COST Action 633 Particulate matter: properties related to health effects

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# Heterogeneities of PM-associated health effects: sources and PM characteristics

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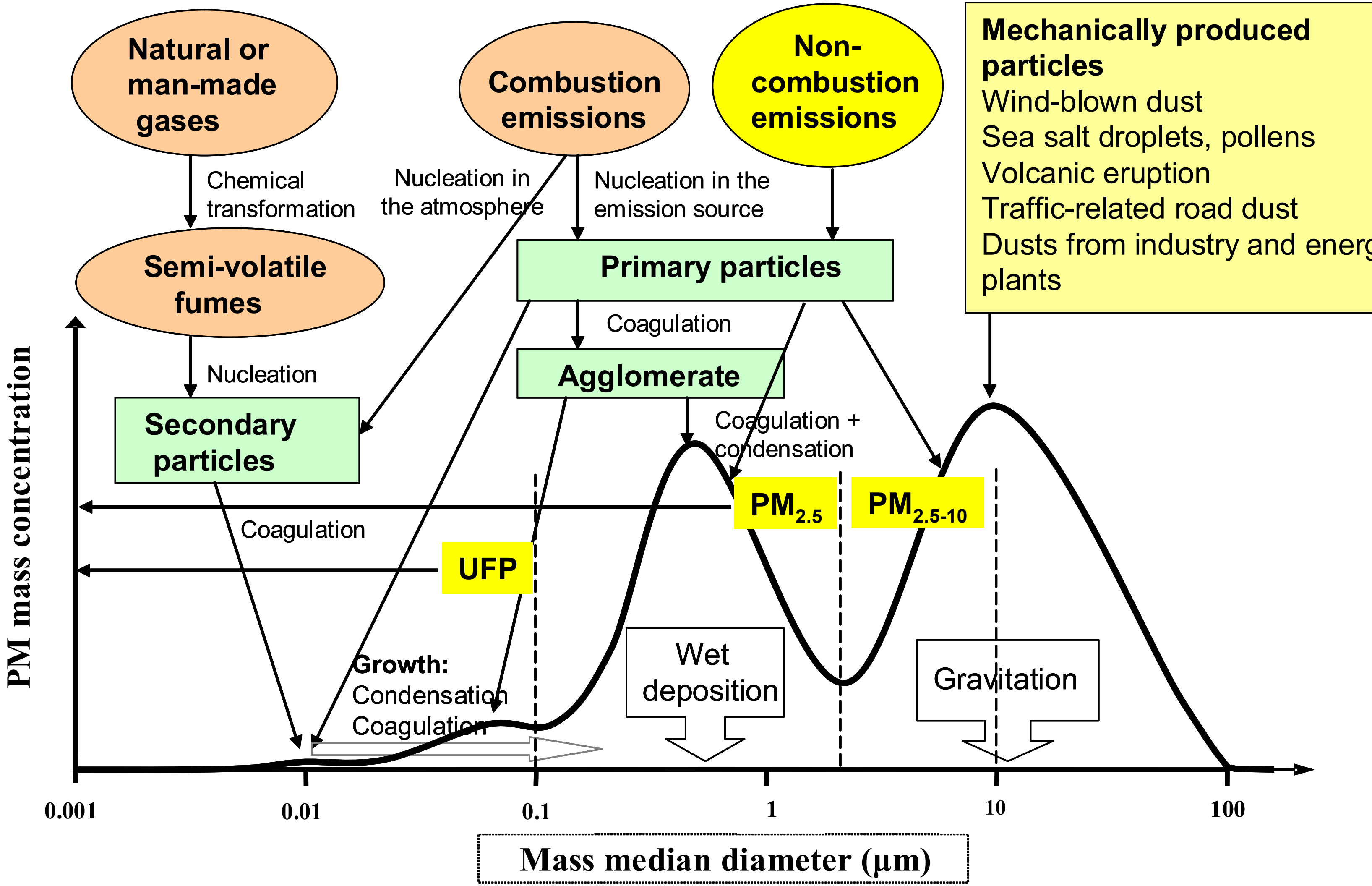
# Presentation contents

- Background information on the complex mixture of urban air particulate pollution and its health effects
- Objectives of the COST 633 Health Working Group
- Heterogeneities in the particulate mass-based exposure-response relationships
- Examples of important gaps in scientific knowledge in connection to particulate sources requiring better control
- Conclusions and recommendations

