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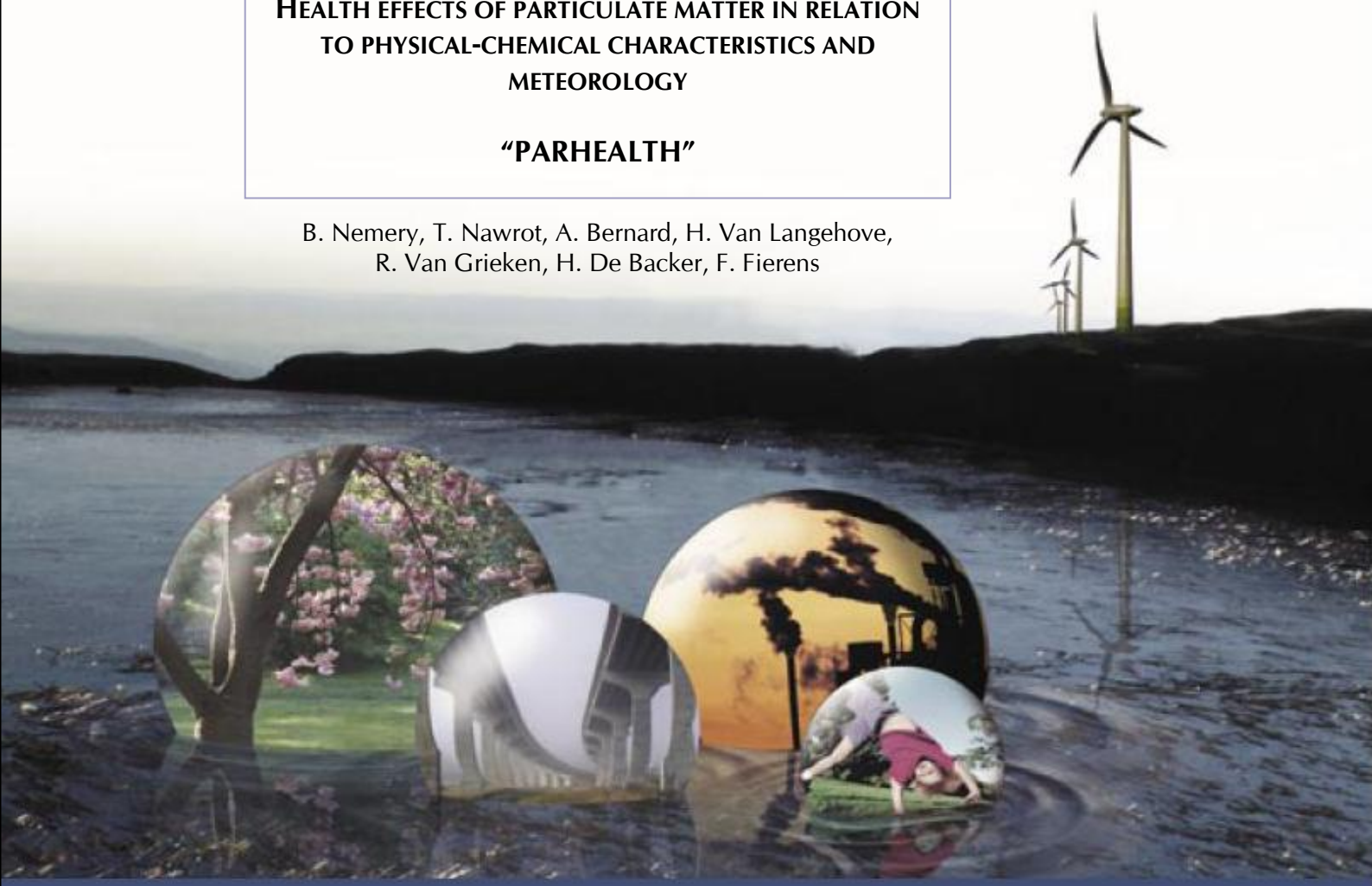
HEALTH EFFECTS OF PARTICULATE MATTER IN RELATION TO PHYSICAL-CHEMICAL CHARACTERISTICS AND METEOROLOGY

"PARHEALTH"

B. Nemery, T. Nawrot, A. Bernard, H. Van Langehove,
R. Van Grieken, H. De Backer, F. Fierens



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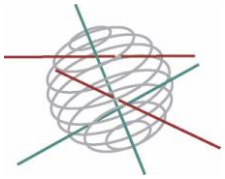

Health and Environment

FINAL REPORT

HEALTH EFFECTS OF PARTICULATE MATTER IN RELATION TO PHYSICAL-CHEMICAL CHARACTERISTICS AND METEOROLOGY "PARHEALTH"

SD/HE/01

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SUMMARY

A. Context

One of the important discoveries of these epidemiological studies was that the increased mortality and morbidity associated with increases in PM was not due only to pulmonary disease, but mainly to *cardiovascular* diseases (Dockery et al. 1993; Künzli et al. 2005; Miller et al. 2007; Peters et al. 2004). Exposure to particulate air pollution is a known trigger for cardiovascular disease (Peters et al. 2004).

One of the major opportunities of PARHEALTH is that we brought together toxicologists, chemists, epidemiologists and clinicians to study the systemic consequences of air pollution related disorders and its underlying mechanisms in susceptible populations (children and elderly).

B. Objectives

1. To investigate the short-term effects of particulates (both mass and physical-chemical characteristics) and ozone using sensitive endpoints of cardiovascular and respiratory responses in two susceptible segments of the population (children and elderly) in which the parameters will be measured in the same person within the same day (before and after ozone peak) and across seasons on days with relatively high and low concentrations of fine particulates in the ambient air this will allow to determine possible threshold levels of PM and ozone in an integrated approach.

2. To bring forward a new multi-compound dataset of highly health-relevant organic and inorganic pollutants. Therefore, an extended set of volatile organic compounds will be sampled in both indoor and outdoor air at the selected sites of study. Next, advanced analytical techniques have to be developed, optimized and validated to identify and quantify specific organic compounds on particulates.

3. To explore the component-specific toxicity (chemical and physical) of particulates in association with meteorological conditions.

The PARHEALTH researches succeeded addressed these objectives that were originally specified.

In addition a cluster between PARHEALTH and SHAPES was established to study subclinical responses in healthy cyclist briefly exposed to traffic related air pollution (PM2TEN)[1]. Moreover, to increase the policy relevance of the project we addressed a public health comparison of established triggers for myocardial infarction and air pollution.

C. Conclusion

- **Epidemiological studies in new-born's and children**

Numerous studies show association between fine particulate air pollutants and mortality in adults. We investigated short-term effects of elevated air pollution levels on infant mortality in Belgium and studied the European Union limit value protects infants form the air pollution trigger. We identified the 2 to 4 weeks of life as a susceptible period for air pollution. In this age class, the risk of death increased by 11% (95% CI: 1 to 22%) per 10 µg/m³ increase in particulate air pollution. Moreover, infants were 1.74 (95% CI: 1.18 to 2.58) times more likely to die on days with a mean a mean PM_{10} above the EU limit value of 50 µg/m³ than on days below this cut-off. Even in an affluent region in Western Europe, where infant mortality is low, days with higher particulate air pollution are associated with an increased risk of infant mortality.

In a cohort of 800 children living in the Southern part of Belgium (lower air pollution concentrations), Clara protein levels were measured and associated with particulate matter to study effects of PM_{10} on lung permeability at low concentrations. There was no evidence of an association between long-term exposure to ambient air pollution, as assessed at each participant's home address and the serum Clara Cell Protein concentration. However, short-term increases in particulate air pollution were associated with Clara cell protein concentration. The effects were strongest taking 7-day averages of air pollution. This could not be explained by other potential important covariates including age, gender, and exposure to environmental tobacco smoke. These findings suggest that acute environmental exposures influence the integrity of the lung epithelium and lead to increased epithelial barrier permeability in the lungs of children.

[1] http://www.belspo.be/belspo/ssd/science/Reports/PM²TEN_FinRep.ML.pdf

- **Epidemiological studies in Elderly**

Is air pollution a relevant trigger for myocardial infarction?

Acute myocardial infarction (MI) has been shown to be triggered by various factors, such as physical exertion, stressful events, heavy meals, or increases in air pollution. However, the relative importance and relevance of each trigger are a matter of debate. We compared triggers at an individual level and at the population level. We calculated population attributable fractions (PAF) for triggers of non-fatal MI based on a systematic literature review. When feasible, we performed a meta-regression analysis for studies of the same trigger. Of the epidemiologic studies reviewed, 36 provided sufficient details to be considered. In the studied populations, the exposure prevalence for triggers in the relevant control time window ranged from 0.04% for cocaine use to 100% for air pollution. The reported odds ratios (OR), ranged from 1.5 to 23.7. Ranking triggers from the highest to the lowest OR resulted in the following order: use of cocaine, heavy meal, smoking of marijuana, negative emotions, physical exertion, positive emotions, anger, sexual activity, traffic exposure, respiratory infections, coffee consumption, air pollution [based on a difference of 30 µg/m³ in particulate matter with a diameter <10 µm (PM_{10}).

Taking into account the OR and the prevalence of exposure, the highest PAF was estimated for traffic exposure [7.4% (95% CI: 4.8 to 10.5%)], followed by physical exertion (7.2%), alcohol (6.3%), coffee (5.0%), a difference of 30 µg/m³ in PM_{10} (4.8%), negative emotions (3.9%), anger (3.1%), heavy meal (2.7%), positive emotions (2.4%), sexual activity (2.2%), cocaine use (0.9%), marijuana smoking (0.8%) and respiratory infections (0.6%). Considering both the magnitude of the risk and the prevalence in the population, air pollution is not a trivial trigger of MI, it is of similar magnitude (PAF: 5-7%) as other well accepted triggers such as physical exertion, alcohol and coffee.

Acute changes in mortality in association with modelled PM constituents

Studies show a strong association between particulate matter with a diameter of < 10 µm (PM10) and daily mortality. Nevertheless, there is also an even stronger dependency between temperature and the daily mortality rate, which is not linear.

Unlike gas phase pollutants that are distinct single chemical compounds such as ozone, PM consists of a mixture of many species, including inorganic acids and salts, low volatility organic compounds, metallic agents and water all in highly variable concentrations.

A database of seven years on a daily basis is available on mortality (n = 354 357) for Belgium, from January 1997 till December 2003. Here, here we present results on the daily mortality rate in function of modelled particulate matter concentrations by use of the chemical transport model CHIMERE. The effect of temperature is taken into account in the analysis.
During winter, sea salt shows a negative correlation with the daily mortality rate. This can be explained by the fact that sea salt is a trigger for south-western regime during winter (less polluted air). During summer, a strong association between mortality and SOA (Secondary Organic Aerosols) was found. Modeled PM constituents might be of value to predict acute risk of air pollution induced mortality at the population level.

Acute changes in blood pressure in association with air pollution constituents
Recent exposure to fine particulate matter air pollution increases the risk for myocardial infarctions and stroke. High blood pressure is known to be an established risk factor for cardiovascular disease. Possibly, increased blood pressure can be an intermediate factor involved in the PM-related cardiovascular morbidity and mortality. Evidence linking exposure to fine particles with increases in blood pressure is still inconclusive. There are studies showing positive associations between recent exposure to PM and blood pressure, but others find negative or no associations. The contrasting findings in these studies may be explained by differences in susceptibility of the participants or inaccurate blood pressure measurements. Also, none of these studies have assessed the effect of the chemical composition of PM on blood pressure. In our study we selected elderly persons because they can be more susceptible to the effects of particulate matter air pollution on cardiovascular and respiratory morbidity and mortality (Goldberg et al. 2000; Gouveia and Fletcher 2000; Morgan et al. 1998; Prescott et al. 1998). These people also spent more time inside their homes, so indoor air quality was also measured in our study.

Elderly were recruited from 5 different nursing homes, located in province Antwerp, Belgium. On the study day, persons completed a questionnaire to obtain information on gender, age, medical history, use of medication, alcohol us. Each person participated in two clinical visits. The clinical visits were scheduled on the same weekday (Wednesday to Friday) and time. Blood pressure was measured on two separate visits according to guidelines of the European Society of Hypertension.

We found that each increase of 10µg/m³ in 24 hour mean outdoor $PM_{2.5}$ was associated with increases in systolic blood pressure and pulse pressure, with 0.89 % (95%CI: 0.15 to 1.6) and 1.8 % (95%CI:0.10 to 3.5) respectively. Iron (Fe), lead (Pb) and zinc (Zn) content of the outdoor $PM_{2.5}$ were most strongly correlated with increases in systolic blood pressure and pulse pressure. With also found significant associations between oxy-PAHs of collected outdoor PM_{10} and systolic blood pressure and pulse pressure.

These results might form a mechanistic pathway linking air pollution as a trigger of cardiovascular events.

Chemical characterisation and development of analytical techniques

In order to obtain a broad and multi-pollutant knowledge on the air quality on the selected sites of study, chemical characterization is done with respect to both (1) organics sorbed on particulate matter (PM_{10}) and (2) gaseous volatile organic compounds (VOCs).

Oxy-PAHs are considered as key compounds in PM-toxicity. Based on a thorough scientific literature search investigating the state-of-the-art knowledge on the occurrence of oxy-PAHs on PM, a set of relevant 3-5 ring oxy-PAHs is selected for further investigation. Given the high complexity of the matrix (PM_{10}) and the trace concentrations of sorbed micropollutants, oxy-PAHs analysis and quantification is a challenging multi-step process (sampling, sample preparation, chromatographic separation and selective/sensitive detection) that needs intensive method development, optimization and validation by use of advanced analytical instruments.

Through a systematic development approach, a fully characterized and validated innovative analytical method is brought forward, making use of accelerated solvent extraction, liquid chromatography and high resolution mass spectrometry (HRMS).

This allowed to provide – as far as we know for the first time – concentration levels of 11 oxy-PAHs in Belgium. Overall, the oxy-PAHs concentration levels are found to be in the lower pg/m³ to ng/m³ level, with phenanthrene-9,10-dione and 7H-benz[a]anthracene-7-one being major compounds. The highest concentrations are measured in February 2008, probably related to the occurrence of stable meteorological conditions.

Next to measuring PM-associated oxy-PAHs, sixty-four volatile organic compounds, representative for indoor air pollution were monitored in both the indoor and outdoor air of the elderly homes, and during children summer camps. The analytical method was based on passive sampling and TD-GC-MS analysis. Real uptake rates were determined to maximize accuracy. Overall, the indoor TVOC concentration ranged from 12 to 320 µg/m³. Comparison with the indoor air guideline values for TVOC concentration (≤200 µg/m³) and benzene (≤ 2µg/m³) reveals that guidelines are exceeded during two sampling campaigns.

D. Contribution of the project in a context of scientific support to a sustainable development policy

PARHEALTH is a multidisciplinary project that incorporates medical, chemical and epidemiological disciplines to unravel the health consequences of air pollution in children and elderly. PARHEALTH integrates both, societal relevance, aiming protecting potential to the most susceptible group (children and children) as well as development of methods that might be relevant to monitor for specific air pollution constituents (oxy-PAH).

- In our epidemiological studies we focused to a large extend to the shape of the association to deliver policy relevant information on possible threshold levels or to provide insight to the impact of particulate air pollution on the current used air pollution standards.

- We deliver information on the shape of the association on the trigger effects of air pollution on infant mortality, showing a linear effect. Assuming causality, of our findings, the current EU limit value for PM_{10} which might be exceeded on 35 day per year, does not prevent PM_{10} from triggering mortality in late neonates.

- We provide information on the use of weather models to model air pollution constituents and its relevance as trigger of mortality at the population level

- Considering both the magnitude of the risk and the prevalence in the population, air pollution is not a trivial trigger of myocardial infarction (MI), it is of similar magnitude (explains 5 to 7% of MI in the population) as other well accepted triggers such as physical exertion, alcohol and coffee.

- At concentrations below the EU daily limit value, serum Clara protein in children, a marker of lung permeability, is associated with 7-day average particulate matter concentrations in the ambient air.

- Regarding pollutant specific compounds, we identified that iron, lead and zinc were most strongly correlated with increases in pulse pressure of elderly. For policy conclusion regarding this aspect the current findings must be interpret within the context of future studies.

E. Keywords

Fine particulate air pollution, ageing, myocardial infarction, poly-aromatic hydrocarbons, metals, children, elderly, biomarkers.

1 INTRODUCTION

Solid and liquid phase material suspended in the atmosphere is referred to as 'particulate matter' (PM) or 'aerosols'. PM consists of a mixture of many species, including inorganic acids and salts, low volatility organic compounds, metallic agents and water all in highly variable concentrations. Our region has the highest annual mean mass PM concentrations of Europe (Aman et al 2004). In this regard, within Europe, Belgium has the highest estimated loss in life expectancy that can be attributed to particulates, being estimated to 13.2 months compared with 9.0 months as the European average (Aman et al 2004).

The major pollution disasters of the Meuse valley in Belgium (1930) (Nemery et al. 2001) and London (1952) (Bell and Davis 2001) were characterized by very high levels of sulphur-containing gaseous and particulate pollutants, which originated mainly from coal burning, and occurred during winter episodes of temperature inversions. Such high peaks of air pollution no longer occur in the industrially developed world. However, from the 1980s onwards more subtle effects of pollution on mortality and morbidity were registered in numerous time-series studies. They showed significant correlations between short-term increases in urban air pollution and increases in daily mortality and morbidity (Brunekreef and Holgate 2002).

PARHEALTH is a multidisciplinary project that incorporates medical, chemical and epidemiological disciplines to unravel the health consequences of air pollution in children and elderly. PARHEALTH integrates both, societal relevance, aiming protecting potential to the most susceptible group (children and children) as well as development of methods that might be relevant to monitor for specific air pollution constituents (oxy-PAH).

2 METHODOLOGY AND RESULTS

2.1 Study 1: Acute mortality and modelled chemical composition of PM [RMI and K.U.Leuven]

2.1.1 Methodology

Forecasting of the air pollutants concentrations was carried out with the CHIMERE model of KMI/RMI and IRCEL.

2.1.1.1 Statistical analysis

Database management and statistical analyses were done with SAS software (V.9.1). First, we tested for an interaction between modelled PM_{10} (and measured PM_{10}) and outdoor temperature (mean daily temperature above and below 16°C). Because of a significant interaction, we studied the dose–response relationship between the mass concentrations of PM_{10} and its modelled composition by the two temperature strata. The reason of testing these two specific strata was based by evidence from the literature of a U-shape mortality curve with outdoor temperature with the lowest mortality observed at a mean daily temperature of 16°C (Huynen et al. 2001). We identified covariates by a stepwise regression procedure with the p-values for variables to enter and to stay in the model set at 0.10. Covariates considered for entry in the model were ammonia, carbon, nitric acid, sodium, secondary organic aerosols and sulphuric acid while PM_{10} mass concentration modelled or measured PM_{10} were forced into the regression model. We additionally tested whether the addition of a squared term of temperature within the temperature strata significantly added to the prediction of mortality.

2.1.2 Results

We used a chemical transport model (CTM) to model the composition of particulate matter for our region of interest with a coarse resolution of 50 km2 in function of the available input data (meteofields, emissions).

The meteorological input fields are gathered from the ECMWF (European Centre for Medium-Range Forecast) operational forecasts. Since the horizontal- and vertical resolution of this operational weather forecast model changed significantly over the years, the input data used is interpolated at a horizontal resolution of about 50 km2.

The emission database we used is the European Monitoring and Evaluation Program (Verstreng 2003) database. We have been using the EMEP emissions for the year 2002.
The modelled composition was linked to the mortality data for Flanders for the period 1997-2004 by partner 1 (K.U.Leuven). Although, the modelling of daily variation in PM composition was done for whole Belgium, mortality data for a long period were only available for Flanders. We hope that mortality data of the Walloon area will become available in the course of the next two years of the project, so that we will be able to provide data for the whole country.

The total number of non-traumatic deaths during the 7-year period was 354 357. Daily mean outdoor temperature and modelled PM_{10} concentrations averaged 11.7°C (standard deviation (SD) 6.1) and 19.9µg/m3 (SD 11.7), respectively. There was an interaction between modelled PM_{10} on days above and below an average daily temperature of 16°C ($p<0.001$). The distribution of the modelled composition differed also between the ambient temperature strata (TABLE I).

