 10μm) or PM2.5 (less than 2.5μm), rather than the larger particles is considered to be responsible for most of the health effects. The number of concentrations of ultrafine particles (0.01 to 0.1μm) is hypothesised to be of particular concern.

It is widely accepted that cardiovascular disorders, especially coronary heart disease, are the most prevalent chronic health conditions affecting both sexes in the western world, entailing enormous health care costs.

STUDY SETTING

Study area: Athens, Augsburg, Barcelona, Helsinki, Rome, Stockholm
Study period: May 2003 – April 2004
Study population: 1200 myocardial infarction survivors
Measurements: Ultrafine particle concentrations will be measured at a central site in each city and traditional air pollution concentrations will be obtained from the local air hygiene networks. Inflammatory markers and single nucleotide polymorphisms will be determined in a core laboratory.
Statistical analyses: Time-series methods; gene-environment interactions will be analysed for subgroups

AIMS OF THE STUDY

- To assess the inflammatory response in association with ambient concentrations of air pollution in myocardial infarction survivors in 6 European cities.
  - To determine dose-response relationships between air pollutants and biomarkers of systemic inflammation in myocardial infarction survivors.
  - To compare the inflammatory response of ultrafine particles to the response of traditional air pollutants.
- To define susceptible subgroups of myocardial infarction survivors based on genotyping.
  - To determine the role of the variation in genes of inflammatory responses by assessing the gene-environment interactions for air pollution exposures.
  - To provide insight into the mechanisms leading from exposure to ambient air pollution to early biological effects in high-risk populations.

APPLICATION OF RESULTS

To provide information that will facilitate the development of appropriate public health strategies to reduce the negative effects of ultrafine particles and traditional air pollutants on the exacerbation of cardiovascular disease.

Germany: Peters A., Brueske-Hohlfeld I., Cyrys J., Henneberger A., Ibald-Mulli A., Illig T., Kirchmair H., Kolz M., Loewel H., Meisinger C., Rueckerl R., Schaffrath Rosario A, Wichmann HE., Koenig W., **Italy:** Forastiere F., Picciotto S., Perucci C., Pistelli R., Santarelli P., **Finland:** Pekkanen J., Lanki T., Tiittanen P., Salomaa V., Eriksson J., Kulmala M. Aalto P. Paatero P., **Sweden:** Bellander T., Nyberg F., Berglind N., Pershagen G., **Spain:** Sunyer J., Marrugat J., Jacquemin B., **Greece:** Katsouyanni K., Chrysohoou C., Panagiotakos D., Antoniades C.

EXPAH Effects of polycyclic aromatic hydrocarbons (PAHs) in environmental pollution on exogenous and endogenous DNA damage – oxidative damage

R Singh[1], B Kaur[1], P B Farmer[1], R J Sram[2], I Kalina[3], T A Popov[4], S Garte[5], E Taioli[5]

E.U. Contract QLK4-CT-2000-00091

AIRNET A Thematic Network on Air Pollution and Health

NERAM Network for Environmental Risk Assessment and Management

Introduction

The project has been evaluating the hypothesis that PAHs are the major source of genotoxic activities of organic mixtures associated with air pollution. The relationship between exogenous DNA damage caused by exposures to these genotoxic compounds and oxidative DNA damage has been evaluated in different human populations exposed to PAHs. The populations monitored were traffic police from Prague (Czech Republic), Kosice (Slovak Republic) and Sofia (Bulgaria), which also included bus drivers. As measures of oxidative DNA damage we have determined 8-oxo-2'deoxyguanosine (8-oxodG) by liquid chromatography-tandem mass spectrometry (LC/MS/MS) and malondialdehyde-2'deoxyguanosine (M_1dG) by the immunoslot blot assay.

8-oxo-2'-deoxyguanosine

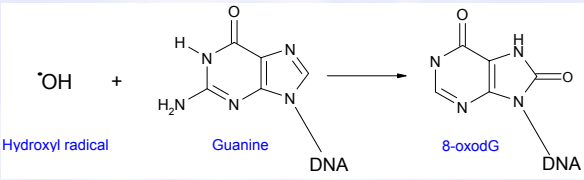


Figure 1 Formation of 8-oxodG by the reaction of hydroxyl radicals with guanine in DNA

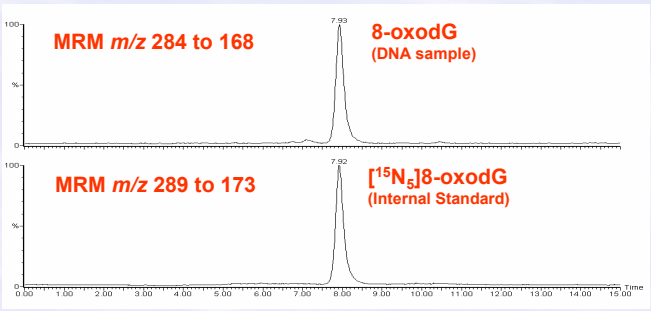


Figure 2 Determination of 8-oxodG in blood DNA samples using LC/MS/MS

Malondialdehyde-2'-deoxyguanosine

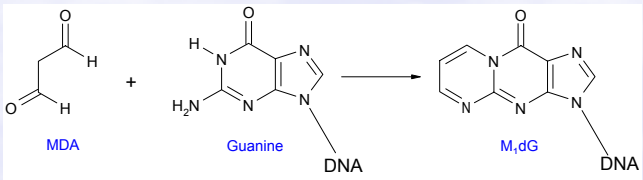


Figure 3 Formation of M_1dG by the reaction of malondialdehyde (MDA) with guanine in DNA

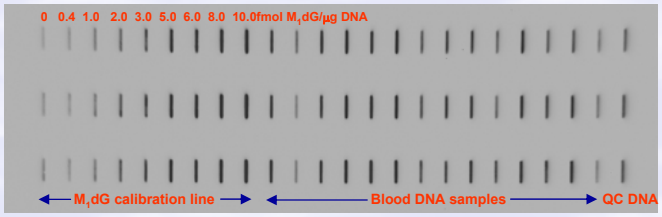
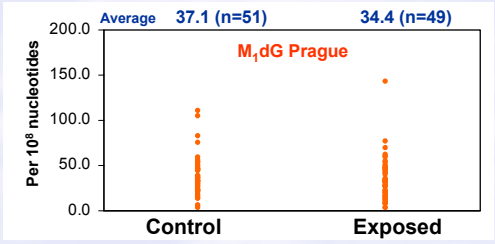
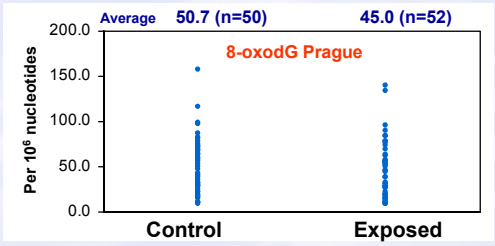
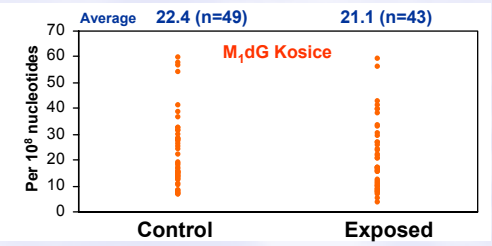


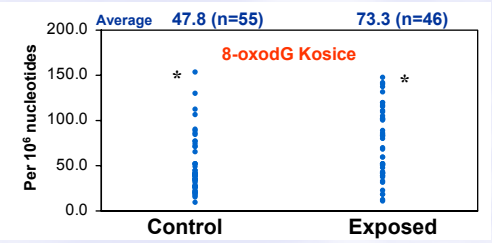
Figure 4 Determination of M_1dG using the immunoslot blot assay

Results









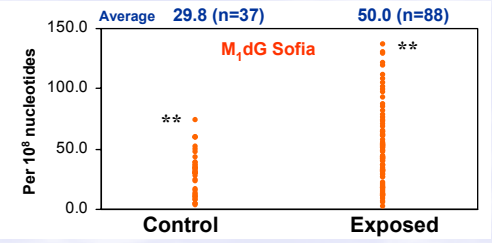


Figure 5 The levels of M_1dG and 8-oxodG in control and exposed groups from Prague, Kosice and Sofia (Statistical analysis was performed using the Student *t*-test).

Conclusions

- There was no significant difference in the level of M_1dG between control and exposed groups from Prague and Kosice and the level of 8-oxodG between control and exposed groups from Prague.
- There was a significant difference (*p = 0.0003) in the level of 8-oxodG between control and exposed groups from Kosice. There was also a significant difference (**p = 0.0005) in the level of M_1dG between control and exposed groups from Sofia.
- The average level of M_1dG was lower in control and exposed groups from Kosice compared to those from Prague and Sofia ($p < 0.0001$).

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5 Genetics Research Institute (ONLUS) Milan, Italy (S. Garte, E. Taioli)

EXPAH: CHROMOSOMAL ABERRATIONS BY FLUORESCENCE IN SITU HYBRIDIZATION (FISH)- BIOMARKER OF EXPOSURE TO CARCINOGENIC PAHs

R. J. Sram[1], O. Beskid[1], B. Binkova[1], Z. Dusek[1], Z. Smerhovsky,[1] I. Kalina[2], T. A. Popov[3], P. B. Farmer[4]

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AIM: To evaluate the effect of ambient air exposure to PAHs on biomarkers of effect - cytogenetic parameters by FISH

STUDIED GROUPS

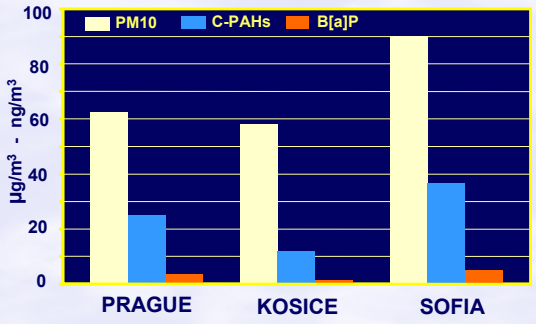
PRAGUE	Exposed :	Policemen
	Controls:	Volunteers > 90 % indoors
KOSICE	Exposed :	Policemen
	Controls:	Volunteers > 90 % indoors
SOFIA	Exposed :	Policemen Bus drivers
	Controls:	Volunteers > 90 % indoors

METHODS

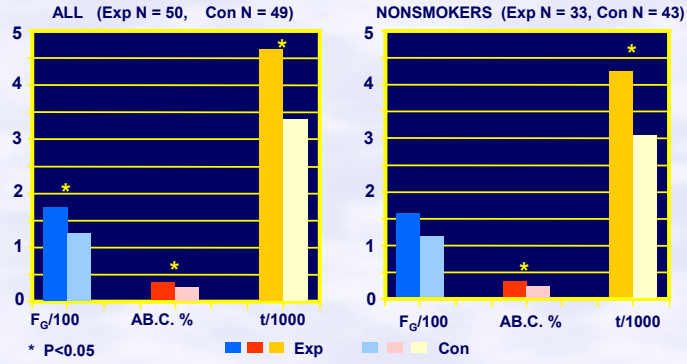
FISH ANALYSIS	1000 metaphases/subject
	Chromosome painting - #1 and #4 (Cambio, UK)
	Metasystem
STATISTICS	Student´s t-test

RESULTS

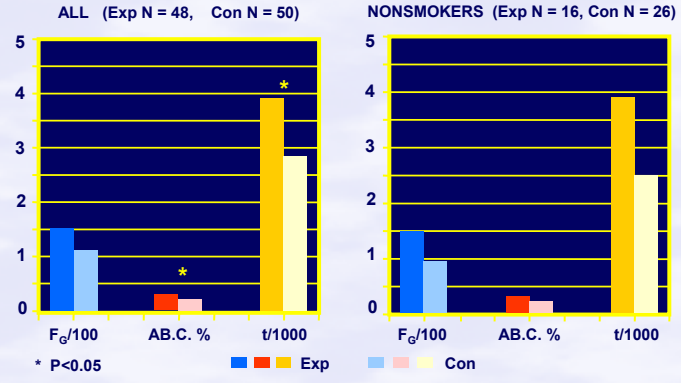
EXPOSURE TO AIR POLLUTANTS DURING WINTER SEASON



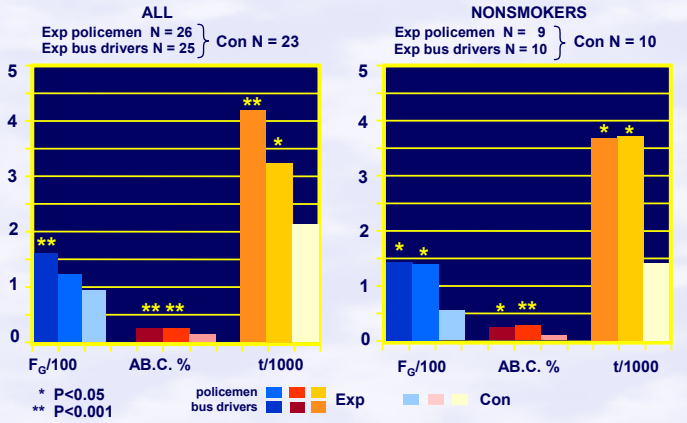
CYTOGENETIC PARAMETERS BY FISH - PRAGUE



CYTOGENETIC PARAMETERS BY FISH - KOSICE



CYTOGENETIC PARAMETERS BY FISH - SOFIA



CONCLUSIONS

Ambient air exposure to carcinogenic PAHs significantly increased FISH cytogenetic parameters in nonsmoking policemen and bus drivers from the center of the city of Prague and Sofia

Chromosomal aberrations detected by FISH seem to be a sensitive biomarker of exposure to carcinogenic PAHs *

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Acknowledgement
Supported by the EC QLRT-2000-00091 and by the Czech Ministry of Environment VaV/340/2/00

EXPAH: EFFECTS OF POLYCYCLIC AROMATIC HYDROCARBONS (PAHs) ON ENVIRONMENTAL POLLUTION ON EXOGENOUS AND ENDOGENOUS DNA DAMAGE – CZECH COHORT

R.J. Sram[1], B. Binkova[1], O. Beskid[1], I. Chvatalova[1], A. Milcova[1], Z. Stavkova[1], Z. Smerhovsky[1], P. Rössner[2]. P. Rössner Jr.[1], P.B. Farmer[3]

AIRNET
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STUDIED GROUPS

Policemen represent a model group, which is highly exposed to ambient air pollution as well as they spend the most of their working hours outdoors. Therefore the effect of PAHs adsorbed on air particles < 2.5 mm was studied in two groups:

policemen from the center of the City spending daily > 8h outdoors

matched controls working indoors

METHODS

- PAHs: VAPS, Personal monitoring
- Cotinine
- Triglycerids
- Total, HDL and LDL cholesterol
- Vitamins A, C. E, folic acid
- Oxidative damage: 8-oxodG, MDA-DNA adducts
- DNA adducts by ^{32}P-postlabelling
- Protein p53 and p21
- Chromosomal aberrations: Conventional, FISH
- Micronuclei
- Genetic polymorphisms: CYP1A1, GSTM1, GSTP1, GSTT1, NAT2, EPHX, MTHFR, MS, p53, XRCC1, XPD, hOGG1

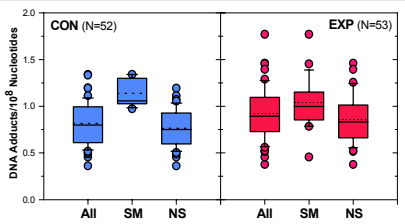
PAHs CONCENTRATIONS Personal monitoring

(median and range)

Group	N	Age (years)	B[a]P ng/m^3	carcPAHs ng/m^3
EXPOSED	53	31.6 ± 7.2	1.6 (0.3 - 8.7)	9.7 (3.1 - 58.2)
Smokers	19	32.9 ± 7.0	1.6 (0.3 - 7.5)	10.8 (3.1 - 43.6)
Nonsmokers	34	30.9 ± 7.3	1.5 (0.3 - 8.7)	8.7 (3.1 - 58.2)
CONTROLS	52	29.6 ± 9.1	0.8 (0.3 - 2.8)	5.8 (3.1 - 19.3)
Smokers	7	37.6 ± 14.2	0.3 (0.3 - 1.4)	3.3 (3.1 - 8.2)
Nonsmokers	45	28.3 ± 7.6	0.9 (0.3 - 2.8)	6.1 (3.1 - 19.3)

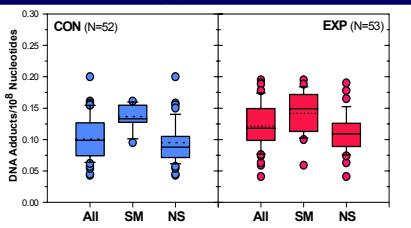
RESULTS

TOTAL DNA ADDUCTS



EXP vs CON: P=0.065
Smokers vs Nonsmokers within groups: P<0.05

„LIKE“ B[a]P-DNA ADDUCT



EXP vs CON: P<0.01
Smokers vs Nonsmokers within groups: P<0.01

MULTIPLE REGRESSION ANALYSIS: DNA - PAH adducts

Model	Coefficients	Unstandardized Coefficients B	Sig.	95 % Confidence Internal for B		R	Adjusted R square	Sig.
				Lower bound	Upper bound			
1	(Constant)	1.072	0.000	0.977	1.167	0.54	0.26	0.000
	Non-exp. + non-smok	-0,277	0.000	-0.389	-0.164			
	Exp. + non-smok.	-0.211	0.001	-0.332	-0.090			
	XRCC1 (+/+)	-0.124	0.012	-0.220	-0.028			
	XPD-23 (+/+)	0.149	0.022	0.022	0.276			
Exploratory model - whole set of variables								
2	(Constant)	1.203	0.000	1.080	1.326	0.54	0.26	0.000
	Non-exp. + non-smok	-0,280	0.000	-0.394	-0.167			
	Exp. + non-smok.	-0.237	0.000	-0.359	-0.114			
	XPD-23 (-/-)	-0.122	0.013	-0.218	-0.027			
	Vit. C (µmol/L)	-0.001	0.017	-0.002	0-000			
Exploratory model - XRCC1 excluded								
3	(Constant)	1.040	0.000	0.932	1.149	0.54	0.27	0.000
	Non-exp. + non-smok	-0.286	0.000	0,397	-0.175			
	Exp. + non-smok.	-0.199	0.001	-0.319	-0.080			
	XRCC1 (+/+)	-0.111	0.025	-0.207	-0.014			
	GSTM1 (null)	0.095	0.040	0.005	0.186			
Exploratory model - XPD-23 excluded								

MULTIPLE REGRESSION ANALYSIS: Percentage of aberrant cels (FISH)

	Bulky DNA Adducts	B[a]P-like DNA adducts	MDA -DNA Adducts
Micronuclei /1000 cells	0.190	0.004	0.180
% AB.C. (conventional)	0.623	0.871	0.590
% AB.C. (FISH)	0.069	0.045	0.807
F_G /100	0.086	0.027	0.916
t/1000	0.114	0.048	0.848

P- vaues for ANOVA, Kruskal-Wallis tests, Pearson and Spearman correlation coefficients

EFFECT OF DNA ADDUCTS ON CYTOGENETIC ENDPOINTS

Model	Coefficients	Unstandardized Coefficients B	Sig.	95 % Confidence Internal for B		R	Adjusted R square	Sig.
				Lower bound	Upper bound			
6	(Constant)	-0.283	-0.019	-0.518	-0.048	0.58	0.29	0.000
	Age (years)	0.012	0.000	0.007	0.017			
	CYP1A*2C (Ile/Val) (+/+)	0.144	0.024	0.020	0.268			
	B[a]P-like adducts/10+08 ncls	1.399	0.018	0.251	2.548			
	EPHX (high activity)	-0.106	0.036	-0.205	-0.007			
	Folates (nmol/l)	-0.004	0.059	-0.008	0.000			
	p53 mspI (+/+)	-0.324	0.098	-0.709	0.061			

PIN=0.10, POUT=0.15

SUMMARY

1. Predictor of total DNA adducts was smoking, vitamin C, polymorphisms of GSTM1, EPHX exon 4, XRCC1 and XPD exon 23.
2. Predictor of „like“B[a]P- derived DNA adduct was smoking, exposure to carcinogenic PAHs, and polymorphisms of GSTM1, XRCC1, MTHFR.
3. Predictor of the frequency of aberrant cells by convention method was polymorphisms of CYP1A1, XPD exon 6 and XPD exon 23.
4. Predictor of the frequency of translocations by FISH was age, smoking, exposure to carcinogenic PAHs, folate, polymorphisms of CYP1A1, GSTM1, GSTP1, EPHX, p53, mspI and MTHFR.

CONCLUSIONS

1. Results of DNA adducts analysis and FISH analysis indicate that city policemen represent a group of the increased genotoxic risk.
2. DNA adducts highly correlated with cytogenetic endpoints by FISH.
3. Polymorphism of metabolic and DNA repair genes identify subjects susceptible to DNA damage by exposure to carcinogenic PAHs.