# The complex mixture of urban air particles in different size ranges




By courtesy of Dr. Markus Sillanpää, Finnish Meteorological Institute

# Clean Air for Europe (CAFE 2005)

- **Fine particulate matter ($PM_{2.5}$; diameter ≤ 2.5 m) causes the largest disease burden of all environmental factors to the European populations**
  - The $PM_{2.5}$ exposures in 2000 were assessed to have been associated with about **350 000 premature annual deaths**, substantial use of health services, and restricted activity in ten millions of children and subjects with chronic cardiovascular ar pulmonary diseases in the EU25
  - The European wide assessment of $PM_{2.5}$ exposure was made **regional scale** (50x50 km) with little data on increments from local emission sources
  - All particulate compositions were regarded **as equally harmfu health**
  - Updated health effect assessment data (WHO 2003 and 2004 were utilized, but **little information was available on source specific $PM_{2.5}$ exposures and health effects**

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# Particle size is an important factor in health effects

- Thoracic particles ($PM_{10}$; diameter < 10 μm) and, more strongly, fine particles ($PM_{2.5}$; diameter < 2.5 μm) consistently associated with mortality, hospital visits and functional decrements among respiratory and cardiovascular patients (WHO 2005)
- Coarse thoracic particles ($PM_{10-2.5}$; diameter 2.5-10 μm) associated mainly with hospital visits and functional decrements among respiratory patients (WHO 2005; Brunekreef and Forsberg 2005)
- Ultrafine particles (UFP; diameter < 0.1 μm) show health effects, including mortality, independent of $PM_{2.5}$ among the respiratory and cardiovascular patients (WHO 2005)
  - Potential to penetrate from the lungs to the systemic blood circulation? (Nemmar et al. 2002; Möller et al. 2008)

# Could the health scientists benefit more fro modern aerosol science in their studies?

## Objectives of the COST 633 Health Working Group

1) To review **European heterogeneities** in epidemiological and toxicological health outcomes associated with ambient air particulate matter
   - Regional differences in health outcomes
   - Seasonal differences in health outcomes
   - Heterogeneities in health outcomes related to specific source
2) To give **inputs** on the health aspects of particulate pollutio
   - To the Working Group on Air Quality and Instrumentation
   - To the Working Group on Sources and Modelling

# Heterogeneities in the particulate mass-base exposure-response relationships

- Mass-based ($PM_{10}$/$PM_{2.5}$) exposure-response relationships i adverse health effects vary, e.g.:
  - **With PM exposure duration:** global data reviewed by Pope an Dockery (2006)
  - **With spatial resolution in exposure assessment:** e.g. long-ter $PM_{2.5}$ exposure and mortality (Los Angeles data by Jerrett et a 2005)
  - **Between different regions in Europe:** hospital admissions (Atkinson et al. 2000) and mortality (Samoli et al. 2005); meta-analysis by Anderson et al. (2004)
  - **With season in the same European location:** myocardial infarction (Lanki et al. 2006a), stroke (Kettunen et al. 2007), respiratory mortality (Halonen et al. 2008)
  - **With contribution to PM mass by different sources** (eg. traffic vs. long-range transport): myocardial ischemia in heart patien (Lanki et al. 2006b) and lung function in asthmatics (Penttine et al. 2006)