TABLE I: Modelled composition of PM_{10} according to temperature strata

		Mean (SD), µg/m^3	P value
Sodium	< 16°	1.32 (1.73)	< 0.0001
	> 16°	0.52 (0.58)	
Secondary organic aerosols	< 16°	0.67 (0.98)	< 0.0001
	> 16°	2.28 (2.02)	
Nitric Acid	< 16°	6.02 (5.61)	< 0.0001
	> 16°	2.66 (3.02)	
Ammonia	< 16°	2.93 (2.08)	< 0.0001
	> 16°	2.12 (1.28)	
Carbon	< 16°	0.57 (0.41)	< 0.0001
	> 16°	0.66 (0.34)	
Sulphuric acid	< 16°	3.55 (2.28)	0.0625
	> 16°	3.73 (1.72)	
PM_{10}	< 16°	13.36 (9.59)	< 0.0001
	> 16°	11.78 (6.63)	

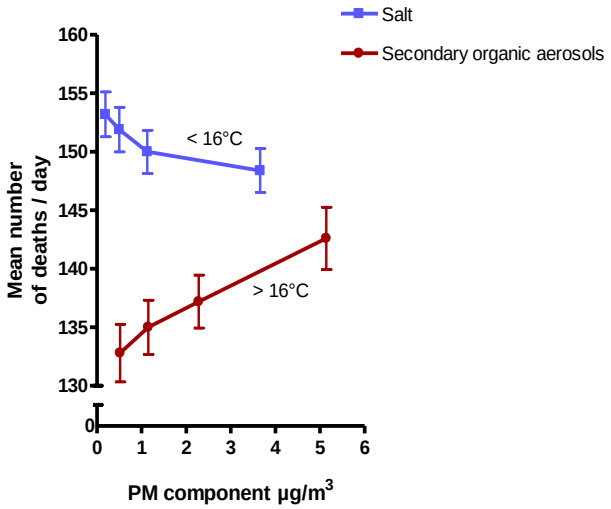

Figure 1: Total daily non -traumatic mortality means (95% CI) plotted against quartiles of PM constituents stratified according to outdoor temperature (< and > 16°C)

Both before (Figure 1) and after (TABLE II) cumulative adjustment for temperature and mass PM_{10}, secondary organic aerosols were positively associated with mortality on days above 16°C while the mass concentration of sodium was inversely associated with mortality on days below 16°C. The other studied components did not enter the regression models.

Independent of temperature and PM_{10} mass concentrations, a 1.6 µg/m³ (IQR-difference) higher sodium concentration on PM_{10} was associated with a 0.8 % decrease in mortality during days with a temperature below 16°C. The corresponding analysis, on days with an average temperature above 16°C showed that a 3.2 µg/m3 (IQR-difference) increase in secondary organic aerosols is associated with 3.4% increase in mortality, TABLE II.

TABLE II: Differences in daily all cause mortality (%) per IQR of concentrations or temperature

	Estimates*	95% CI	P value
≥16°C			
PM_{10}, +14 µg/m³	+ 3.4%	0.1 to 9.0	0.043
Sec. org. aerosols, + 3.2 µg/m³	+ 3.4	0.6 to 8.8	0.025
Temperature, +3.2°C	+ 1.4%	-0.2 to 4.2	0.075
<16°C			
PM_{10}, +14 µg/m³	+ 0.8%	-1.0 to 3.4	0.29
Sodium, + 1.6 µg/m³	- 0.8 %	-2.0 to -0.2	0.014
Temperature, +6.4°C†	- 4.9	-11.3 to -3.5	0.0002

*Estimates calculated for an IQR difference.

†Including both a linear and quadratic term.

Significant covariates were selected by stepwise regression analysis in which PM_{10} was forced into the model.

In this time-series analysis of modelled PM concentrations of several constituents, we showed that secondary organic aerosols were the most potent trigger for mortality during warm days (>16°C) while with an average temperature below 16°C there is a marginal decrease in mortality on days with higher fraction of sodium on PM_{10}. These effects could not be explained by differences in temperature or total mass. These novel results will merit future research on acute effects and composition of PM on mortality. The use of regression coefficients based only on PM_{10} mass constituents only may underestimate the effects of some its components.

This analysis has been extended with $PM_{2.5}$ modelled components and modelled maximum ozone concentrations.

Because of the known significant interaction between temperature and mortality, described above, the dataset is also for this analysis split up for two different temperature strata (> 16 °C and < 16°C). Therefore, temperature has been introduced into the regression models, together with the modelled species under consideration. For the lower temperatures, a quadratic temperature term was additionally added. The averaged modelled $PM_{2.5}$ concentrations were respectively 15.7 µg/m3 (temp ≥16°C) and 15.2 µg/m3 (temp < 16°C).

Also the influence of an event, until two days, has been investigated by introducing lagged variables.

No correction was made for total $PM_{2.5}$ mass for this analysis.

The next two figures show the results of this analysis as an excess risk [mean (95% CI)] of mortality per IQR of concentrations, stratified according to outdoor temperature. Figure 2 shows that all modelled species are significantly covariates for the stratified data with an average daily temperature higher than 16 °C. Also ozone is a very important trigger for mortality. All the other components also show a significant excess risk for the next day and day+2, while ozone is only a significant trigger for the day of the event itself. Figure 2 shows that he influences of the components of $PM_{2.5}$ are also detectable two days after an event.

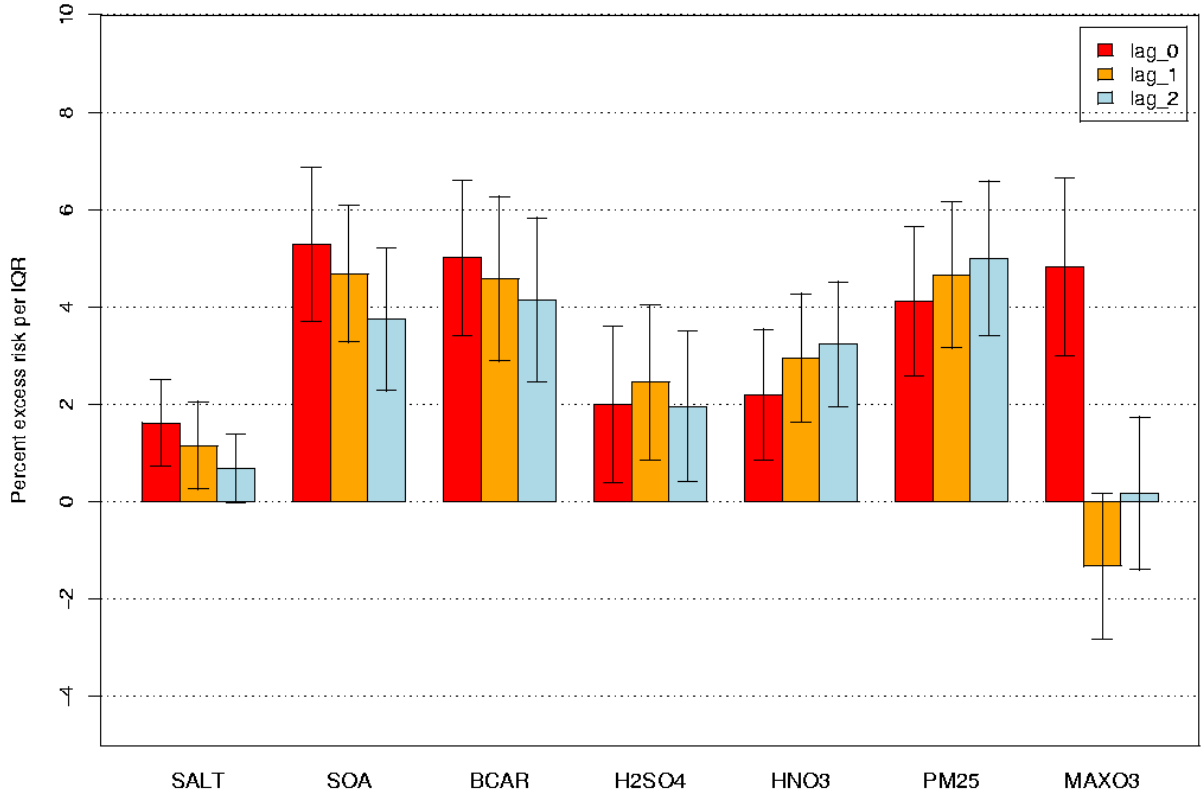

Figure 2: Excess risk [mean (95% CI)] of mortality per IQR of concentrations, stratified according to outdoor temperature (> 16°C). Lag_0 is the excess risk of modelled concentrations on the day of the event. Lag_1 and Lag_2 are the influences of an event on respectively day +1 and day+2.

Figure 3 summarizes the analysis for the cold period. This shows, besides a non-linear highly significant correlation with temperature, only for ammonia (NH3) and total $PM_{2.5}$ mass (PM25) a significant trigger for mortality.

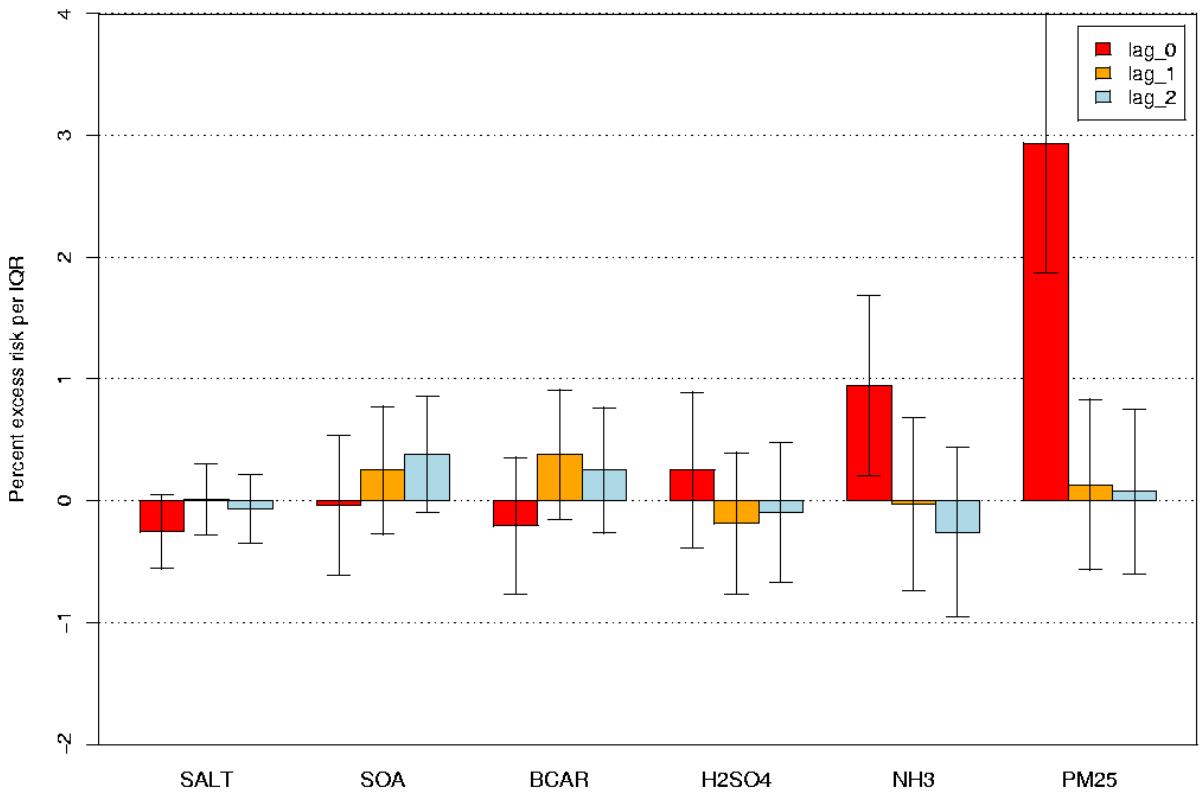

Figure 3: Excess risk [mean (95% CI)] of mortality per IQR of concentrations, stratified according to outdoor temperature (> 16°C). Lag_0 is the excess risk of modelled concentrations on the day of the event. Lag_1 and Lag_2 are the influences of an event on respectively day +1 and day+2.

We will extend the analysis by including mortality data from the Walloon region when they will become available.

2.2 Study 2: Public health importance of triggers of myocardial infarction [K.U.Leuven]

Acute myocardial infarction (MI) has been shown to be triggered by various factors, such as physical exertion, stressful events, heavy meals, or increases in air pollution. However, the relative importance and relevance of each trigger are a matter of debate. We compared triggers at an individual level and at the population level.

2.2.1 Methods

We calculated population attributable fractions (PAF) for triggers of non-fatal MI based on a systematic literature review. When feasible, we performed a meta-regression analysis for studies of the same trigger. We conducted a literature search to identify all studies on triggers for MI that would enable a computation of population attributable fractions (PAFs). Using PubMed at the National Library of Medicine and the Web of Science citation databases, we compiled all studies on trigger events defined as stimuli or activities occurring within a relevant period before the onset of acute MI.

We initially used "myocardial infarction", and "trigger", as key terms. We also searched for studies including both terms "myocardial infarction" and "case-crossover" because the latter study design is typical for evaluating triggers. We further performed additional searches that included the following terms in which we replaced trigger by "onset" or "preceding". We also considered references found in our literature search and review articles.

In the case-crossover design, the study population consists of subjects who experienced an episode of the health outcome of interest. The case-crossover design thus consists only of "cases", with each subject serving as his or her own control. As in a matched case-control study, the inference is based on a comparison of exposure distributions. The case-crossover study is most suitable for studying associations having the following characteristics: 1) the individual exposure varies within short time intervals; 2) the disease has an abrupt onset and short latency for detection; and 3) the induction period is short, as is the case for MI.

We included all studies published in English before January 2010 on triggers of non-fatal myocardial infarction. We excluded studies done exclusively at an ecological level but we included all population-based or hospital-based case-control and case-crossover studies with sufficient information about number of subjects and exposure. We selected only studies using PM_{10} or $PM_{2.5}$ as indicators of air pollution. We identified in total 36 studies (Barnett et al. 2006; Baylin et al. 2006; Baylin et al. 2007; Cendon et al. 2006; Clayton et al. 2008; Gerlich et al. 2009; Hallqvist et al. 2000; Koken et al. 2003; Lanki et al. 2006; Linn et al. 2000; Lipovetzky et al. 2004; Lipovetzky et al. 2007; Mann et al. 2002; Meier et al. 1998; Mittleman et al. 1993; Mittleman et al. 1995; Mittleman

et al. 1999; Mittleman et al. 2001; Moller et al. 1999a; Moller et al. 2001; Moller et al. 2005; Muller et al. 1996; Peters et al. 2001; Peters et al. 2004; Peters et al. 2005; Pope, III et al. 2006; Smeeth et al. 2004; Steptoe et al. 2006; Strike et al. 2006; Sullivan et al. 2005; von Klot et al. 2008; Willich et al. 1993; Ye et al. 2001; Zanobetti and Schwartz 2005; Zanobetti and Schwartz 2006; Zanobetti et al. 2009) that evaluated 13 triggers and met the selection criteria (Figure 4).

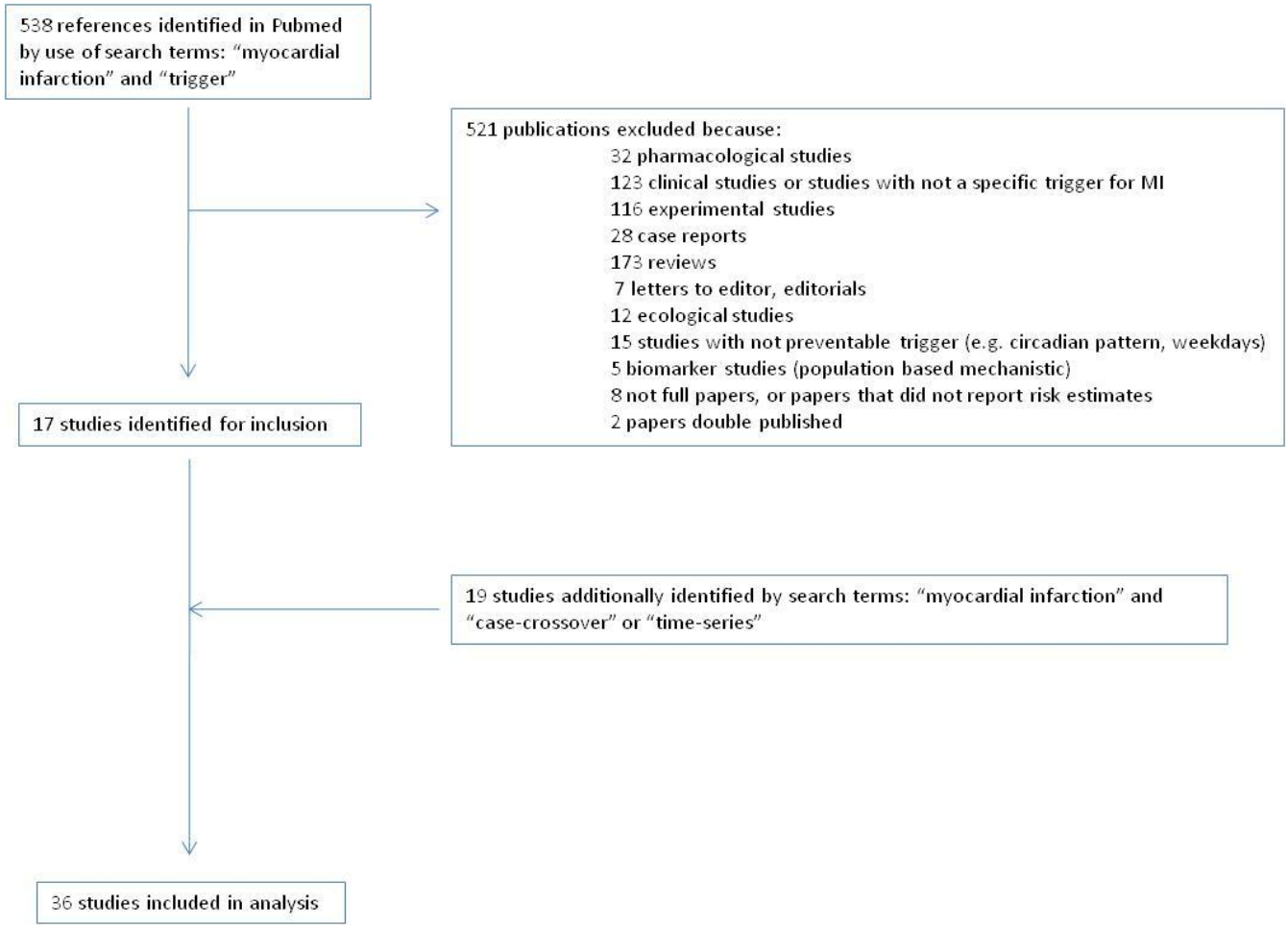

Figure 4: Flow chart

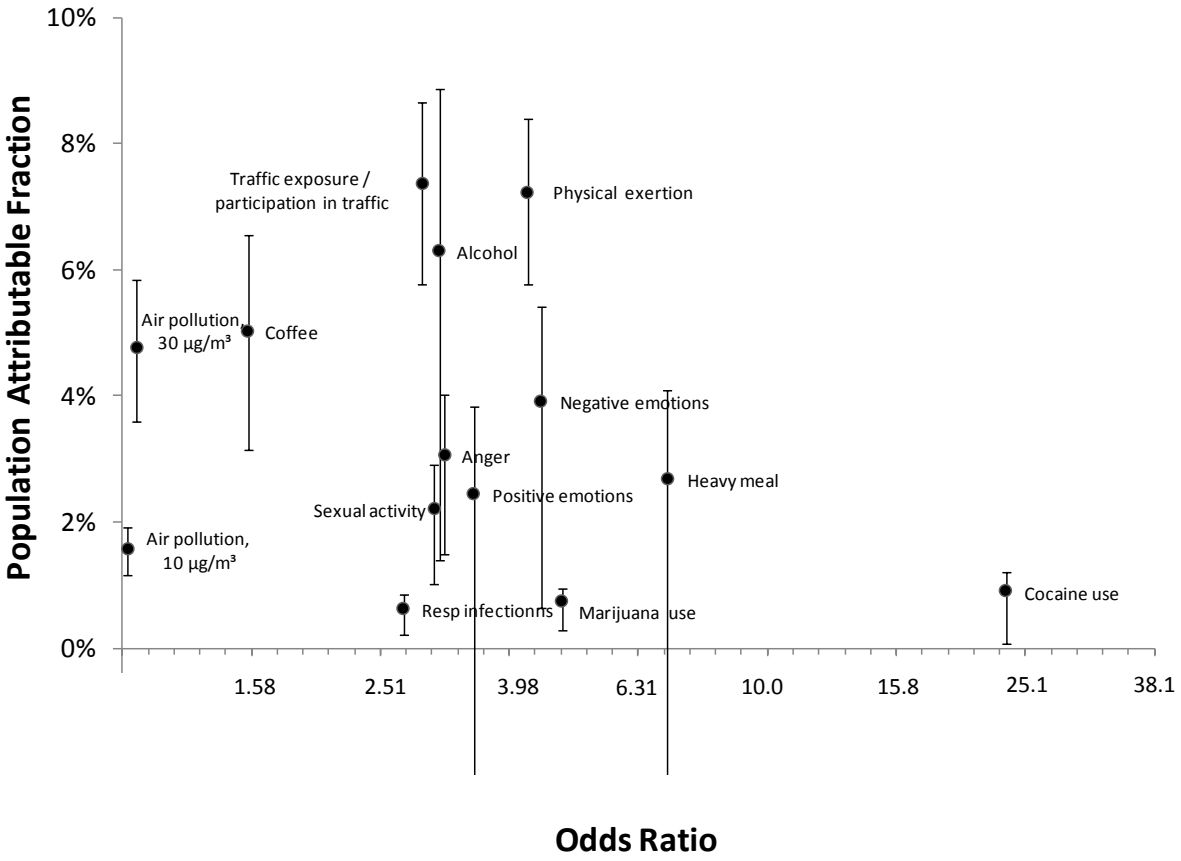

Figure 5: Relation between odds ratio and the Population Attributable Fraction for each studies trigger. Population attributable fractions was calculated and reported along with their 95% CI. Not significant triggers show 95% CI that are lower than 0%. X-axis is log scale, odds ratios are given as anti-logs.