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Acknowledgement
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SHORT-TERM EXPOSURE TO PM2.5, PM1 AND BLOOD COAGULATION IN HUMANS.

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Background

Health effects of short- and long-term exposure to airborne particulate matter (PM) are only partially known. In addition to respiratory effects, effects on the cardiovascular system have been repeatedly described. Epidemiological studies consistently showed an increased cardiovascular mortality and increased rates of cardiovascular hospital admissions following acute episodes of urban pollution. After acute exposure, the following changes in healthy adult subjects have been described: increased plasma viscosity; elevated reactive protein C; heartbeat frequency alterations; increased risk of implanted cardioverter-defibrillator discharges (implying a response by the autonomous nervous system).

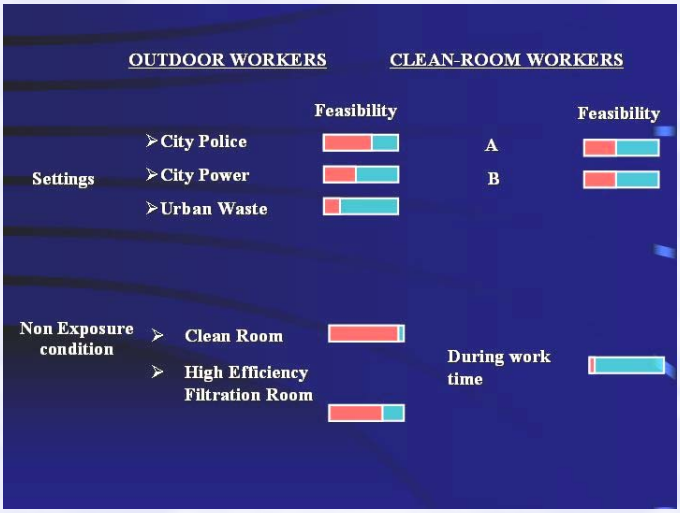
Sources and images of urban pollution

Study Hypothesis

Two main explanatory hypotheses have been proposed: 1) the particulate matter deposited in the airways elicits the release of inflammatory mediators capable of affecting the cardiac function and the coagulation process; 2) the smallest particles (aerodynamic diameter <100nm) reach the blood stream from the alveolar space and directly affect the coagulation processes. In addition to particle size, the number of inhaled and deposited particles appears important (although often overlooked in favour of mass measures). It should be noted that, given the same particles mass, the finest particles occupy a more extended surface.

Design

We designed a study in order to evaluate acute effects of exposure to PM on the coagulation process in humans. We planned to examine 50 healthy adult subjects, age 20-55 years, smokers and non-smokers, free from blood coagulation (possibly) related disorders and living in urban polluted areas. The candidates hold jobs in clean industrial environments, virtually free from PM exposure due to production needs (e.g., semiconductors, and pharmaceutical). Their basal blood coagulation function is assessed before the work shift. They later spend six hours in clean rooms, PM "free". Coagulation markers are then assessed at the end of the "non-exposure" period. Subsequently, the outdoor/indoor exposure period (commuting, social and domestic life) begins. For a 14-hour period, PM1 and PM2.5 exposure is evaluated through 1) selection of granulometric fraction and gravimetric analysis (mass definition); 2) elemental analysis (inorganic elements composition); ultra-fine particle counting (from which to derive an exposure estimate). At the end of the exposure period, a further blood coagulation markers assessment is planned.



Two possible study designs. The clean-room workers approach was selected. Feasibility: red, poor; blue, good.

Perspectives

Analyses explore possible changes of coagulation function markers in relation to exposure/non-exposure periods, and to varying levels/types of exposure. This observation may provide a plausible biological explanation for the epidemiologically established link between air pollution and acute myocardial infarction, with obvious bearing on prevention policy and primary health care.

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THE METAL CONTENT OF AIRBORNE PARTICLES: APPLICATION TO EPIDEMIOLOGICAL RESEARCH

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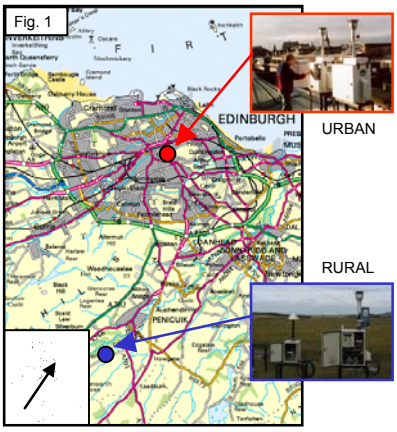
Introduction

- This poster describes the first long-term study in the UK of the quantitative relationship between elevated respiratory & cardiovascular mortality and morbidity rates and the variance in daily metal composition of PM_{10} in urban background air.
- **Research question addressed:** Do observed and/or modelled variations in daily metal concentrations explain additional variance in health outcomes compared to PM_{10} mass concentration alone?

Interpretation of atmospheric science aspects of these data are presented separately

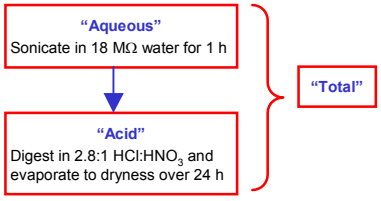
Sample collection

Daily PM_{10}, $PM_{2.5}$, and Black Smoke samples collected for 1 year at an urban background and rural location (Fig 1.)



Metal Analyses

Analysis for Fe, Cu, Ni, V, Zn, Mn, Cd, Cr, As, Ti and Pb by ICP-MS in two sequential extractions of each sample



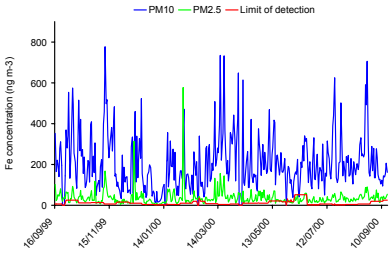


Fig 2 Example time series of daily Fe concentrations 1999-2000

Experimental design

1999-2000: Measure metal concentrations in PM_{10}, $PM_{2.5}$, BS

↓

Poisson regression analyses of 1999-2000 PM_{10}, $PM_{2.5}$, BS & [metal]

↓

Compute & categorise air mass trajectories 1999-2000
For each trajectory category: compute [metal] per particle mass enrichment factors (EF)

↓

Compute trajectories 1992-2000
Multiply EF & observed PM_{10}, BS to estimate [metal] time series

(Also alternative estimate of [metal] time series using multiple regression)

↓

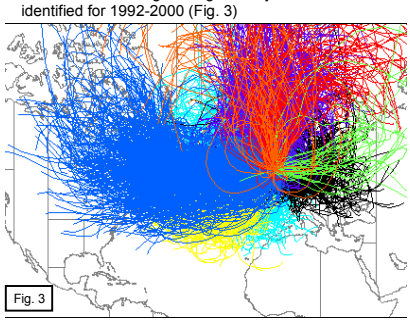
Poisson regression analyses of 1992-2000 PM_{10}, $PM_{2.5}$, BS & [metal]

↓

Do analyses using metrics of metal exposure explain more of the variance in health outcome than PM_{10} metrics?

Influence of air-mass back-trajectory on metal content

- Daily 5-day air-mass back trajectories were calculated and clustered hierarchically using Euclidian squared distance and average linkage. 8 major clusters were identified for 1992-2000 (Fig. 3)



- PM_{10} and $PM_{2.5}$ mass concentrations in air masses from east/central Europe, or centred on the UK were, respectively, 40-40 % or ~25 % higher compared with air masses from the N, W or SW.
- Water soluble metal concentrations of UK and central Europe air masses were up to double those from the W, SW and N
- Metal enrichment factor (ng μg^{-1}) differed significantly between air-mass back-trajectory cluster for most metals (Table 1, Kruskal-Wallis P values).

TABLE 1

	Aqueous	Total
Fe	**0.012**	**<0.001**
Cu	**0.014**	**<0.001**
Ni	**<0.001**	0.31
V	**<0.001**	**<0.001**
Zn	0.10	0.26

Retrospective extrapolation of [metal] time series

- Metal enrichment factors showed large variation within trajectory clusters, hence cluster analysis did not provide very precise estimates for retrospective extrapolation.
- Therefore we also used forward stepwise multiple regression to predict EF from combination of trajectory co-ordinates and local meteorological & co-pollutant observations:

Metal	Trajectory cluster	Regression A	Regression B
Fe	0.07	0.02	0.14
Cu	0.18	0.07	0.49
Ni	0.05	0.10	0.24
V	0.19	0.23	0.39
Zn	0.09	0.07	0.25

- **Table 2** R^2 indicating proportion of variance in EF explained by prediction methods involving trajectory clusters; multiple regression using trajectory co-ordinates (A); and coordinates plus local meteorological and co-pollutant variables (B). n~365.

Epidemiological analyses

- Generalised additive Poisson regression models were used for the time series analyses.
- To reduce multiple testing, subset of 60 'primary' analyses defined: cardiovascular admissions, respiratory deaths and all-cause deaths in subjects aged > 65 in relation to Fe, Cu, Ni, V, Zn in aqueous and total (water plus acid) filter extracts of PM_{10}

Epidemiological results

- We found a significant association between cardiovascular admissions and total PM_{10} (95% confidence limits of +1,+6 (P<0.01) for percent changes in relative risk associated with a 10 $\mu g\ m^{-3}$ range of total PM_{10}, consistent with previous analyses).
- Similar associations were noted for some of the metal fractions using both extrapolation techniques. However, further multiple Poisson regressions showed that metal effects were no longer significant after adjusting for total PM_{10}.

Conclusions

- Using an objective statistical analysis of air mass back trajectories **we did not find that extrapolated concentrations of Fe, Ni, V, Cu, or Zn explained more of the variance in 3 primary health outcomes than PM_{10} mass concentration alone**.
- Further analyses using multiple regression techniques improved the precision of our exposure estimates but still did not show evidence of stronger effects on health from specific metal fractions than from total PM_{10}.
- At present these data do not provide evidence justifying separate regulation of metal constituents of urban PM_{10}.

Acknowledgement

This work was supported by the UK Department of Health

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PARTICLE SPECIES AND DAILY MORTALITY AND ADMISSIONS IN THE WEST MIDLANDS, UK

S.A. Bremner, et al., St. George's Hospital Medical School

Qualitative differences in particulate air pollution at different locations throughout Europe (RAIAP)

Flemming R. Cassee, Henk J.Th. Bloemen, A. John F. Boere, Paul H.B. Fokkens, Daan L.A.C. Leseman, Gergio Catani, Bjørn V. Johansen, Tadeusz Halatek and Erik Dybing

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Introduction

Given the widely different prevalence rates of respiratory allergies and asthma between the countries of Europe and the substantial exposure to ambient particles in urban environments, the EU project Respiratory Allergy and Inflammation Due to Ambient Particles (RAIAP) aimed to relate the chemical composition of collected ambient particulate matter (PM) to different health end-points. With the aid of high-volume cascade impactor (HVCI), coarse (2.5-10 µm) and fine (0.1-2.5 µm) PM samples were collected in Amsterdam (NL), Rome (I), Lodz (PL), Oslo (N) and the Dutch sea-side background (De Zilk) during spring, summer and winter. The sampling campaign has been successfully performed in the period of March 2001 - April 2002.

Figure 1: RAIAP PM collection device with the HVCI on top of a box with the pump pull the air through the impactor (right panel) and the inside of the HVCI showing the pink PUF with collected PM (black) of the fine mode stage (left panel).

Results

• PM yields were usually higher in winter compared to the other two seasons, which may reflect contributions of combustion processes for heating purposes.

• In addition, higher PM concentrations were observed in Lodz and Rome compared to Oslo and Amsterdam. In general less secondary aerosol was measured in the coarse fraction when related to the fine fraction.

• Significant contrast in chemical composition was observed. Metal concentrations were high in Rome, with the exception of zinc, which was high in Lodz.

• The location in Amsterdam is characterized by relatively high magnesium (Mg) and vanadium (V) levels. As expected, sea-spray aerosol was significantly present in samples taken in Amsterdam and De Zilk.

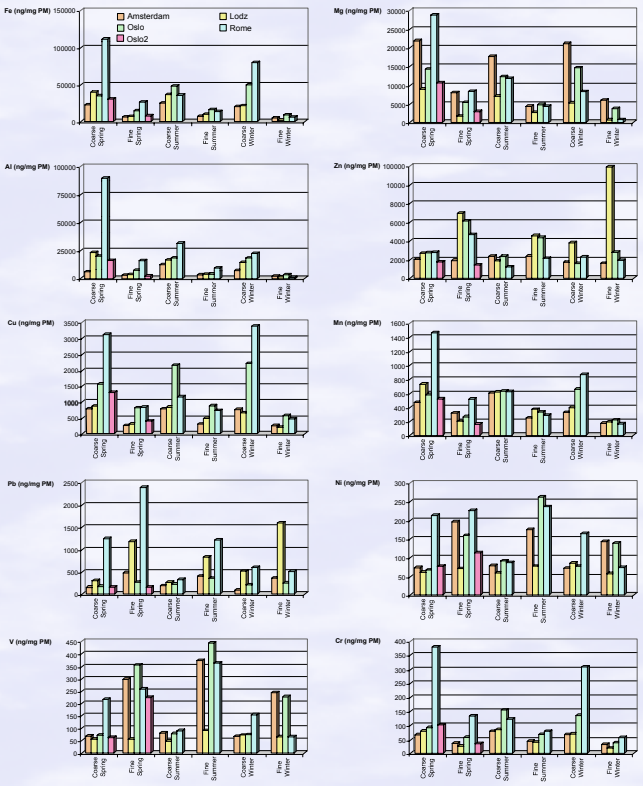

Figure 2: Transition metal contents of PM of spring, winter and summer PM samples from Amsterdam, Lodz, Oslo and Rome.

• Relatively high levels of PAHs were measured in Lodz, in particular during winter season. A more diverse pattern is found for the traffic markers (hopanes ans steranes).

• Traffic markers: Although generally higher amounts are found in the fine fraction, relatively high amounts of steranes were observed for both winter and summer samples of the location in Oslo, as well as the winter sample from Rome and the summer sample from Lodz. This pattern is not reflected in the hopane levels, which seem to be more dominant in both the spring and winter from Lodz, Oslo and Rome.

These data will provide valuable information for *in vitro* an *in vivo* toxicity studies performed within this project.

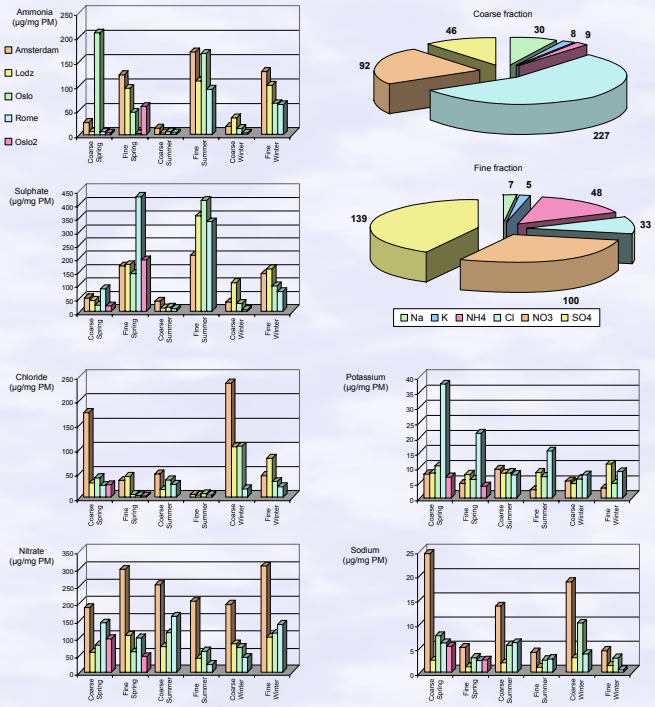

Figure 3: Inorganic contents of PM of spring, winter and summer PM samples from Amsterdam, Lodz, Oslo and Rome. The pie charts are for De Zilk.

SEASONAL TREND OF THE PHYSICO-CHEMICAL CHARACTERISTICS OF PM2.1: A STUDY BY SEM /EDX AND XPS IN AN URBAN AREA OF ROME

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INTRODUCTION

Epidemiological studies have shown a clear association between effects on health and environmental concentrations of particulate matter, especially its fine component, that is, below 2.5 µm (PM2.5). Some studies seem to indicate a larger number of deaths and hospitalizations during summer rather than winter in correspondence with the same particulate exposition.There is wide agreement regarding the hypothesis that the factor which are determinant for the effect of particulate on public health include the chemical composition of particles and their capacity to carry potentially toxic substances (such as organic substances or metallic composts) adsorbed on their surface. The aim of the present research was to study the PM2.1 composition and its physico-chemical characteristics in the city area of Rome over the course of a year by analysing, specifically the role of pollution carrier of carbonaceous particulate particularly for sulphates and organic compounds.

Materials and methods

Particulate was collected between December 2000 and December 2001 at a site near the city centre of Rome, with heavy motor traffic, by an eight-stage cascade impactor (Andersen particle fractionating sampler) with a pre-separator stage able to eliminate particles with aerodynamic diameter > of 10 µm. In this study the particulate matter with aerodynamic diameter ranging from 2.1 to 0.4 µm was utilised. Together with the particulate, and at the same site, acid aerosol was collected using an annular denuder system in order to evaluate the environmental concentration of NO_3^-, NO_2^-, Cl^- and SO_4^{--} ions. Scanning electron microscopy (SEM) equipped with a thin-window system for X-ray microanalysis by energy dispersion spectrometry and photoelectron spectroscopy techniques (XPS) were used. The ion chromatography (IC) was used to measure the environmental concentration of soluble ions.

Results

The X-ray microanalysis data of single particles were analysed using hierarchical cluster analysis (HCA) to classify the particles into groups with similar chemical composition and to determine the principal components of the particulate.It was possible to classify the particulate into four principal groups or particle "clusters": carbon rich particles, soil erosion particles, sulphates, and metallic particles. Figure 1 shows the average abundances of the identified "clusters" calculated in summer and winter.

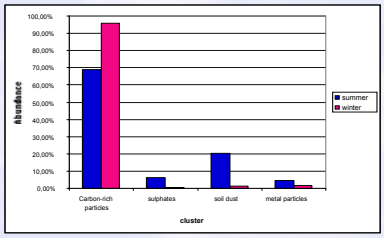

Figure 1. Average abundances of principal components in the PM2.1

Particles from soil or building material erosion (silica, silicates, carbonates) represented 4% in winter and more than 20% in summer because of the alumino-silicates carried by the wind over the sea from North Africa. Sulphates, mainly composed by Ca-sulphates can reach at most 7-8% of the total amount. Carbon rich particles, mainly resulting from vehicular traffic and, during winter, from heating systems, made up 66 to98% of the particulate depending on the season, and was more abundant in winter. Observations performed with high resolution SEM and TEM show that such particles constitute of a large number-hundreds of thousands- of aggregates of carbonaceous microsferules (Figure 2)

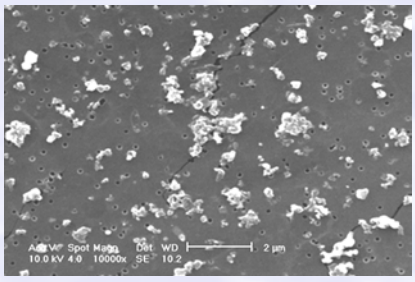

Figure 2. Carbon- rich particle aggregates. SE image (10000x)

On a varying percentage of such particles On a varying percentage of such particles, the X-ray microanalysis showed traces of S and more rarely, of alkaline metals, such as Na and K. This results was attributed to the ability of carbonaceous particles to act as vehicles for sulphur compounds, i.e. ammonium and alkaline metal sulphates, produced by SO_2 photochemical oxidation catalysed by the particles themselves. The XPS spectrum in the S_{2p} region (169-170 eV) confirm that the sulphur composts present in the PM2.1 are constituted essentially by the SO_4^{--} anion. Data for S concentration in PM2.1 obtained by XPS show that such concentrations increase from winter to summer, more than doubling from about 4% in weight of fine particulate to over 9%. (Figure 3a).

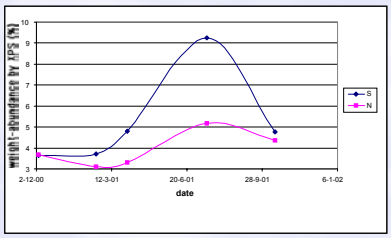

Figure 3a. Trends of sulphur and nitrogen weight-abundance in PM2.1 estimated by XPS

A similar increase is seen in the SEM data for the abundance of sulphate and carbon particles which carry S (Figure 3b). Sulphur and nitrogen abundances, estimated by XPS in the solid phase, appear closely correlated (R=0.88) each other and highly seasonal with an evident maxim in the summer (Figure 3a); this results suggest that the nitrogen observed could be present in the PM2.1 mainly as stable ammonium sulphate.