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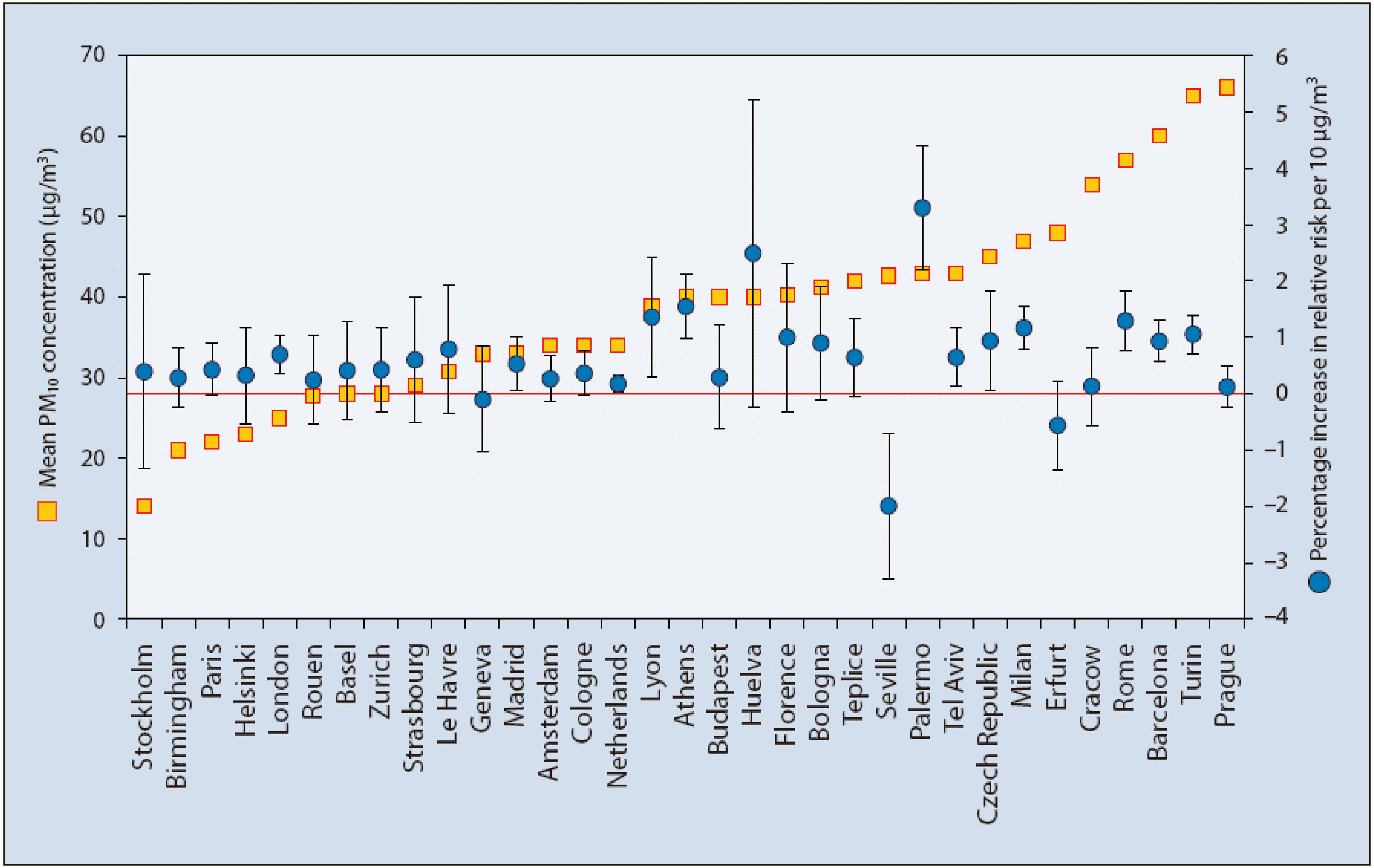
# Mortality estimates of urban air $PM_{10}/PM_{2.5}$ va with different exposure assessment

| PM parameter (each change 10 μg/m³) | PM monitoring site | Time scale in concentration change | Significant increase in all cause mortality |
|---|---|---|---|
| $PM_{10}$ | Central | 24 hours | 0.2 – 0.8 % |
| $PM_{10}$ | Central | 5-40 days | 1.3 – 1.8 % |
| $PM_{2.5}$ | Central | 24 hours | 0.6 – 1.2 % |
| $PM_{2.5}$ | Central | Years | 6 – 16 % |
| $PM_{2.5}$ | High resolution modelling | Years | 17 % |

**Mortality estimates increase with exposure time and higher resolution PM exposure assessment as reviewed by Pope and Dockery (2006).**

# Urban background $PM_{10}$ concentration and mortality estimates in European cities




*Source*: Anderson et al. 2004 *(2)*.

**Southern European cities show generally higher all cause mortality estimates per 1 µg/m³ PM10 and have higher annual concentrations at urban sites than Northern European cities (Anderson et al. 2004 in WHO Global AQGs 2005).**

# $PM_{2.5}$ concentration and stroke mortality in Helsinki (1998-2004)

| | Increase in mortality (%) | |
|---|---|---|
| Lag time | Warm season | Cold season |
| 0 days | 6.86* | - 0.19 |
| 1 days | 7.40* | - 0.17 |
| 2 days | 4.01 | 0.59 |
| 3 days | -1.72 | 0.46 |

**Mortality estimates for the interquartile range in $PM_{2.5}$ concentratio[n] consistently higher during the warm season (Kettunen et al., 2007).**