For triggers studied in more than one study, a meta-analytical pooled effect estimate was derived from the point estimate of each separate study weighted by the inverse of the variance ($1/SE^2$). We used random effect estimates. The association between outdoor air pollution and health outcome is usually described by an exposure-response function that expresses the relative increase in adverse health for a given increment in air pollution. So, we calculated the pooled relative risk (with upper and lower 95% CI) for two scenarios of change in PM_{10}, namely increases by 30 and 10 µg/m³. When only $PM_{2.5}$ was available (Barnett et al. 2006; Zanobetti and Schwartz 2006; Zanobetti et al. 2009),(REF 1-35-36) we converted the ORs assuming that PM10 consists of 70% of PM2.5 (Ballester et al. 2008) (REF37).

However, other conversion factors were also considered as part of sensitivity analyses.

Sensitivity of the findings was examined by recalculating the pooled association sizes after excluding studies one by one. If the parameters of a "non-significant" air pollution effect were not reported, the authors of the paper were contacted in order to avoid bias resulting from the exclusion of non-significant studies, an important problem in any meta-analysis (Easterbrook et al. 1991; Nawrot et al. 2002) 38-39. If no additional information was made available, the non-significant odds ratios were assumed to be 1 and the non-significant P-values to be 0.50 (Nawrot et al. 2002; Needleman and Gatsonis 1990) 38-40.

In the case of air pollution, the prevalence of exposure among the population was estimated as 100% which is in line with the assumptions made by the epidemiological studies providing the effect estimates. For consistency between other triggers, and in the absence of detailed population surveys, the prevalence of exposure among the general population was estimated from the control group (in the case of case-control studies) or the control period (in the case of case-crossover studies) from the identified studies as reported in TABLE III. When several studies existed for a same trigger, the average prevalence of the risk factor was calculated by weighting by the sample size of each study.

As opposed to most other triggers for which the excess risk is expressed for a binary exposure (yes/no), the effects of air pollution need to be expressed on a continuous scale. We, therefore, present three scenarios to estimate the impact of PM_{10} on the incidence of MI in the population, namely the impact of lowering PM_{10} by 30, 10, and 1 $\mu g/m^3$.

TABLE III: Triggers for non-fatal myocardial infarction (continuance next page)

Trigger	Author	N	Mean age, y	Hazard period prior to MI episode	Exposure metric	Exposure frequency in controls or control period†	Odds ratio (95% CI)	Attributable risk of the exposed
Alcohol	Gerlich, '09	250	60	12 hours	Drinking any alcohol.	3.2%	3.1 (1.4-6.9)	67.7% (28.6-85.5)
Anger	Mittleman, '95	1623	61	2-hour	Anger scale above 5 (very angry, furious, or enraged)	1.0%	2.3 (1.7-3.2)	56.5% (41.2-68.8)
	Möller, '99 (Moller et al. 1999b)	660	60	2-hour	Anger scale above 5 (very angry, furious, or enraged)	0.26%	5.7 (3.0-10.6)	52.3% (66.7-90.6)
	Strike, '06	295	60	2-hour	Anger scale above 5 (very angry, furious, or enraged)	6.7%	2.06 (1.12-3.92)	51.5% (10.7-74.5)
	Lipovetzky, '07	209	52	1-hour	Anger scale above 4 (moderately anger, hassled in voice) at workplace	0.58[2]	9.0 (1.1-71.0)	88.9% (9.1%-98.5)
Cocaine use	Mittleman, '99	38	44	1-hour	person-time exposed to cocaine (average annual exposure x 1-hour)	0.04%	23.7 (8.1-66.3)	95.8% (87.7-98.5)
Coffee	Baylin, '06	503	57	1-hour	Drinking coffee	10.6%	1.5 (1.2-1.9)	33.3% (16.7-47.4)
Emotions (positive)	Lipovetsky, '07	209	52	1-hour	Standardized mood scale, PANAS questionnaire. Emotions at workplace	1.0%	3.5 (0.7-16.8)	71.4% (0-94.0)

Emotions (negative)	Lipovetsky, '07	209	52	1-hour	Standardized mood scale, PANAS questionnaire. Emotions at workplace	0.6%	14 (1.8-106.5)	92.8%(44.4-99.1)
	Steptoe, '06	295	60	2-hour	Depressed mood was assessed on a 5-point scale	6.1%	2.5 (1.05-6.5)	60.0% (4.8-84.6)
	Möller, '05	1381	59	24- hour	Work related stress: deadline	0.21%	6.0 (1.8-20.4)	83.3% (44.4-95.1)
Heavy meal	Lipovetzky, '04	209	52	1-hour	Did you eat a meal much larger than usual	0.45%	7.0 (0.8-66)	85.7% (0-98.5)
Marijuana	Mittleman, '01	124	44	1-hour	Smoking marijuana	0.20%	4.8 (2.9-9.5)	79.1% (65.5-89.5)
Physical exertion	Mittleman, '93	1228	62	1-hour	6 or more metabolic equivalents	0.74%	5.9 (4.6-7.7)	83.1% (78.3-87.0)
	Willich, '93	1194	61	1-hour	6 or more metabolic equivalents	3.9%	2.1 (1.1-3.6)	74.3% (9.1-72.2)
	Hallqvist, '00	660		1-hour	6 or more metabolic equivalents	1.9%	3.3 (2.4-4.5)	69.7% (58.3-77.8)
	Baylin, '07	530		1-hour	6 or more metabolic equivalents	2.30%	4.94 (3.73-6.54)	79.6% (73.0-84.6)
	Strike, '06	295	60	1-hour	6 or more metabolic equivalents	2.85%	3.5 (1.4-10.6)	71.4% (28.6-90.6)
	Von Klot, '08	1301	61*	2-hour	6 or more metabolic equivalents	~3%	5.7 (3.6-9.0)	82.4% ((72.2-89.0)

Respiratory infection	Meier, '98	1922	~60	1 to 10 days	acute bronchitis, pneumonia and productive cough	1.04%	2.7 (1.6-4.7)	62.9% (37.5-78.7)
	Smeeth, '04	20921	72*	1-3	Acute bronchitis, pneumonia, chest infections, influenza	0.31%	4.95 (4.43-5.53)	79.8% (76.7-81.8)
	Baylin, '07	499	57	1 to 6 days	n.r.	1.3%	1.48 (0.92-2.38)	32.4% (0-58.3)
	Clayton, '08	11155	71	1 to 7 days	acute bronchitis, pneumonia and productive cough	0.3%	2.55 (1.71-3.80)	60.7% (41.2-73.7)
Sexual activity	Muller, '96	1633	61	2-hour	Frequency of sexual activity	1.2%	2.5 (1.7-3.7)	60.0% (41.2-73.0)
	Baylin, '07	470	57	2-hour	Frequency of sexual activity	0.31%	5.47 (2.71-11.0)	81.7% (63.0-90.9)
	Möller, '01	699	n.r.	1-hour	Frequency of sexual activity	1.30%	2.1 (0.7-6.5)	52.4% (0-84.6)
Traffic exposure	Peters, '04	625	60	1-hour	time spent in cars, on public transportation, and on motorcycles and bicycles	4.12%	2.92 (2.22-3.83)	65.8% (54.5-73.7)

*median, n.r: not reported. †In the absence of detailed population surveys, the prevalence of exposure among the general population was estimated from the control group (in case of case-control studies) or the control period (in case of case-crossover studies.

TABLE IV: Prevalence of exposure, pooled Odds Ratio (OR), and Population Attributable Fraction (PAF) for the studied triggers of MI

Trigger	Prevalence of exposure among the population†	OR* (95%CI)	PAF (95%CI)
Air pollution, 10 µg/m³ reduction (n=11)*	100%	1.02 (1.01-1.02)	1.57% (0.89-2.15%)
Air pollution, 30 µg/m³ reduction (n=11)*	100%	1.05 (1.03-1.07)	4.76% (2.63-6.28%)
Alcohol	3.2%	3.1 (1.4-6.9)	5.03% (2.91-7.06%)
Anger, (n=4)*	1.5%	3.11 (1.8-5.4)	3.07% (1.19-6.16%)
Cocaine use	0.04%	23.7 (8.1-66.3)	0.90% (0.28-2.55%)
Coffee	10.6%	1.5 (1.2-1.9)	5.03% (2.08-8.71%)
Emotions. Positive	1%	3.5 (0.7-16.8)	2.44% (-0.30-13.64%)
Emotions negative, (n=3)*	1.18%	4.46 (1.85-10.8)	3.92% (0.99-10.4%)
Heavy meal	0.46%	7.00 (0.8-66)	2.69% (-0.09-23.00%)
Marijuana	0.2%	4.8 (2.9-9.5)	0.75% (0.38-1.67%)
Physical exertion, (n=6)*	2.45%	4.25 (3.17-5.68)	6.16% (4.20-8.64%)
Respiratory infection, (n=4)*	0.36%	2.73 (1.51-4.95)	0.57% (0.17-1.29%)
Sexual activity, (n=2)*	1.07%	3.11 (1.79-5.43)	2.21% (0.84-4.53%)
Traffic exposure	4.14%	2.92 (2.22-3.83)	7.36% (4.81-10.49%)

*odds ratio (OR) based on pooled OR and prevalence based on weighted means. Individual estimates given in Table III and Table V.

†Prevalence based on control time window, was estimated from the control group (in case of case-control studies) or the control period (in case of case-crossover studies. When several studies existed for a same trigger, the average prevalence of the risk factor was calculated by weighting by the sample size of each study. For triggers studied in more than one study, the prevalence was based on the weighted average

2.2.2 Findings

Of the epidemiologic studies reviewed, 36 provided sufficient details to be considered. In the studied populations, the exposure prevalence for triggers in the relevant control time window ranged from 0.04% for cocaine use to 100% for air pollution. The reported odds ratios (OR), ranged from 1.5 to 23.7 (TABLE III, IV). We identified 14 studies (Barnett et al. 2006; Cendon et al. 2006; Koken et al. 2003; Lanki et al. 2006; Linn et al. 2000; Mann et al. 2002; Peters et al. 2001; Peters et al. 2005; Pope, III et al. 2006; Sullivan et al. 2005; Ye et al. 2001; Zanobetti and Schwartz 2005; Zanobetti and Schwartz 2006; Zanobetti et al. 2009) relating particulate matter air pollution with non-fatal MI. Seven studies (Cendon et al. 2006; Koken et al. 2003;Lanki et al. 2006;Linn et al. 2000;Mann et al. 2002;Ye et al. 2001;Zanobetti et al. 2009) were time-series analyses and 7 studies (Barnett et al. 2006; Peters et al. 2001; Peters et al. 2005; Pope, III et al. 2006; Sullivan et al. 2005; Zanobetti and Schwartz 2005) were case-crossover studies (TABLE V). For the 14 studies, the combined risk estimate involved 593,480 subjects and was 1.016 (95% CI: 1.009-1.022; p≤0.0001) for an increase of 10 µg/m³ in PM_{10}. The corresponding pooled OR for an increase of 30 µg/m³ was 1.049 (1.027-1.067).

Estimates were not influenced by excluding two studies (Koken et al. 2003;Ye et al. 2001) for which the association size was reported as non-significant, but details of the statistical parameters were not available. Heterogeneity was further addressed by excluding each study one by one: excluding the study with the highest OR (Zanobetti et al. 2009) led to a drop in the pooled OR to 1.013 (95% CI: 1.007-1.019; p≤0.0001), while the pooled estimated increased to 1.019 (95% CI: 1.010-1.027; p≤0.0001) after excluding the study with the lowest OR (Zanobetti and Schwartz 2005). The combined estimate expressed for a 10 µg/m³ increase in particulate air pollution (PM_{10}) was 1.011 (95% CI: 1.003 to 1.018) for time-series studies and 1.027 (1.009-1.027) for case-crossover studies.

Ranking triggers from the highest to the lowest OR resulted in the following order: use of cocaine, heavy meal, smoking of marijuana, negative emotions, physical exertion, positive emotions, anger, sexual activity, traffic exposure, respiratory infections, coffee consumption, air pollution [based on a difference of 30 µg/m³ in particulate matter with a diameter <10 µm (PM_{10})].

Taking into account the OR and the prevalence of exposure, the highest PAF was estimated for traffic exposure [7.4% (95% CI: 4.8 to 10.5%)], followed by physical exertion (7.2%), alcohol (6.3%), coffee (5.0%), a difference of 30 µg/m³ in PM_{10} (4.8%), negative emotions (3.9%), anger (3.1%), heavy meal (2.7%), positive emotions (2.4%), sexual activity (2.2%), cocaine use (0.9%), marijuana smoking (0.8%) and respiratory infections (0.6%).

TABLE V: Characteristics of the studies on particulate air pollution and non-fatal myocardial infarction

Author	Design	N	Hazard period before MI episode	Odds ratio (95% CI) for 10 µg/m³ increase
Linn 2000	Time-series	~51465	24h	1.006 (1.001 – 1.012)
Peters 2001	Case-crossover	772	2h 24h	1.147 (1.020 – 1.291) 1.180 (1.035 – 1.355)
Ye 2001	Time-series	~7380	24h	n.s.
Mann 2002	Time-series	19 690	24h	1.00 (0.99 – 1.012)
Koken 2003	Time-series	~4073	24h	n.s.
Sullivan 2005	Case-crossover	5 793	24h:	1.014 (0.986 -1.049)
Zanobetti 2005	Case-crossover	302 453	24h	1.007 (1.003 to 1.01)
Peters 2005	Case-crossover	851	24h	1.017 (0.97 -1.065)
Pope 2006	Case-crossover	4818	24h	1.025 (1.007-1.050)
Zanobetti 2006	Case-crossover	15 578	24h	1.10 (1.014-1.196)
Cendon 2006	Time-series	724 717	24h (ICU) 24h (Infirmary)	1.032 (1.022-1.086) 1.050 (1.001-1.098)
Lanki 2006	Time-series	26 854	24h	1.003 (0.995-1.011)
Barnett 2006 *	Case-crossover	~30 660	24h (age ≥ 65)	1.049 (1.023 – 1.077)
Zanobetti 2009 *	Time-series	121 652	48h	1.016 (1.008 - 1.024)
Combined estimate		593 480		1.016 (1.009 - 1.022)

Bold indicates estimate included in meta-analysis. *Based on $PM_{2.5}$ and converted into PM_{10} assuming that 70% of PM_{10} consists of $PM_{2.5}$.
n.s. means not significant but no details were reported

2.2.3 Interpretation

Considering both the magnitude of the risk and the prevalence in the population, air pollution is not a trivial trigger of MI, it is of similar magnitude (PAF: 5-7%) as other well accepted triggers such as physical exertion, alcohol and coffee.

2.3 Study 3: Acute health effects of particulate air pollution in elderly [K.U.Leuven, U.C.Louvain, U.Gent, U.Antwerpen, IRM/KMI, IRCEL]

2.3.1 Methodology

2.3.1.1 Design and population

Partner 1 (K.U.Leuven) studied residents living in facilities of six retirement communities in the province of Antwerp. Partner 5 and 6 (RMI/KMI and IRCEL) provided forecasting on meteorological conditions and air pollution levels to plan our campaigns. Exclusion criteria included: being outside of the service flats for a longer period, psychiatric disorders, dementia, or medical conditions that would place the subject at risk from the blood donation. Persons were included if they were 65 years or older, nonsmoker and able to give informed consent. The study was conducted between June 2007 and October 2009. The ethical board of the K.U.Leuven approved the study protocol.

On the study day, persons completed a questionnaire to obtain information on gender, age, medical history, use of medication, alcohol use.

Each person participated in two clinical visits, on average 30 days apart. The clinical visits were scheduled on the same weekday (Wednesday to Friday) and time to control for circadian rhytm. 89 persons participated in the first examination, of which 75 also participated in the second examination. No PM data were available for 5 persons. So our final analysis included 84 persons.

2.3.1.2 Health measurements

2.3.1.2.1 Blood collection and analysis

We drew a venous peripheral blood sample. Blood was immediately analysed for platelet activity. Platelet function was measured with the platelet function analyzer (PFA-100). The PFA-100 test cartridge consists of a capillary, a blood sample reservoir, and a membrane coated with collagen/epinephrine with a central opening (Figure 6). Blood is aspirated through the capillary and flows through the opening in the collagen-coated membrane thus exposing platelets to high shear rates, causing platelet activation. A platelet thrombus forms at the opening, thus gradually diminishing and finally arresting blood flow. The time from the start of aspiration until the opening completely closes, i.e., the closure time, reflects platelet aggregation in a shear stress–dependent way (Kundu et al. 1995; Nemmar et al. 2003).

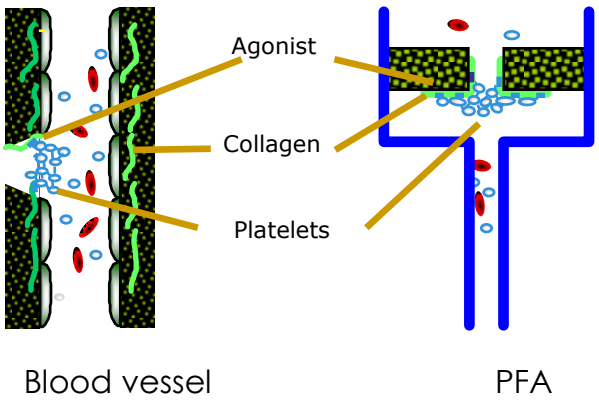

Figure 6: Principle of the PFA-100. Platelet activation is measured as the closure time (CT). This is the time from the start of blood flow until the platelets form a thrombus and blood flow stops ex-vivo.

Blood cell counts and differential leukocyte counts were determined using an automated cell counter with flow differential (Cell Dyn 3500, Abbott Diagnostics, Abott Park, IL, USA).
For other planned analysis, blood was rapidly separated (less than 20 minutes after blood draw) into serum or plasma by using an onsite field laboratory. After centrifugation, each fraction was aliquoted, coded, transported frozen on dry ice from the field to our unit, and stored at -80°C.

I. Plasma endothelin

Plasma endothelin concentrations were measured with a commercially available -immunoassay (Human endothelin Quantiglo, R&D Systems, UK).

II. Clara cell protein

Clara cell protein was measured on serum samples with an in house latex immunoassay, using the rabbit anti-protein 1 antibody from Dakopatts (Glostrup, Denmark) (Bernard and Lauwerys 1983) by U.C.Louvain.

2.3.1.2.2 Blood pressure measurement

Blood pressure was measured on two separate visits according to guidelines of the European Society of Hypertension (Parati et al. 2008) with an automated device (Stabilograph, Stolberg, Germany).
The measurements were done five times consecutively. The average of last three measurements was calculated and used in the analyses.

2.3.1.2.3 Exhaled NO measurement

Fractional exhaled NO was measured with an electrochemistry-based NIOX MINO device (Aerocrine, Sweden). This instrument complies with ATS/ERS recommendations (ATS/ERS recommendations for standardized procedures for the online and offline measurement of exhaled lower respiratory nitric oxide and nasal nitric oxide, 2005 2005). The device contains a scrubber which makes the inhaled air NO-free. The procedure consists of maximal inhalation. Subjects were instructed to monitor a flow rate as visualized on a display to maintain a flow rate of 50 ml/second.

2.3.1.3 Ambient air monitoring

PM_1, $PM_{2.5}$ and PM_{10} mass concentrations were measured with an optic device (Aerocet 531, Met One). These measurements all were taken on the site (indoors and outdoors) (K.U.Leuven). We further obtained $PM_{2.5}$ and PM_{10} concentrations measured at a fixed monitoring station (from VMM) in Borgerhout, nearby serviceflat Cavell. From IRCEL we obtained interpolated PM_{10} values.

Ambient ozone was continuously monitored using a UV photometric O3 analyser Model 427 (Signal Instrument Company Limited, England) (UCLouvain). The equipment was calibrated before the start of the field study by an official laboratory of the national air monitoring network (lab of Mr Alain Derouane of IBGE-BIM). UGent and UAntwerpen sampled gaseous inorganic and volatile organic compounds (VOC) as well as particulate matter (PM) in ambient air. Next, these research groups performed physical-chemical analysis of the collected air and PM samples, with focus on both organic (UGent) and inorganic (UAntwerpen) pollutants. The IRM/KMI provided data on weather conditions. In order to obtain a broad and multi-pollutant knowledge on the air quality on the selected sites of study, both organics sorbed on particulate matter (PM) and gaseous volatile organic compounds (VOCs) are considered (Partner 3, UGent).

2.3.1.3.1 Oxy-PAHs analysis on particulate matter: method development, sampling and analysis

I Selection of target oxy-PAHs

A thorough scientific literature search was performed to reveal the state-of-the-art knowledge concerning the occurrence of oxy-PAHs on PM and analytical methods to determine these target compounds. The literature search is validated as a review article published in Atmospheric Environment which reports on (i) the main sources and atmospheric pathways of oxy-PAHs, (ii) their available physical-chemical properties and (iii) their health effects. Next to that, a thorough discussion is given on the entire analytical sequence necessary to identify and quantify oxy- PAHs on atmospheric PM (Walgraeve et al. 2010). As far as we know, no concentrations of oxy-PAHs are available for Belgium.