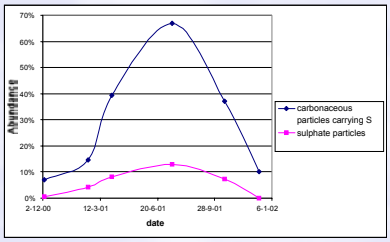

Figure 3b. Seasonal trend of sulphates and carbonaceous particles carrying S obtained by SEM

Moreover sulphate and nitrate ion concentration, determined by IC appear significantly higher during winter and the trend observed in the gaseous phase mirrors the results obtained for the sulphur compounds in the solid particulate phase (Figure 4)

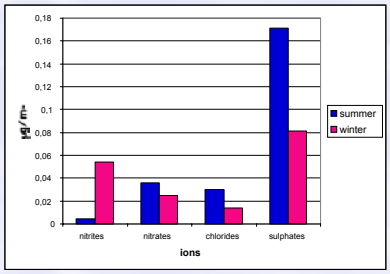

Figure 4. Average concentrations of nitrates, nitrites, sulphates and chlorides identified by IC

The XPS spectrum in the C1s region (286-290 eV) (Figure 5) show the presence of several form of oxidised carbon (organic carbon) driven by graphitic particles.

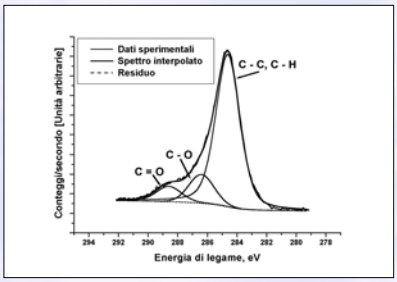

Figure 5. Three different states of Carbon obtained by deconvolution of high-resolution XPS spectra of C1s region

Figure 6 shows the trend of the ratio of Oxcarbon to total Oxygen (Oxcarbon/Oxtotal) together with the oxygenated carbon trend evaluated directly by deconvolution of C signal. Both trends show an increase of the oxygenated carbon in PM2.1 during winter.

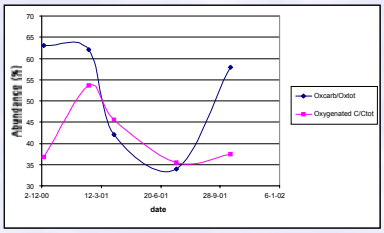

Figure 6. Seasonal trend of the ratio of oxygen bound to the carbon to the total oxygen together with the oxygenated carbon trend

Conclusions

In the urban area of Rome the principal source of pollutants emissions is the vehicular traffic and, during winter, the heating systems, mainly burning fuel or methane gas. The SEM, XPS and IC results obtained in this work seem to indicate that the antropic factors and climate pattern determine the physico-chemical PM2.1 characteristics and particularly the abundance and quality of the pollutants carried by the carbonaceous particles. The antropic factors contribute to the quality of pollutants particularly in winter by a rise of the C-rich particles together with those of the oxygenated carbon observed in the carbonaceous particulate. The climate pattern seems to determine the behaviour of the secondary pollutants carried by the solid atmospheric particulate: in summer, characterised by atmospheric stability, high pressure and humidity, we observed an increasing concentration of sulphate ions and a rise of the abundance of sulphate and carbon particles which carry S

Effects of Rome (Italy) winter urban air particles on monocytic macrophagic RAW 264.7 cell line.

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AIRNET
A Thematic Network on Air Pollution and Health

NERAM
Network for Environmental Risk Assessment and Management

Introduction

Several epidemiological studies have shown statistical associations between exposure to increased particulate matter levels with aerodynamic diameter <10μm (PM10) and increased morbidity and mortality at various geographical urban areas (1,2). In the present study we have used as a model system the macrophage cell line RAW 264.7 and we sought to compare the induction of proinflammatory mediators in the cell-line by Rofa, Carbon Black, fine fraction and coarse fraction of an air sample collected in the city of Rome, during the winter season. We have evaluated cytotoxicity, arachidonic acid (AA) release, and the production of Tumor Necrosis Factor Alpha (TNF-α) induced by the same concentration of the different particles. By comparing the production of proinflammatory mediators induced by all the kinds of particles, we should elucidate the importance of particle core versus the organic compounds and transition metals adsorbed on urban particles.

Materials and methods

The airborne particulate was collected continuously for 15 days, in Rome, Italy, during February 2001. The sampling site was located in the central urban area characterised by moderate or heavy traffic. In winter season the principal source of the atmospheric pollution in this area are the vehicular traffic and heating systems.
The single particles constituting the coarse and fine fractions were characterised by Scanning Electron Microscopy (SEM), equipped with a system for X-ray microanalysis of the elemental determination by energy dispersion spectrometry and the data were analyzed by cluster analysis methods.
The mouse monocyte/macrophage cell line RAW 264.7 was maintained in RPMI 1640 medium supplemented with 100 U/ml penicillin, 100 μg/ml streptomycin, 10% heat-inactivated fetal bovine serum, 2 mM of Glutamax I (complete RPMI 1640 medium). All experiments were performed using RPMI with 1% heat inactivated fetal bovine serum. RAW 264.7 cells were plated at a density of 1.3 x 10^5 cells/well in 96 wells plates. Cell cultures were allowed to adhere overnight, and stimulated with the particles the day after.

Results

The cluster analysis method allowed us to identify in the particulate matter seven groups (clusters) of similar particles in both the coarse and the fine fractions: C-rich particles, Ca-carbonates, Ca-sulphates, silica, silicates, Fe-rich particles and metals (figure 1a,b).
The most significant source of the carbonaceous particulate, C-rich particles,in the urban area of Rome consists of the motor vehicle exhausts. C-rich particles were more abundant in the fine fraction (71.5%) than in the coarse fraction (29.4%).

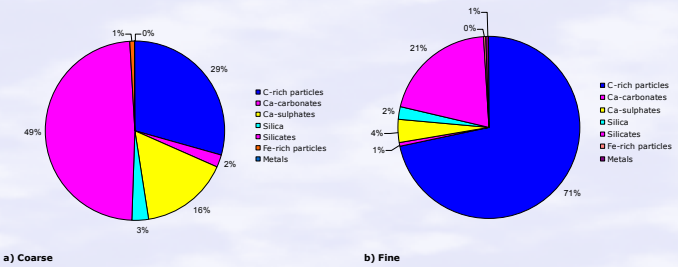

Fig.1. Principal clusters identified by Cluster Analysis in **a)** coarse fraction and in **b)** fine fraction

AA release after 5 h of cell treatment (figure 2) showed that fine fraction induced at 30 μg/ml and 120 μg/ml a significant ($p<0.05$) release of 199 ± 39% and of 208 ± 25.8% respectively. Coarse fraction–induced AA release was not significant. Rofa-induced of AA was lower than the level of both urban fractions, whereas CB had no effect on AA release. LPS at a concentration of 1 μg/ml significantly ($p<0.001$) induced a release of [^{3}H]AA of 280 ± 35.7 % (n=6).

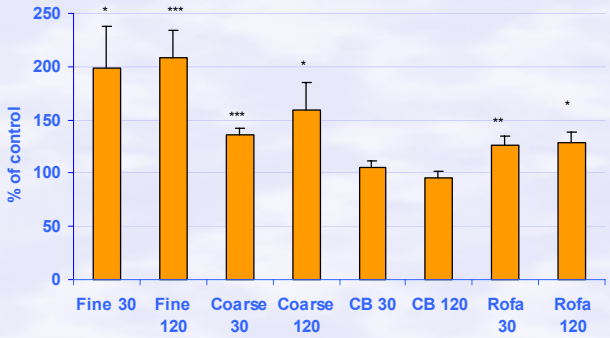

Fig. 2. Effect of Carbon Black, Rofa, Fine and Coarse urban particles on [^{3}H]AA release in RAW 264.7 cells. Cells were prelabelled with [^{3}H]AA and then incubated for 5 h with the particles (30 μg/ml and 120 μg/ml). The radioactivity released by untreated cells was taken as 100%. Values are means ± S.E.M. of six independent experiments assayed in triplicate. Values with asterisk are significantly different from corresponding control cells.

As shown in figure 3, both urban fractions dose-dependently increased TNF-a production at both time point. After 24 h of treatment TNF-α production induced by coarse particles at 30 μg/ml decreased significantly ($p<0.01$). At 120μg/ml the decrease was not significant from 2404 ± 377 pg/ml after 5 h to 1502 ± 263 pg/ml after 24 h.
Fine fraction markedly induced a TNF-α release ($p<0.005$) at 30 μg/ml and ($p< 0.05$) at 120 μg/ml after 5 h of treatment. After 24 h of incubation fine fraction at both concentration did not show any significant decrease of TNF-α production. Carbon black and Rofa both had a lower effect on TNFα release and decreased significantly ($p<0.005$) after 24 h of incubation.

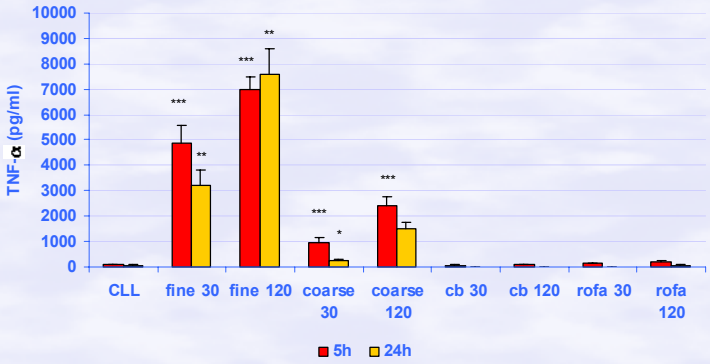

Fig. 3. Effect of Carbon Black, Rofa, Fine and Coarse urban particles on TNF-α production in RAW 264.7 cells.
The cells were incubated in the presence of the particles A: 30 μg/ml and B: 120 μg/ml for 5 h and 24 h.
Values are means ±S.E.M. of six independent experiments assayed in duplicate.
Values with asterisk are significantly different from corresponding control cells.

Conclusions

In conclusion, our data indicate that fine urban particles collected during the winter season in the city of Rome induced in vitro an inflammatory reaction more than the coarse urban particles. The use of carbon black and Rofa, with granulometric size within the range of fine particles evidentiated an inflammatory reaction less marked than that induced by the fine urban fraction, indicating that organic compounds adsorbed on the particles surface are responsible for cytokines and inflammatory mediators production.

1) Katsouyanni, K., Touloumi, G., Samoli, E., Gryparis, A., Le Tertre, A., Monopolis, Y., Rossi, G., Zmirou,D., Ballester, F., Boumghar, A., Anderson, H.R., Wojtyniak, B., Paldy, A., Braunstein, R., Pekkanen, J., Schindler, C. and Schwartz J. 2001. Epidemiology 12, 521-531.

2) Zanobetti, A., Schwartz, J., Dockery, D.W. 2000. Environ. Health Perspect. 108, 1071-77.

European Directives for Air Quality Analysis of the new limits in comparison with asthmatic symptoms in children

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Objectives

To analyse the air quality in Oporto Metropolitan Area (Oporto-MA) according to the previous legislation yet in application, as well as according to the new European Directives, aiming to evaluate i) if risks associated to SO_2 and particles are considered differently by the two analysed legislations; and ii) if the delay on the application of the EU Directives is associated to health risks. Special attention was given to the necessity of reducing pollutant concentrations, mainly of those that were more drastically reduced in European Directives (SO_2 and particles). The incidence of asthmatic symptoms in children was selected as indicator.

Method

Selected sites for air quality monitoring

Site I **(S_I)** is situated about 5 km far from the coastline, in an open area of a suburban industrial zone of Oporto city. It is situated on the west relative to the refinery and petrochemical plants, being influenced mainly by their atmospheric emissions as well as by other industrial emissions transported by prevailing winds from W and NW.

Site II **(S_{II})** is located 6 km far from the coastline in a suburban rural area. This site is not significantly influenced by traffic, urban and industrial emissions in a direct way, being considered with background behaviour for atmospheric pollution in Oporto-MA.

Site III **(S_{III})** is located in a reference area, clearly rural, without significant influences of anthropogenic emissions of atmospheric pollutants.

Analysis of asthmatic symptoms and asthma rates in children living in the area of the selected sites

Written questionnaires similar to those used for ISAAC were completed by the child parents or tutors. The children were studding on primary or secondary schools where the monitoring sites were installed (S_{II} and S_{III}) or at 300 m of the monitoring place (S_I).

Asthmatic children were identified if dyspnea and wheezing was referred in the absence of upper respiratory infections; this first asthma identification was confirmed through tests of bronquic reactivity with methacoline.
Three groups were considered: **children without asthmatic symptoms**, with **asthmatic symptoms** and **with asthma.**

A random sample of 720 children aged 6 to 11 years was analyzed.

Results

Exceedances relative to the previous legislation and to the new European Directives (1999 - 2001)

Legislation		Limits (μgm^{-3})			S_I	S_{II}
Previous	SO_2	All			No	No
	Particles (total)	All			No	No
European Directives	SO_2	Hourly:	350 (1)		No	No
		Daily:	125 (2)		Yes	No
	Particles (PM_{10})	Daily:	50	1st phase (2005) (3)	Yes	Yes
				2nd phase (2010) (4)	Yes	Yes
		Annual:	40	1st phase (2005)	Yes	Yes
			20	2nd phase (2010)	Yes	Yes

(1) Not to be exceeded more than 24 times a year
(2) Not to be exceeded more than 3 times a year
(3) Not to be exceeded more than 35 times a year
(4) Not to be exceeded more than 7 times a year

Rates of asthmatic symptoms and asthma in children

		S_I	S_{II}	S_{III}
Sample size		300	156	264
Age		10-12	6-10	6-10
Sex	Female (%)	60.2	59.5	51.2
	Male (%)	39.8	40.5	48.8
Asthmatic symptons (%)		36.8	20.4	3.5
Asthma (%)		10.3	6.1	1.2

Considering the 95% confidence level, all the rates in Table 2 are significantly different, with the exception of asthma rates for S_I and S_{II} that, according to the sample size of asthmatic children, are not significantly different.

Conclusions

It was concluded that all kind of limits of the previous legislation were obeyed, either for SO_2 or particles. Nevertheless, the limits settled by EU were exceeded both for SO_2 and PM_{10} revealing that even suburban areas have a background concentration of particles that does not guarantee the protection of public health, against to the conclusions obtained through the analysis of the previous legislation. Therefore, it can be concluded that the previous legislation and the EU Directives lead to completely different conclusions about the obedience to limits for the protection of public health.

The results showed that the EU limit for PM_{10} was not obeyed even at the reference site, which suggests that it is exaggeratedly low, being expectable a lot of difficulties for implementing its obedience. Accordingly, effects on asthmatic symptoms and asthma were not observed at the reference site even when the limits were not obeyed. The pollen effect in spring and the traditional viral respiratory infectious of winter were more important than the PM_{10} concentrations, if they belong to the range of the restrictive limits settled by EU Directives, or if they even are slightly higher.

The results showed that SO_2 can influence the rate of asthmatic symptoms, but PM_{10} can influence also the rate of asthma confirmed through the methacoline tests. The background concentration of PM_{10} in Oporto-MA can influence the worsening of asthma in children not guarantying the protection of public health.

Studies involving much more schools should be carried out to increase the sample sizes, aiming to confirm some of the conclusion obtained. Nevertheless, it could be concluded that the risks associated to SO_2 and particles are considered differently by the two analysed legislations, which means that the delay on the application of the much more restrictive EU Directives can be associated to health risks. In spite of the exaggeratedly low EU limit for particles, according to the effects observed on asthmatic symptoms and asthma in children, the concentrations of SO_2 and particles allowed in the previous legislation must be reduced to protect the public health.

LEPAE
LABORATORY OF PROCESS, ENVIRONMENT AND ENERGY ENGINEERING

Universidade do Porto
FEUP Faculdade de Engenharia

Developing Risk-Based Priorities for Reducing Air Pollution in Ukraine

Michael Brody[1], Jane Caldwell[1], Alexander Golub[2]
[1] US Environmental Protection Agency, [2] Environmental Defense

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Ukraine: Current Status

Population in 2000
- 49.5 million

GDP in 2000
- 33.4 billion USD1995

Area
- 604,000 square km

Air Pollution Management in Ukraine

- Based on the Soviet system of standards;
- Too many controlled pollutants;
- No prioritization;
- Almost no economic incentives for private sector to reduce emissions;
- Small level of state environmental protection expenditures.

Programs and Limitations

1980's	• Ambient and Emission standards • Complicated system of pollution permits with too many pollutants
1991	• Pollution fees to create environmental funds • Environmental funds were small, fees did not keep pace with inflation ➢ IMF criticizes "off-budget" funds

Reforms Needed

- Nature and scope of pollution problem needs to be defined;
- Identification and prioritization of risks lacking;
- Efficient risk management approaches to be determined;
- Incentives needed for polluter to reduce emissions. Steps Towards Reform

Steps Towards Reform

- Partnership between USEPA and Ukrainian environmental protection authorities;
- Capacity building project (CPB);
- Use screening human health risk assessment for prioritization;
- Apply western methods, share experiences for risk analysis for Ukraine;
- Utilize experience in other NIS countries.

CBP Draws Upon:

- Analytical tools, information, expertise of the US EPA;
- Approaches used in Europe (e.g., EU Tacis, Denmark) and technical assistance programs in Ukraine;
- Work of multi-lateral organizations such as the Organization of Economic Cooperation and Development (OECD);
- Relevant experience and tools from neighboring countries (Russia, and Poland).

Russian Example for Screening Assessment

- Significant US EPA training in risk assessment;
- Qualified epidemiologists, long-term experience of air dispersion modeling, modest requirements for input data, and clear and understandable results;
- Health risk analysis in Russia- pilot stage 1996-1998;
  - Successively implemented in 6 cities
  - Results were broadly published
- Russian government officially recognized the method.

Russian Health Risk Analysis 1998 - 2002

- Health risk resulted from industrial pollution in Russia was analyzed in up to 20 cities;
  - Volgograd, Novokuznetsk, Perm, Angarsk, Krasnouralsk, Ekaterinburg, Samara, Novokuibyshevsk, Velikii Novgorod, Voronezh, Serpukhov, Moscow, Klin, Cherepovets, Verkhniaa Pyzhma, Orenburg and others
- Carcinogenic and non-carcinogenic risk;
- Stationary and mobile pollution sources;
- From air pollution to multimedia health risk analysis based on USEPA methods and standards.

Air Pollution in Ukraine

CBP – Beginning the Process

- Broad consultations –kick-off seminar
  - Build understanding of the approach by illustrating many of the principles of modern environmental policy
  - Facilitate partnership and to exchange experience between the two governments and share best practices available in multilateral international institutions, academic and NGOs
- Pilot cities for risk analysis
  - Zaporizhzhia
  - Kharkiv
- Bridge to policy making
  - Prioritization and environmental finance
  - Modernization of air pollution management system

Next Steps for CPB

- Acquire official emissions data for Zaporizhzhia and Kharkiv oblasts;
- Digitize available population data;
- Model dispersion of emissions;
- Screening-level cancer and non-cancer risk assessments;
- Risk-based priorities based on USEPA and WHO standards.
- Comparative Risk Assessment:
  - Identification of risk reduction opportunities that provide the greatest health benefits;
- Use of economic analysis to support efficient decisions;
- Encourage use of national pollution fee-based environmental funds to pay for high priority opportunities.

Acknowledgements

Ukraine Ministry of Environment and Natural Resources
Counterpart International • Kiev • Washington DC

ENVIRONMENTAL DEFENSE
finding the ways that work

Making the Linkage between Air Issues and Human Health: An ENGO Perspective on Sustainable Transportation Policy in Central Ontario.

Q. Chiotti

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Background and Context

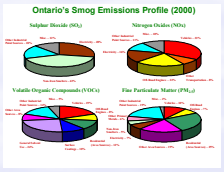
The identification, development and implementation of strategies for effective air quality (and climate change) management that leads to measurable improvements in human health and the environment is a challenging task. While it undoubtedly involves a number of scientific, technical and economic considerations, the pathway from recognizing a significant environment and health hazard to the implementation of policy is rarely smooth and straightforward. While sound scientific data on the health risks associated with air issues may be necessary to inform policy decisions, in practice such information is insufficient to generate desired policy outcomes. This is certainly the case for transportation, one of the leading emission sources contributing to smog and climate change. A number of obstacles and challenges exist, including uncertainty in health and atmospheric science, the complexity of the transportation problem, limitations in technology, jurisdictional conflicts, competing interest groups, among others. To address this problem, Pollution Probe and the York Applied Centre for Applied Sustainability organized a national conference on Transportation, Air Issues (air quality and climate change), and Human Health, which attracted over 140 participants including 25 speakers representing government, industry, health and environmental groups.