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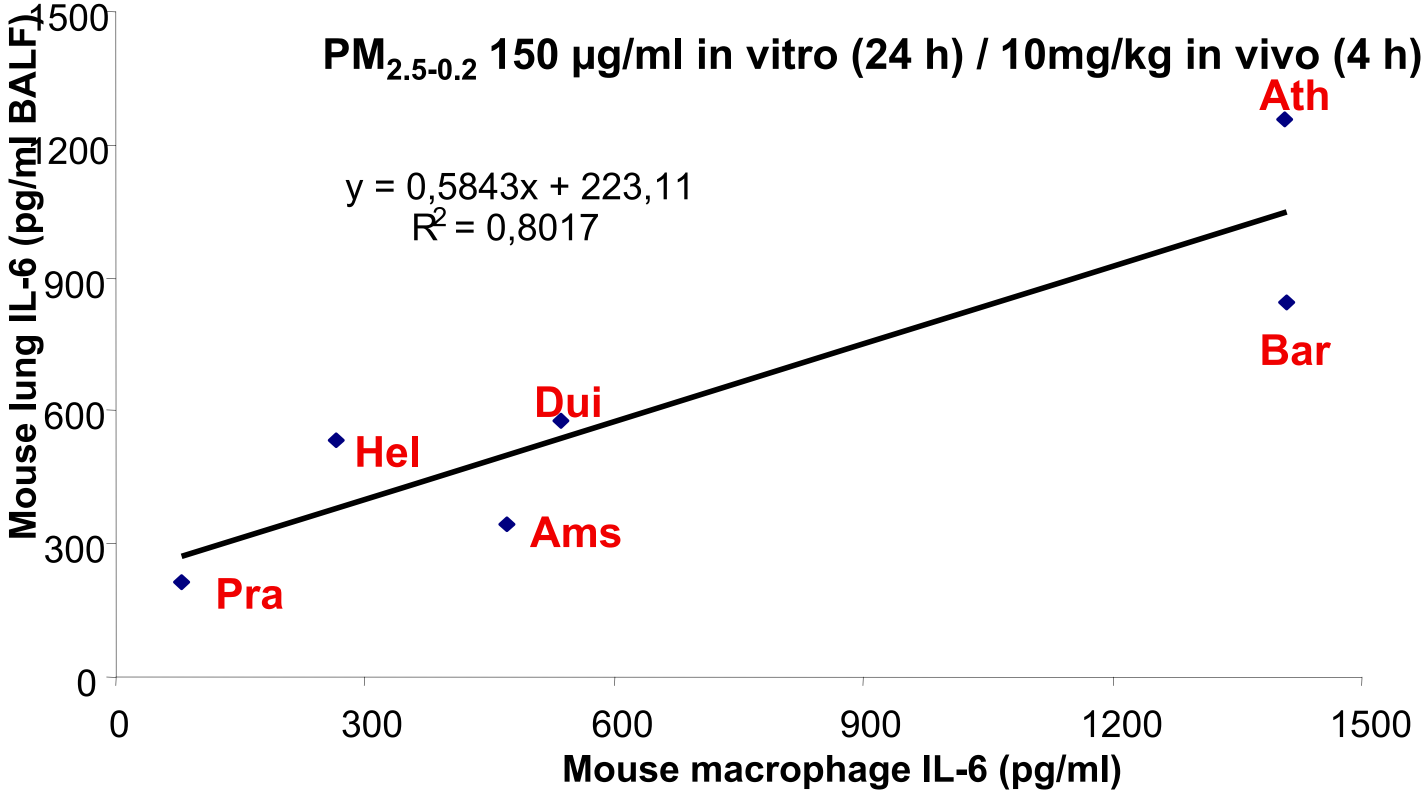
# Differences in the inflammatory activity of urban background fine particulate ($PM_{2.5-0.2}$) samples from six European field campaigns




**Samples from Southern European warm season field campaigns showed higher atmospherically transformed organics (Sillanpää et al. 2005), and inflammatory activity per mg $PM_{2.5-0.2}$ mass *in vitro* in a mouse macrophage cell line (Jalava et al. 2007) and *in vivo* in the mouse lung (Happo et al. 2007) than the correspondi samples from Central and Northern European campaigns of the PAMCHAR proj**

# Association of the source-specific $PM_{2.5}$ witl the risk of myocardial ischemia during exercise test in Helsinki

| Source category | OR | C.I. Lower | C.I. highe |
|---|---|---|---|
| Long range | 1,02 | 0,98 | 1,06 |
| **Local traffic** | **1,22** | **1,05** | **1,41** |
| Soil | 1,04 | 0,82 | 1,31 |
| **Oil combustion** | **1,19** | **0,98** | **1,44** |
| Salt | 0,76 | 0,56 | 1,04 |

**Local traffic-related and oil combustion-related PM compositions showed much higher risk estimates than the other sources per 1 µg/n of daily variation in the source-specific $PM_{2.5}$ (Lanki et al. 2006b).**

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# Major uncertainties and gaps in scientific knowledge from the EU policy point-of-view