For this project, a selection of target oxy-PAHs is made based on this literature search and the commercial availability of analytical standards. The final compounds selected are: benzo[*a*]pyrene-4,5-dione, benzo[*a*]pyrene-1,6-dione, benzo[*a*]pyrene-3,6-dione, benzo[*a*]pyrene-6,12-dione, chrysene-5,6-dione, phenanthrene-9,10-dione, 4-oxa-benzo[*def*]chrysene-5-one, pyrene-1-carboxaldehyde, napthacene-5,12-dione, benz[*a*]anthracene-7,12-dione, and 7H-benzo[*de*]anthracene-7-one. These are mainly 4- to 5-ring oxy-PAHs, with the exception of phenanthrene-9, 10-dione, being a 3-ring oxy-PAH compound.

II. Sampling of particulate matter

Between 2007 and 2009, particulate matter samples (n =69) were collected in the outdoor environment of six 6 elderly homes located in both urban and suburban sited nearby the city of Antwerp. In cooperation with the Flemish Environmental Agency (VMM) (Dr. E. Wauters, member of the following-up committee), PM_{10} was sampled on glass or quartz filters by use of high-volume samplers (sampling time: 24h, sampling flow: ± 500 L/min). The loaded filters were deep frozen for storage before analysis. PM_{10} was collected during the sampling campaigns at the elderly homes RVT Cavell (June 2007), RVT Czagani (February 2008, October 2009), RVT Compostella (September 2008), and RVT Cantershof (February 2009). TSP was collected during the sampling campaign in RVT Den Olm (June 2009, September 2009).

III. Analytical method development for identification and quantification of selected oxy-PAHs on PM

Given the high complexity of the matrix (PM_{10}) and the trace concentrations of sorbed micropollutants, oxy-PAHs analysis and quantification is a challenging multi-step process that needs method development, optimization and validation by use of advanced analytical instruments.

The first step is the development of a suitable sample preparation, including an extraction and concentration step. Two advanced extraction methodologies are investigated: ice-cooled ultrasonic extraction (USE) and accelerated solvent extraction (ASE). Extraction dependent parameters such as extraction temperature (0-40°C), ASE cycle time (3-5-cycles), and rinse volume (40-60%) were optimized during method development, and both techniques are evaluated based on the obtained recoveries, reproducibility and matrix effects. After filtration, volume reduction and reconstitution, the extracts were transferred into autosampler vials for further instrumental analysis.

Next, the individual compounds in the extract mixture have to be separated through gas or liquid chromatography. Due to the low vapour pressure of the 4- to 5-ring oxy-PAHs, separation with high performance liquid chromatography (HPLC) appeared to be the most appropriate and was performed on a Surveyor HPLC system (Thermo Finnigan) making use of a C18 column and a binary mobile phase gradient.

Phenanthrene-9,10-dione suffers from analytical artefacts when analysed with gas chromatography, as during the injection this compound decomposes to 9H-fluorene-9-one. Therefore, this 3-ring oxy-PAH is also included in our group of target compounds. For identification and quantification, highly sensitive and selective detection instruments are needed. In this project, we have developed an innovative method making use of the Thermo Finnigan MAT95XP-TRAP high resolution mass spectrometry (HRMS) equipment available at AMBER-Lab (UGent, http://www.amberlab.ugent.be). Ionization is done by APCI using nitrogen gas as the auxiliary and sheath gas, and the temperature of both the vaporizer and the heated capillary were optimized during method development.

The optimized method including both sample preparation, separation and detection steps, was fully validated by determining the recoveries, method detection limits, precision, linearity and matrix effects.

2.3.1.3.2 Sampling and analysis of VOCs

I. Selection of target compounds

A set of 64 LC–MS grade VOCs are selected for monitoring, based on the availability of standards and their presence/relevance as indoor air pollutants. These compounds involve (i) 9 (cyclo)-alkanes (ii) 17 aromatic hydrocarbons, (iii) 23 oxygen-containing hydrocarbons, (iv) 6 chlorinated hydrocarbons, (v) 3 terpenes, and (vi) 6 compounds that could not be attributed to the previous groups.

II. Air sampling for VOC analysis

During the entire project, up to 120 air samples have been collected for VOC analysis at two types of sampling sites:

1) (Sub)urban environment: in parallel with PM sampling in the elderly homes (2007-2009), airborne VOCs are collected in four inhabitant rooms, the recreational room and the garden of each elderly home. During the sampling period, a selected group of inhabitants was medically investigated.
2) Rural environment: during summer 2007 (July-August), sampling of the ambient air at 3 different rural locations (Waismes, Torgny, Ramegnies-Chin) was performed, while children participated in summer camp activities.

III. Analytical methodology

Because of the expected low concentrations of VOCs in ambient air, analyte enrichment is necessary. Therefore, target compounds were pre-concentrated on 200 mg of the polymeric sorbent Tenax TA hold in stainless steel sorbent tubes (Markes).

The monitoring in the elderly homes was performed using diffusive axial-sampling sorbent tubes, with an exposure time between 6 and 9 days. Samplers were equipped with an aluminum diffusion cap during exposure. At each sampling location two passive samplers were exposed next to a blank (sorbent tube that was closed at both sides with a brass cap). In order to calculate the time weighted average (TWA) concentrations by passive sampling, a compound dependent uptake rate is required. Using ideal uptake rates (calculated from the sampler geometry and diffusion coefficient) may result in an underestimation of the TWA concentrations. Therefore, there is a need for real uptake rates applicable for VOCs monitoring at indoor air conditions. Literature data, however, are scarce. Therefore, uptake rates of 25 VOCs were determined on site at the recreational room by performing simultaneous active and passive sampling at the same location.

For monitoring campaigns in Waismes, Torgney and Ramegnies-Chin, sorptive VOCs sampling and enrichment is done in an active way, using the same type of tubes. Sampling time was 30 min with 3-4 samples per sample location. Calibration of the sampling pump was performed before and after the sampling using a Gilian Gilibrator2 flow calibration system.

The instrumental analysis was performed by thermal desorption gas chromatography-mass spectrometry (TD-GC-MS). A 30m FactorFour VF-1ms low bleed bounded phase capillary GC column (Varian, 100% polydimethylsiloxane, internal diameter 0.25mm, film thickness 1µm) was used for separation. Masses from m/z 29 to 300 were recorded in full scan mode on a Trace DSQ Quadrupole MS (Thermo Finnigan), hyphenated to the GC, and operating at an electron impact energy of 70 eV. Criteria for detection and quantification are based on a signal to noise ratio (S/N) of 3 and 10 respectively. Blank correction was done where necessary.

2.3.1.3.3 Passive sampling and analysis of gaseous inorganic and BTEX compounds

I. Sampling methodology

Gaseous compounds (NO2, SO2 and O3) were collected by means of Radiello® passive samplers (Fondazione Salvatore Maugeri, Padova, Italy). This sampler comprises a compound-specific adsorbing cartridge, surrounded by a cylindrical microporous diffusive body; mounted on a supporting plate. Sampling was done both outside and inside of service flats, the exposure time of a single cartridge was 7 days; the mean concentrations of pollutants were calculated for these periods of time.

II. Analytical procedure

NO2, SO2

The analytical procedure involved the recovery of NO2-, SO32- and SO42- from the chemiadsorbed cartridges by means of water extraction (5 mL of milli-Q water) followed by two 1-minute mechanical stirring steps, between which the extracts were left to settle for one hour. NO2-, SO32- and SO42- were determined by means of ion chromatography (IC).

The analytical procedure involves ozonolysis of 4,4'-dipyridylethylene (during exposure), while sample preparation involves the formation of a yellow-coloured azide, which intensity is proportional to the amount of sampled ozone. Subsequently analysis is performed by means of UV-VIS spectrophotometry.

2.3.1.3.4 Active sampling and inorganic and single particle analysis of particulate matter

I. Sampling methodology

Bulk aerosol samples

Harvard-type impactors (MS&T Area Samplers, Air Diagnistics and Engineering, Inc. Harrison, ME, USA), equipped with very quiet oil-free pumping units (Air Diagnistics and Engineering, air sampling pump, model SP-280E) were used to collect $PM_{2.5}$ in: service flats Cavell, June 2007, children camps July-August 2007 (Waimes-Malmedy, Torgny-Virton and Ramegnies-Chin) and service flats Czagani, February 2008). PM_1 was sampled in service flats (Czagani, February 2008). Total suspended matter (TSP) was sampled in Cavell with the use of stacked filter units (Polycarbonate Open Filter Holder type SM 16509B, produced by Sartorius).

Samples were collected indoors and outdoors; additionally in Cavell, the samples were taken in two apartments as well. Aerosols were collected on Nuclepore polycarbonate filters (pore size 0.4 µm, Nuclepore Whatman International Ltd., England) or teflon filters (PALL Life Sciences, USA). Sampling time was approximately 24 hours, each filter was exchanged just before the medical test performed by UAntwerpen, and stored in the freezer upon analysis.

Elemental carbon (EC)

The soot (or elemental carbon) concentration in air was determined with a dual wavelength aethalometer (Magee Scientific Company, USA) in Czagani (February 2008) indoors and outdoors during the whole sampling period.

The aethalometer is an optical method that measures continuously the attenuation of a beam of light at a near-infrared wavelength (880 nm) that is transmitted through the sample when collected on a fibrous filter. Another wavelength of 370 nm was used simultaneously to determine so-called UV-absorbing Particulate Material (UVPM). This is a sum of EC and a mixture of organic compounds that absorb UV photons. The UVPM is expressed in units of "EC Equivalent". In case this value is higher than the EC concentration, it shows that there is an additional optical absorption that is of the same magnitude as if it was produced by the presence of black carbon in the amount that is equal to the difference between them.

II. Analysis procedure

Bulk aerosol samples

The mass concentration of dust was determined gravimetrically by weighing the filters before and after the sampling. Both times the filters were conditioned for 24 hours before weighing in controlled temperature and relative humidity, in order to minimise possible influences on the determined mass concentrations, due to adsorbed H2O on the aerosols. Each time weighing was performed in three repetitions. Afterwards, the elemental composition of aerosols was determined by Energy Dispersive X-ray Fluorescence (EDXRF; Epsilon 5, PANalytical, Almelo, The Netherlands).

Subsequently, the contribution of the water soluble fraction (concentrations of ions such as sulphates, nitrates, chlorides plus sodium, potassium, ammonium, magnesium and calcium cations) was measured by ion chromatography (IC Dionex, USA). For anion separation, the eluent was composed of 3.5 mM Na2CO3 / 1.0 mM NaHCO3 and the flow rate was set at 1.2 mL.min-1. For cation separation, the eluent was 20 mM H2SO4, with a flow rate of 1.0 mL.min-1. Calibration was performed using certified standard multi-ion solutions (Combined Seven Anion Standard II, Combined Six Cation Standard II, Dionex, USA). The gravimetric analysis and ED-XRF analysis of aerosol filters are so-called "non-destructive" analysis steps, for they don't require any special sample treatment; however determination of their water-soluble ion content demands leaching out in a minimal quantity of Milli-Q water through ultrasonic leaching before introducing the sample to ion chromatograph.

2.3.1.4 Statistical analysis

We performed pollutant-specific, exposure-response analysis using mixed models (Verbeke and Molenberghs 2000) as implemented in the SAS system. A random effect for individual participants accounted for similarities across the two clinical examinations for each person. In other words this method allows each subject to serve as his or her control over time and controls for potential confounding from between-subject covariates that do not change over time. Furthermore, associations can be studied in a relatively small sample size.

2.3.2 Results

In a cohort of elderly, we (K.U.Leuven) measured cardiovascular and respiratory parameters in the same person within the day and across seasons and evaluate their relationship with both physical properties and specific inorganic and organic components associated with particulates. This specific experimental design allows us to study the particulate induced effects, in association with ozone peaks, independently of the direct meteorological effects.

We measured the participants' sitting blood pressure, platelet function (PFA-100 closure time), and total number of platelets at baseline and at a follow-up clinical visit 1 to 2 week(s) apart. At the two clinical visits blood was taken and immediately deep-frozen for future analyses. We (K.U.Leuven) gathered information on smoking, drinking, social class, energy spent in physical activity and use of medication by the use of a standardized questionnaire.

Air pollution measurements included collecting TSP, $PM_{2.5}$ and PM_1 on polycarbonate filters with low volume samplers (UAntwerpen) and PM_{10} collection with high volume samplers (UGent) for analysis of oxy-PAHs (for details see previous chapter on implementation of the methodology).

2.3.2.1 Characteristics of the population

The study population consisted mainly of women. Mean age of the participants was 83 years (range 68 to 95 years). Medication variables were stable during the two to three week follow-up. TABLE VI summarizes the characteristics of the participants at baseline.

TABLE VI: Subject characteristics (at baseline), n=84

Anthropometric data	Mean or number	SD
Age, years	83.3	5.2
Women, %	59	70%
Height, m	1.61	0.08
Weight, kg	71.1	12.5
BMI, kg/m³	27.7	4.6
Lifestyle		
Past smoking	35	42%
Pack years	10.3	20.9
Passive smoking	39	46%
Regular alcohol use	47	56%
Diabetes	7	8%
Past cardiovascular disease	29	35%
Medication use		
Lipid-lowering medication	17	20%
Antiplatelet medication	35	42%
Antihypertensive medication	54	64%
Health measurements		

Exhaled NO, ppb	25.2	21.1
PFA closure time, sec	142.3	53.7
White blood cell count, /µL	6.6	1.8
Clara cell protein,	16.0	8.8
Systolic blood pressure, mmHg	140.6	21.2
Diastolic blood pressure, mmHg	75.1	12.0
Pulse pressure, mmHg	65.5	16.2
Endothelin, pg/mL	1.65	0.69

2.3.2.2 Exposure levels

2.3.2.2.1 Determination of the mass concentration of particulate matter

The data on mass concentrations of PM_{10}, $PM_{2.5}$ and PM_1 arranged per sampling campaign are summarized in TABLE VII. PM_{10} was sampled starting from the third sampling period and PM_1 staring from the second. This was due to the equipment limitations at the early phase of the project. $PM_{2.5}$ was sampled in every location both indoors and outdoors. The highest PM concentrations were registered during the winter campaign in Czagani, Broechem, in 2008. The $PM_{2.5}$ in ambient air reached the maximum value of 100.6 µg m^{-3} on 19th February. This corresponded to the indoor value of 53.6 µg m^{-3}. The lowest concentrations were observed in Bonheiden in 2009. On the average (taking into account all sampling campaigns) the ratio of $PM_{2.5}/PM_{10}$ was equal to 0.7 both indoors and outdoors, whilst the ratio of $PM_1/PM_{2.5}$ was equal to 0.6 outdoors and 0.7 indoors. The higher ratio of $PM_1/PM_{2.5}$ indoors may indicate more effective infiltration of the fine particles from the outside air; it may also be a result of higher settling efficiency of coarser particles, on windows and other indoor surfaces.

TABLE VII: Summary of the indoor and outdoor PM mass concentrations (µg/m³)

	INDOOR			OUTDOOR		
	PM_{10}	$PM_{2.5}$	PM_1	PM_{10}	$PM_{2.5}$	PM_1
Cavell 2007						
Minimum	n.d.	9.4	n.d.	n.d.	11.3	n.d.
Mean	n.d.	15.1	n.d.	n.d.	20.1	n.d.
Median	n.d.	15.0	n.d.	n.d.	15.9	n.d.
Standard deviation	n.d.	2.8	n.d.	n.d.	8.6	n.d.
Maximum	n.d.	18.9	n.d.	n.d.	32.1	n.d.
Czagani 2008						
Minimum	n.d.	10.6	5.8	n.d.	11.4	5.3
Mean	n.d.	24.8	15.7	n.d.	43.4	22.1
Median	n.d.	17.6	11.3	n.d.	32.8	18.4
Standard deviation	n.d.	14.6	8.7	n.d.	27.7	13.1
Maximum	n.d.	53.5	31.9	n.d.	100.6	46.1
Compostella 2008						
Minimum	15.5	11.5	7.8	16.2	9.3	7.2
Mean	18.5	13.4	9.6	25.7	18.5	12.2
Median	17.9	13.1	9.2	20.7	11.3	8.5
Standard deviation	2.3	1.9	1.3	10.0	10.9	5.8
Maximum	22.2	16.2	11.4	42.0	34.2	21.1
Cantershof 2009						
Minimum	n.d.	9.9	6.9	18.9	10.7	n.d.
Mean	n.d.	12.8	9.1	28.6	20.8	n.d.
Median	n.d.	12.0	8.1	26.4	19.2	n.d.
Standard deviation	n.d.	3.4	2.5	8.7	8.1	n.d.
Maximum	n.d.	19.8	14.4	43.0	32.8	n.d.
Den Olm 2009						
Minimum	9.0	6.7	3.8	15.9	8.4	4.4
Mean	12.5	9.9	6.6	20.6	12.8	7.5
Median	12.1	9.3	6.4	19.8	12.4	7.1
Standard deviation	3.8	2.8	1.8	3.8	3.0	2.0
Maximum	19.8	16.2	9.9	26.4	17.3	10.3
Czagani 2009						
Minimum	11.7	7.5	4.3	15.7	9.1	5.3
Mean	17.1	11.7	7.8	26.2	18.1	11.2
Median	14.7	10.4	6.6	24.0	15.6	10.7
Standard deviation	6.4	4.7	3.5	9.7	8.2	4.7
Maximum	32.2	22.2	15.0	49.0	34.8	22.5

n.d. – not determined

The indoor/outdoor ratios of particulate matter for every campaign are shown in TABLE VIII. Slightly higher indoor concentrations in summer (Cavell 2007, Compostella 2008 and Den Olm 2009) compared to winter (Czagani 2008, Cantershof 2009 and Czagani 2009) are observed; the possible reason could be the higher infiltration of particulate matter from outdoors during summer months (when windows and doors are usually opened for a longer time and more frequently than in the winter). Most of the ratios are below 1, only with the exception for Borsbeek, what implies no significant sources of PM in the indoor environments.

TABLE VIII: Indoor/outdoor ratios of different mass fractions averaged over the duration of specified campaign

	Cavell 2007	Czagani 2008	Compostella 2008	Cantershof 2009	Den Olm 2009	Czagani 2009
PM_{10}	-	-	0.79	-	0.63	0.67
$PM_{2.5}$	0.74	0.60	0.86	0.68	0.81	0.65
PM_1	-	0.76	1.01	0.52	0.88	0.70

2.3.2.2.2 Determination of the water soluble fraction of particulate matter

The ionic composition of particulate matter (NO_3^-, Cl^-, SO_4^{2-}, Na^+, NH_4^+, K^+, Mg^{2+}, Ca^{2+}) is summarized in TABLE IX. Average values per sampling location in different mass fractions were calculated. In case the specific ion was below the detection limit few times only per the whole campaign, random numbers below the detection limit were generated in order to calculate the averages. In case only one or two or none of values per sampling campaign, were above the detection limit, the "<LOD" indication was used.

TABLE IX: Average ionic concentrations in different fractions of PM per sampling location in µg m^{-3}

	NO_3^-	Cl^-	SO_4^{2-}	Na^+	NH_4^+	K^+	Mg^{2+}	Ca^{2+}
Indoors								
PM_{10}								
Cavell 2007	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
Czagani 2008	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
Compostella 2008	0.71	0.09	2.13	0.60	1.37	0.26	0.03	0.32
Cantershof 2009	0.77	0.24	1.73	0.85	0.88	0.15	0.04	0.21
Den Olm 2009	0.49	0.07	1.04	0.16	0.40	0.06	<LOD	<LOD
Czagani 2009	0.76	0.19	1.03	0.29	0.35	0.10	0.01	<LOD

$PM_{2.5}$								
Cavell 2007	0.19	<LOD	3.04	0.31	0.80	0.14	<LOD	<LOD
Czagani 2008	1.22	0.20	5.49	0.74	1.72	0.64	<LOD	<LOD
Compostella 2008	0.36	0.04	1.97	0.27	1.39	0.18	0.01	0.14
Cantershof 2009	0.55	0.11	1.72	0.63	0.92	0.13	0.02	<LOD
Den Olm 2009	0.27	0.03	0.97	<LOD	0.40	0.04	<LOD	<LOD
Czagani 2009	0.38	0.05	0.94	<LOD	0.32	0.06	<LOD	<LOD
PM_{1}								
Cavell 2007	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
Czagani 2008	0.56	0.04	3.53	0.29	1.05	0.45	0.002	<LOD
Compostella 2008	0.16	0.03	1.92	0.15	1.38	0.18	0.01	0.16
Cantershof 2009	0.17	0.02	1.53	0.24	0.87	0.12	0.01	0.11
Den Olm 2009	0.11	<LOD	0.93	<LOD	0.40	0.03	<LOD	<LOD
Czagani 2009	0.19	0.02	0.92	<LOD	0.32	0.08	<LOD	<LOD
Outdoors								
PM_{10}								
Cavell 2007	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
Czagani 2008	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
Compostella 2008	3.16	0.19	2.71	0.80	2.91	0.44	0.08	0.69
Cantershof 2009	5.42	0.89	3.54	2.42	3.53	0.34	0.20	0.69
Den Olm 2009	2.55	0.29	1.77	0.51	1.11	0.09	0.04	0.26
Czagani 2009	3.50	0.79	1.73	0.77	1.26	0.17	0.06	0.19
$PM_{2.5}$								
Cavell 2007	1.06	<LOD	5.16	0.30	1.64	0.29	0.02	0.18
Czagani 2008	11.21	0.83	8.24	0.73	5.00	1.59	0.01	<LOD
Compostella 2008	2.15	0.03	2.41	0.26	2.75	0.23	0.03	0.36
Cantershof 2009	2.93	0.03	2.50	0.51	2.87	0.22	0.03	0.21
Den Olm 2009	1.70	0.02	1.81	<LOD	1.27	0.10	<LOD	<LOD
Czagani 2009	2.24	0.07	1.68	<LOD	1.18	0.13	0.01	<LOD
PM_{1}								
Cavell 2007	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
Czagani 2008	5.82	0.37	3.11	0.27	2.18	0.83	<LOD	<LOD
Compostella 2008	1.30	0.01	1.70	<LOD	1.81	0.17	0.02	0.21
Cantershof 2009	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
Den Olm 2009	1.02	<LOD	1.28	<LOD	0.87	0.03	<LOD	<LOD
Czagani 2009	1.13	0.04	1.00	<LOD	0.70	0.10	0.004	<LOD

The highest concentrations of ions were registered during the winter 2008 (Czagani, Broechem). The concentrations of Mg^{2+} and Ca^{2+} were often below the limit of the detection, thus the partitioning below fractions (TABLE X) and percentage contribution calculations (Figure 7) were only done for nitrate, sulphate, chloride, ammonium, sodium and potassium ions.