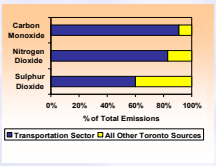
Convergence of Understanding and Policy Response

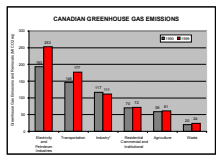
Since Pollution Probe first transportation conference in 1996, there has been growing scientific and medical evidence in attributing emissions from transportation and other sectors to air quality problems and adverse human health effects. The problem is no longer making the link, but rather what transportation actions need to be taken to reduce emissions causing smog and climate change. Indeed, there are numerous national and provincial emission commitments that are due by the end of the decade (e.g. Ozone annex, Kyoto Protocol). Following successful reductions in SO_2 emissions, it is forecast that due to improvements in fuels and technology, emissions of NO_2, VOCs and $PM_{2.5}$ are expected to decrease substantially from current levels by 2010. As part of the National Climate Change Strategy, the Federal Government has pledged to work with the auto industry to obtain a 25% improvement in fuel efficiency. Sales of fuel inefficient SUV's, light trucks and mini-vans however continue to out pace more fuel efficient compact passenger vehicles. Urban sprawl, population and economic growth are also forecast to increase substantially over the next 20-30 years in many regions of Ontario, especially the Greater Toronto Area. This has prompted the Provincial Government of Ontario to consider "Smart Growth" concepts into the planning process, such reshaping where and how people live, unlocking gridlock and protecting the environment. There are some inherent contradictions to these policy goals however, and it is uncertain, if not unlikely, that such measures will lead to substantial emission reductions.

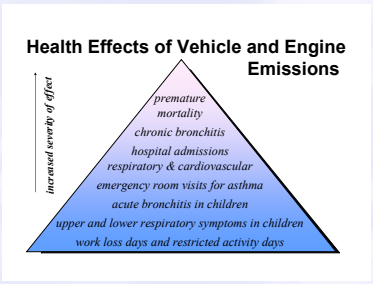
Future policies that will help achieve improved air quality and health include regulated corporate average fuel efficiency, an urban gas tax that is used to improve public transit, the adoption of the net gain approach as a framework from which to inform land use and transportation decisions, and the implementation of a national TDM strategy. However, despite best intentions, there is some degree of air pollution and climate change that is inevitable, and adaptation will be necessary at the individual level and for health infrastructure. Future success will require a collective and collaborative effort by government, industry and NGOs that is committed to achieving environmental sustainability.

The Evidence



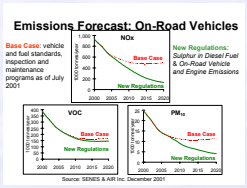


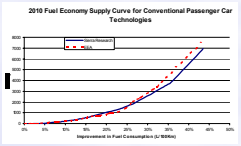




30% of GHG emissions in Ontario are from transportation sources; for Canada, GHG emissions from transportation have increased 21% from 1990 to 1999

Forecasts





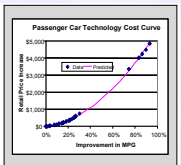
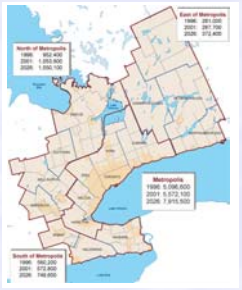
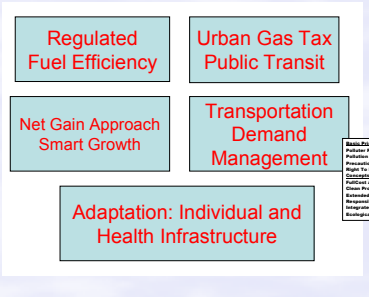


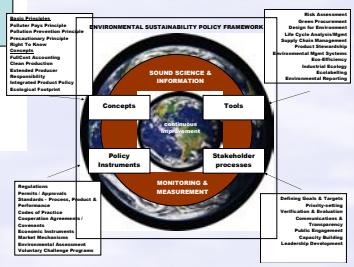
Figure 1. Passenger Car Fuel Economy Technology Cost Curve, EEA Industry Average Data, 2010 - 2015



- **64% increase in auto trips by 2021**
- **Triple the number of congested roads and double the commute time**
- **$3 Billion congestion costs and 2,500 premature deaths due to air pollution**

Pathway Forward





Notes: This poster draws heavily from the proceedings to the *Transportation, Air Issues and Human Health Conference,* by Keating, M., Chiotti, Q., Ogilvie, K. and Bell, D. (2003), which is available online: www.pollutionprobe.org

POLLUTION PROBE
CLEAN AIR. CLEAN WATER.

Predicted health impacts of congestion pricing in Stockholm
– a local assessment

Forsberg B*, Johansson C#, Segerstedt B*, L Burman#
* Dept of Public Health and Clinical Medicine, Umeå University, Umeå, Sweden, # Environment and Health Protection Administration of Stockholm"

AIRNET A Thematic Network on Air Pollution and Health

NERAM Network for Environmental Risk Assessment and Management

Aim and background

Traffic congestion leads to substantial waste of time, extra fuel combusted, polluted air and negative health effects. Using price to allocate space on congested roads involves charging relatively higher prices for travel during periods of peak hours than in other periods. The introduction of such a system is planned in Stockholm, and we have estimated the effects on air pollution levels and health.

Methods

Traffic models, a dynamic emission database and an air quality dispersion model have been used to calculate the effect on NO_2 and PM_{10} levels of a system for congestion pricing.

Air pollutant concentrations have been combined with spatial distribution of the population (100×100 m resolution for the inner city, 500 x 500 for outskirts) to obtain population weighted means and extreme values.

The modelled exposure reductions have been combined with reported exposure-response functions and local base-line frequencies to quantify health benefits expected from the pricing system.

Results and conclusions

-For approximately 330000 inner-city residents it is estimated that the reduction in long-term exposure corresponds to 46 deaths per year based on NO_2 as exhaust exposure indicator (Dutch study by Hoek et al).

- If instead PM10 is used as indicator (US coefficients as used by Künzli et al and in APHEIS) the reduction is estimated to 18 deaths per year.

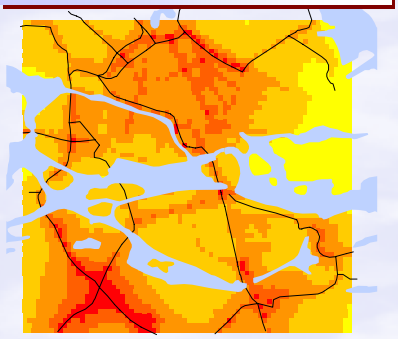
Estimated long term effects on the inner-city mortality

Indicator	Assumed RR per µg/m³	Reduction deaths per year	With day time population
PM_{10}	0,43 %	17,7	18.3
NO_2	1,2 %	46,3	48.1

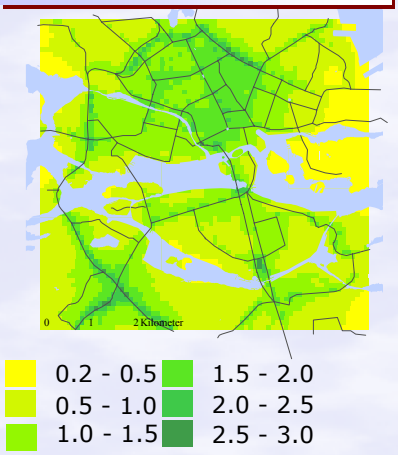
In both cases the population weighted annual mean concentration was estimated to be reduced by 1.2 µg/m³. If the effects on concentration reduction in the outskirts (a population of about one million people) is included, the total reduction in mortality is estimated to be 65 cases using NO2 as indicator. "Reductions in hospital admissions were also estimated, but only as a decrease in short-term effects.

Applied relative risks are associated with uncertainties. Other factors cause additional uncertainties, why we see it necessary to further develop this kind of assessments.

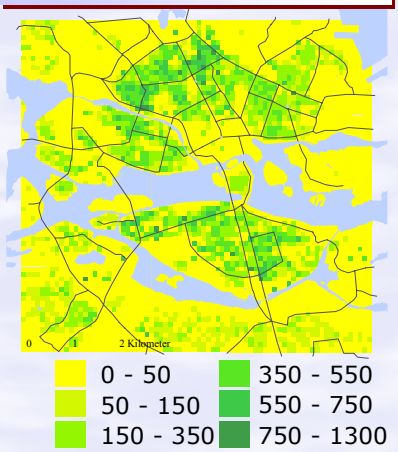
Annual mean NO_2 background concentration (µg/m³) in the inner city



Estimated reduction in annual mean NO_2 concentration (µg/m³) in the inner city



Estimated reduction in NO_2 annual mean population exposure (µg/m³ ×persons) in the inner city



ALBERTA'S PM AND OZONE MANAGEMENT FRAMEWORK

G. C. (GEOFF) GRANVILLE, Shell Canada Limited

AIRNET
A Thematic Network on Air Pollution and Health
NERAM
Network for Environmental Risk Assessment and Management

Introduction

National Standards (called Canada Wide Standards [CWS]) were established for PM2.5 and Ozone in June, 2000.

CWS NUMERIC STANDARDS BY 2010

PM2.5:

30 µ/m3, 24 hour avg., achievement based on 98th percentile annual ambient measurements, averaged over 3 consecutive years

Ozone:

65 ppb, 8 hour avg., achievement based on 4th highest annual ambient measurement, averaged over 3 consecutive years

OTHER COMPONENTS OF THE CWS:

"Keeping Clean Areas Clean" and "Continuous Improvement". The CWS includes a mechanism to analyze exceedances caused by transboundary or naturally occurring levels

In Alberta, the Clean Air Strategic Alliance (CASA) is the multi-stakeholder, consensus-driven process used to manage ambient air quality issues in the province[1]. CASA was asked by the Provincial Government to set up a Team to develop consensus recommendations for an *implementation plan* to achieve the provisions of the CWS. Specifically, they were requested to:

- Recommend strategies to achieve the CWS for PM and ozone
- Recommend key components of strategies
- Achieve stakeholder support for the implementation plan

The Implementation Plan

To deliver the Implementation Plan, the Team developed a "management framework", comprising a series of "Trigger Levels" (ie ambient air concentrations - see Table) at which various actions would be taken a specific "structiure" (see Figures 1 and 2), plus a series of accompanying "principles" for guidance purposes.

FIGURE 1: FINE PARTICULATE MATTER AND OZONE MANAGEMENT FRAMEWORK

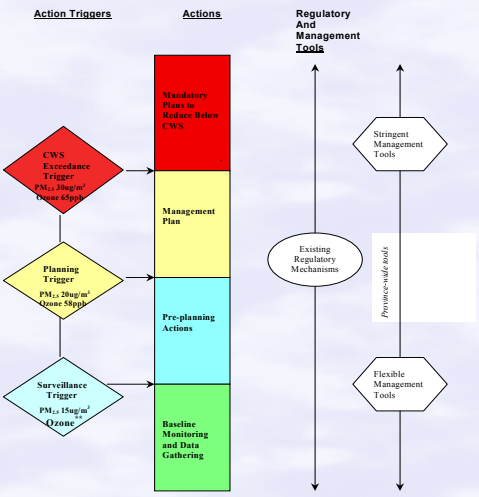
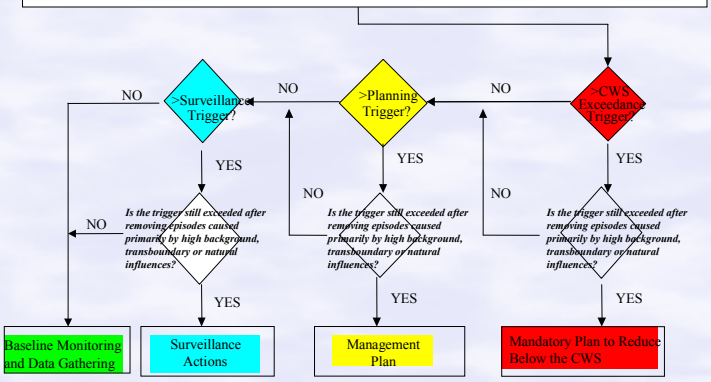


Fig 2: Annual Analysis of Monitoring Data for PM, O_3 in Each Air Zone



The trigger levels shown in the Table below required extensive and prolonged discussion, and were chosen in parallel with agreements on specific management actions identified at each trigger level[3]

Level	PM2.5	Ozone
Exceedance	30 micrograms/m^3	65 ppb
Management	20.0 micrograms/m^3	58.0 ppb
Surveillance	15.0 micrograms/m^3	Determined by AENV[2]
Baseline	< 15.0 micrograms/m^3	Determined by AENV

The Principles include the following:

The trigger concentrations are neither "pollute up to" levels nor "not to exceed" levels.

Activities should be prioritized according to available resources and contextual factors, including:

- •population density
- •trends in ambient levels
- •the predicted impact of existing activities and initiatives
- •economic growth forecasts
- •age of facilities, and
- •any factors related to the overall practicality of actions

stringent management tools are to be used as levels approach the CWS, more flexible management tools are to be used when ambient levels are at baseline or surveillance levels

Trigger Levels will be used for airshed planning and will not be applied as "point of impingement" concentrations in relation to approval limits and conditions.

Examples of Management Tools include:

Regulatory

- approvals, regulations, prohibitions

Standards, codes of practice

- air quality guidelines, new source performance stds

Programs

- pollution prevention planning, education

Agreements, Targets and Objectives

- emissions trading programs (could be regulated)

Incentives

- economic instruments, recognition programs

Conclusions

The Team's recommendations have been accepted and are being implemented within the province. The framework may be adopted in other jurisdictions due to its flexibility and broad stakeholder buy-in.

Notes

1. See: www.casahome.org
2. AENV: Alberta Environment
3. Details are included in the final report, see: http://www.casahome.org/uploads/PMO3_ManagementFrameworkSEP-18-2003.pdf

Air Quality Improvements with European Environment Policies: SO_2 Case Study in a Coastal Region in Portugal

M.C. Pereira, M.C.M. Alvim-Ferraz and R.C.Santos

AIRNET
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Objectives

To evaluate the first reflexes in the air quality of Oporto Metropolitan Area (Oporto-MA) of the "Auto-Oil" Directives implemented in Portugal. Therefore, SO_2 data from three selected monitoring sites: urban/traffic (Paranhos) , industrial (Custóias) and rural (VN Telha), were analysed between 1999 and 2002.

"Auto-Oil" Directives

- **98/69/EC** relating to measures to be taken against air pollution by emissions from motor vehicles
- **98/70/EC** relating to the quality of petrol and diesel fuels

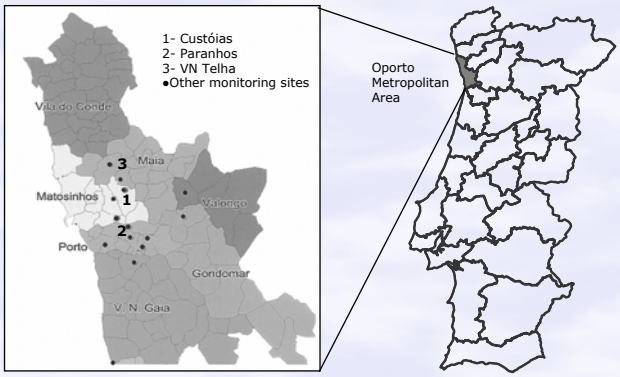
Oporto Metropolitan Area

Oporto is the 2nd Portuguese main city

Close proximity of Atlantic Ocean with 50 km of coastline

Population density: 540 hab/km²

Most important stationary sources of atmospheric pollutants: one oil refinery, one petrochemical plant, one thermoelectric plant working with natural gas, one incineration unit and one international shipping port



Results

Exceedances of SO_2 hourly and daily limits for protection of human health according to EC legislation 1999/30/EC in Oporto-MA

site	Number of hours [1]				Number of days [2]			
	1999	2000	2001	2002	1999	2000	2001	2002
Urban/Traffic	4	0	*	*	0	0	*	*
Industrial	15	9	3	1	**5**	0	0	0
Rural	2	0	2	1	0	0	0	0

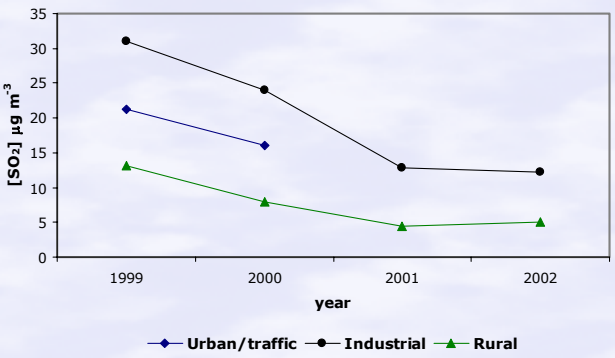
* Not enough data for statistical treatment
[1] hourly limit (350 μg m^{-3}) not allowed more than 24 exceedances per year
[2] daily limit (125 g μm^{-3}) not allowed more than 3 exceedances per year

The recommended SO_2 hourly limit value was not surpassed between 1999-2002.

The hourly limit value in the industrial area was reached 15 times in 1999 against 1 time in 2002 showing a significant decrease in the number of high values along the years.

The daily limit value for protection of human health was exceeded once in 1999 in the industrial site not being exceeded presently.

SO_2 annual average concentrations in Oporto-MA



The annual averages of SO_2 concentration have decreased considerably for all types of monitoring sites.

For the industrial and rural sites the decrease was around 60%.

The rural site presented the lowest SO_2 annual concentrations and in the last two years the values are similar with those usually found in other rural areas of Europe.

Conclusions

The improvement of fuel quality, the emission control programmes and the technological innovations in the past years are responsible for the significant decrease in the SO_2 concentrations and consequently the exceedances of EC SO_2 limits in Oporto-MA.

The potential human health risk still persists, particularly, in industrialized areas. It is expected that the tendency of SO_2 levels in ambient air of Oporto-MA will continue downwards in the coming years following the present European trends.

The "Auto-Oil" directives clearly improved the air quality of Oporto-MA but other pollutants namely PM_{10} and O_3 are critical in the area and deserve special attention. **Policies based in the same principles are welcome to solve the problems related with these pollutants.**

LEPAE
LABORATORY OF PROCESS, ENVIRONMENT AND ENERGY ENGINEERING

Universidade do Porto
FEUP Faculdade de Engenharia

HEALTH EFFECTS OF PM IN DENMARK

- DO PARTICLE FILTERS ON HEAVY-DUTY VEHICLES SOLVE THE PROBLEM ?

AIRNET
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Aims

1) To quantify the health effects related to particulate air pollution in Denmark, and

2) To estimate the health gains from supplying particle filters to all heavy-duty vehicles (>3.5 tons) in Denmark.

Methods

We used methods similar to those of Künzli et al. (1): Relative risk estimates in relation to PM_{10} were estimated from the epidemiological literature. Population exposure to PM_{10} was calculated. Cases attributable to man-made PM_{10} was calculated for mortality, hospital admissions, chronic and acute bronchitis, restricted activity days and asthma attacks.

Health gains of equipping all Danish heavy-duty vehicles with particle filters were estimated under different assumptions:

1) Health effects of particles depend entirely on their number concentration, and

2) Health effects of particles, also ultra fine particles (UFP), depend entirely on their mass concentration (PM_{10}).

Two examples of the many commercially available particle filters: Johnson Matthey CRT (left) and Engelhard DPX (right). In general, these filters are all claimed to be more than 80 % effective under field conditions. See for example www.dieselnet.com for further information.

Results

In 2000, the average exposure of the 5 million Danes to PM_{10} was estimated to 22.26 µg/m³. Particle filters would reduce this PM_{10} exposure only marginally, namely to 22.17 µg/m³.

In contrast, particle filters on all heavy-duty vehicles in Denmark would reduce population exposure to UFP by about 20%.

Health gains (per year) for three scenarios of reduced PM concentrations in Denmark. Data first published in (2).

			Particle filters on all heavy-duty vehicles in Denmark	
Health effect	Age group (y)	No man-made PM (PM_{10} = 7.5µg/m³)	Assumption 1: Health effects depend on particle number (~UFP)	Assumption 2: Health effects depend on particle mass (~PM_{10})
Total mortality	≥30	3,400	1,000	22
Cardiovascular hospital admission	All	2,200	650	14
Respiratory hospital admission	All	1,500	450	10
Chronic bronchitis incidence	≥25	3,300	1,000	22
Acute bronchitis	≤15	11,600	3,500	76
Restricted activity days	≥20	1,800,000	550,000	12,000
Asthma attacks	≥15	141,000	40,000	920
Asthma attacks	<15	18,700	5,500	120

Conclusion

Large numbers of health effects can be related to atmospheric PM. Particle filters on all heavy-duty vehicles in Denmark are expected to reduce these numbers substantially (app. 20%) if the health effects depend entirely on the number concentration, but only marginally (app. 0.5%) if the effects depend entirely on the mass concentration (PM_{10}). The truth probably lies somewhere between these two extreme assumptions.

Policy

The health effects of particulate air pollution has been discussed intensely in Denmark during the last years, and the Government has acknowledged the problem.

Policy-making has focused on 1) enforced use of particle filters on all heavy duty vehicles in Denmark and 2) environmental zones in densely populated parts of larger Danish cities.

Although the government wishes to reduce particle pollution, Danish legislation enforcing the use of particle filters on heavy-duty vehicles seems clash with that of the European Union. On the other hand, the government is keen to promote environmental zones in larger Danish cities with exclusive access to heavy-duty vehicles equipped with particle filters.

The municipality of Copenhagen has already taken up the challenge and aims to establish an environmental zone in the central part of Copenhagen by the autumn of 2004.