- Reasons for the observed heterogeneities in PM exposure-response relationships in Europe are not convincingly know
  - Are they due to differences in PM sources, meteorology and/or population / exposure characteristics?
- Causal associations between PM sources, chemical composition and health effects are poorly understood
  - Are the newly-generated local combustion source PM (e.g. traff domestic heating with coal and wood, industries) more harmful than the aged PM from regional or long-range transport?
  - The largest gaps in knowledge by organic PM compositions
- Little known about cumulative health impacts from long-term PM exposures in Europe
  - Respiratory and cardiovascular disease prevalence and mortali developmental effects on children
  - Important to know causative PM sources and compositions

# Physico-chemical and biological PM properties health interest for future integrated studies

| PM property | Determinants |
|---|---|
| Size class / distribution | Number, length, surface area, volume / mass |
| Fraction by solubility | Non-soluble solid fraction vs. water-soluble and lipid-soluble fractions |
| Chemical composition | Transition metals (e.g. Cu, Ni, V, Fe)<br>Reactive organic compounds (e.g. PAHs, quinones) |
| Bioavailability | Active or passive penetration to key defence cells (e.g. macrophages, other inflammatory cells, epithelial cells |
| Biological reactivity | Oxidative potential; inflammatory, cytotoxic and genotoxic activities |

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# Important source environments for new studi
# Urban automotive traffic 1

- Complex emission source of ultrafine (UFP; diameter < 0.1 µ and fine ($PM_{2.5}$; diameter ≤ 2.5 µm) particles
  - Primary particulate (EC, OC, metals) and gaseous (CO, $NO_x$, VC emissions from vehicle engines
  - Gas-to-particle transformation of secondary organics (e.g. PAH and inorganics (e.g. $NO_3^-$) of combustion origin in the atmosph
- Complex emission source of coarse thoracic particles ($PM_{10-}$ diameter ≤ 2.5-10 µm) size range
  - Primary metal particle emissions from the brakes (e.g. Cu) and tires (e.g. Zn) and the body (e.g. Fe) of the car
  - Secondary resuspended road dust particles: *mineral particles* containing several elements (e.g. Si, Ca, Al, Fe, Mn) in varying ratios; *soil-derived organic debris from plants*, and *microbial contaminants* (bacteria, molds, toxins - e.g. endotoxin)

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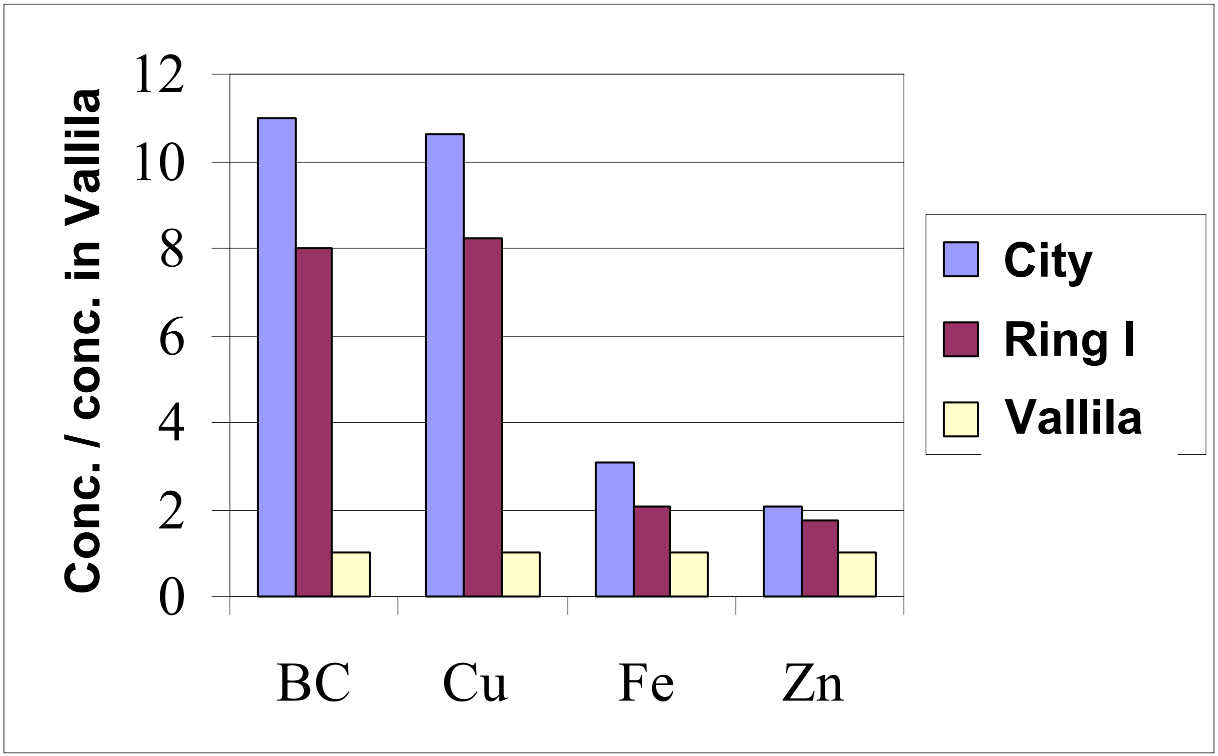
# Important source environments for new studi
## Urban automotive traffic 2