TABLE X: Partitioning of ionic species in PM fractions

	NO_3^-	Cl^-	SO_4^{2-}	Na^+	NH_4^+	K^+
$PM_{2.5}/PM_{10}$ Indoors	**0.57**	**0.43**	**0.91**	**0.56**	**1.00**	**0.71**
$PM_{2.5}/PM_{10}$ Outdoors	**0.56**	**0.15**	**0.86**	**0.38**	**0.90**	**0.65**
$PM_1/PM_{2.5}$ Indoors	**0.43**	**0.33**	**0.87**	**0.39**	**0.91**	**0.97**
$PM_1/PM_{2.5}$ Outdoors	**0.50**	**0.51**	**0.63**	**0.43**	**0.64**	**0.65**

Sodium and chloride accumulate mainly in the coarse fraction (>$PM_{2.5}$) and are characterised by the lowest $PM_{2.5}/PM_{10}$ and $PM_1/PM_{2.5}$ ratios (0.38 and 0.15 for $PM_{2.5}/PM_{10}$ in the ambient air, respectively). On the other hand sulphate and ammonia show the highest ratios (0.86 and 0.90 for $PM_{2.5}/PM_{10}$ in ambient air respectively), what suggest that both of them are accumulated in fine fraction (<$PM_{2.5}$). As it can be seen from the table X, the ratios increase in indoor air with respect to outdoor. This again suggests that fine particulate matter can penetrate to the indoor environments more easily than the coarser particles.

Figure 7 shows the percentage contribution of ionic species to the total ionic concentration in $PM_{2.5}$ with regard to the annual season. As mentioned above magnesium and calcium were excluded from the calculations. Secondary origin ions (nitrate, sulphate and ammonium) constitute on average 89% of the total ion concentration.

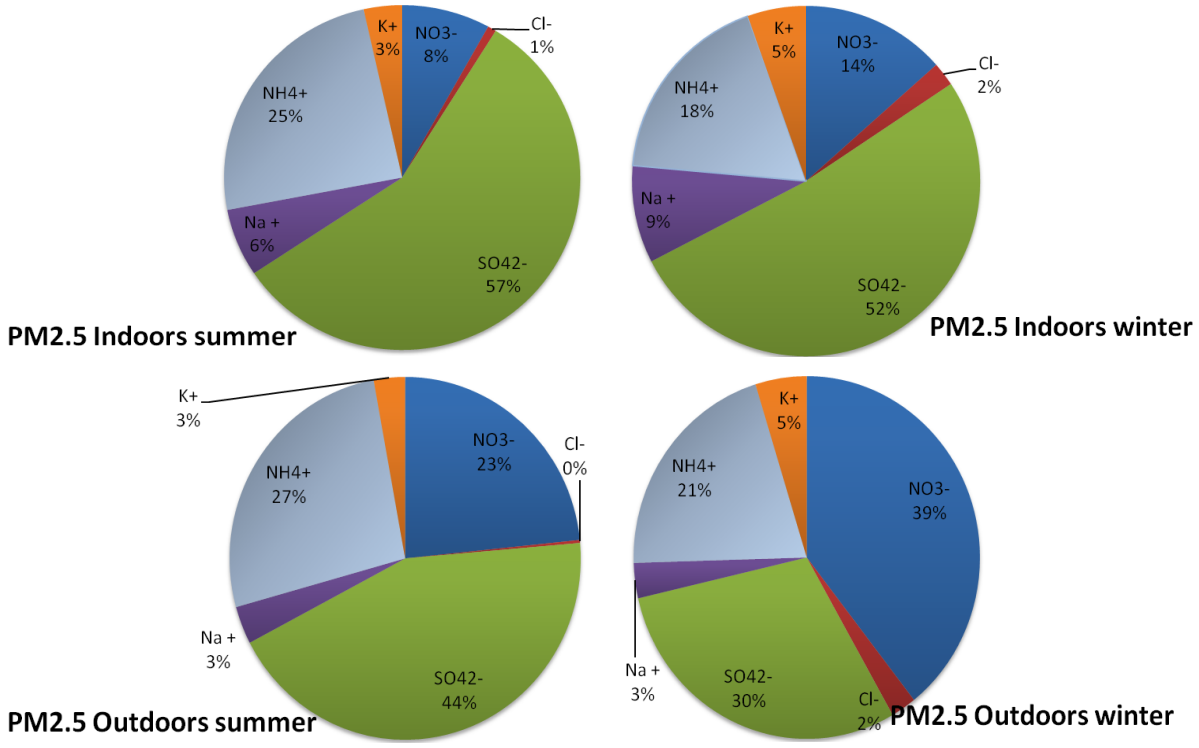

Figure 7: Percentage contribution of ionic species to their total concentration

2.3.2.2.3 Determination of the total elemental composition in the bulk samples

Concentrations of both light (from Z=12) and heavy (to Z=82) elements were measured. Among light elements, the highest contents were found for S, Si and Ca – both outside and inside the service flats. Fe and Al levels were comparable, pointing a crust source of these elements. Cd and Se content were always below the detection limit.

The tables below (TABLE XI, XII, XIII and XIV) contain the statistical characteristics of the measured elements (min, max and mean concentrations) in indoor (TABLE XI and XII) and outdoor air (TABLE XIII and XIV).

Sr, Cr and As were often found below the detection limit, thus they were excluded from further calculations, i.e. partitioning ratios calculations (table XIII). Al was excluded for the same reason from the calculations of the fine fractions ratio ($PM_1/PM_{2.5}$).

TABLE XI: The elemental concentrations in indoor air in different PM fractions during summer sampling campaigns

	Cavell 2007			Compostella 2008			Den Olm 2009		
Element	Min	Max	Average	Min	Max	Average	Min	Max	Average
PM_{10}									
Al	n.d.	n.d.	n.d.	144.3	238.4	188.5	<LOD	61.4	31.2
As	n.d.	n.d.	n.d.	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
Ca	n.d.	n.d.	n.d.	443.4	744.5	542.7	122.2	222.7	179.2
Cl	n.d.	n.d.	n.d.	51.9	187.3	96.0	11.8	262.2	109.6
Cr	n.d.	n.d.	n.d.	<LOD	10.6	5.2	<LOD	<LOD	<LOD
Cu	n.d.	n.d.	n.d.	44.6	64.8	52.5	<LOD	43.5	22.4
Fe	n.d.	n.d.	n.d.	234.4	320.8	275.4	48.9	160.9	100.6
K	n.d.	n.d.	n.d.	185.1	282.5	219.5	84.6	153.3	112.1
Mn	n.d.	n.d.	n.d.	20.4	26.1	24.0	<LOD	16.9	9.1
Ni	n.d.	n.d.	n.d.	<LOD	5.0	3.6	<LOD	3.2	1.6
Pb	n.d.	n.d.	n.d.	35.2	53.2	44.0	<LOD	31.2	17.0
S	n.d.	n.d.	n.d.	436.6	1127.6	727.7	208.6	774.6	416.4
Si	n.d.	n.d.	n.d.	370.9	528.3	459.3	97.6	237.2	158.1
Sr	n.d.	n.d.	n.d.	1.0	5.1	3.5	<LOD	<LOD	<LOD
Ti	n.d.	n.d.	n.d.	17.7	22.3	19.4	3.0	19.2	7.5
V	n.d.	n.d.	n.d.	<LOD	7.4	2.0	<LOD	4.1	1.7
Zn	n.d.	n.d.	n.d.	22.6	61.9	47.2	8.2	25.4	14.4
$PM_{2.5}$									
Al	63.5	166.7	108.8	104.8	130.8	118.9	<LOD	<LOD	<LOD
As	4.6	13.2	7.3	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
Ca	72.4	309.7	182.7	209.0	324.8	260.4	46.2	120.0	73.5
Cl	133.6	260.3	199.9	31.0	74.3	41.0	<LOD	126.6	42.5
Cr	<LOD	<LOD	<LOD	1.0	10.0	5.4	<LOD	<LOD	<LOD
Cu	6.2	13.6	11.5	40.1	57.0	47.2	<LOD	27.8	16.2
Fe	82.3	195.5	147.1	124.5	214.4	174.3	23.6	90.5	51.2
K	33.8	107.2	75.1	109.2	206.8	159.1	47.0	127.8	76.2
Mn	1.6	3.5	2.6	18.7	22.2	20.8	<LOD	10.9	6.3
Ni	<LOD	<LOD	<LOD	<LOD	4.3	2.6	<LOD	2.2	1.4
Pb	9.8	15.3	12.0	30.7	53.5	41.5	<LOD	25.9	12.9
S	480.4	1571.7	840.2	433.6	1132.6	692.1	212.1	697.0	390.4
Si	105.3	327.6	208.8	248.8	275.3	261.8	35.8	158.0	74.7
Sr	1.4	3.9	2.5	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
Ti	2.7	10.2	7.0	10.8	12.6	11.5	1.5	10.1	4.5
V	<LOD	6.4	2.6	1.5	5.8	2.4	<LOD	3.7	2.1
Zn	18.3	77.6	34.3	16.6	53.5	39.2	4.5	22.1	10.7

PM_1									
Al	n.d.	n.d.	n.d.	46.2	126.3	65.9	<LOD	<LOD	<LOD
As	n.d.	n.d.	n.d.	<LOD	14.6	4.7	<LOD	<LOD	<LOD
Ca	n.d.	n.d.	n.d.	82.7	345.4	151.2	13.5	26.1	20.3
Cl	n.d.	n.d.	n.d.	15.6	52.1	27.5	4.1	21.9	10.2
Cr	n.d.	n.d.	n.d.	2.9	6.7	3.8	<LOD	<LOD	<LOD
Cu	n.d.	n.d.	n.d.	17.3	47.2	25.1	0.3	13.3	7.9
Fe	n.d.	n.d.	n.d.	55.2	181.5	100.6	10.0	29.6	18.1
K	n.d.	n.d.	n.d.	62.7	292.0	128.2	22.4	94.4	51.4
Mn	n.d.	n.d.	n.d.	7.7	19.3	11.0	<LOD	5.5	3.3
Ni	n.d.	n.d.	n.d.	<LOD	3.2	2.1	<LOD	1.8	1.1
Pb	n.d.	n.d.	n.d.	17.2	51.3	27.0	<LOD	18.2	8.0
S	n.d.	n.d.	n.d.	354.1	1349.1	634.6	161.0	627.9	340.5
Si	n.d.	n.d.	n.d.	111.0	314.9	165.8	10.6	39.8	25.4
Sr	n.d.	n.d.	n.d.	<LOD	3.3	1.3	<LOD	<LOD	<LOD
Ti	n.d.	n.d.	n.d.	4.4	13.1	6.7	<LOD	2.2	1.1
V	n.d.	n.d.	n.d.	0.8	5.7	1.9	<LOD	3.4	1.9
Zn	n.d.	n.d.	n.d.	9.9	52.8	28.7	2.8	13.6	6.8

TABLE XII: The elemental concentrations in indoor air in different PM fractions during winter sampling campaigns

	Czagani 2008			Cantershof 2009			Czagani 2009		
Element	Min	Max	Average	Min	Max	Average	Min	Max	Average
PM_{10}									
Al	n.d.	n.d.	n.d.	94.2	166.0	116.5	31.5	94.0	60.1
As	n.d.	n.d.	n.d.	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
Ca	n.d.	n.d.	n.d.	289.7	504.3	393.7	172.7	369.3	269.9
Cl	n.d.	n.d.	n.d.	68.9	782.7	300.0	53.0	843.4	261.0
Cr	n.d.	n.d.	n.d.	<LOD	7.0	4.4	<LOD	4.2	2.4
Cu	n.d.	n.d.	n.d.	10.2	38.2	28.3	7.4	44.5	24.4
Fe	n.d.	n.d.	n.d.	134.0	225.8	171.4	107.8	318.5	156.3
K	n.d.	n.d.	n.d.	126.0	230.0	172.5	143.0	419.4	237.8
Mn	n.d.	n.d.	n.d.	<LOD	18.9	14.5	3.5	13.4	9.2
Ni	n.d.	n.d.	n.d.	2.4	5.1	3.8	<LOD	4.2	1.9
Pb	n.d.	n.d.	n.d.	12.8	35.4	28.4	10.2	39.8	25.7
S	n.d.	n.d.	n.d.	340.7	1456.9	615.8	282.5	1169.7	445.8
Si	n.d.	n.d.	n.d.	295.9	515.1	424.5	126.9	383.1	272.9
Sr	n.d.	n.d.	n.d.	1.0	5.9	3.9	<LOD	<LOD	<LOD
Ti	n.d.	n.d.	n.d.	14.0	27.3	18.3	8.6	14.8	11.4
V	n.d.	n.d.	n.d.	1.3	6.8	3.8	<LOD	5.0	2.3
Zn	n.d.	n.d.	n.d.	18.3	40.6	28.9	22.8	80.8	43.8

$PM_{2.5}$									
Al	<LOD	133.3	68.4	<LOD	101.8	75.6	<LOD	<LOD	<LOD
As	0.5	9.0	3.1	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
Ca	141.2	422.4	231.7	176.3	288.3	228.6	57.7	108.0	82.5
Cl	30.0	811.6	155.7	29.9	405.2	121.4	19.6	208.7	81.1
Cr	0.1	6.3	2.4	1.4	7.7	4.3	<LOD	<LOD	<LOD
Cu	6.4	39.9	22.0	<LOD	37.4	30.9	<LOD	35.6	27.0
Fe	68.2	328.5	165.4	90.9	142.9	115.7	46.9	162.7	74.4
K	144.8	524.4	292.1	85.1	192.2	138.9	79.1	346.4	171.8
Mn	<LOD	24.1	10.7	11.6	20.5	16.9	<LOD	14.7	10.2
Ni	<LOD	10.3	4.0	<LOD	5.1	3.6	<LOD	4.0	1.6
Pb	7.4	53.2	28.3	14.2	47.0	32.2	13.3	41.5	28.1
S	357.5	5398.0	1508.3	333.4	1484.7	611.9	186.3	1148.9	382.6
Si	104.7	342.8	222.5	199.7	299.8	260.1	39.5	137.8	89.0
Sr	0.5	5.5	2.5	0.1	4.9	1.6	<LOD	<LOD	<LOD
Ti	5.6	17.6	11.7	8.7	13.0	11.2	2.4	6.0	3.9
V	0.8	13.9	4.9	<LOD	7.8	3.7	<LOD	4.5	1.7
Zn	19.0	141.7	62.3	13.2	36.7	23.8	13.7	73.3	34.4
PM_{1}									
Al	<LOD	72.3	31.0	32.5	55.2	40.3	<LOD	<LOD	<LOD
As	<LOD	4.2	2.2	<LOD	7.9	2.3	<LOD	<LOD	<LOD
Ca	58.0	166.3	83.0	100.8	164.7	124.2	10.8	24.7	18.1
Cl	9.9	86.0	43.0	19.3	70.4	36.2	9.2	28.7	19.9
Cr	<LOD	3.5	1.7	<LOD	3.4	1.3	<LOD	2.2	1.0
Cu	4.5	17.1	10.5	0.7	18.3	13.2	2.1	19.4	10.1
Fe	24.1	144.5	66.7	46.6	77.1	60.3	11.9	52.3	24.6
K	88.5	362.6	206.0	53.7	148.3	99.7	52.2	271.1	136.8
Mn	<LOD	9.8	5.2	<LOD	9.6	6.3	<LOD	6.6	3.9
Ni	0.9	5.6	2.7	1.8	4.1	2.6	<LOD	3.2	1.3
Pb	5.9	31.9	18.8	6.8	27.7	18.2	4.8	30.3	17.3
S	269.6	3115.5	959.4	292.4	1262.4	518.9	162.9	840.6	318.2
Si	55.1	229.1	100.3	123.3	186.5	150.0	13.7	59.4	31.9
Sr	<LOD	1.2	0.7	<LOD	1.9	0.9	<LOD	<LOD	<LOD
Ti	3.1	9.3	4.7	4.9	8.2	6.3	<LOD	3.2	1.1
V	0.5	9.2	3.6	1.2	6.9	3.4	<LOD	4.5	1.7
Zn	11.4	76.4	38.4	10.0	28.4	17.7	10.2	52.9	25.7

TABLE XIII: The elemental concentrations in ambient air in different PM fractions during summer sampling campaigns

	Cavell 2007			Compostella 2008			Den Olm 2009		
Element	Min	Max	Average	Min	Max	Average	Min	Max	Average
PM_{10}									
Al	n.d.	n.d.	n.d.	109.6	394.7	234.9	<LOD	234.6	105.7
As	n.d.	n.d.	n.d.	<LOD	18.0	6.4	<LOD	<LOD	<LOD
Ca	n.d.	n.d.	n.d.	473.3	2810.2	1033.7	136.9	811.2	447.1
Cl	n.d.	n.d.	n.d.	29.9	718.2	169.4	<LOD	1853.9	325.4
Cr	n.d.	n.d.	n.d.	7.4	12.0	9.4	<LOD	4.1	3.0
Cu	n.d.	n.d.	n.d.	36.9	61.2	47.1	22.4	123.4	77.8
Fe	n.d.	n.d.	n.d.	305.2	727.3	467.4	91.4	567.8	315.1
K	n.d.	n.d.	n.d.	184.0	399.4	261.8	79.1	292.5	175.7
Mn	n.d.	n.d.	n.d.	22.4	39.5	29.2	<LOD	24.9	14.8
Ni	n.d.	n.d.	n.d.	<LOD	5.7	4.0	<LOD	3.9	2.6
Pb	n.d.	n.d.	n.d.	29.0	61.2	48.0	<LOD	34.6	21.0
S	n.d.	n.d.	n.d.	561.1	1307.1	836.1	412.8	1011.0	724.6
Si	n.d.	n.d.	n.d.	321.3	1163.5	700.5	183.0	992.2	513.5
Sr	n.d.	n.d.	n.d.	<LOD	6.1	3.9	<LOD	4.0	2.6
Ti	n.d.	n.d.	n.d.	15.1	46.8	26.1	6.1	29.4	16.8
V	n.d.	n.d.	n.d.	2.3	9.1	4.0	<LOD	7.0	4.2
Zn	n.d.	n.d.	n.d.	21.1	90.5	59.6	14.3	41.7	23.6
$PM_{2.5}$									
Al	73.1	228.8	129.7	78.6	216.4	133.7	<LOD	47.4	26.5
As	2.2	19.4	6.3	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
Ca	150.1	492.6	359.1	174.6	1751.3	609.0	34.7	141.6	81.4
Cl	134.0	149.8	143.1	<LOD	116.0	43.3	<LOD	110.7	19.9
Cr	<LOD	<LOD	<LOD	1.3	9.3	6.5	<LOD	<LOD	<LOD
Cu	5.5	9.7	6.9	33.8	48.8	39.8	23.3	75.4	45.6
Fe	140.9	303.3	214.6	139.1	359.0	234.1	32.5	132.4	81.3
K	50.5	150.8	98.0	99.3	279.7	176.1	39.2	153.3	90.2
Mn	4.8	9.5	6.6	20.2	30.8	24.2	<LOD	12.5	7.1
Ni	<LOD	8.6	3.5	<LOD	4.9	2.6	<LOD	2.5	2.0
Pb	<LOD	20.5	12.9	30.7	59.5	45.9	<LOD	29.7	17.4
S	504.8	2143.7	1179.5	477.5	1287.8	764.0	291.6	1005.0	686.3
Si	172.2	718.3	367.8	216.2	595.3	378.4	<LOD	315.4	124.2
Sr	1.2	3.8	2.5	<LOD	5.6	2.5	<LOD	<LOD	<LOD
Ti	6.0	23.6	12.0	7.8	24.7	13.9	<LOD	9.2	4.1
V	1.3	23.1	8.6	<LOD	7.4	2.8	1.1	6.0	3.5
Zn	19.9	54.6	37.0	15.2	70.9	44.6	8.8	27.1	14.9