References

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2. Raaschou-Nielsen O, et al. Health effects of ambient particulate matter – a quantitative assessment (in Danish, English summary). Ugeskr Laeger 2002; 164:3959-63.

Clean Air and Health Policy Considerations in South Caucasian Countries

Dr. Ketevan Samadashvili

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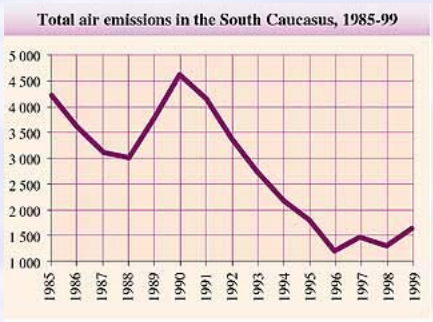
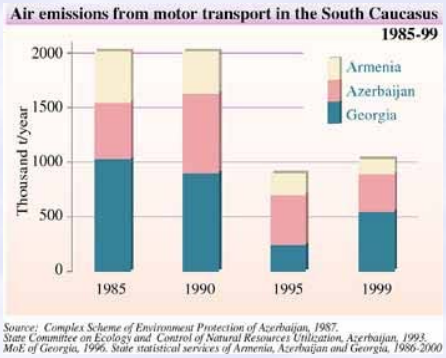
Major Sources of Air Pollution and their Emissions.

During the 1970s and 1980s, transport and industry were the major sources for air pollution in the Caucasus region. In the early 1990s total emissions fell due to the general economic decline (Table 1).



Source: Complex Scheme of Environment Protection of Azerbaijan, 1987. State Committee on Ecology and Control of Natural Resources Utilization, Azerbaijan, 1993. MoE of Georgia, 1996. State statistical services of Armenia, Azerbaijan and Georgia, 1986-2000

Historically, the percentage of emissions from transport, with some exceptions, was higher than stationary source emissions in most of parts of the region. For example, whereas in Georgia and Armenia mobile sources contributed over 60% of total emissions, in Azerbaijan with large industrial capacities, the figure varied from 30% to 40% (Table 2).



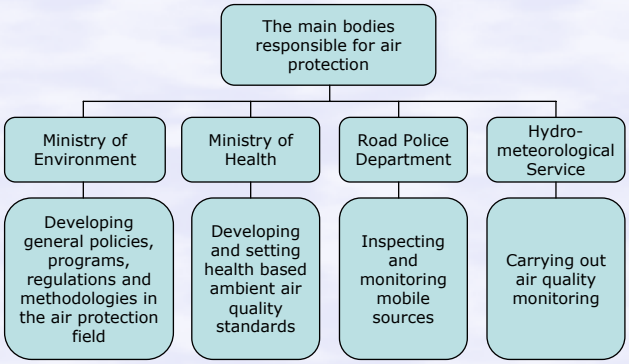
Source: Complex Scheme of Environment Protection of Azerbaijan, 1987. State Committee on Ecology and Control of Natural Resources Utilization, Azerbaijan, 1993. MoE of Georgia, 1996. State statistical services of Armenia, Azerbaijan and Georgia, 1986-2000

In the early 1990s, industrial emissions declined even more dramatically, increasing the transport share of total emissions to 80%. The reasons for high vehicle emissions were heavy traffic in urban areas and high emissions from cars lacking pollution control devices.

Urban Air Quality

High ambient concentrations of CO, NO_x, phenol and formaldehyde indicate a significant impact from traffic. The problem with ground level ozone is a concern. Cities such as Tbilisi, Yerevan, Vanadzor, Ararat, etc. with valley type terrain or/and poor ventilation may suffer the most.

It is expected that in the future sulphur dioxide and nitrogen oxide emissions will soar, as the number of vehicles and industrial activities increase. Moreover, the problem may become very acute, taking into account the inefficient and out of date technologies employed. In the short-term, the major threat can be expected from rapidly growing road transport with its obsolete fleet and poorly maintained vehicles. Hence, the problem with NOx emissions is becoming acute. The implementation of TRACECA project will significantly increase traffic in major highways and may highly contribute to trans-boundary air pollution as well.



Policy Measures and Responses

After independence, all the South Caucasus countries adopted framework laws on environmental protection. At present, Georgia is ahead from other South Caucasus countries in terms of harmonisation Georgian and EU legislation. National air quality legislation is proposed for Georgia which will include a list of air pollutants, limit values and a definition of monitoring requirements based on the EU Air Framework Directive. These provisions will come into force on the 1st of January 2005 by a specific order of the Environmental Minister, taking into consideration all recent amendments made in EU Directives. Fuel quality and mobile source emission standards shall be developed on the basis of appropriate EU Directives and introduced by 1 January 2003.

Even if the legislation were perfect, poor enforcement system would preclude compliance of existing laws and regulations. At present, the countries lack finances to develop modern compliance assurance monitoring and control systems. Environmental law enforcement officers are untrained and poorly equipped with measuring devices and there is no legal basis for the frequency and quality of inspections and emission measurements. Administrative penalties imposed on violators, including permit conditions, are symbolic, encouraging illegal activities. On a whole, the Caucasus countries lack legislation and practical experience related to environmental damage, liability and compensation issues, and public court suits.

REC CAUCASUS

Composition of and exposure to $PM_{2.5}$ while commuting in the metro and on the street

P. Aarnio, A. Kousa, T. Yli-Tuomi, M. Jantunen, T. Koskentalo, R. Hillamo

AIRNET
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Introduction

Contribution of traffic to personal $PM_{2.5}$ exposure is important for the assessment of PM health effects (1, 2, 3). We have recently started research activities aiming at improving our knowledge on the mass and number concentrations as well as size distributions and composition of fine particles in different traffic environments. So far we have conducted a short preliminary survey in different vehicles and a more comprehensive measurement campaign in the metro in Helsinki, Finland.

Methods

In this preliminary study in different vehicles we monitored the $PM_{2.5}$ mass concentrations (MIE personalData-RAM-1000) as well as particle number concentrations (P-TRAK Ultrafine Particle Counter Model 8525) while commuting in the city either by train, metro, bus or on foot.

In the Helsinki metro we collected $PM_{2.5}$ samples on teflon and quartz fibre filters at two underground and at one surface metro station. The $PM_{2.5}$ concentrations were also recorded continuously using a photometric method. Exposure to fine particles in commuting was measured inside a metro car using the same methods as at the stations. Samples were collected during three to five days from 6.30 am to 6.30 pm.They were analysed for their elemental (BC) and organic carbon (OC) content with thermal-optical transmission (TOT) method. The elemental composition was analyzed with ED-XRF.

Results

The mass and particle number concentrations showed large variability in different traffic environments and were in many occasions clearly higher than in urban background environment (Fig 1).

During the measurement campaign in the metro high concentrations of $PM_{2.5}$ were observed at the stations and also in the metro car, where the concentrations increased whenever the car was in the tunnel and decreased when the metro car was on surface in open air (Fig 2).

The $PM_{2.5}$ samples collected in the metro contained very high concentrations of iron, and also e.g. manganese, copper, chromium and nickel concentrations were clearly elevated. The origin of these elements is probably the rails and wheels. At Station1 the effect of the tunnel construction work in the area were reflected in the concentrations of e.g. silicon, aluminum, and potassium (Table 1).

Conclusions

- Additional measurements in trains, trams, buses and personal cars are needed to obtain a more representative comprehension of the exposure to PM in traffic.
- The composition of $PM_{2.5}$ in the metro differs greatly from that of ambient PM. The size distribution of particles is likely to be different, too (2)
- We need more information about the mass and number concentrations as well as the size distributions of particles in the metro.
- Before making conclusions about the health effects of exposures to PM in metro we need more information about
  - the relevance of short exposures to high concentrations for the health effects
  - epidemiological and/or toxicological evaluation of the health risk of PM exposure in metro relative to the risks of PM exposure in street traffic and in ambient urban air.

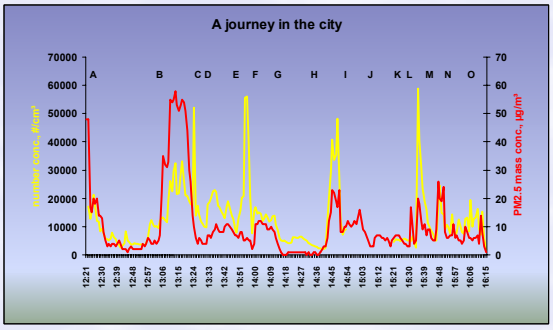

Figure 1. Particle mass ($PM_{2.5}$) and number (0.02 - > 1 µm) concentrations in different environments A: metro station, AB: commuting in a metro, BC: metro/railway station CD: street, DE: bus1, EF: street, FG: bus2, GH: cafe, HI: railway station, IJ: local train1, JK: station2, KL: train2, LM: train3, MN: railway station, NO: street.

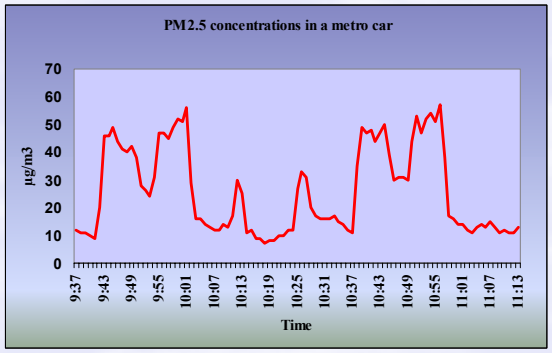

Figure 2. $PM_{2.5}$ concentrations (1 min values) inside a metro car while commuting

Table 1. The concentrations of some elements, $PM_{2.5}$ mass, BC, and OC in the metro (averages of 2 or 3 twelve hour samples collected between 6.30 am and 6.30 pm in March 2003) and at the urban traffic monitoring site (annual averages in 1997, BC and OC in 2002)

	Station1 undergr.	Station2 undergr.	Station3 surface	Metro car	Urban traffic 1997/2000
ng/m³					
Al	3100	300	90	200	59
Ca	810	210	82	99	71
Cl	190	270	96	110	43
Cr	42	88	2.0	13	
Cu	100	110	10	42	3.1
Fe	19000	36000	1300	6900	96
K	1600	150	65	140	85
Mn	220	420	16	81	3.3
Ni	19	35	2.7	10	2.0
S	400	440	210	500	830
Si	11000	690	280	660	
Ti	71	47	6.9	8.8	0.8
µg/m³					
PM2.5	84	58	8.5	18	12
BC		2.8			1.2
OC		6.6			3.0

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Acknowledgements

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HEPMEAP: *In vitro* and *in vivo* toxic potency of ambient fine and coarse PM across Europe: the influence of traffic exhaust emissions

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Introduction

Ambient particulate matter (PM) may be responsible for serious respiratory and cardiovascular health effects or even premature mortality especially among susceptible sub-populations. To assess the inflammatory and toxic potential of ambient suspended particles collected at places across Europe with contrasts in traffic intensity and to link these properties with chemical composition data on the one hand and epidemiological health observations on the other hand, different EU PM samples fine (PM0.1-2.5) and coarse (PM10-2.5) were tested *in-vitro and in-vivo* (both rats and humans).

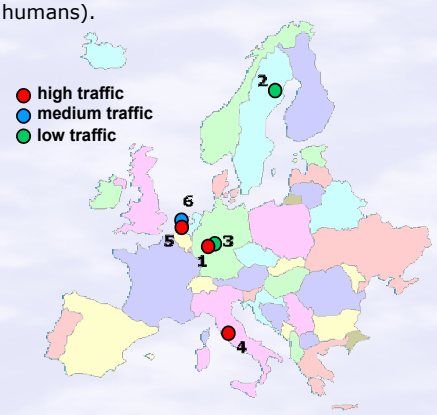


Figure 1: Site selection for *in vivo* study.

1 - D, Ostbahnhof; **2** - S, Lycksele; **3** - D, Grosshadern; **4** - I, Rome; **5** - NL. HIA; **6** - NL, Dordrecht.

To choose a selection of the EU PM samples for the *in-vivo* study, samples were ranked based on ascorbate depletion, arachidonic acid release, IL-6, and DNA damage (Table 1). Furthermore the site selection (Figure 1) and chemical characterization were taken into account. The most interesting samples were selected based on 4 categories (most or least reactive in both fractions and greatest contrast between fractions with fine or coarse most reactive).

Materials and Methods

Spontaneous hypertensive rats (SHR; 12-weeks-old) received a single PM dose (3 or 10 mg PM per kg bodyweight). Health effects were determined at 24 hours after this treatment using lung histopathology, bronchoalveolar lavage fluid and blood analysis.

Results

PM site	*In vitro* ranking results
1	high ascorbate, AA release, DNA damage; mainly coarse
2	high AA release, IL-6;mainly fine - high hop/ster/V/B(a)P;coarse/fine
3	high ascorbate, IL-6;mainly fine - low hop/ster/V/B(a)P;coarse/fine
4	high AA release;coarse/fine - high Ni/Cu/Zn;coarse/fine
5	high AA release, IL-6;coarse/fine
6	high ascorbate, IL-6;mainly coarse

Table 1: *In vitro* ranking results

Figure 2 shows a marked, dose-dependent increase in cytotoxicity (LDH), the coarse fraction being more potent than the fine fraction. All samples induced increased lung permeability (albumin) with the exception of the sample from Rome.

In general, exposures to PM lead to an dose dependent oxidative stress response (anti-oxidants such as glutathione and uric acid) (Figure 3).

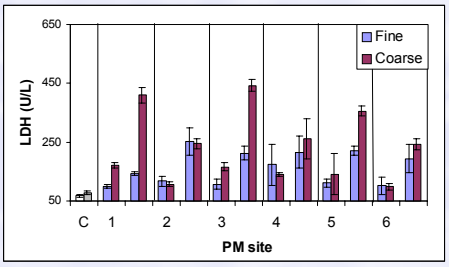


Figure 2: LDH concentrations in BALF of SHR exposed to saline control (C) or EU PM samples at 24 hrs after a single intratracheal instillation of 3 respectively 10 mg/kg bodyweight. Values are presented as means ± standard errors of the mean, *N*=7-8

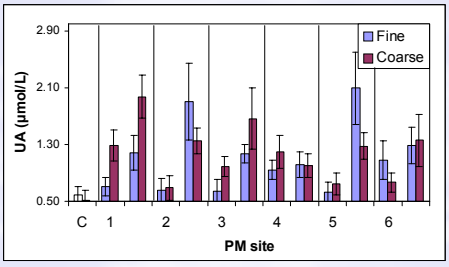


Figure 3: Example of a anti-oxidant induction in BALF of SHR at 24 hrs post-exposure.

Significant inflammatory response were observed as indicated by an increased neutrophil-influx (Figure 4) and increased cytokine levels in the lungs. This effect was most prominent for coarse fraction PM whereas fine resulted primarily in increased macrophages (Figure 5) showing an increased host defense.

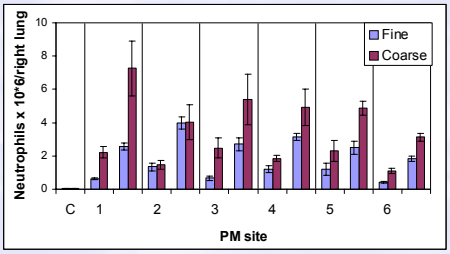


Figure 4: Neutrophil concentration in BALF of SHR at 24 hrs post-exposure.

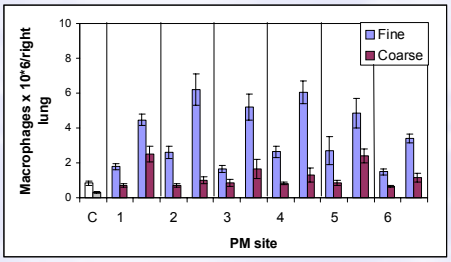


Figure 5: Macrophage concentration in BALF of SHR at 24 hrs post-exposure.

Particularly the PM sampled in Germany at the Ostbahnhof and the HIA PM from the Netherlands show severe pathological changes. Both these samples and the PM from Grosshadern also resulted in increased cell proliferation compared to the other samples.

Conclusions

The still incomplete database of results indicate that

- All selected ambient fine and coarse PM samples express toxic potency.
- PM from locations with a high density of road traffic induce more effects than those from low density traffic areas.
- Both coarse and fine PM fractions induce adverse health effects, but affect different indicators.
- The correlation between *in vitro* data and *in vivo* outcomes has not yet been studied.

This research has been performed within the framework of the European project entitled 'Health effects from motor engine exhaust and ambient air pollution (HEPMEAP; QLK4-1999-01582).

More information at WWW.HEPMEAP.ORG

Exposure to traffic-generated VOCs: Total exposure in urban environments and the specific contribution of exposure while in traffic

Vito Ilacqua and Matti Jantunen
KTL - National Public Health Institute, Kuopio, Finland

AIRNET
A Thematic Network on Air Pollution and Health

NERAM
Network for Environmental Risk Assessment and Management

Introduction

Traffic emissions include a wide array of VOCs that pose potential threats to human health, not only individually, but also as a mixture. Urban populations have heavier exposures due to the high density of both population and traffic. This study separates out the contribution to personal exposure due to traffic emissions in Athens, Basel and Helsinki.

Methodology

Personal, ambient, and indoor (home and workplace) exposure concentrations from the EXPOLIS database were used to perform a microenvironmental apportionment of 8 traffic-related VOCs, as well as Black Smoke. Individuals with exposure to Environmental Tobacco Smoke (ETS) were excluded from the analysis. This first step yielded the indoor- and outdoor-generated fractions, as well as the fraction attributable to other locations (mainly commuting).

The latter two fractions were subjected to Maximum-Likelihood principal factors analysis to determine the traffic contributions. Traffic emissions were separated by other fuel emissions (evaporative) by including Black Smoke in the analysis. The communalities of the traffic factors were used to quantify the traffic contributions.

The sampling design did not allow a complete separation of workplace contributions, which may therefore include a quota of outdoor contributions, including some from traffic.

The full separation of traffic sources was possible only for the city of Helsinki, due to a larger sample size. Less specific information could be obtained for Athens and Basel.

Results

Tailpipe VOC emissions appear to be the largest contributor to personal exposure for 6 of the VOCs analyzed, including benzene, as shown in the figure.

In all cases, the majority (≥ 2/3) of exposure to traffic VOCs takes place indoors.

Other outdoor sources are generally of minor importance. with the notable exception of benzene.

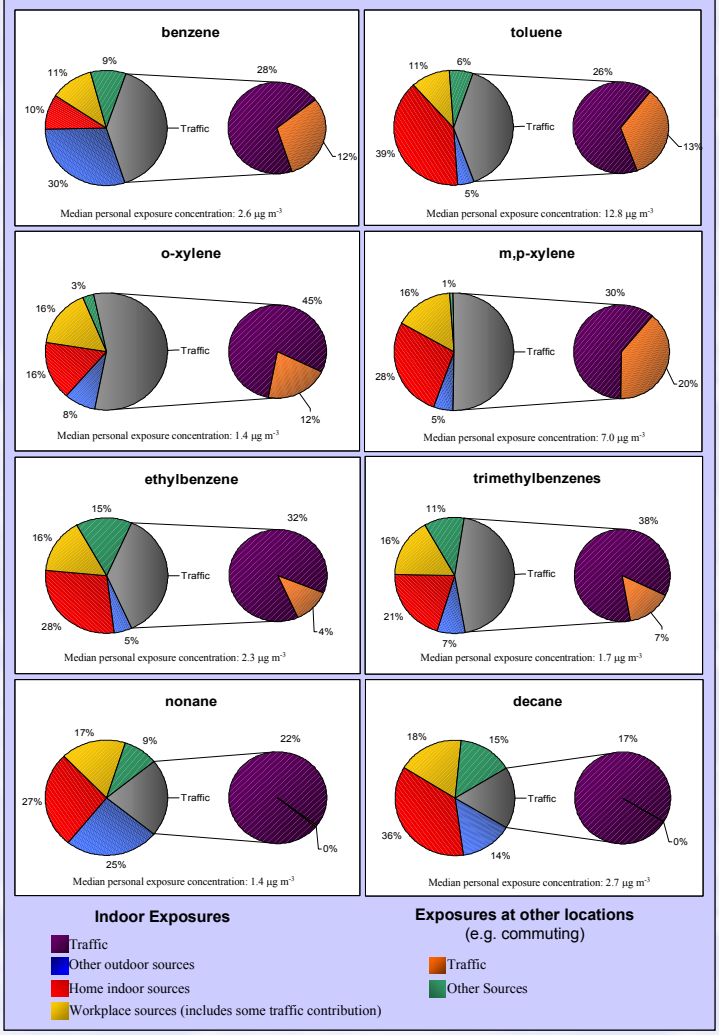
Indoor sources - presumably from evaporative emissions and consumer products - on the other hand, contribute a very large fraction of personal exposure.

The make-up of indoor/outdoor and traffic/non-traffic source contributions is quite specific to each urban environment considered (Table 1).

Table 1. Comparisons of contributions to benzene exposure across cities.