**Concentration in busy traffic lane vs. central monitoring site**




**Black carbon (BC) and copper (Cu) concentrations in $PM_{2.5}$ were 8-11 times and the iron (Fe) and zinc (Zn) concentrations twice as high as the average concentrations in the Vallila station (Yli-Tuomi et al. 2005).**

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# Important source environments for new studi
## Urban automotive traffic 3

- Personal exposure to traffic-derived pollutants underestimat
  - Several times higher exposures to primary particulate and gaseous pollutants of combustion and non-combustion origin the immediate vicinity to busy traffic (Riediker et al. 2004, Yli-Tuomi et al. 2005)
  - Co-exposure with noise may cause additional risks
- Vicinity to traffic of home, school and nursery is associated with increased risk of adverse health impacts
  - Premature deaths and atherosclerosis among the residents (e.g Hoek et al. 2002; Hoffmann et al. 2007), increased asthmatic symptoms in school-children (e.g. Janssen et al. 2003)
- Need for new integrated studies on air pollution abatement a reduction of personal exposures
  - Environmental zones, congestion fee (reduction of vehicles)
  - Improved air filtering in ordinary homes, schools, nurseries, an homes for the aged

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# Important source environments for new studie

# Decentralized biomass heating 1

- Bioenergy use is promoted in decentralised heating systems but they are currently less energy efficient and cause more a quality problems than large power plants
  - Domestic wood combustion is responsible for 25% of all $PM_{2.5}$ emissions, 65% of PAH emissions and 25% of all NMVOC emissions in Finland – situation similar to many other EU-countries
  - The main reasons for large $PM_{2.5}$ emissions from domestic heating appliances (commonly $\leq$ 20 KW) are poor combustion technology, poor biomass fuel quality and operational errors o the users (e.g., overloading, restriction of air supply)
  - Small heating plants (commonly $\leq$ 20 MW) using biomass or heavy fuel oil have no obligation to use the most efficient flue g after-treatment technology - no PM emission limits!

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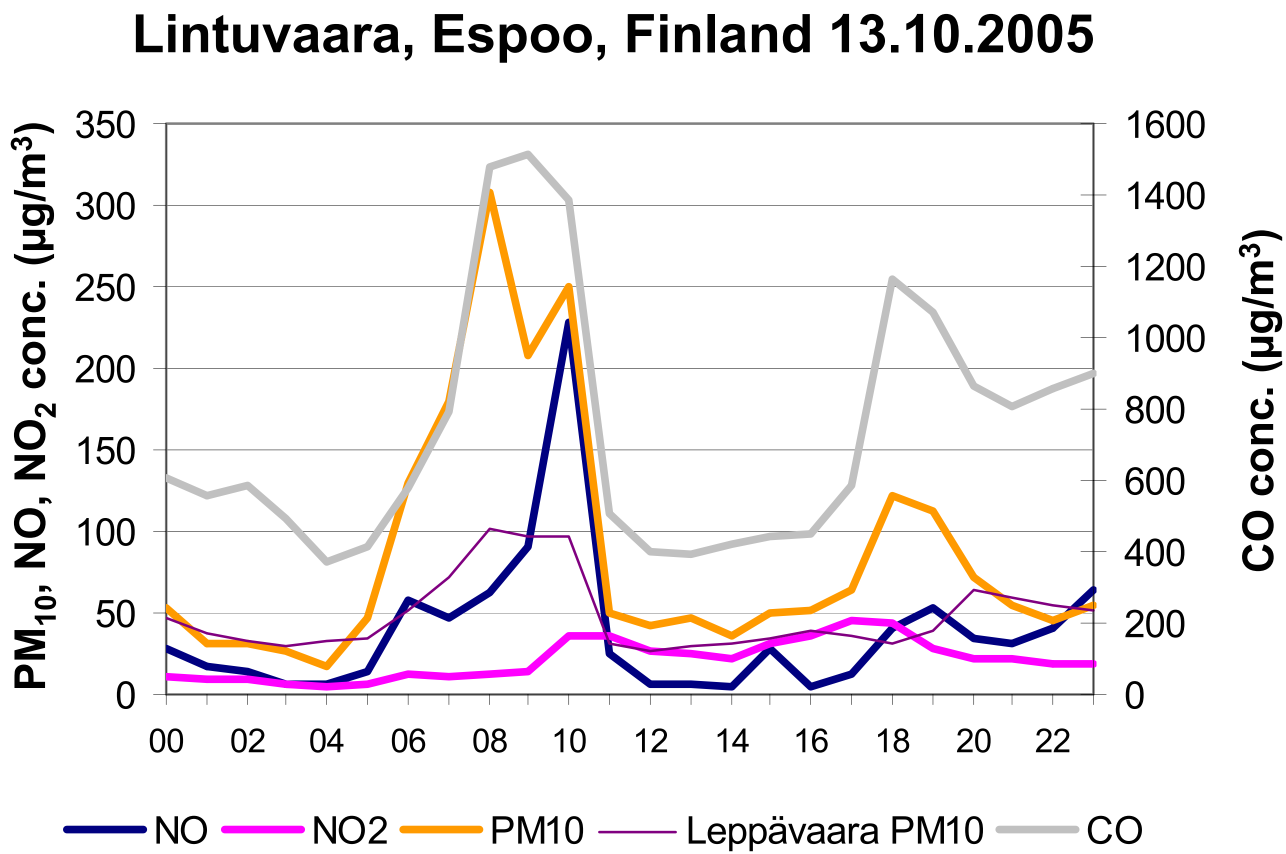
# Important source environments for new studie