PM_1									
Al	n.d.	n.d.	n.d.	46.1	101.7	62.4	<LOD	<LOD	<LOD
As	n.d.	n.d.	n.d.	<LOD	9.2	4.7	<LOD	<LOD	<LOD
Ca	n.d.	n.d.	n.d.	112.1	817.9	332.7	4.2	11.9	8.6
Cl	n.d.	n.d.	n.d.	6.8	40.1	18.3	<LOD	7.2	3.3
Cr	n.d.	n.d.	n.d.	<LOD	4.0	2.8	<LOD	<LOD	<LOD
Cu	n.d.	n.d.	n.d.	12.9	23.0	16.8	10.5	30.0	22.0
Fe	n.d.	n.d.	n.d.	53.0	163.1	102.8	12.4	26.8	18.6
K	n.d.	n.d.	n.d.	48.3	179.1	110.5	18.6	102.1	56.5
Mn	n.d.	n.d.	n.d.	7.6	12.6	10.4	<LOD	6.6	4.5
Ni	n.d.	n.d.	n.d.	1.2	3.5	2.1	1.1	2.2	1.8
Pb	n.d.	n.d.	n.d.	14.4	29.5	24.2	<LOD	21.1	12.2
S	n.d.	n.d.	n.d.	346.7	802.9	525.1	223.3	824.4	488.7
Si	n.d.	n.d.	n.d.	122.4	315.7	189.7	<LOD	39.2	14.7
Sr	n.d.	n.d.	n.d.	<LOD	2.6	1.1	<LOD	<LOD	<LOD
Ti	n.d.	n.d.	n.d.	4.2	11.0	6.6	<LOD	1.4	0.6
V	n.d.	n.d.	n.d.	0.7	5.8	2.2	1.8	4.5	3.4
Zn	n.d.	n.d.	n.d.	8.0	35.4	25.4	4.1	14.2	8.8

TABLE XIV: The elemental concentrations in ambient air in different PM fractions during winter sampling campaigns

	Czagani 2008			Cantershof 2009			Czagani 2009		
Element	Min	Max	Average	Min	Max	Average	Min	Max	Average
PM_{10}									
Al	n.d.	n.d.	n.d.	91.0	211.8	160.5	<LOD	284.1	99.4
As	n.d.	n.d.	n.d.	<LOD	18.9	8.4	<LOD	<LOD	<LOD
Ca	n.d.	n.d.	n.d.	420.7	1097.1	655.0	188.8	800.0	428.6
Cl	n.d.	n.d.	n.d.	120.3	4565.9	1301.6	160.5	2337.8	1074.9
Cr	n.d.	n.d.	n.d.	<LOD	9.3	4.9	<LOD	7.1	4.0
Cu	n.d.	n.d.	n.d.	<LOD	45.4	27.3	8.4	110.0	38.5
Fe	n.d.	n.d.	n.d.	271.1	742.6	422.4	196.2	878.2	385.2
K	n.d.	n.d.	n.d.	118.0	459.9	256.3	172.4	596.9	316.0
Mn	n.d.	n.d.	n.d.	14.9	25.0	20.3	4.6	30.1	16.3
Ni	n.d.	n.d.	n.d.	3.9	14.5	6.2	<LOD	7.4	2.8
Pb	n.d.	n.d.	n.d.	13.7	51.2	37.7	16.4	63.1	36.3
S	n.d.	n.d.	n.d.	485.8	2818.3	1068.4	385.4	1685.4	637.6
Si	n.d.	n.d.	n.d.	382.5	980.0	654.8	125.8	733.4	412.9
Sr	n.d.	n.d.	n.d.	<LOD	10.3	5.2	<LOD	4.1	2.6
Ti	n.d.	n.d.	n.d.	14.7	44.4	22.8	8.1	33.4	17.5
V	n.d.	n.d.	n.d.	2.5	23.3	8.3	<LOD	8.0	3.6
Zn	n.d.	n.d.	n.d.	26.3	82.2	45.1	24.3	122.5	66.7

$PM_{2.5}$									
Al	<LOD	199.1	67.8	<LOD	109.2	44.6	<LOD	33.8	22.9
As	<LOD	9.9	4.3	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
Ca	118.2	565.6	240.6	65.3	363.9	169.5	47.0	100.4	72.2
Cl	74.0	2147.1	892.1	26.2	827.7	185.0	56.0	510.5	206.0
Cr	0.9	9.3	4.0	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
Cu	<LOD	50.6	21.2	<LOD	33.8	17.0	10.7	48.8	29.4
Fe	67.1	531.5	249.5	73.3	194.8	131.2	68.2	267.0	123.3
K	154.8	624.3	342.1	50.7	204.5	140.9	96.1	503.6	226.0
Mn	<LOD	29.3	14.0	10.6	19.1	14.6	5.7	16.2	12.0
Ni	<LOD	12.3	5.6	3.6	6.3	4.6	<LOD	4.4	2.3
Pb	7.4	65.7	37.5	13.8	48.2	30.7	14.4	61.4	40.5
S	431.8	6445.9	1921.5	398.1	1987.6	812.1	299.0	1537.9	569.5
Si	48.6	645.0	271.3	106.9	420.5	235.6	<LOD	129.4	74.1
Sr	<LOD	3.8	2.3	0.2	4.7	2.4	<LOD	<LOD	<LOD
Ti	4.3	26.2	12.4	5.3	13.1	8.4	<LOD	6.0	3.6
V	1.3	16.5	6.6	1.9	9.6	5.2	<LOD	6.8	3.2
Zn	17.8	186.3	86.0	15.4	42.1	29.3	19.7	103.5	53.6
PM_{1}									
Al	<LOD	<LOD	<LOD	n.d.	n.d.	n.d.	<LOD	<LOD	<LOD
As	<LOD	5.6	2.8	n.d.	n.d.	n.d.	<LOD	2.8	1.4
Ca	4.5	59.6	31.1	n.d.	n.d.	n.d.	6.4	24.8	11.1
Cl	45.3	876.2	382.8	n.d.	n.d.	n.d.	30.9	282.0	104.3
Cr	<LOD	3.3	1.1	n.d.	n.d.	n.d.	<LOD	<LOD	<LOD
Cu	0.4	19.5	7.6	n.d.	n.d.	n.d.	1.0	19.9	8.9
Fe	13.1	138.5	55.3	n.d.	n.d.	n.d.	12.9	51.5	25.8
K	70.3	413.7	216.8	n.d.	n.d.	n.d.	61.8	352.9	159.7
Mn	<LOD	9.4	4.9	n.d.	n.d.	n.d.	1.7	6.0	3.7
Ni	<LOD	7.1	3.3	n.d.	n.d.	n.d.	<LOD	3.0	1.6
Pb	4.4	41.6	21.8	n.d.	n.d.	n.d.	9.5	38.7	22.3
S	295.9	2014.6	782.4	n.d.	n.d.	n.d.	221.8	642.7	367.7
Si	14.3	83.3	43.1	n.d.	n.d.	n.d.	<LOD	34.0	18.4
Sr	<LOD	1.6	0.6	n.d.	n.d.	n.d.	<LOD	<LOD	<LOD
Ti	0.1	3.8	2.0	n.d.	n.d.	n.d.	<LOD	1.2	0.7
V	0.4	11.9	4.7	n.d.	n.d.	n.d.	0.4	4.4	2.3
Zn	9.9	90.5	41.2	n.d.	n.d.	n.d.	12.5	62.1	33.3

Elements such as Si, Al, Ca, Fe, Cl and Ti are characterized by very low PM2.5/PM10 and PM1/PM2.5 ratios, they range between 0.24-0.56. These elements are often of crustal origin, thus they are found mainly in the coarse fraction. On the contrary, elements such as: Cu, K, Mn, Ni, Pb, S, V and Zn accumulate mainly in the fine fraction; they are characterized by high PM2.5/PM10 ratios in the range of 0.61 for K to 0.98 for Pb in the ambient air. The ratios of PM1/PM2.5 are somewhat lower.

Both ratios generally increase in indoor air compared to the outdoor since fine particles infiltrate much easier from outside than coarse particles.

2.3.2.2.4 Determination of the elemental carbon (EC) concentration

The concentration of elemental carbon was measured continuously starting from the second campaign (Czagani, 2008). In two locations (Cantershof and Den Olm in 2009) due to the equipment limitations, the EC was only measured indoors. The time interval was 5 minutes. The values are the arithmetic mean from these periods of time. In Figure 8, the graphs from each campaign are shown. In these graphs one data point is an average of 30 minutes, which were calculated using instrumental averages of 5 min intervals. As it can be seen in graphs from locations where the EC was sampled both indoors and outdoors, the indoor concentration reflected almost perfectly the ambient concentrations. Somewhat lower concentrations were registered inside and a lag of up to two hours was observed. In Figure 9 one day from sampling campaign in Czagani old-age home is shown for an in detail view. Generally the concentrations were elevated in the evening hours. This might be ascribed to either transport from urban centers (Allen et al., 1999) or a local influence, i.e. emission from the heating of nearby houses as far as winter periods are concerned. Furthermore, the elevated concentrations can be also seen during morning hours 7-10 a.m., which can be a result of increased traffic and subsequently increased emissions (Allen et al., 1999). Because of its large contribution from traffic, EC, it is often used as a surrogate for traffic-related particles and usually continuous measurements show good correlation with concentrations of Diesel Exhaust Particles (DEP) or with traffic counts (Wu et al, 2007). The highest outdoor peak concentration in campaigns where outdoor concentrations were measured, was registered in winter 2008 (Czagani) and amounted to 26 µg m^{-3} whilst in indoor air, the highest peak was registerd in winter 2009 (Czagani) and reached up to 38.8 µg m^{-3} . The lowest indoor concentrations were measured in June 2009 in Den Olm old-age home. Generally, in all sampling campaigns, the lowest daily concentrations of BC were registered around 14-16 h.

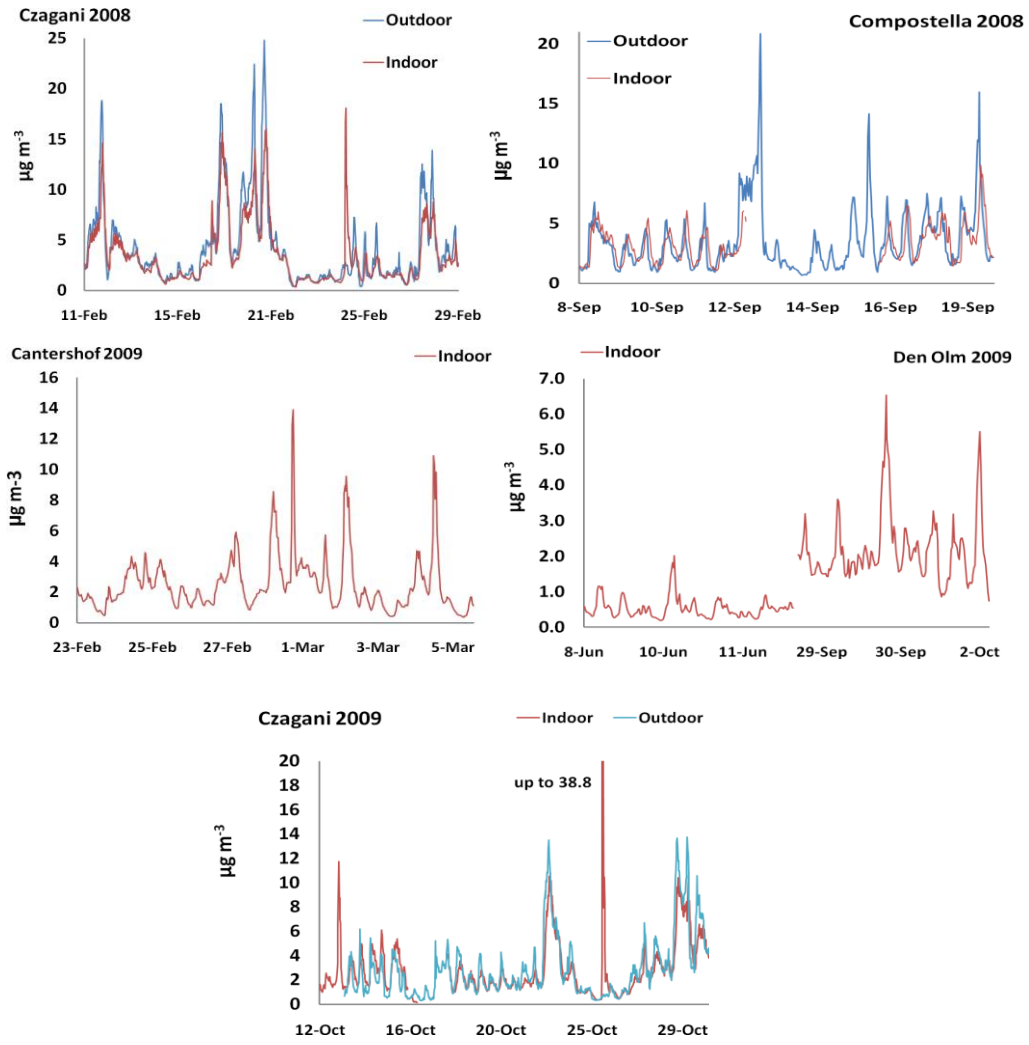

Figure 8: Elemental carbon (EC) concentrations (µg m^{-3}) in different sampling locations

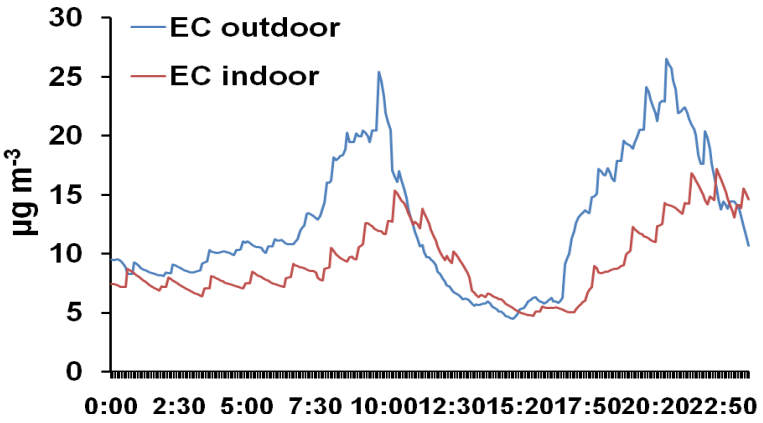

Figure 9: Outdoor and Indoor concentration of elemental carbon (EC) on the 20/02/2008 in Czagani old age home

2.3.2.2.5 Determination of gaseous organic and inorganic pollutants

Gaseous pollutants, both inorganic (SO2, NO2) were collected. The content of NO_2 content was ca. 10-fold higher than SO2, and this ratio was observed both indoors and outdoors during sampling campaigns. Figure 10 shows the weekly average concentrations of NOx, whilst Figure 11, weekly average concentrations of SO_2.

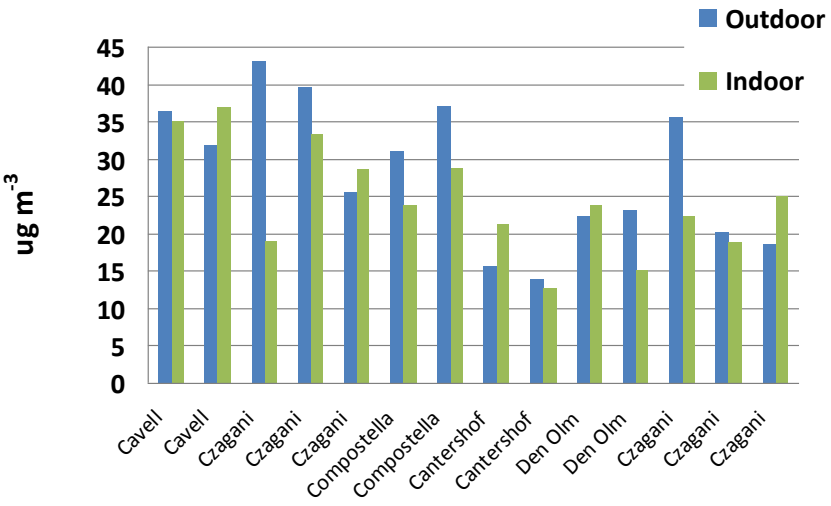

Figure 10: Concentration of NOx indoors and outdoors during the sampling period

The concentration range of NOx outside was 14 - 43 µg m^{-3}, whilst indoors it was between 13 - 37 µg m^{-3}. SO_2 concentration range was 0.5 - 5 µg m^{-3} outside, whilst it ranged from below the limit of detection up to 5.4 µg m^{-3} in the inside of the buildings.

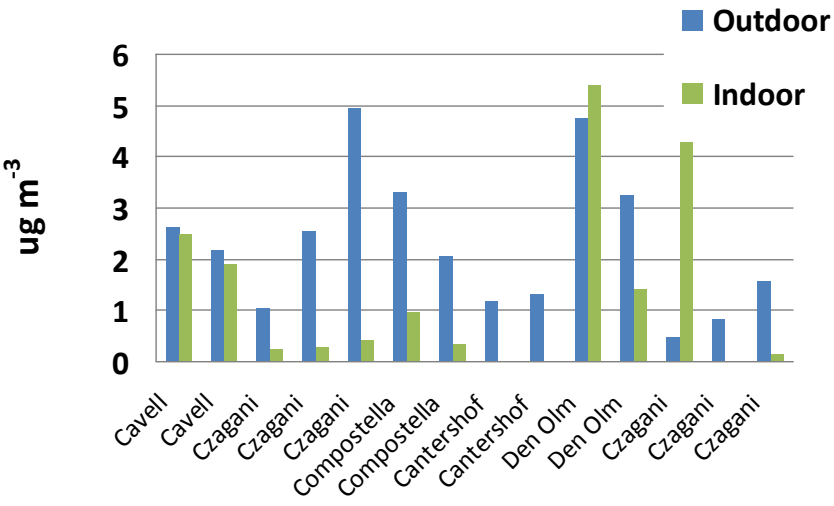

Figure 11: Concentration of SO_2 indoors and outdoors during the sampling period

2.3.2.2.6 Oxy-PAHs analysis on particulate matter: method, sampling and analysis

I. Method development

To evaluate the extraction techniques, recoveries of the selected oxy-PAHs from quartz fiber filters were determined (Figure 12). The filters were spotted with a known amount (11-172 ng) of the target oxy-PAHs and after the extraction, the extract was analysed with the optimized HPLC-HRMS method. The same standard solution was analysed after direct injection. The ratio of the peak intensities gives the recovery. The results indicate that the recoveries for both extraction techniques are high (ASE: 79-98% (n=10); USE: 78-90% (n=9) and not significantly different (a=0.05) from each other. Since the number of manipulation steps is higher and a dirtier extract is obtained for the USE procedure, ASE extraction was selected as the extraction technique for further analysis.

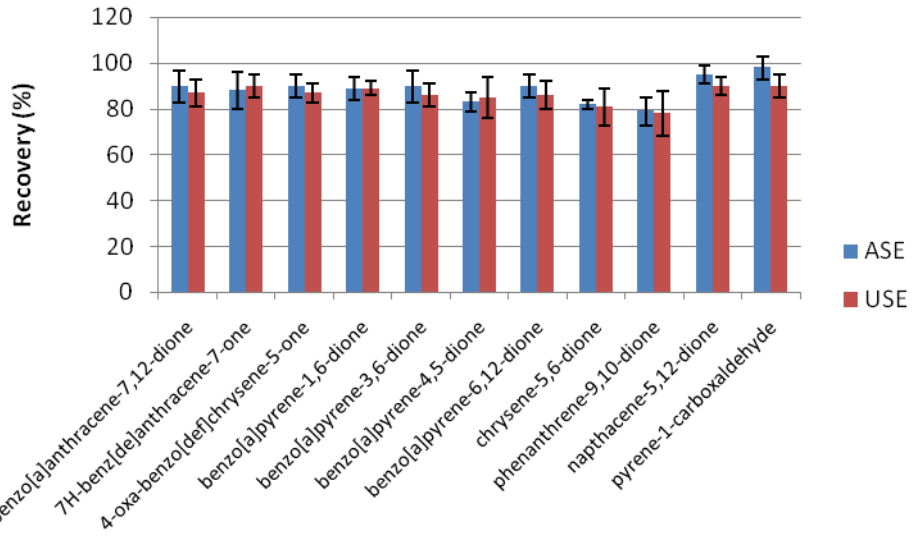

Figure. 12: Recoveries (%) for USE and ASE extraction of oxy-PAHs from quartz (n=9-10)

Particulate matter is an extremely complex matrix containing a wide range of chemical species, including inorganic acids and salts, metals, water, and a complex mixture of low volatility organic compounds, all in highly variable concentrations. It is possible that these matrix constituents can influence both the recovery and ionization process. The response of a sample-extract can be higher (ion enhancement) or lower (ion suppression) when compared to a standard solution containing the same amount of target compounds.

Therefore, the influence of the matrix on both the ionization process and the recovery was investigated. Matrix dependent recoveries were in the range between 76 and 107%, and matrix enhancement/suppression factors were between 89 and 123%.

Before each analytical run calibration was performed by injection of 5 standard solutions. Linearity was good as indicated by r^2 values ranging from 0.95 to 0.9999. Instrumental detection (IDL) limits calculated based on a signal to noise ratio of 3 were dependent of the compound under study and ranged from 8 pg to 1265 pg injected.