Benzene	Median personal exposure concentration ($\mu g\ m^{-3}$)	Indoor generated	Indoor exposure to traffic	Indoor exposure to other outdoor sources	Exposure in other locations
Athens	10.3	10% ± 5%	65% ± 11%	10% ± 2%	17% ± 9%
Basel	2.6	53% ± 17%	26% ± 5%	24% ± 5%	20% ± 7%
Helsinki	2.5	22% ± 4%	29% ± 1%	31% ± 1%	23% ± 3%

Contributions to total personal exposure in Helsinki (non ETS exposed population)



Implications

- The observation that the majority of exposure to traffic byproducts takes place indoors at home, presents a major challenge in terms of exposure reduction strategy. While office and commercial buildings equipped with HVAC systems might implement some air treatment, the situation for individual homes is hardly viable to a treatment option.
- Thus, the available approaches must necessarily aim to reduce ambient levels of these compounds and/or to reduce the proximity of heavily populated residential areas to major traffic routes.
- Controlling exposure in commercial areas or while commuting would apparently have a less important, though by no means negligible, impact.
- The generally important contributions from indoor sources, however, point to a careful evaluation of the most cost-effective strategy. Unfortunately, whether limited resources should be allocated to reducing exposure from traffic or from consumer products, building materials, etc. can only be determined for each city individually.

Acknowledgments

Support by EU Contracts ENV4-CT96-0202 (DG12–DTEE) and ERBIC20CT96-0061, Academy of Finland N36586, KTL projects 2169, 2127; *Bundesamt für Bildung und Wissenschaft BBW Nr. 95.0894; Schweizerischer Nationalfonds 32-048922.96*

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Two years fine and ultrafine particles measurements in Rome, Italy

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[1]Italian National Institute of Health, Rome, Italy
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AIRNET
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Introduction

The main objective of this study were to establish validated and consistent data sets of particle number concentrations in Rome. These data-sets will be used for quantifying the contributions of various pollution sources of ultrafine particles and to develop a method to estimate the previous number concentration of ultrafine particles. These data were collected and will be used in the framework of the research project : "Health effects of air pollution on susceptible sub-population – traditional air pollutants, ultrafine particles and myocardial infraction: data base and health assessment" which main objectives are to quantify the risk of hospitalisation and of death due to air pollution, in particular airborne ultrafine particles, in individuals with coronary heart disease and to quantify the attributable risk of environmental exposures among a sensitive subgroup in order to facilitate appropriate public health strategies for the prevention of air pollution related health effects.

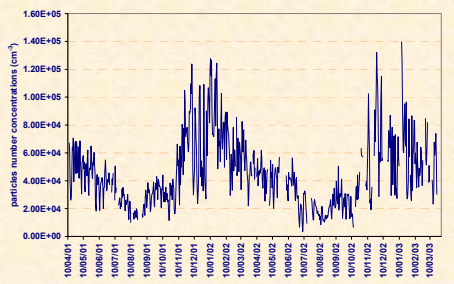
Material and Methods

Two-year measurement of ultrafine particles using condensation particle counter (CPC 3022A, TSI) have been performed, together with others classic pollutants (PM_{10}, $PM_{2.5}$, CO, NO_2, NO, NO_x, O_3 collected only at the primary site).

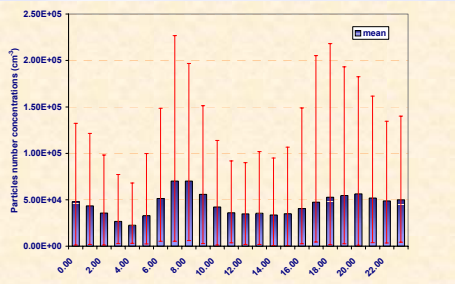
Secondary site (Orto Botanico - Urban background) from February 2002 to March 2003).

Primary site: (ISS - traffic oriented) from April 2001 to March 2003.

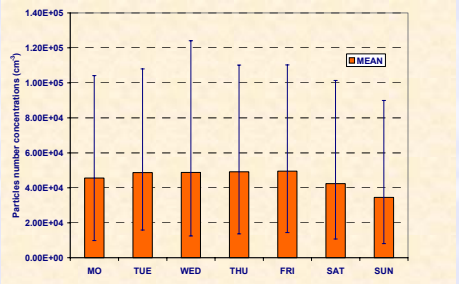
Results



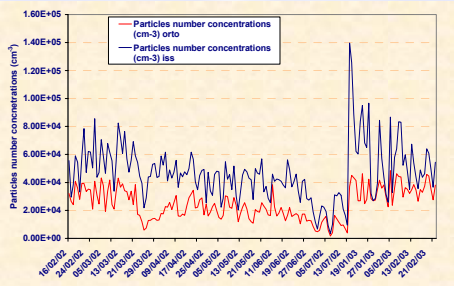
Daily trend trough the years are reported Higher values were found during winter period.



Hourly averages trend trough the years are reported. Particles concentrations were very well correlated with traffic, with the higher mean values during the hours of higher traffic volume.



Week-day typical trough the year are reported. Mean concentrations for the different days of the week are very close, with a minimum, as expected, during Sunday.



The concentration of particles at the background site were about 50% lower with respect to the traffic oriented site. In both sites particles concentrations were very well correlated with traffic, with highest mean values during rush hours. Otherwise, peak concentrations decreased sharply with the distance from the sources. Comparison between the particles number concentrations measured at the two sites showed a good correlation (r = 0.74).
In the most polluted cities the problem of the spatial variability of UF is a relevant issue.

	Valid N	Mean	S.D.	Min	25th Perc	Median	75th Perc	Max
$PM_{2.5}$ ($\mu g/m^3$)	387	24.0	12.2	4.4	16.2	21.3	29.0	87.9
PM_{10} ($\mu g/m^3$)	389	42.0	18.7	6.7	30.2	38.6	48.5	124.9
$PM_{2.5}/PM_{10}$	314	0.58	0.13	0.21	0.49	0.58	0.67	0.99
CO (mg/m^3)	659	1.4	0.8	0.3	0.8	1.2	1.7	6.3
NO_x ($\mu g/m^3$)	661	86.8	54.7	14.4	49.6	70.7	108.5	344.9
NO ($\mu g/m^3$)	661	41.9	44.9	1.6	12.7	25.7	52.3	288.9
NO_2 ($\mu g/m^3$)	661	44.9	14.5	12.1	34.4	44.5	53.9	89.2
O3 ($\mu g/m^3$)	688	32.8	18.1	2.9	17.3	32.9	46.8	86.2
Particles iss (pp/cm^3)	630	4.56E+04	2.47E+04	3.50E+03	2.76E+04	4.10E+04	5.80E+04	1.40E+05
Particles orto (pp/cm^3)	178	2.50E+04	1.11E+04	1.73E+03	1.65E+04	2.41E+04	3.43E+04	4.83E+04

Mean of the 24 hours daily mean over the whole period and descriptive statistics of total particles number concentrations, $PM_{2.5}$, PM_{10}, CO, NO_x, NO_2 and O_3 are reported. Mean values trough the year were lower than the target value recommended by the UE Directive, except for PM_{10}. No target limit values were still proposed by UE for $PM_{2.5}$ and particles number concentration.

Variable	$PM_{2.5}$	PM_{10}	CO	NO_X	NO	NO_2	O_3	Particles
$PM_{2.5}$	1.0000							
PM_{10}	0.8511	1.0000						
CO	0.6452	0.5729	1.0000					
NO_X	0.7052	0.6269	0.8954	1.0000				
NO	0.6710	0.6037	0.8970	0.9767	1.0000			
NO_2	0.5550	0.4598	0.5852	0.7456	0.5851	1.0000		
O_3	-0.4044	-0.3080	-0.7039	-0.7082	-0.6756	-0.5851	1.0000	
Particles	0.5534	0.4976	0.7688	0.8336	0.8168	0.6234	-0.6341	1.0000

CO, NO and NO_X are all highly correlated with ultrafines (577, 576 and 576 obs. respectively). CO could be used as a retrospective exposure parameter.

Contact person at the conference: Achille Marconi, Italian National Institute of Health - Viale Regina Elena 299 00181 Rome, Italy + 39 06 49902202 marconi@iss.it

ASP

ISTITUTO SUPERIORE DI SANITÀ

PM_{10}- Exposure Assessment For A City In Europe From 1950 - 2050

N. Metz

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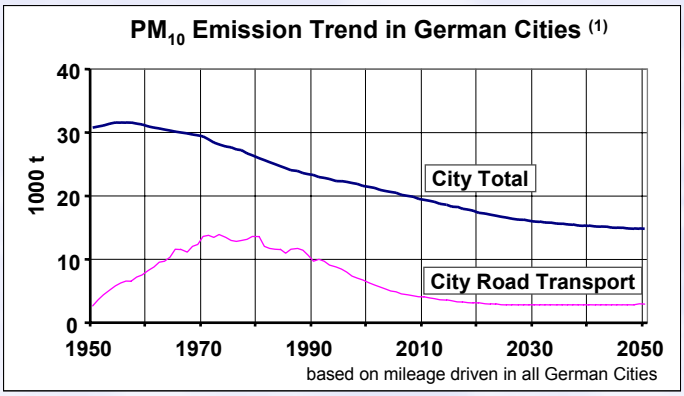
NERAM
Network for Environmental Risk Assessment and Management

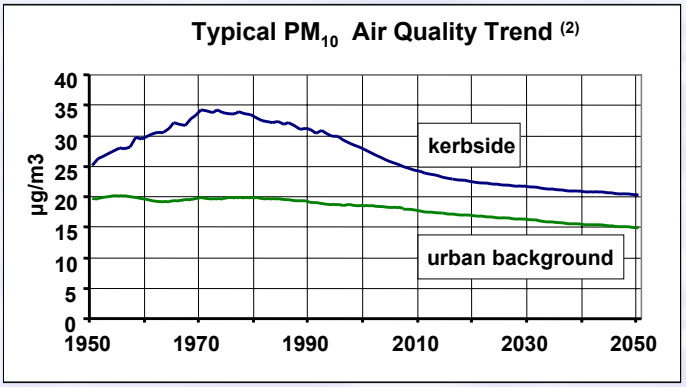
Background

For most epidemiological studies the exposure pattern in the past is unknown since measurement of air quality data in Germany started 1980. To close the gap for the time before and the development in the future an air quality assessment is made by taking the main emitters into account. Road transport is one of the main emitters. Therefore the development of the emissions of two wheelers, passenger cars, light and heavy duty vehicles and busses are considered. The contribution of other emitters is important for the development of the urban background levels. With the correlation of measured air quality and emission data in the time from 1980 until 2000 it is possible to recalculate the air quality in the past and to predict the air quality from 2003 onwards according to the expected emissions.



Features

Emission data for most sources are available from the German Umweltbundesamt (UBA). For road transport the German UBA has developed an emission model TREMOD which allow the forecast in the future until 2020. This model is expanded until 2050 and also for the time before 1980 back to 1950. PM_{10} is chosen as component and Munich is chosen as a typical city. PM_{10} air quality data are available from "Bayerisches Landesamt für Umweltschutz" in Munich. A simple box model allows to derive the air quality in the missing time period from correlations in the periode where both emission and air quality data exist.

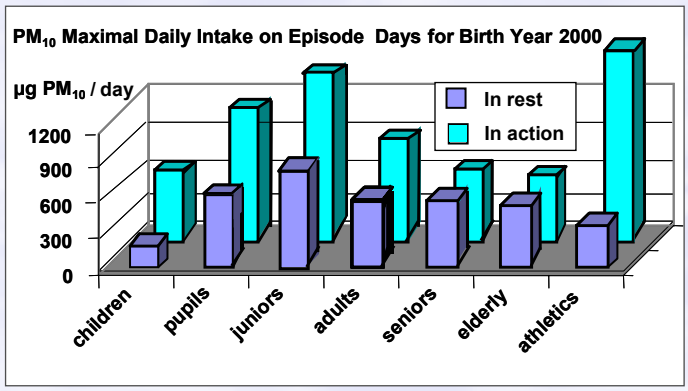


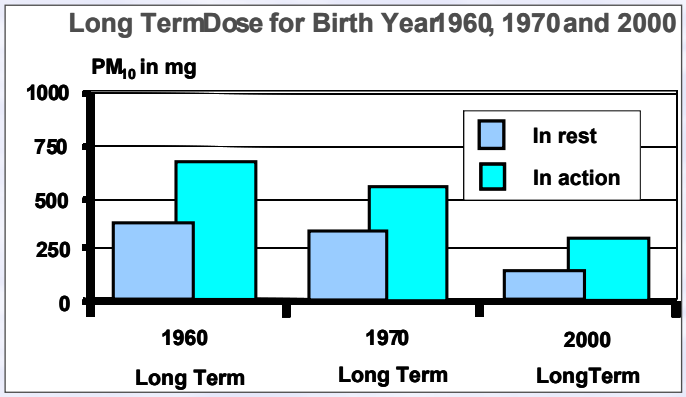
Findings and Conclusions

PM emissions from road transport before 1980 were lower than in the time from 1970 to 1980. PM emission of other sources, e.g. coal burning, were higher in the fifties and declined with the increasing use of heating oil. With the increase of vehicles since 1950 also PM_{10} air quality concentrations increased near kerbside, while urban background levels were almost constant until 1990. Later on the introduction and permanent improvement of diesel technology and diesel fuel quality led to declining PM-emissions from passenger cars and duty vehicles. PM-emissions reduction have an ongoing trend in the future. The contribution of road transport is declining and therefore PM_{10} air quality in the future is slowly improving as well. Even at sites where most inhabitants are exposed the EU air quality standard derived by WHO coming into force in 2010 will be met at normal meteorological conditions.

PM_{10} Exposure for different age groups is dependent from the birth year. With short term (98%-Percentiles)-Values in 2000 as an example Juniors in action have the highest daily intake within age groups. Athletics during their exercise do have the highest daily intake. Based on annual means the max. daily intake (not shown here) is about a third of that.

With the assumption that 20% of the inhaled particles of this size class are deposited and 90% are cleared with time the whole life dose (90 years) for somebody with the birth years 1960, 1970 and 2000 on the basis of annual means is calculated. In 2000 the long acting PM_{10} dose is less than ½ compared with 1960.





(1) based on UBA emission inventories, Berlin

(2) Based on Measurements of Bayerisches Landesamt für Umweltschutz, Augsburg

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Review of the Health Effects from Motor Vehicle Traffic in Tbilisi

Dr. Ketevan Samadashvili

AIRNET
A Thematic Network on Air Pollution and Health

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The Factors Contributing to Air Pollution in Tbilisi

In Georgia atmospheric air pollution has always been the environmentally most sensitive issue. Despite the sharp decline in transportation and industrial activities in recent years, Tbilisi continues to show signs of environmental stress - poor air quality, excessive noise, traffic congestion, loss of green areas and degradation of historical buildings and monuments. Many stresses, especially from transport, are increasingly leading to deterioration in the quality of life and human health.

Vehicles overload the capital nowadays. Most of them are 15-20 years old. The key common feature is that Soviet-made cars are designed to run relatively low octane petrol. They are characterized with low efficiency of fuel consumption; obsolete burning system, lack of any emission control equipment and thus very high level of emissions.

Table 1 The Quantity of Motor Vehicles in Tbilisi (31.10.02).

#	Type of vehicle	Total		Properly maintained	
		Soviet-made	Foreign	Soviet-made	Foreign
1.	Cars	81763	12377	68327	9933
2.	Buses	453	-----	348	-----
3.	Trolley-buses	17607	100	31107	70
4.	Trucks	1455	322	1319	243
5.	Vans		-----		-----

Four fifths of Tbilisi air pollution results from motor transport. Some part of it origins due to deficient traffic management and the other part is due to the poor technical condition of vehicles.

According to HYDROMET information air quality in Tbilisi in 2002 was represented by the following data:

Table 2 Air Pollution Substances in Tbilisi

Pollutants	Dust	CO	NO_2	Formaldehyde	Phenol	NO
Mean ann. con. [mg/m^3]	0.55	4.8	0.04	0.015	0.005	0.03
Max. ann. con. [mg/m^3]	1.00	11.5	0.10	0.028	0.01	0.07

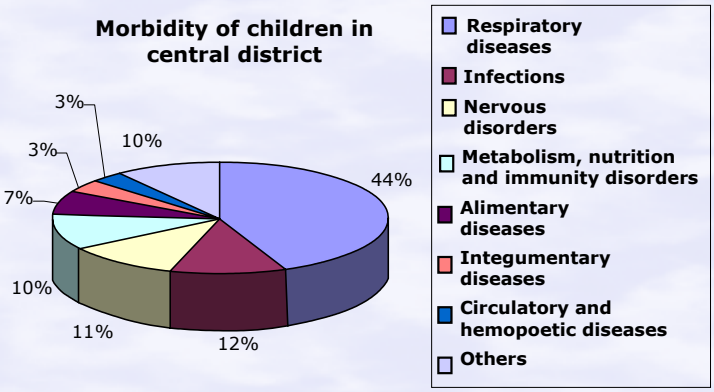
According to the data mean annual concentration of dust exceeds MPC (maximum permissible concentration) 2.2 times, mean annual concentration of CO is 1.18 MPC, concentration of formaldehyde is 5 MPC, concentration of phenol – 1.6 MPC. Mean annual concentrations of nitrogen oxides are within the allowed limits. Maximum annual concentration of dust is 4 MPC, concentration of CO is 2.8 MPC, concentration of NO2 – 3.78 MPC. Maximum annual concentrations of phenol, formaldehyde and NO are within the allowed limits.

Health Effects

High instances of respiratory and circulatory diseases in the capital of Georgia appear to be related to high levels of air pollution.

We carried out a special investigation of the state of health of 7-17 years old children. Two districts have been selected for study – central, with permanently heavy traffic and high air pollution and suburb, where the traffic is less intensive, as control district for comparison. Following results has been obtained:

Level of respiratory illness higher among the children from central district than among those from the suburb. Also the skin diseases are obtained 2.5 times more. Some changes in immune system are investigated too.



Approximation of EU and Georgia legislation

There is an obvious necessity of development of the traffic circulation management system in the city. Traffic management and road improvements thus should be studied simultaneously, and both should have high priority in any strategy to reduce air pollution.

Georgian authorities have stated a long-term objective for our country to become an EU member. This will require approximation of Georgian law to the Council Directive 96/62/EC on Ambient Air Quality Assessment and Management.

It is worthwhile to mention that some progress towards harmonization has already been achieved in Georgia. The use of leaded petrol was completely banned in 1999 and new standard specifications for petrol were developed and introduced in 2000

Notes

----- - Information not available

REC CAUCASUS

AIR POLLUTION: BANGLADESH PERSPECTIVE

M.S. Alam, Institute for Environment and Development Studies
Dhaka, Bangladesh

AIRNET Science-Policy Interface Work Group

AIRNET
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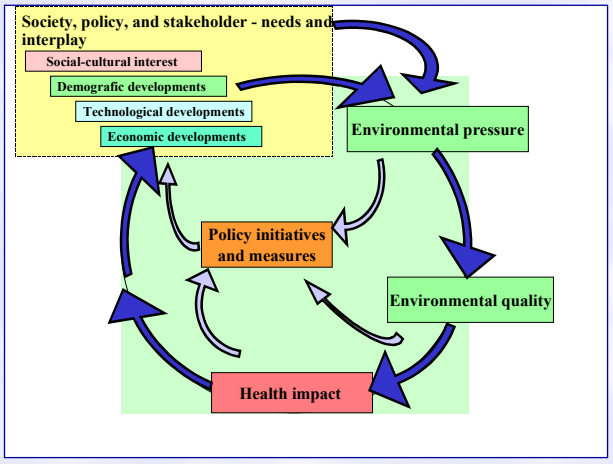
Objectives

- Discuss and interpret the outcomes of air pollution and health research. Link these outcomes with policy issues, abatement strategies and control implications.
- Identify research agenda based on the needs of policy makers and stakeholders.
- Perform an overall analysis of the AIRNET project (originally task of WG6). Evaluate to what extent the Network has achieved its aims and suggest whether follow up activities may be useful.

Science-Policy Interface Work Group aims to achieve this by:

- Identifying information and research needs relevant to end-users such as policy makers, industry, NGOs and the public.
- Improving linkages and developing a close interaction between the scientific community and the stakeholders (i.e. the users of AIRNET's output).



Report structure

Science-Policy Interface end-report

Part 1 Introducing the interface

1. Concept of science-policy-society interface and its meaning for air pollution and health *(J. Tuomisto, A. Petersen, D. Briggs, F. Langeweg)*
2. End-user needs and issues *(E. Rameckers, P. van den Hazel, CONCAWE, N. Fudge, A. Totlandsdal)*
3. Current policy strategies and decision framework *(CONCAWE, K. Katsouyanni, M. Amann)*

Part 2 Application of the interface

4. Integrated assessment and cost-benefit modelling in air quality management *(M. Amann, F. Hurley, R.Maas)*
5. Public health benefits of reduced air pollution levels and implemented control measures *(A. Totlandsdal, N. Stilianakis, P. Schwarze, F. de Leeuw, L. van Bree)*
6. Health risk communication *(E. Rameckers, P. van den Hazel, N. Fudge, W. Tuinstra)*
7. Policy, society and stakeholder driven research priorities *(to be confirmed)*
8. Health-beneficial clean air policy options and tools *(A. Rabl, F. Hurley, R. Maas)*

Overall summary and integration of the AIRNET project

Proposed outline…

1. Input of the evidence from working groups 1-5
2. Air pollution and health – key information and policy implications
3. Health-effective clean air strategies, options and priorities
4. Policy recommendations to the CAFE programme
5. Most prominent end-user driven research priorities and strategies
6. Value of integrating network projects and information capacity building
7. Value of AIRNET as an integrated network project; how does AIRNET contribute to developing/improving the science-society-stakeholder interface?