# Decentralized biomass heating 2




**One day with weak winds: higher $PM_{10}$ concentrations than with busy tr in Leppävaara (Espoo) or Helsinki downtown (YTV, Helsinki, 2006).**

# Important source environments for new studie

## Decentralized biomass heating 3

- There is a high probability of elevated exposures in the neighbourhoods to $PM_{10}$, $PM_{2.5}$, PAH and NMVOC
  - The release of combustion emissions from domestic heating appliances and small power plants is close to the ground:

    ⇒ poor aerosol mixing in cold season days leads to elevated local outdoor pollutant concentrations ⇒ penetration indoor

    ⇒ the use domestic heating appliances most active in the evenings, when also the neighbouring residents are at home
  - Little research done until now on the air quality problems, and the human exposure and health effects related to poor biomass combustion in residential areas

    ⇒ increased asthma attacks have been reported as reviewed by Boman et al. (2003) and Naeher et al. (2007)
  - *Are these particulate compositions as harmful to the health of children and cardiac patients as those from traffic exhaust?*

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# Important source environments for new studi
## Wildfire smoke episodes 1

- Smoke-haze episodes from forest, bush and peat fires are common features in Southern and Northern Europe
  - Incomplete biomass combustion gives rise to large amounts of potentially harmful particulate ($PM_{2.5}$, PAH) and gaseous (CO, NMVOC) emissions
  - The combustion-derived pollutants can episodically cause profound increases in $PM_{10}$ and $PM_{2.5}$ concentrations, and in the carbonaceous composition of the particulate mass
  - The smoke-haze episodes appear annually in certain months an can last for weeks each time, and the episodes are likely to beco more prevalent with climate change in Europe

    ⇒ smoke aerosols affect populations not only locally but also at distances hundreds or thousands of kilometres away

    ⇒ emissions from incomplete biomass combustion increase the radiative forcing that enhances global warming (IPCC 20

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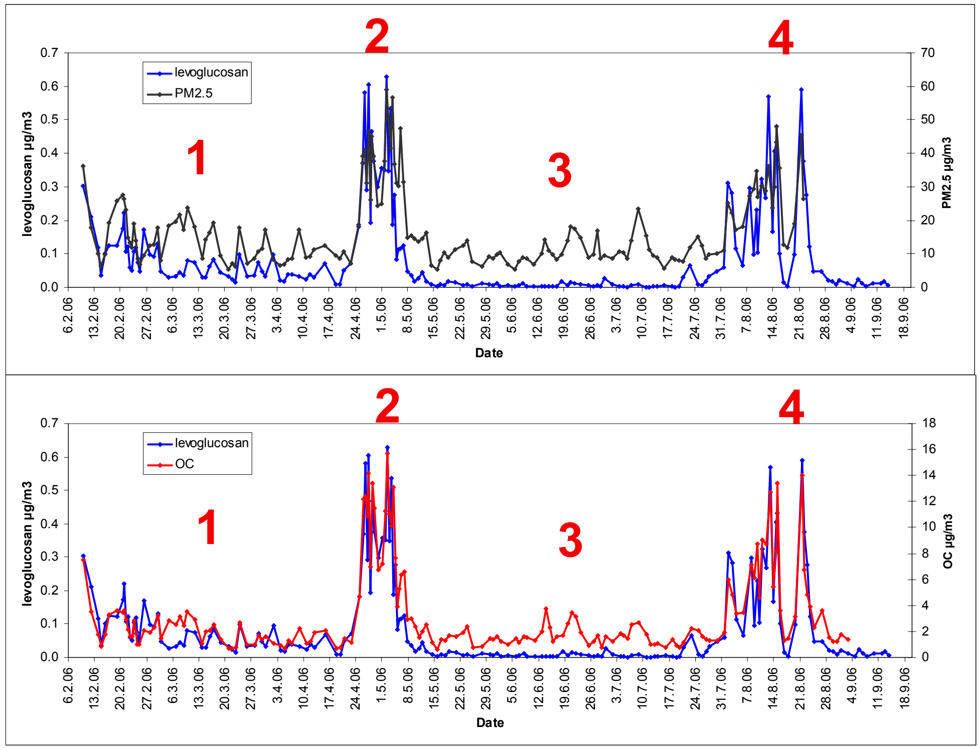
# Important source environments for new studi