II. Concentration levels of oxy-PAHs

In TABLE XV the minimum and maximum concentration levels for the target oxy-PAHs are given for all sampling campaigns. Concentrations are expressed in mass per volume of sampled air (pg/m^3). All given concentrations are corrected both for recovery and ion suppression or enhancement. To the best of our knowledge, these are the first data available about oxy-PAHs concentrations in Belgium.

TABLE XV: Oxy-PAHs concentrations (pg/m³) measured at the elderly homes

	RVT Cavell (n=8) June 2007 PM10		RVT Czagani (n=18) February 2008 PM10		RVT Compostella (n=11) September 2008 PM10	
	min	max	min	max	min	max
Phenanthrene-9,10-dione	22	97	52	896	92	140
Chrysene-5,6-dione	1	3	8	62	6	20
Benzo[a]pyrene-4,5-dione	1	2	3	74	13	13
Benzo[a]pyrene-1,6-dione	2	5	24	131	5	39
Benzo[a]pyrene-3,6-dione	1	5	23	144	8	42
Benzo[a]pyrene-6,12-dione	2	7	41	424	19	53
4-oxa-benzo[def]chrysene-5-one	ND		3	55	ND	
7H-benzo[de]anthracene-7-one	-		-		46	601
1-pyreencarboxaldehyde	-		-		ND	
benz[a]anthracene-7,12-dione	-		-		ND	
napthacene-5,12-dione	-		-		ND	

	RVT Cantershof (n=10) February 2009 PM10		RVT Den Olm (n=7) June - september -october 2009 TSP		RVT Czagani 2 (n=15) October 2009 PM10	
	min	max	min	max	min	max
Phenanthrene-9,10-dione	329	329	87	5785	68	1018
Chrysene-5,6-dione	4	25	12	12	2	62
Benzo[a]pyrene-4,5-dione	11	13	ND		10	52
Benzo[a]pyrene-1,6-dione	6	38	54	235	8	84
Benzo[a]pyrene-3,6-dione	5	42	6	21	7	100
Benzo[a]pyrene-6,12-dione	5	58	7	34	23	162
4-oxa-benzo[def]chrysene-5-one	ND		ND		ND	
7H-benzo[de]anthracene-7-one	54	591	17	668	105	2126
1-pyreencarboxaldehyde	ND		ND		ND	
benz[a]anthracene-7,12-dione	ND		ND		ND	
napthacene-5,12-dione	ND		ND		ND	

-: not measured; ND: compound signal to noise ratio less than 3.

Overall, the oxy-PAHs concentration levels are found to be in the lower pg/m³ to ng/m³ level. Concentrations of phenanthrene-9,10-dione and 7H-benz[a]anthracene-7-one are higher compared to the other target compounds. The concentration levels observed are in accordance with the literature data about these compounds (Walgraeve et al. 2010). The median of the total oxy-PAH concentrations - calculated as the sum of the individual concentrations of the oxy-PAHs monitored during all sampling campaigns - was the highest during the sampling campaign at RVT Czagani (February 2008). A median total oxy-PAH concentration of 486 pg/m³ is measured, which is a factor 2 to 11 times higher compared to the other sampling campaigns.

2.3.2.2.7 VOCs concentrations in the ambient air of the elderly homes

Using passive sampling and TD-GC-MS analysis, 64 VOCs (representative for indoor air pollution) were monitored in indoor and outdoor air. TABLE XIV gives an overview of the obtained concentration levels (µg/m³) in the different elderly homes, expressed as a concentration range (minimum-maximum concentration), both for indoor and outdoor sampling locations. The total volatile organic compound (TVOC) concentration is defined as the sum of the concentrations of all target compounds that could be quantified. Overall, the indoor TVOC concentration ranged from 12 to 320 µg/m³. In Figure 13, box-plots of the concentration levels measured during the different sampling campaigns are plotted for both the TVOC concentration and a set of target compounds (BTEX and limonene). Comparison with the Flemish indoor air guideline values for TVOC concentration (≤200µg/m³) and benzene (≤2µg/m³), reveals guidelines are exceeded during the sampling campaigns in February 2008 and 2009. Three compounds are mainly responsible for the increased TVOC levels: limonene, ethyl acetate, and toluene. Stable meteorological conditions during the sampling campaign in February 2008 might have contributed to the benzene guideline exceeding in RVT Czagani. TVOC concentrations in the gardens of the elderly homes ranged from 7 to 28 µg/m³.

TABLE XVI: VOCs concentration levels measured during the elderly home sampling campaign (NQ = below quantification limit)

	Indoor														Outdoor	
	Cavell (n=10)		Czagani (n=10)		Compostella (n=10)		Cantershof (n=10)		Den Olm (n=10)		Czagani (n=10)		all sampling campaign (n=12)	
Component Name	min	max	min	max	min	max	min	max	min	max	min	max	min	max
n-hexane	2.91	6.65	0.51	1.98	NQ		0.89	14.80	NQ		NQ		0.73	3.94
ethyl acetate	3.74	34.76	3.17	59.46	1.80	36.93	3.19	114.91	3.90	21.35	1.62	63.39	0.47	1.64
methylcyclopentane	0.32	0.96	0.30	1.78	0.25	0.82	0.40	15.39	0.48	0.53	NQ		0.45	0.86
1,2-dichloroethane	0.17	0.37	NQ		NQ		0.22	0.41	NQ		NQ		0.12	0.16
benzene	0.71	1.61	2.22	3.00	0.27	0.31	1.10	1.40	0.32	0.47	0.53	0.85	0.37	3.70
cyclohexane	0.86	3.09	0.32	2.02	0.46	3.52	0.47	51.69	NQ		1.61	9.26	0.14	1.15
n-heptane	0.79	2.93	0.61	4.35	0.35	3.49	0.55	67.88	0.31	0.48	0.40	0.76	0.15	1.10
toluene	5.51	26.69	5.11	61.36	0.68	11.73	2.96	6.10	1.04	10.09	2.40	7.38	0.95	6.74
hexanal	7.75	29.54	18.80	30.24	6.63	40.90	4.62	94.58	9.20	21.29	NQ		NQ	
octane	2.78	5.80	0.98	3.02	0.77	1.36	0.90	8.67	0.89	1.50	0.64	1.29	0.25	1.71
tetrachloroethylene	0.21	0.91	0.26	0.70	0.13	0.79	0.17	0.98	0.06	0.12	0.20	3.02	0.06	0.30
ethylbenzene	0.56	1.46	0.69	3.22	0.19	0.44	0.36	0.54	0.13	0.18	0.23	0.41	0.11	0.89
p-xylene	0.43	1.18	0.75	6.74	0.53	1.28	0.96	1.40	0.34	0.45	0.67	1.17	0.31	2.50
styrene	0.13	6.16	0.55	1.45	0.16	0.49	0.29	2.80	0.10	0.44	0.24	0.47	0.04	0.13
phenol	1.45	4.51	NQ		NQ		0.49	1.83	0.50	1.00	1.39	21.62	0.44	3.37
n-nonane	1.57	2.86	0.78	4.45	0.39	7.14	0.47	1.49	0.51	1.02	0.54	1.24	0.17	1.71
alpha-pinene	0.77	9.52	2.00	6.69	0.34	15.89	0.90	19.33	0.56	2.30	0.54	2.38	0.06	0.63
benzaldehyde	2.21	5.30	1.58	3.19	1.43	3.51	0.81	4.98	0.78	2.13	0.64	1.58	1.60	4.50
propylbenzene	0.16	0.52	0.13	1.62	0.37	0.40	0.07	0.15	0.03	0.09	0.06	0.12	0.02	0.16
1,2,4-trimethylbenzene	0.77	2.49	0.74	9.34	NQ		0.42	1.13	0.15	0.41	0.32	0.59	0.12	0.83
n-decane	1.71	3.01	1.25	11.56	1.07	36.22	1.12	3.66	1.33	5.20	NQ		0.40	1.20
limonene	0.53	21.09	18.93	102.47	3.69	47.21	3.99	142.70	10.26	45.93	2.58	45.70	NQ	
n-undecane	2.00	2.45	0.66	5.55	0.46	17.04	0.80	1.87	NQ		3.66	3.66	0.36	0.43
n-dodecane	NQ		0.93	11.37	1.75	6.33	2.15	2.22	NQ		1.50	1.50	NQ	

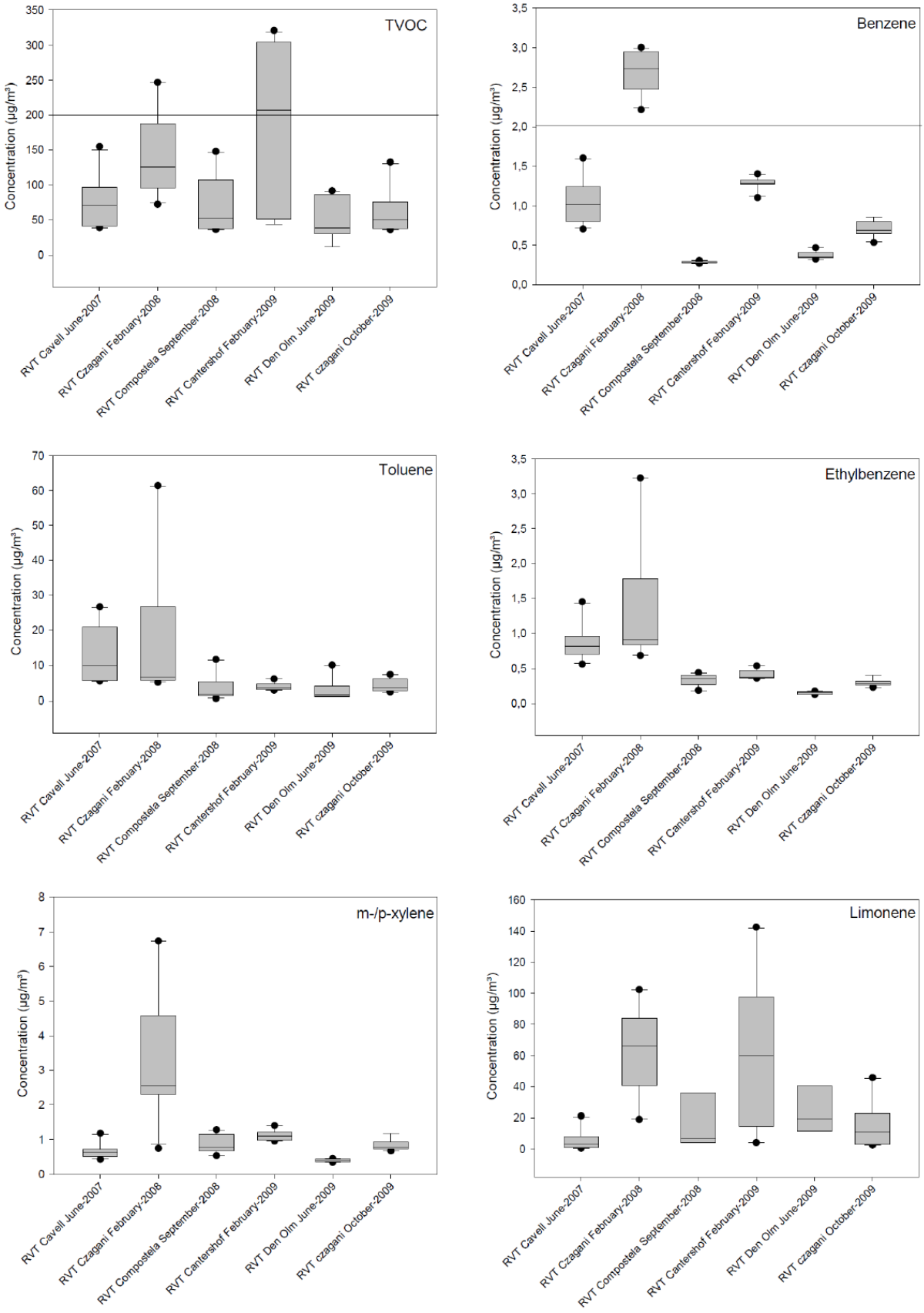

Figure 13: Box plots representing the concentration levels observed in the indoor air of the different elderly-homes.

2.3.2.3 Health effects

2.3.2.3.1 Effect on platelet function

I. Gravimetric $PM_{2.5}$

At baseline, the closure time of the PFA-100 averaged 142.3 (SD: 53.7) seconds. Between two clinical examinations in the same person, no significant effect of in- and outdoor 24 hour average $PM_{2.5}$ levels on the closure time of PFA-100 were seen (Figure 14).

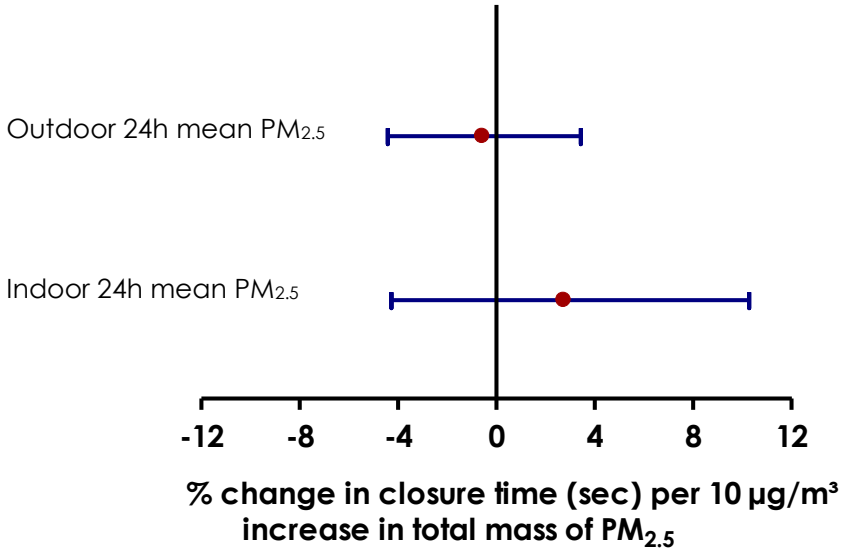

Figure 14: Point Estimates and 95% CI of the percent change in closure time per 10 µg/m³ increase in indoor and outdoor 24 hour mean $PM_{2.5}$.

II. Elemental composition of $PM_{2.5}$

Median concentrations of the different metals in outdoor $PM_{2.5}$ over the study period (54 days) are given in TABLE XVII.

TABLE XVII: Median concentration of elemental composition of outdoor $PM_{2.5}$ (over 54 days)

$PM_{2.5}$ component, ng/m3	Median	Interquartile range	Percent of $PM_{2.5}$ mass
Sulfur (S)	502.3	482.6	4.1
Potassium (K)	105	83.5	0.81
Iron (Fe)	104.2	75.3	0.77
Silicium (Si)	97.0	125.4	0.86
Calcium (Ca)	82.9	157.6	0.65
Chlorine (Cl)	40.3	247.6	0.92
Zinc (Zn)	21.5	19.8	0.19
Lead (Pb)	20.0	13.0	0.15
Aluminium (Al)	12.1	52.1	0.15
Copper (Cu)	9.5	5.4	0.15
Manganese (Mn)	8.5	5.8	0.07
Titanium (Ti)	4.6	4.0	0.03
Nickel (Ni)	2.3	1.3	0.02
Vanadium (V)	2.1	1.9	0.02
Chromium (Cr)	1.7	3.2	0.02
Strontium (Sr)	1.1	0.98	0.01
Arsenic (As)	0.98	10.3	0.02

No significant effect of the different metal concentrations in outdoor 24 h mean $PM_{2.5}$ concentration on platelet function was seen (Figure 15A and 15B).

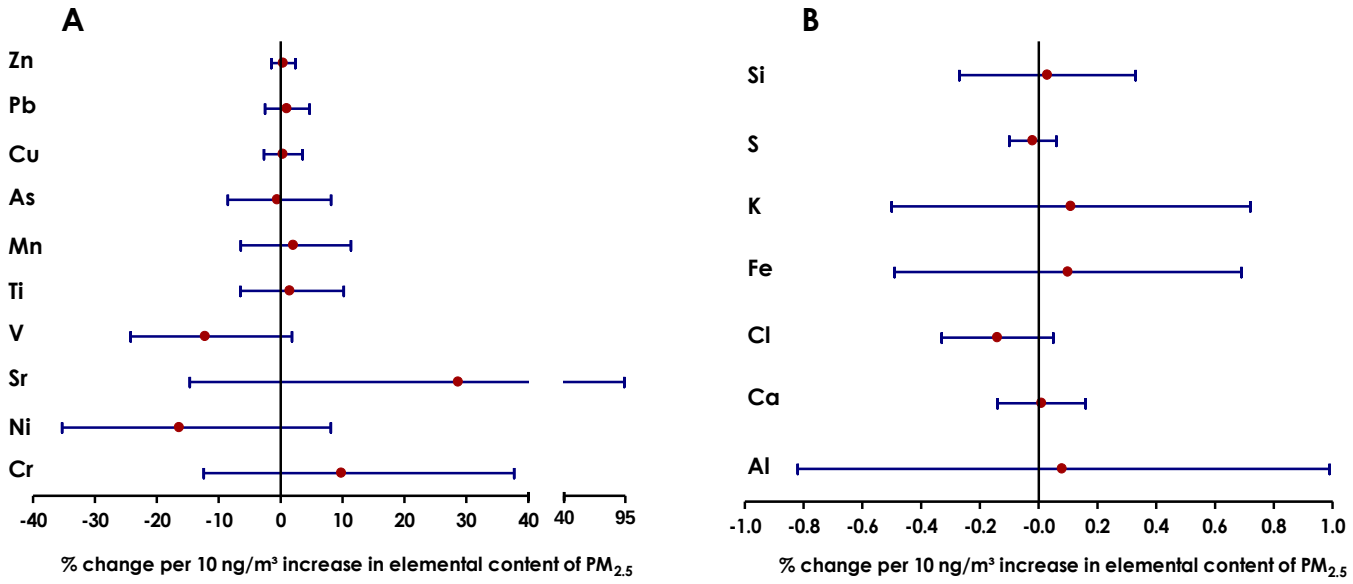

Figure 15: Point Estimates and 95% CI of the percent change in closure time per 10 ng/m³ increase in elemental content of outdoor 24 hour mean $PM_{2.5}$.

III. Oxy-PAHs

No significant effect of the different oxy-PAHs on platelet function was observed (Figure 16).

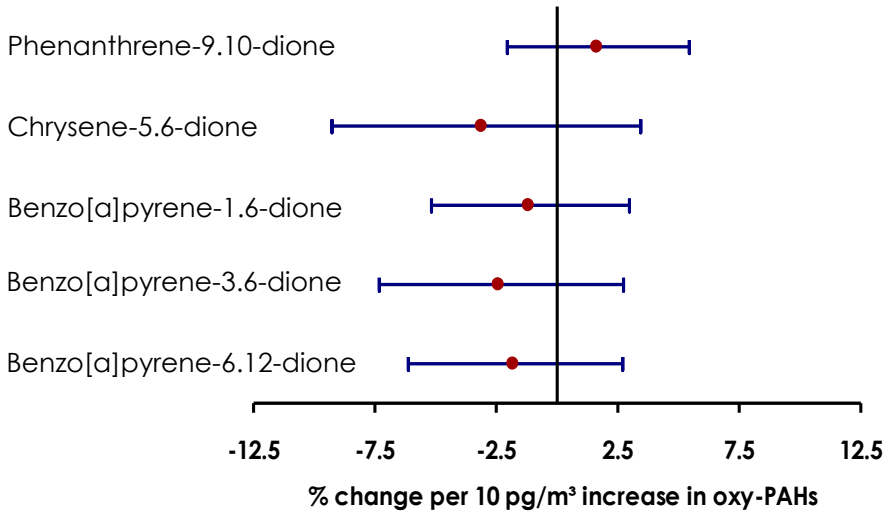

Figure 16: Point Estimates and 95% CI of the percent change in closure time per 10 pg/m³ increase in oxy-PAHs of outdoor 24 hour mean PM_{10}

2.3.2.3.2 Effects on blood pressure (BP)

I. Gravimetric $PM_{2.5}$

Increases in systolic blood pressure and pulse pressure were associated with both outdoor 24 hour mean $PM_{2.5}$ exposure while diastolic blood pressure was not changed. Each 10µg/m³ increase in outdoor $PM_{2.5}$ was associated with a 0.89% increase in systolic BP and a 1.8% increase in pulse pressure.

Indoor 24 hour mean $PM_{2.5}$ levels were not significantly associated with changes in blood pressure (Figure 17).