Proposed authors: AIRNET management team and work group leaders

Timeline

November 2003	AIRNET/NERAM Conference, Rome 3rd SPI work group meeting – d iscussion of end-report and selection of 'frequently asked questions' (FAQs)
November 2003 – April 2004	Preparation of SPI end-report and answering of FAQs
Spring 2004	4th SPI work group meeting – discussion of draft end-report
May 2004	Drafts of reports from work groups 1-5 available
May – September 2004	Development and preparation of integrated report
September 2004	Finalise SPI end report and AIRNET integrated report
October 2004	AIRNET Conference, Prague Presentation of end-reports
Ongoing	• Identification of key scientific papers within air pollution and health for AIRNET Alert! • Writing of non-specialist summaries of recent important science-policy related papers for AIRNET Alert! • Preparation of SPI workshops. Output from these workshops will be incorporated into the end-report.

For more information…

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  → L.van.Bree@rivm.nl
- Contact Nina Fudge within the AIRNET management team
  → nina.fudge@rivm.nl
- Visit the AIRNET Science-Policy Interface web page
  → http://airnet.iras.uu.nl

Institutes and organisations involved in the science-policy interface work group

- CEFIC (Conseil Européen de l'Industrie Chimique / European Chemical Industry Council)
- Centre for Energy Studies, Energy and Technical Innovation, Paris, France
- CONCAWE (International Association of Oil Companies with Refining Capacity in Europe), Brussels, Belgium
- Department for Environment, Food and Rural Affairs (DEFRA), London, UK
- Department of Environmental Medicine, National Institute of Public Health, Oslo, Norway
- Department of Epidemiology and Public Health, Imperial College, London, UK
- Department of Hygiene and Epidemiology, Medical School, University of Athens, Athens Greece
- Division of Environmental Health, National Public Health Institute, Kuopio, Finland
- EEA (European Environment Agency), Copenhagen, Denmark
- EFA (European Federation of Allergy, Airways Diseases Patients Association)
- Environmental Health Department, National Institute of Public Health Surveillance (Institut de Veille Sanitaire), Saint Maurice, France
- Eurometaux (Association of the European non-ferrous metals industry), Brussels, Belgium
- EuroNickel
- European Academy Science Advisory Council
- European Commission, DG Environment CAFE programme, Brussels, Belgium
- European Commission, DG Research, Brussels, Belgium
- European Commission, DG SANCO (Health and Consumer Protection), Brussels, Belgium
- European Commission Joint Research Centre (JRC), Ispra, Italy
- Ministry of Housing, Spatial Planning and the Environment, (VROM), The Netherlands
- Netherlands Environmental Assessment Agency (RIVM), Bilthoven, The Netherlands
- Institute of Occupational Medicine, Napier University, Edinburgh, UK
- Institute for Risk Assessment Sciences (IRAS), Utrecht University, The Netherlands
- International Institute for Applied Systems Analysis (IIASA), Laxenburg, Austria
- International Society of Doctors for the Environment (ISDE), Corcelles-sur-Chavornay, Switzerland
- SEAaT (Shipping Emissions Abatement and Trading), UK
- Swiss Agency for the Environment, Forest and Landscape (SAEFL), Air Pollution Control Division
- Umweltbundesamt (Federal Environmental Agency), Berlin, Germany
- VITO (Flemish Institute for Technological Research), Mol, Belgium
- WHO European Centre for Environment and Health, Bonn, Germany
- WHO European Centre for Environment and Health, Rome, Italy

AIRNET Epidemiology Work Group

Introduction

The objective of this work group is to facilitate an interactive communication and review forum to gather, discuss, and interpret the findings arising from epidemiological research on air pollution and how this will impact the risk and health assessment.

Thereby this work package, together with the package on exposure assessment and toxicology will contribute to a better understanding of health effects of air pollution and related exposure conditions important for this.

The Work Group will deliver a report "Epidemiology" containing the output of the Working Group. Short interim reports will be prepared based on the Annual Conferences, website information, and Working Group meetings.

Activities of the workgroup

1. Identifying the questions asked by end-users.

2. Identifying all European research in the field and put it into context with research from outside Europe. Provide non-specialist summarization of research findings which are most relevant to end-users.

3. Assess the potential policy implications of these findings and identify research needs and open questions.

The starting point for the report is relevant research funded by E.C. FP4 and FP5.

Time path

AIRNET covers the years 2002-2004.

- The first meeting has taken place in Utrecht in July, 2002.
- The second meeting in December 2002 in London (1st annual Conference) where a a draft Work Plan was discussed
- The third meeting was in Utrecht in June 2003 when first draft text outlines of the report were discussed
- Rome meeting: discussion of draft texts
- September 1 2004 Completion of the report

Report Structure

1. Introduction (Katsouyanni)

2. Role of Epidemiological studies (Katsouyanni)

3. End-user information needs

4. Health effects of pollutants

4.1.1 Introduction of particle fractions (Pekkanen)

4.1.2 Particle mass (Hoek)

4.1.3. Traffic and other combustion particles (Peters)

4.1.4 Road dust (Forsberg)

4.1.5 Wood burning (Forsberg)

4.1.6 Ultrafine particles (Pekkanen)

4.1.7 Secondary particles (Peters, Wichmann)

4.1.8 PAH (Sram)

4.2 Ozone (Hoek)

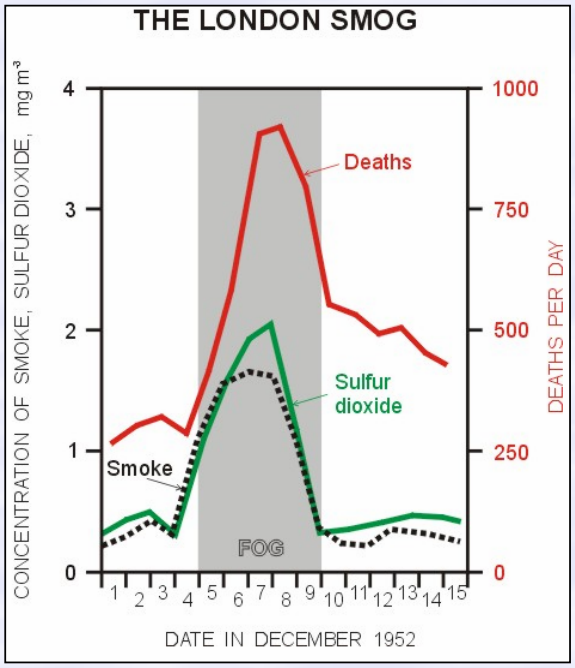
4.3 Nitrogen dioxide (Forastiere)

4.4 Sulphur dioxide (Sunyer / Downs)

4.5 Carbon monoxide (Fletcher)

5. Assessment of key issues and future research needs (Katsouyanni)

6. Potential policy implications epidemiology findings (Katsouyanni)



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AIRNET Exposure Assessment Work Group

Introduction

The objective of this work group is to facilitate an interactive communication and review forum to gather, discuss, and interpret the findings arising from research studying and assessing exposure of the general population and specific risk groups to air pollution and how this will impact the risk and health assessment. Thereby will this work package, together with the package on epidemiology and toxicology, contribute to a better understanding of health effects of air pollution and related exposure conditions important for this.

The Working Group will deliver a report "Exposure" containing the output of the Working Group. Short interim reports will be prepared based on the Annual Conferences, website information, and Working Group meetings.

Activities of the workgroup

1. Identifying the questions asked by end-users.
2. Identifying all European research in the field and put it into context with research from outside Europe. Provide non-specialist summarization of research findings which are most relevant to end-users.
3. Assess the potential policy implications of these findings and identify research needs and open questions.

The starting point for the report is relevant research funded by E.C. FP4 and FP5.

Contrast in exposure to (traffic-related) air pollution

Time path

AIRNET covers the years 2002-2004.

- The first meeting has taken place in Utrecht in June, 2002.
- The second meeting in December 2002 in London (1st annual conference) where a a draft Work Plan was discussed
- The third meeting was in Utrecht in June 2003 when first draft text outlines of the report were discussed
- Rome meeting: discussion of draft texts
- Discussion of 2nd draft report at WG meeting (Spring 2004)
- September 1 2004: Completion of the report
- Presentation at 3rd AIRNET conference in Prague (Oct. 2004)

Air pollution measurement at the roof of a school near a major motorway

Report Structure

1. Preface (*Janssen*)
2. Definitions of concentrations, exposure, dose (*Janssen*)
3. End-user information needs
4. State of the art in Exposure Assessment of PM, PAH, $N0_2$, O_3, C0 and $S0_2$

4.1. Ambient concentrations

4.1.1. Concentration levels throughout Europe (*Urbanus*)

4.1.2. Temporal and spatial variation (*Briggs, Ziomas, Amann*)

4.2. Modelling of exposure: determinants and surrogates (*Briggs, Ziomas, Amann*)

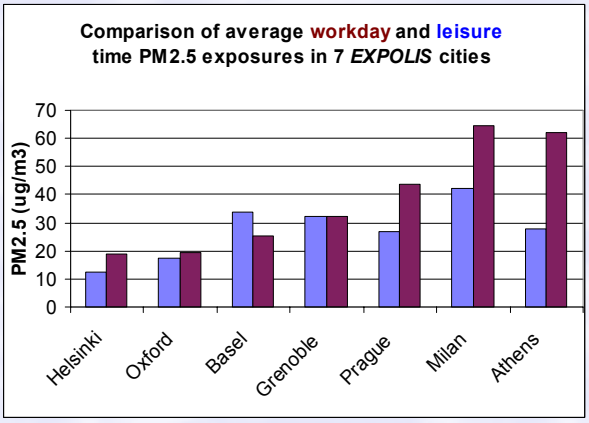
4.3. Personal exposure to the ambient fraction (*Bayer-Oglesby*)

4.4. Biological markers of exposure (*Sram*)

4.5. Exposure misclassification (*Janssen*)

4.6. Implications for epidemiological studies and risk assessment (*Hoek*)

5. Assessment of key issues and future research needs (*Jantunen*)
6. Potential policy implications of exposure (*Jantunen*)



Institutes involved in the exposure assessment work group

International Institute for Applied Systems Analysis (IIASA), Laxenburg, Austria
TotalFinaElf, Research center BP, Solaize Cedex, France
National Institute of Public Health and the Environment (RIVM), Bilthoven, The Netherlands
Institute for Risk Assessment Sciences (IRAS), Utrecht University, The Netherlands
Institute for Risk Assessment Sciences (IRAS), Utrecht University, The Netherlands
Imperial College of Science, Technology and Medicine, Department of Epidemiology and Public Health, London, United Kingdom
KTL - Environmental Health, Kuopio, Finland
University of Basel, Institute of Social and Preventive Medicine, Basel, Switzerland
Laboratory of Genetic Ecotoxicology, Institute of Experimental Medicine Acad, Prague, Czech Republic
CONCAWE, International Association of Oil Companies with Refining Capacity in Europe, Brussels, Belgium
Department of Chemical Engineering, National Technical University of Athens, Athens, Greece
BP International Ltd, Middlesex, UK

AIRNET Health Impact Assessment Work Group

E. Sanderson and M. Krzyzanowski (Work Group Leaders)
M. Amann G. Büchele, P. van Hazel, F. Hurley, G. de Jong, A. Karakatsani, A. Le Tertre, S. Medina, B. Miller, E. Rameckers, C. Roythorne, J. Tuomisto, S. Weiland, and G. Weinmayr (Contributors)

Introduction

The objective of this work package is to facilitate an interactive communication and review forum to gather, discuss, and interpret the outcome of air pollution risk and health impact assessments using the findings from exposure assessments, epidemiology, and toxicology. A particular focus on the elements of the HIA and the uncertainties at each level will be addressed for the various end users (i.e. policy makers).

Thereby this work package will form a bridge-building network to optimally and inter-actively link all the scientific disciplines from which the input is needed for an adequate and sound health risk and impact assessment of air pollution (see Figure 1).

The Work Group (WG) will deliver a report "HIA" containing the output of the contributing authors. Short interim reports will be prepared based on the Annual Conferences, website information, and Working Group meetings.

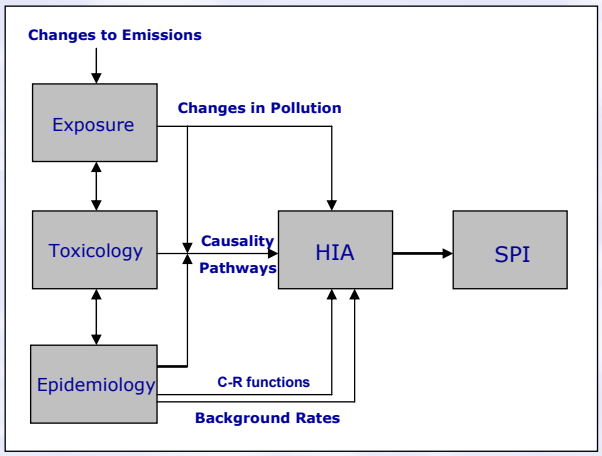


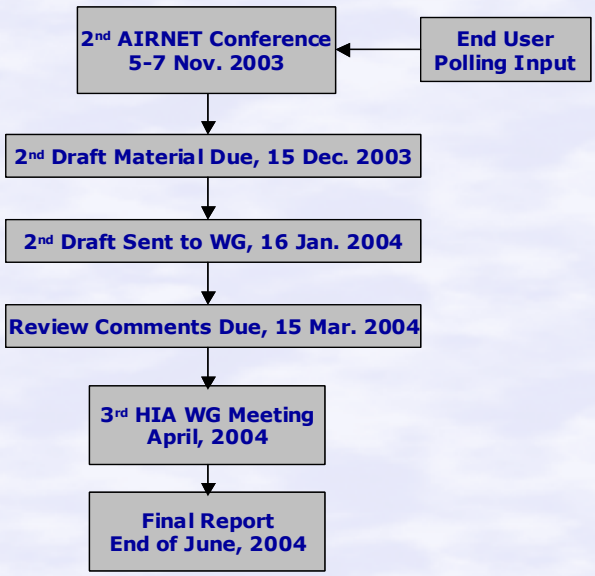
Figure 1. How other WGs link with HIA: a simplified schema.

Activities of the WG

1. Identifying the questions asked by end users.
2. Identifying all European research in the field and put it into context with research from outside Europe. Provide non-specialist summarization of research findings which are most relevant to end-users.
3. Assess the potential policy implications of these findings and identify research needs and open questions.

The starting point for the report is relevant research funded by E.C. FP4 and FP5.

Time Line



Report structure

1. Preface
2. Purpose of HIA [G. de Jong, C. Roythorne]
2.1 Assessment of the baseline situation
2.2 Impacts of changing pollution level
2.3 Changes of pollution due to certain decisions and alternative actions (i.e. traffic changes, urban development, etc.)
2.4 Links with further steps assisting decision making (i.e. economic analysis)
3. Results of HIA (attributable cases, attributable YLL, DALYs, QUALYs, etc.) [G. Büchele, G. Weinmayr, S. Weiland, P. van Hazel]
4. Attributable and Avoidable Impacts (i.e. time scale of the impacts, reversibility of effects) [M. Krzyzanowski, B. Miller, F. Hurley]
5. Information Necessary for HIA (link with European research) [B. Miller, F. Hurley]
5.1 Exposure-response function
5.2 Exposure information
5.3 Underlying frequency of health outcome
6. Uncertainty Analysis [G. de Jong, C. Roythorne, F. Hurley]
6.1 Statistical issues
6.2 Sensitivity analysis
7. Case Studies/Examples [S. Medina, A. Karakatsani, A. Le Tertre]
7.1 Impacts of present situation (e.g. APHEIS2)
7.2 Impacts of changing pollution level (e.g. APHEIS3 with new AirQ)
7.3 Impacts of changes of pollution due to certain decisions (i.e. traffic changes, urban development, etc.) [J. Tuomisto]
8. Assessment of Key Issues and Future Research Needs (Everyone)
9. Potential Policy Implications of Health Impact Assessment Findings [M. Amann]

http://airnet.iras.uu.nl/

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AIRNET Toxicology Work Group

Objectives

• Facilitate an interactive communication and review forum to gather, discuss and interpret the findings arising from toxicology research on air pollution and how this will impact risk and health assessment

• Contribute to a better understanding of causality and the biological plausibility of health effects of air pollution and components and exposure conditions important for this together with the exposure and epidemiology work groups

HOW?

1. Identification of issues relevant to end-users such as policy makers, industry, NGOs and the public
2. Publication of non-specialist summaries of research findings that are especially relevant for end-users
3. Assessments of the potential policy implications of those research findings

Report structure

1. Preface (0.5 page)

2. End-user information needs
- co-ordinated with other AIRNET WG groups - 0.5 page

3. State-of-the-art in toxicology
3.1 PM (12 pages)
3.1.1 Dosimetry (Kreyling)
3.1.2 Human clinical studies (Sandström)
3.1.3 Animal studies (Cassee)
3.1.4 Cellular and mechanistic studies (Donaldson)
3.1.5 Health implications (Kyrtopoulos)

3.2 Ozone (8 pages)
3.2.1 Dosimetry (Kyrtopoulos)
3.2.2 Human clinical studies (Sandström)
3.2.3 Animal studies (Bernard)
3.2.4 Cellular and mechanistic studies (Bernard)
3.2.5 Health implications (van Bree)

3.3 Nitrogen dioxide (8 pages)
3.3.1 Dosimetry (Kyrtopoulos)
3.3.2 Human clinical studies (Sandström)
3.3.3 Animal studies (Schwarze)
3.3.4 Cellular and mechanistic studies – (Schwarze)
3.3.5 Health implications (Schwarze/ Sandström)

3.4 PAH (6 pages)
3.4.1 Dosimetry (Botsivali/ Farmer)
3.4.2 Human clinical studies (Botsivali/ Farmer)
3.4.3 Animal studies - (Botsivali/ Farmer)
3.4.4 Cellular and mechanistic studies (Botsivali/ Farmer)
3.4.5 Health implications (Botsivali/ Farmer)

3.5 CO (4 pages)
3.5.1 Dosimetry (Refsnes/ Låg)
3.5.2 Human clinical studies – (Refsnes/ Låg)
3.5.3 Animal studies (Refsnes/ Låg)
3.5.4 Cellular and mechanistic studies (Refsnes/ Låg)
3.5.5 Health implications (Refsnes/ Låg)

3.6 SO2 (4 pages)
3.6.1 Dosimetry (Hagemann)
3.6.2 Human clinical studies (Hagemann)
3.6.3 Animal studies (Hagemann)
3.6.4 Cellular and mechanistic studies (Hagemann)
3.6.5 Health implications (Hagemann/ Borm)

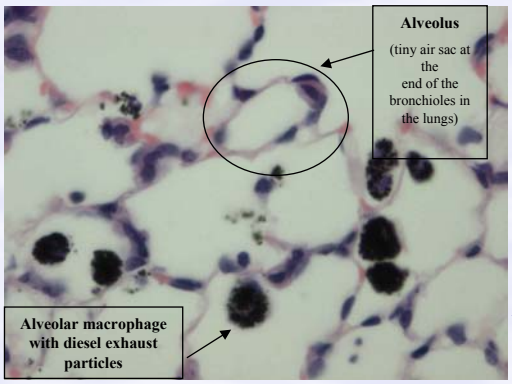
4. Assessment of key issues, additional concerns and future research needs (2.5 pages) (Dybing, Cassee, Donaldson, Gephart, Salonen)

5. Potential policy implications of toxicology findings (2.5 pages) (van Bree, Dybing, Cassee)

6. Frequently asked questions (total 10 questions) (2 pages)

Animal models are being widely used to measure the effects of exposure to air pollution. This figure shows a device used to expose rats to ambient air particles. Afterwards the rats are examined to investigate the possible effects of the exposure.

Photo: MGO, RIVM



This figure shows a cross section of a mouse lung instilled with diesel exhaust particles (50µl of 3 mg DEP/ml, intranasally instilled)

Photo: Dormans, RIVM

Upcoming events

- Discussion of 1st draft report (5 November 2003)
- Selection and answering of frequently asked questions
- Discussion of 2nd draft report at WG meeting (Spring 2004)
- Finalise report (September 2004)
- Presentation at 3rd AIRNET annual conference in Prague (October 2004)
- Identification of key scientific papers within air pollution and health for AIRNET alert
- Writing of non-specialist summaries of recent important science-policy related papers for AIRNET Alert! (ongoing)

For more information...