# Wildfire smoke episodes 2

## Helsinki, Finland – annual data 2006

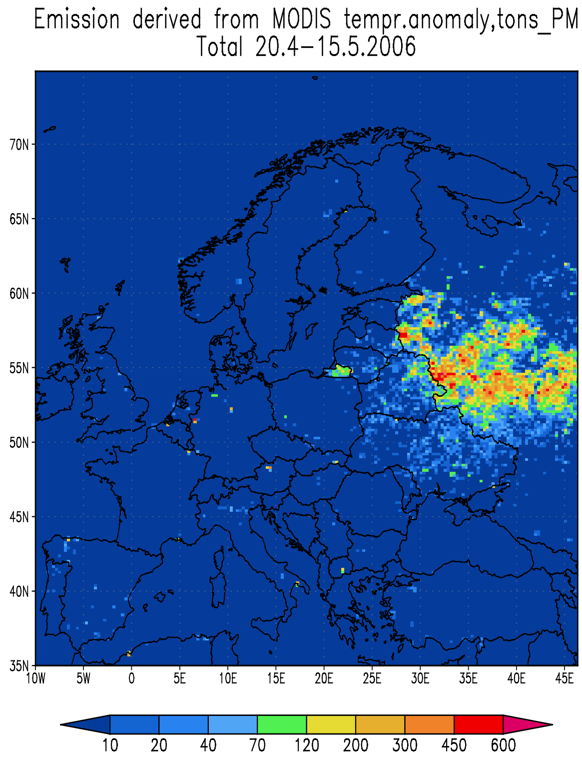



**The impacts of (1) local small-scale wood combustion and (2 and 4) episodes wild fire smoke on the PM$_{2.5}$ (upper panel) and organic carbon (lower panel) concentrations were identified with the help of specific marker – levoglucosa (blue lines); (3) the low biomass-smoke June-July (Saarnio et al., submitted).**

# Important source environments for new studie

# Wildfire smoke episodes 3

## Helsinki, Finland – 10 August 2006




**The location of wildfires and their emissions can be assessed from satellite images, and the transportation of smoke-haze can be modell (Saarnio et al., submitted).**

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# Important source environments for new studi
# Wildfire smoke episodes 4

- There is a high probability that the populations with elevated exposures to wildfire smoke $PM_{2.5}$ in Europe outnumber many in premature deaths and hospital emergency visits the local victims of fire burns
  - Little research done until now on the air quality problems, and th human exposure and health effects related to wildfire smoke

    ⇒ increased asthma attacks and activation of systemic inflamma have been shown as reviewed by Naeher et al. (2007)
  - Satellite images, dispersion modelling and specific aerosol trace could be used for prevention of the adverse health outcomes:

    ⇒ forecasting, early warning systems to the populations

    ⇒ European collaboration in more effective fire fighting
  - *Are these particulate compositions as harmful to the health of children and cardiac patients as those from traffic exhaust?*

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# Conclusions and recommendations

- Integrated toxico-epidemiological studies with participation of modern aerosol science are needed
  - Better characterization of PM exposures including source- and chemical composition-specific phenomena can be achieved
  - Research on new PM measures, e.g. oxidative potential, may open new possibilities for monitoring of health risks
  - Intervention studies can give direct inputs to the development of EU-policies (e.g. targeting of emission control and air quality monitoring) and to product development in SMEs and larger enterprizes
- Several PM source environments would urgently need new EU-funded studies, e.g.
  - Traffic-derived pollution at large (incl. noise)
  - Decentralized biomass vs. fuel oil combustion for heating
  - Smoke-haze from massive vegetation fires