- Contact the chair: Erik Dybing

  => *erik.dybing@fhi.no*
- Contact Annike Totlandsdal within the AIRNET management team

  => *annike.totlandsdal@rivm.nl*
- Visit the AIRNET Toxicology webpage

  => http://airnet.iras.uu.nl

Institutes involved in the toxicology work group

Cancer Biomarkers and Prevention Group, Biocentre, University of Leicester, UK
CONCAWE, International Association of Oil Companies with Refining Capacity in Europe, Brussels, Belgium
Division of Environmental Medicine, Norwegian Institute of Public Health, Oslo, Norway
Department of Occupational and Environmental Medicine Sahlgrenska University Hospital and Academy Göteborg, Sweden
Division of Toxicology and Neurotoxicology, Karolinska Institutet, Stockholm, Sweden
Edinburgh Lung and the Environment Group Initiative (ELEGI) University of Edinburgh, Edinburgh Scotland
European Commision Joint Research Centre, Ispra, Italy
ExxonMobil Chemicals Europe, Europe Headquarters, Machelen, Belgium
Fraunhofer Institute of Toxicology and Experimental Medicine, Clinical Asthma- and Inhalation Research, Hannover, Germany
GSF - National Research Center for Environment and Health, Institute for Inhalation Biology, Neuherberg/Munich, Germany
Institute for Risk Assessment Sciences (IRAS), Utrecht University, Utrecht, The Netherlands
Institute of Biological Research and Biotechnology, National Hellenic Research Foundation, Greece
Istituto Superiore di Sanità (Italian National Institute of Health) , Rome, Italy
IUF - Environmental Health Research Institute (Institut für umweltmedizinische Forschung), Heinrich-Heine University Düsseldorf, Germany
National Public Health Institute, Unit of Environmental Epidemiology, Kuopio, Finland
Netherlands Environmental Assessment Agency - RIVM, Bilthoven, The Netherlands
Public Health and Clinical Medicine, Umeå University, Umeå, Sweden
School of Life Sciences, Napier University, Edinburgh, Scotland
Statoil Research Centre, Trondheim, Norway
Unit of Toxicology Faculty of Medicine, Catholic University of Louvain, Brussels, Belgium
University of Vienna Medical School, Division of Immunology, Allergy and Infectious Diseases (DIAID), Department of Dermatology, Vienna, Austria
VITO - Flemish Institute for Technological Research, Mol, Belgium

AIRNET/NERAM 2nd ANNUAL CONFERENCE IN ROME 5-7 NOVEMBER 2003

AIRNET stakeholder survey

A. Totlandsdal, N. Fudge, E. Sanderson, E. Rameckers, L. van Bree, B. Brunekreef, E. van Otterloo, G. Hoek, N. Janssen and M. Tewis

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Why?

Why organise a stakeholder polling?

- To ensure that stakeholders views are considered in AIRNET
- To ensure that AIRNET output meets end-users needs

What did we want to find out?

- Stakeholders' questions and issues of concern relating to air pollution and health
- How stakeholders like their information to be presented
- Stakeholders' expectations of AIRNET

How?

Input

1. Stakeholder presentations at the 1st AIRNET annual conference
2. Qualitative questionnaire to stakeholders in the field of air pollution and health

Questionnaire population

- Stakeholders within AIRNET
- Members of CAFE steering committee
- Stakeholders from APHEIS

Stakeholder categories

- Government agencies (international, national and local)
- European parliament
- Private sector (representatives form industry)
- NGO/ advocacy group
- Research institutes

Response rate (overall): 24% (65/264)

Note: only 3 out of 105 members of the European Parliament responded

Stakeholders prefer information that...

- is presented as short overviews of about 1-2 pages, synthetic executive summaries and non-specialist summaries
- is ready for policy use (including a practical linkage between the research findings and implementation of public protection)
- that can be passed on to other users

Stakeholders' expectations of AIRNET

- **Policy support role**
  Assist policy makers in the policy making process, by providing scientific information in a form that is usable in debating policy options
- **Information role**
  Provide information on the actual health status of European citizens, new research activities and findings, reports and conferences
- **Network role**
  Improve the co-ordination and focus of European research and bring it closer to air quality legislation and policy development, link experts to exchange experience and views, and link European research with research in other parts of the world
- **Stakeholder views**
  Identify and consider the views of stakeholders

Stakeholder questions...

- are very general compared to the ones that scientists have and like to answer
- cover a wide range of issues (health impact, policy, monitoring,communication, pollutants, etc)
- will be used as guidelines for the outline and content of the AIRNET work group reports

For more information...

If you would like to read the AIRNET stakeholder needs report please sign up for a hard copy at the conference registration desk. You can also download a pdf of the stakeholder report from the AIRNET website (http://airnet.iras.uu.nl)

"We need more information about the effects of bad odours on health, because people from the region of the ore mountains complain about their bad health and connect this to bad odour and substances" (Local government agency, Germany)

"It is of interest to know what other countries/organisations are focusing on. Information sharing and "lessons learned" is important" (National government agency, Norway)

"Much scientific literature is hard to interpret, and needs much more attention than should be necessary to understand the key results. Rarely is it presented with useful real world comparisons" (NGO, UK)

"There is a wealth of information and sources and it is in fact a challenge to keep up with all of the new information" (Private sector stakeholder, Belgium)

" Specific practical information to pulmonary doctors on how to manage and advise respiratory patients with regard to air pollution are lacking." (NGO - health care professionals, Switzerland)

"We are also concerned that clinical, toxicological, mechanistic and non-ecological exposure data are not being taken into account in any serious manner in assessing the effects of air pollution" (Private sector stakeholder, Belgium)

"What is the significance of hot spots of the pollution burden (e.g. busy street canyons) for exposure and compliance with limit values? (Local government agency, Germany)

"The lack of sufficient monitoring stations at the moment is compromising health impact assessment" (National government agency, Portugal)

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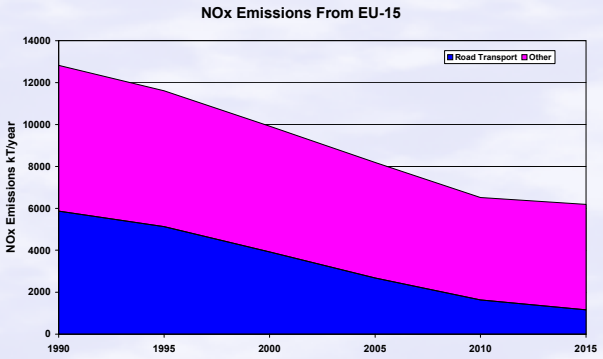
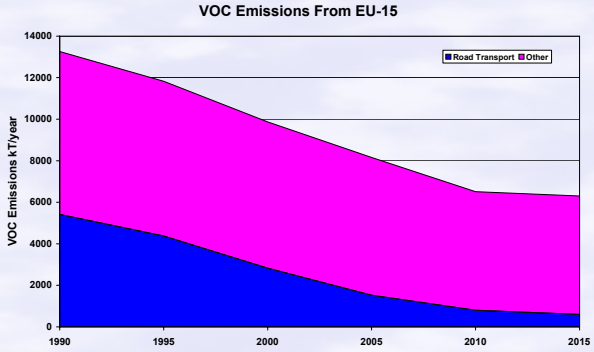
Trends in European Ambient Air Quality

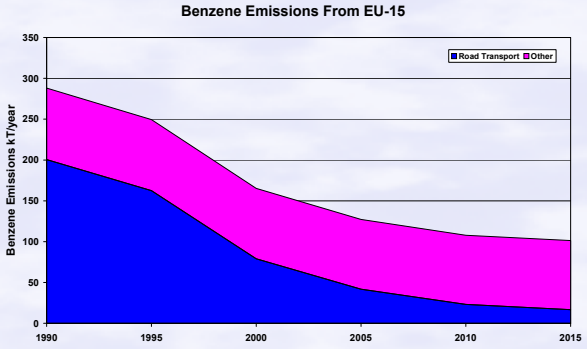
The concern with air quality was part of the original remit of CONCAWE and has been, over the last four decades, possibly the most prominent of all environmental issues in Europe. Particularly since the early 90's, the pace of legislation regulating emissions to air has been relentless. CONCAWE has been directly involved in all aspects linked to the activities of the downstream oil industry. The impact of the legislation has been impressive, achieving dramatic reductions in the levels of all main air pollutants in spite of the increase in activity levels and energy usage.

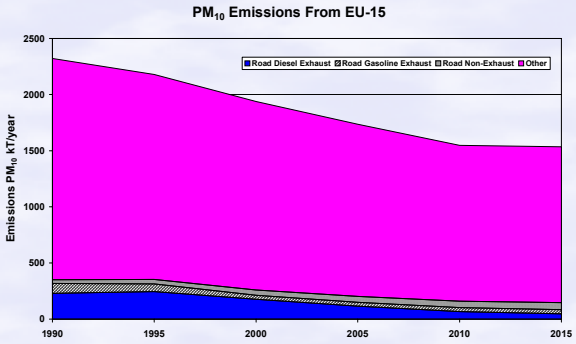
Air quality is a complex field and it is essential that legislation promotes actions that make a real difference. CONCAWE has consistently supported the "environmental-quality-driven" approach whereby a measure is judged by its capacity to produce results in air quality terms, at a reasonable cost as opposed to a "technology-driven" approach where the best technology is forced in, irrespective of its cost and its contribution to the achievement of air quality targets.

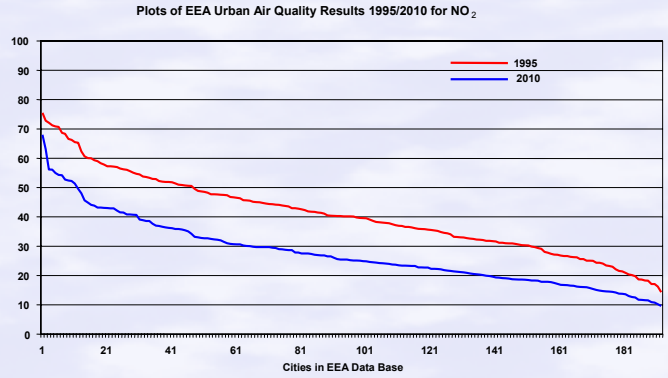
Legislation has so far focussed on one specific issue at a time such as sulphur in liquid fuels or control of VOC emissions. The CAFE (Clean Air For Europe) programme represents a change in emphasis in that it focuses on air quality and health targets and seeks to determine the most cost-effective measures likely to allow these targets to be reached. CONCAWE has welcomed this development and is actively contributing to the programme.

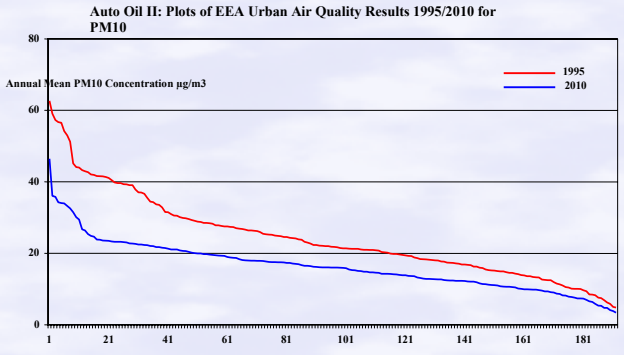












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Trends in European Automotive Fuels Quality and Emissions

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CONCAWE started its activities on fuels qualities and related automotive emissions in the late 70's. These last 25 years have seen momentous changes in the levels of vehicle emissions, with a substantial contribution from improvements in the quality of fuels.

In the early years work focussed on issues such as reduction of lead content of gasoline and definition of the optimum octane for unleaded gasoline. The introduction of the latter in the late 80's enabled the first generation of catalyst cars to enter the market.

In the 90's the emphasis shifted towards the interrelationships between vehicle technologies, fuels and emissions. The European Programme on Engines, Fuels, and Emissions (EPEFE) ushered in an era of cooperation between the auto industry, the fuels industry and the regulator, and paved the way for the Auto-Oil programmes in which CONCAWE played a leading role on behalf of the oil industry. With the introduction of 10 ppm sulphur fuels during this decade, the sulphur content of European road fuels will have been reduced by two orders of magnitude in less than 15 years, enabling a number of innovative vehicle technologies to further reduce emissions and improve fuel efficiency. CONCAWE has made regular contributions to the understanding of the interactions between vehicles and fuels by conducting a series of test programmes.

As the focus shifts towards greenhouse gas emissions, the quest for lower pollutant emissions remains an essential objective. Discussions on the definition of Euro-5 (light duty) and Euro-6 (heavy duty) vehicle emission standards have already started and so has the review of the 2000 Fuels Directive. New technologies are holding much promise to meet the ambitious goals of virtually eliminating pollutants emissions while increasing vehicle efficiency. Fuel improvements can play a role in this when they enable new technologies that allow a step change in performance.

Figure 1 Progress towards elimination of lead from gasoline by year 2000

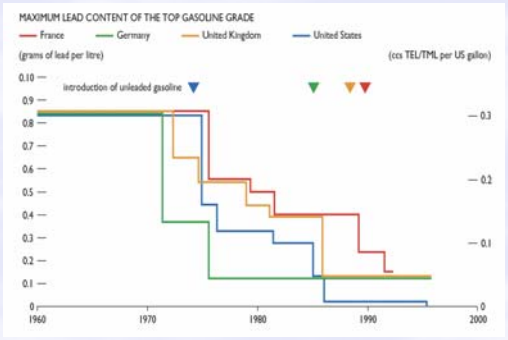


Figure 2 RUFIT study supported 95 RON as optimum octane level

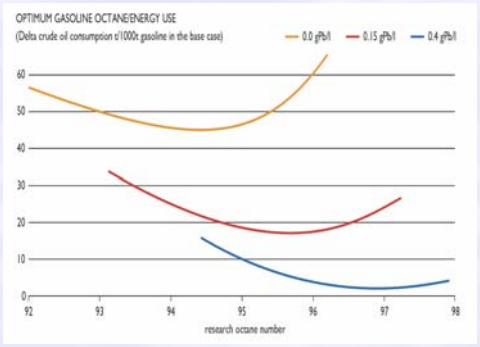


Figure 3 Impressive reductions in regulated emissions limits achieved

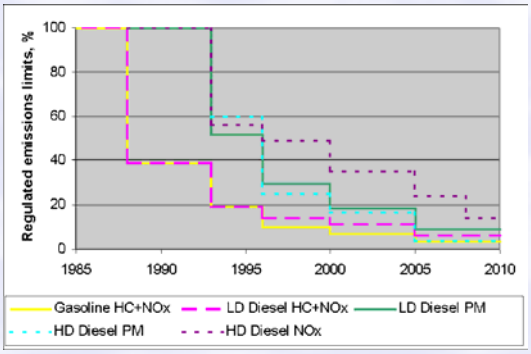
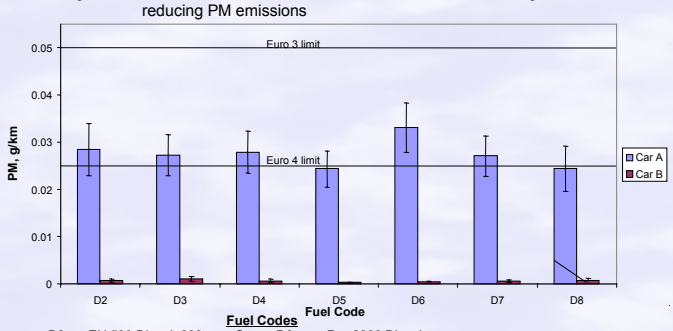


Figure 4 Summary of European gasoline and diesel fuel specification developments

Year			1993	1995	1996	2000	2005	2009
Gasoline Unleaded 95/85			EN228					
Sulphur	ppm m/m	max	1000	500		150	50/10	10
Benzene	% v/v	max	5			1		
Aromatics	% v/v	max				42	35	
Olefins	% v/v	max				18		
Oxygen	% m/m	max	2.5[1]			2.7		
RVP (summer)	kPa	max	up to 80			60[2]		
E100	% v/v	min	40(s)/43(w)			46		
FBP	°C	max	215			210		
Year			**1993**	**1995**	**1996**	**2000**	**2005**	**2009**
Diesel (standard grade)			EN590					
CI		min	46					
CN		min	49			51		
Sulphur	ppm m/m	max	2000		500	350	50/10	10[3]
Density	kg/m^3	min	820			360		
		max	860			845		
T95	deg C	max	370					
PAH	% m/m	max				11		
Lubricity	µm @ 60ºC	max			460			

(1) Up to 3.7% at Member State discretion. Individual limits apply to specific compounds
(2) 70 kPa max allowed in Member States with arctic or severe winter conditions
(3) End date for full introduction of 10 mg/kg S max diesel remains subject to further review

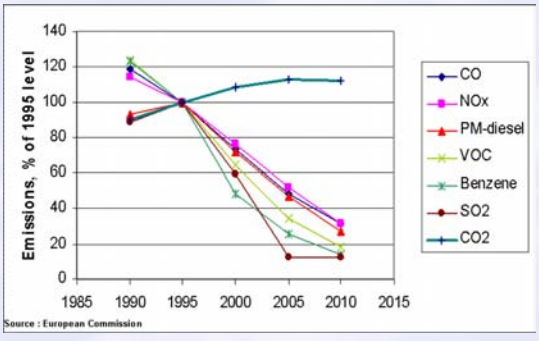
Figure 5 Particulate filter are much more effective than fuel changes in reducing PM emissions



Fuel Codes

D2	EN 590 Diesel: 280 ppm S	D6	Pre-2000 Diesel
D3	EN 590 Diesel: 38 ppm S	D7	D4 + 5% RME
D4	EN 590 Diesel: 8 ppm S	D8	Fischer-Tropsch Diesel
D5	Swedish Class 1 Diesel		

Figure 6 Current challenge is to reduce GHG emissions while also achieving very low regulated emissions



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Some special problems of improving air quality strategies for Russian cities. – View from the inside

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AIRNET
A Thematic Network on Air Pollution and Health

NERAM
Network for Environmental Risk Assessment and Management

Monitoring and AQ in cities:

- National AQ monitoring network **lunched in 60-es**
- Regular observations in more than **650 stations**
- More than **250 cities**
- Approx. **30 pollutants**
- **Annual exposure is very high in 35 cities**
- Among **5 major contributors** to very high exposure

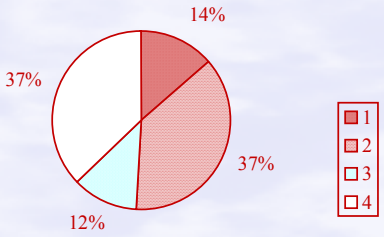
in these cities **are**:

- BaP in **33** cities, rank 1in 6 cities
- Formaldehyde in **31** cities, rank 1 in 12 cities
- Nitrogen dioxide in **26** cities, rank 1 in 5 cities
- TSP in **14** cities, rank 1 in 7 cities
- Phenol in **4** cities, rank 1 in 2 cities
- Hydrogen fluoride in **4** cites, rank 2 in 2 cities
- Carbon bisulfide in **4** cities, rank 1 in 2 cities

- **trends to higher annual average levels** in largest cities:
- nitrogen dioxide - in 5 cities
- formaldehyde - in 7 cities
- BaP - in 13 cities

Major current factors:

- A huge number (598) of pollutants under legal control with no prioritizing
- A legal status of AQ limit values is not high enough to enforce effectively
- AQ limit values are too strong to be met in the foreseeable future
- Hygienic criteria are regulatory ones at the same time
- Enormous growth of urban traffic in 90- is (hundreds per cent) growth of cars amount
- Infrastructure of cities is not correspond actual traffic
- High intensity of transit transportation through cities
- Low quality of fuel combustion in automobiles
- Industry sited in living districts leads to "specific" AP
- Energy losses (transportation, home heating)
- Low level of public awareness in AQ&Health issues



Air Pollution Index &Number of Cities (%):

1: API > 13 (very high)

2: API = 7 to 13 (high)

3: API = 5 to 6 (higher)

4: API < 5 (low) **(Normal AQ at API=5)**

Total population in cities under (1) and (2) is 58.1 mill. in 2002

What should be done?

What could be done?

- To answer the question: is clean air needed indeed or there exist different national/regional/municipal priorities?
- To answer the question: what is the acceptable AQ?
- To answer the question: what is the achievable AQ in the time fixed?
- To answer the question: could national/regional/municipal economy be converted to meet clean air everywhere?
- To answer the question: who/what should be responsible for clean air to be purchased to everyone?
- To develop and to declare to the nation clear AQ policy
- To determine pollutants of highest priority at national/regional/municipal level
- To establish AQ standards for priority pollutants as national law/governmental act and terms&rates to meet them as well
- To elaborate cooperation of authority, busyness, and society in AQ&Health issues
- To construct the bridge between decision makers and scientific community on AQ&Health issues
- To restore public transport
- To strengthen pressure upon industry (land price, technologies, products/goods
- To harmonize national approaches in activity on improving air quality with international ones
- To strengthen activity on improving policy makers/public awareness

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