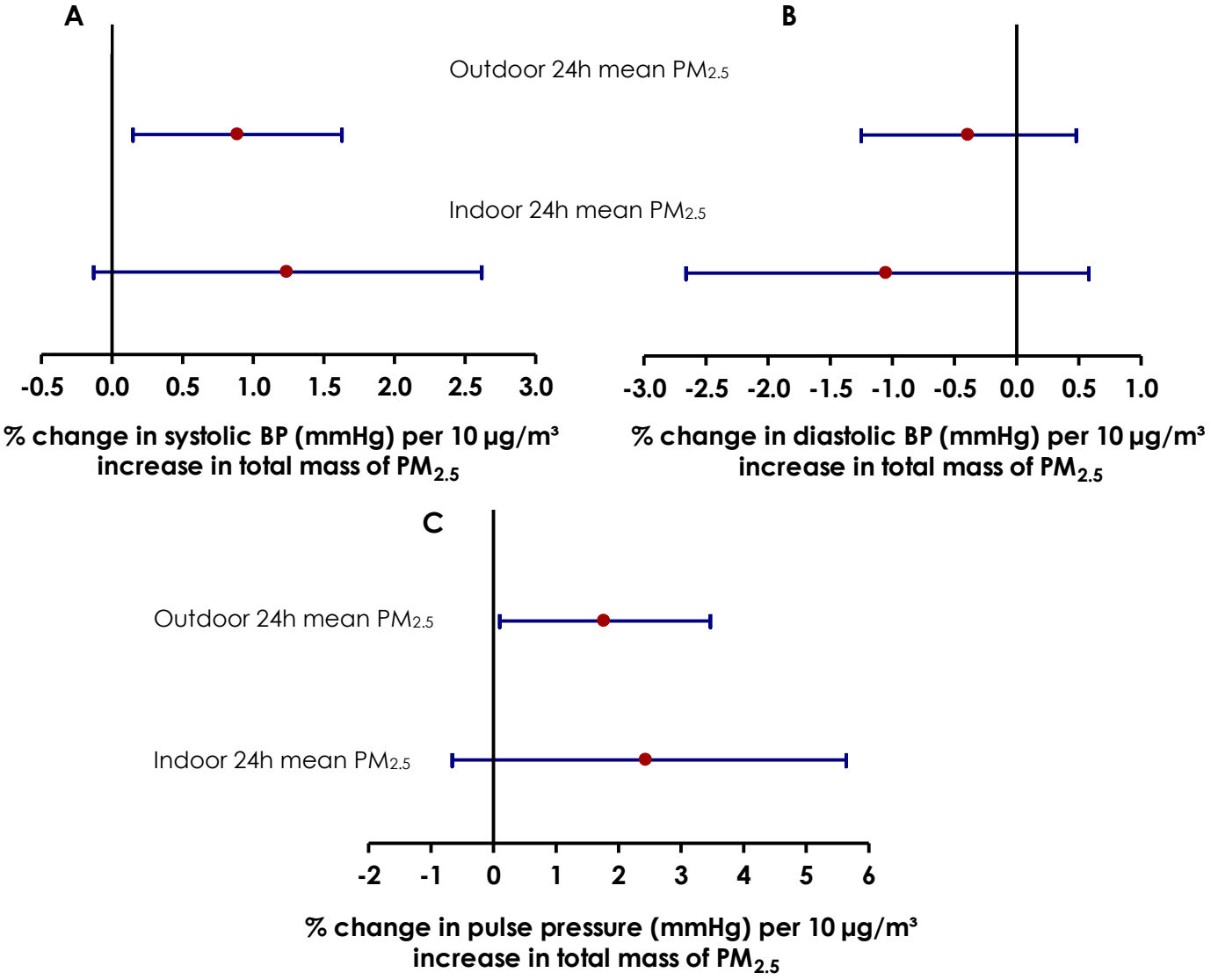

Figure 17: Point Estimates and 95% CI of the percent change in systolic BP (A), diastolic BP (B) or pulse pressure (C) per 10 µg/m³ increase in indoor and outdoor 24 hour mean $PM_{2.5}$

II. Metal composition of $PM_{2.5}$

Potassium (K), iron (F), zinc (Zn) and lead (Pb)content of outdoor $PM_{2.5}$ were significantly associated with systolic blood pressure (Figure 18) and pulse pressure (Figure 19). We did not observe significant associations between the metal content and changes in diastolic blood pressure.

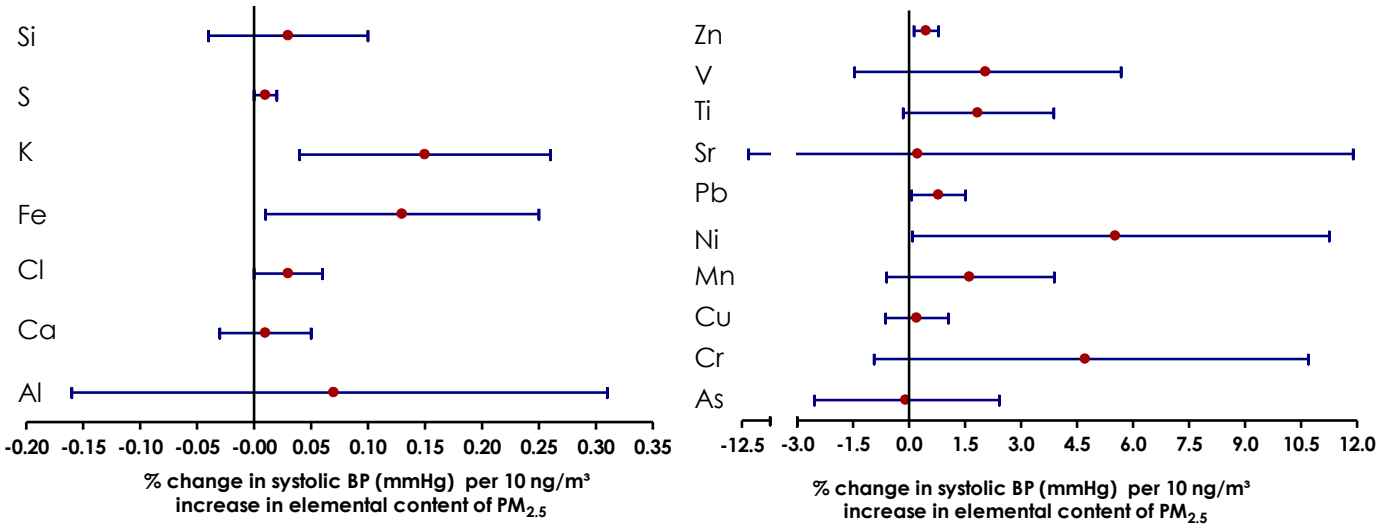

Figure 18: Point Estimates and 95% CI of the percent change in systolic blood pressure per 10 ng/m³ increase in elemental content of outdoor 24 hour mean $PM_{2.5}$.

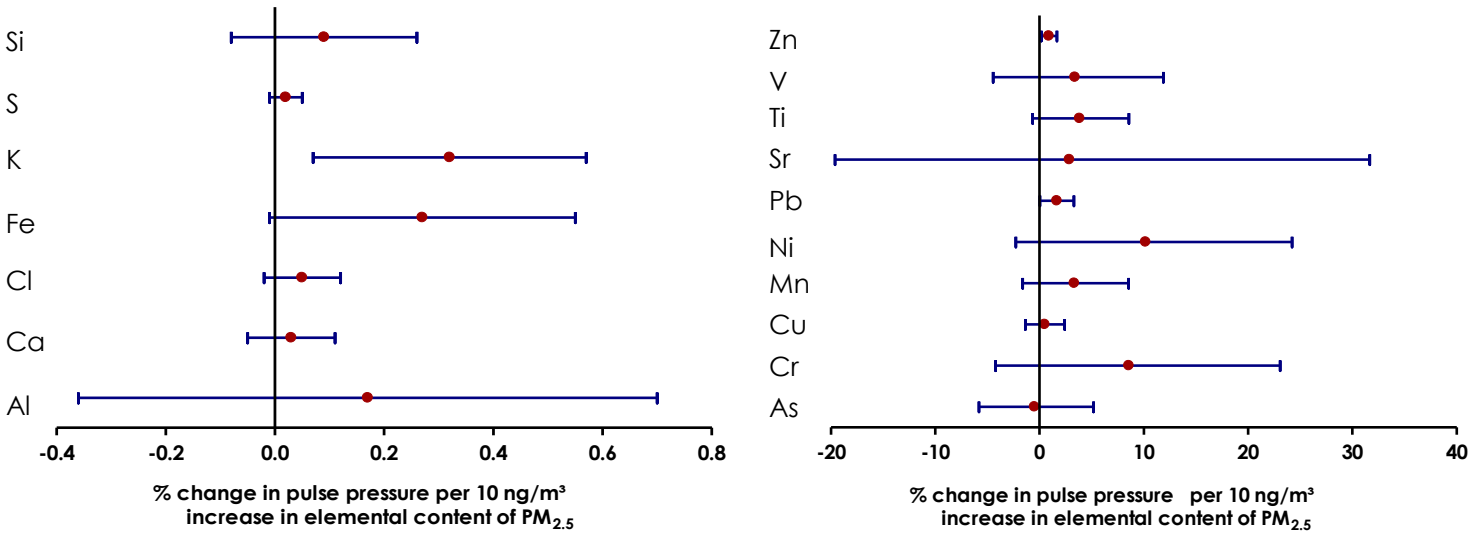

Figure 19: Point Estimates and 95% CI of the percent change in pulse pressure per 10 ng/m³ increase in elemental content of outdoor 24 hour mean $PM_{2.5}$.

III. Oxy-PAHs

The different oxy-PAHs were significantly associated with increases in systolic blood pressure (Figure 20) and pulse pressure (Figure 21), but not with diastolic blood pressure.

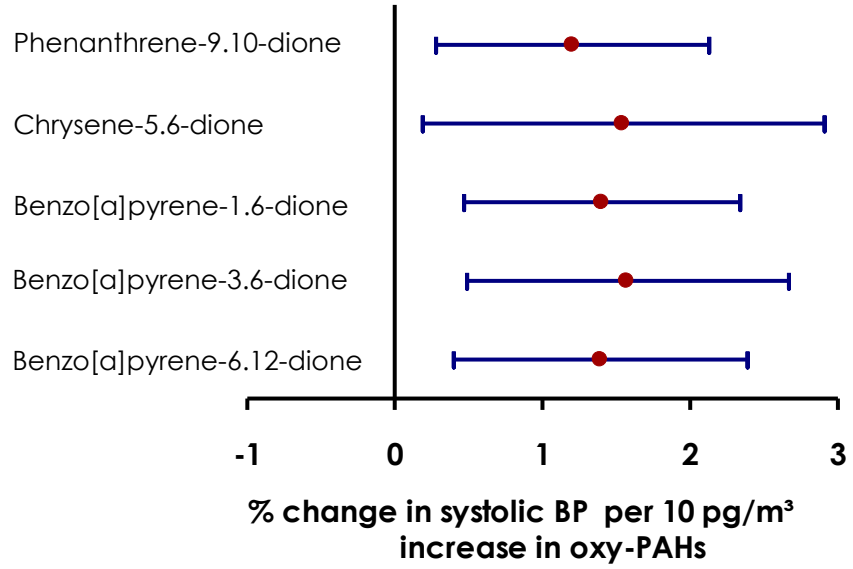

Figure 20: Point Estimates and 95% CI of the percent change in systolic blood pressure per 10 pg/m³ increase in oxy-PAHs of outdoor 24 hour mean PM_{10}

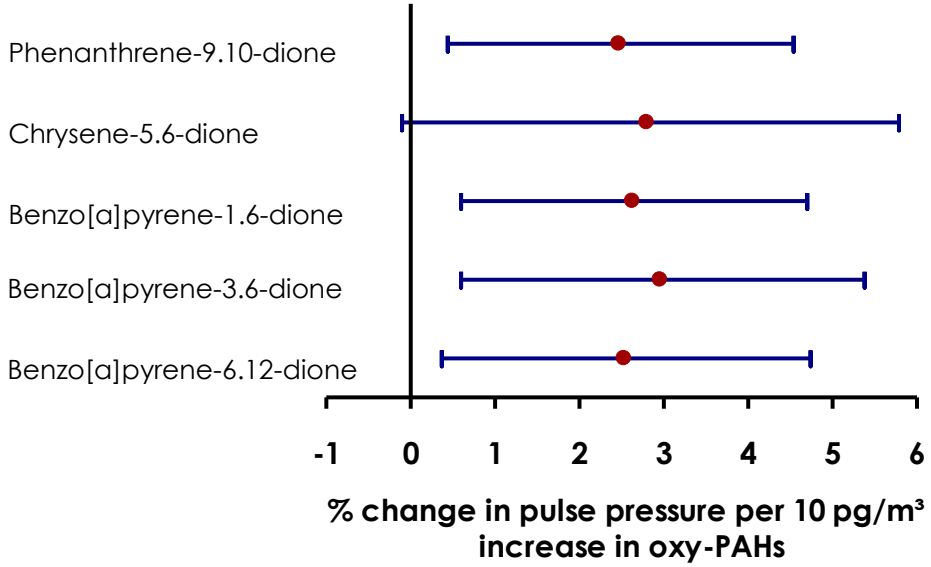

Figure 21: Point Estimates and 95% CI of the percent change in pulse pressure per 10 pg/m³ increase in oxy-PAHs of outdoor 24 hour mean PM_{10}.

2.3.2.3.3 Effects on plasma endothelin-1

I. Gravimetric $PM_{2.5}$

No significant effect of in- or outdoor $PM_{2.5}$ concentrations on serum levels of CC16 were seen (Figure 22).

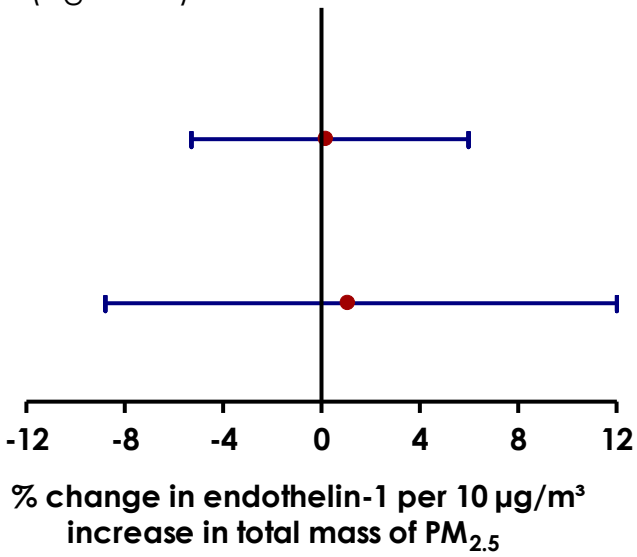

Figure 22: Point Estimates and 95% CI of the percent change in endothelin-1 per 10 µg/m³ increase in indoor and outdoor 24 hour mean $PM_{2.5}$

II. Metal composition of $PM_{2.5}$

Calcium (Ca) and aluminium (Al) content of outdoor 24 h mean $PM_{2.5}$ was associated with changes in plasma level of endothelin-1 (Figure 23).

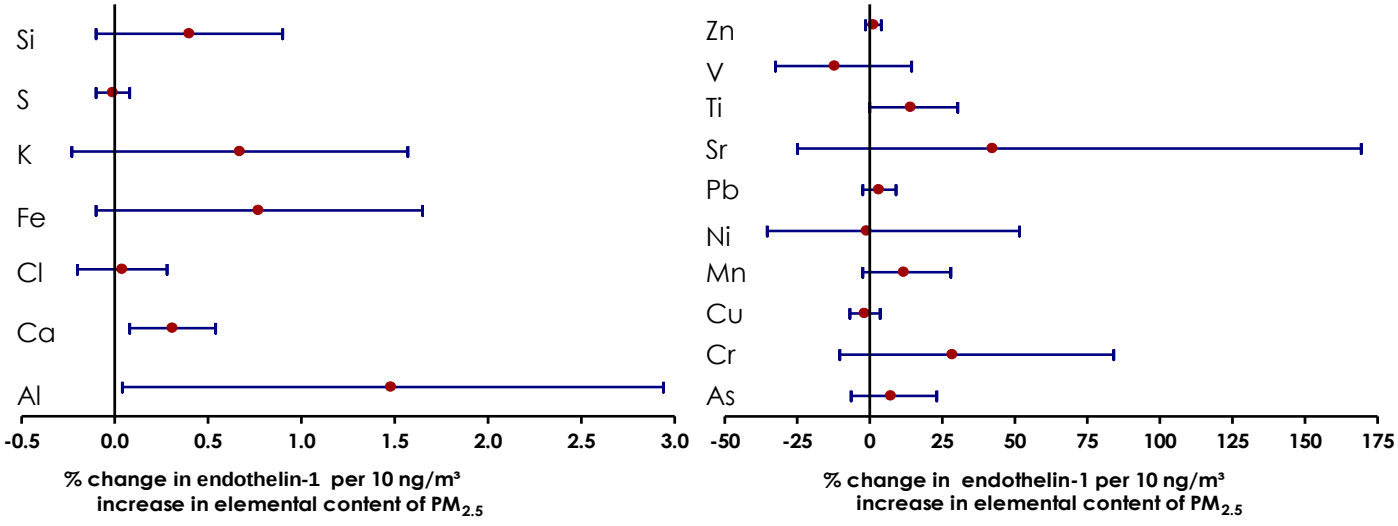

Figure 23: Point Estimates and 95% CI of the percent change in endothelin-1 per 10 ng/m³ increase in elemental content of outdoor 24 hour mean $PM_{2.5}$.

III. Oxy-PAHs

Each increase of 10pg/m³ in benzo[a]pyrene-3.6-dione and in benzo[a]pyrene-6.12-dione was associated with an increase of 8.6% and 8.0% in plasma endothelin-1 concentrations, respectively. The other oxy-PAHs were not significantly associated with white blood cell counts (Figure 24).

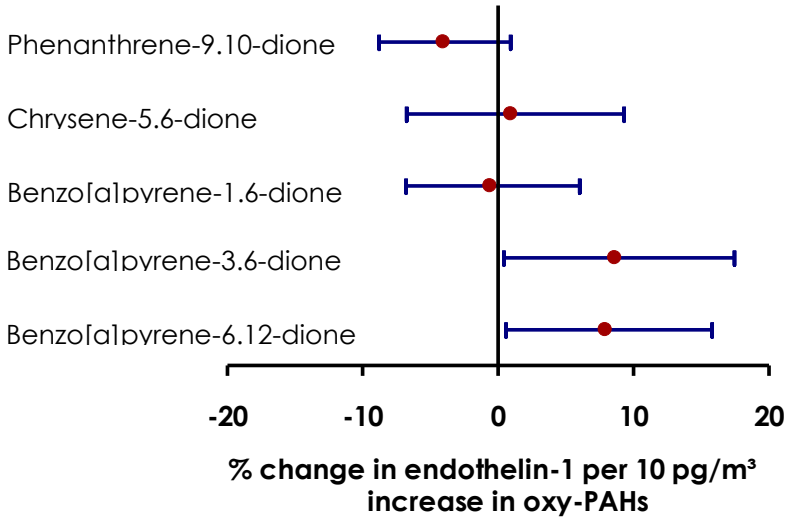

Figure 24: Point estimates and 95% CI of the percent change in endothelin-1 per 10 pg/m³ increase in oxy-PAHs of outdoor 24 hour mean PM_{10}

2.3.2.3.4 Effects on exhaled NO

I. Gravimetric $PM_{2.5}$

$PM_{2.5}$ exposure was associated with increases in exhaled NO, but only reached statistical significance with outdoor concentrations (Figure 25). For each increase in 24 hour means outdoor $PM_{2.5}$ with 10µg/m³, we observed an increase in exhaled NO of 8.6% (95%CI: 2.6 to 14.9%).

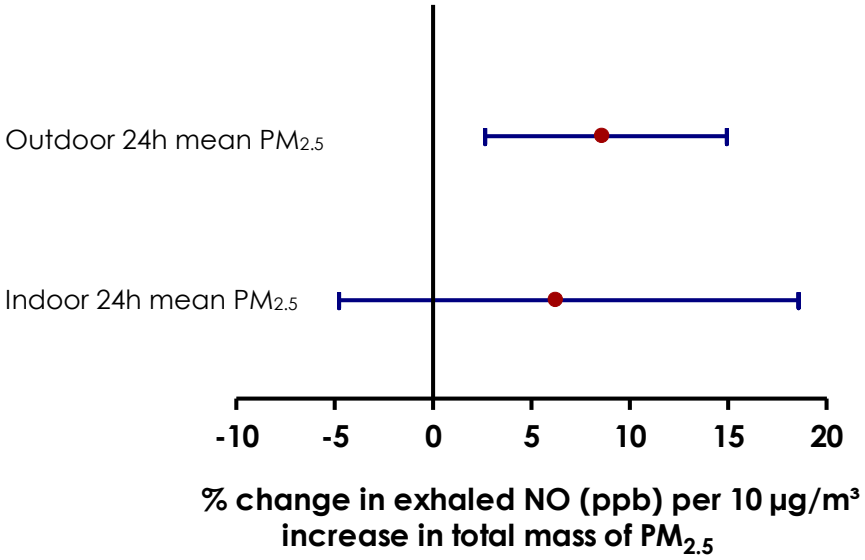

Figure 25: Point Estimates and 95% CI of the percent change in exhaled nitric oxide per 10 µg/m³ increase in indoor and outdoor 24 hour mean $PM_{2.5}$

II. Metal composition of $PM_{2.5}$

Each 10 ng/m³ increase in potassium (K), iron (Fe), chloride (Cl), zinc (Zn), vanadium (V), lead (Pb), nickel (Ni), manganese (Mn), chromium (Cr) and arsenic (As) content of outdoor 24h mean $PM_{2.5}$ was associated with increases in exhaled NO (Figure 26).

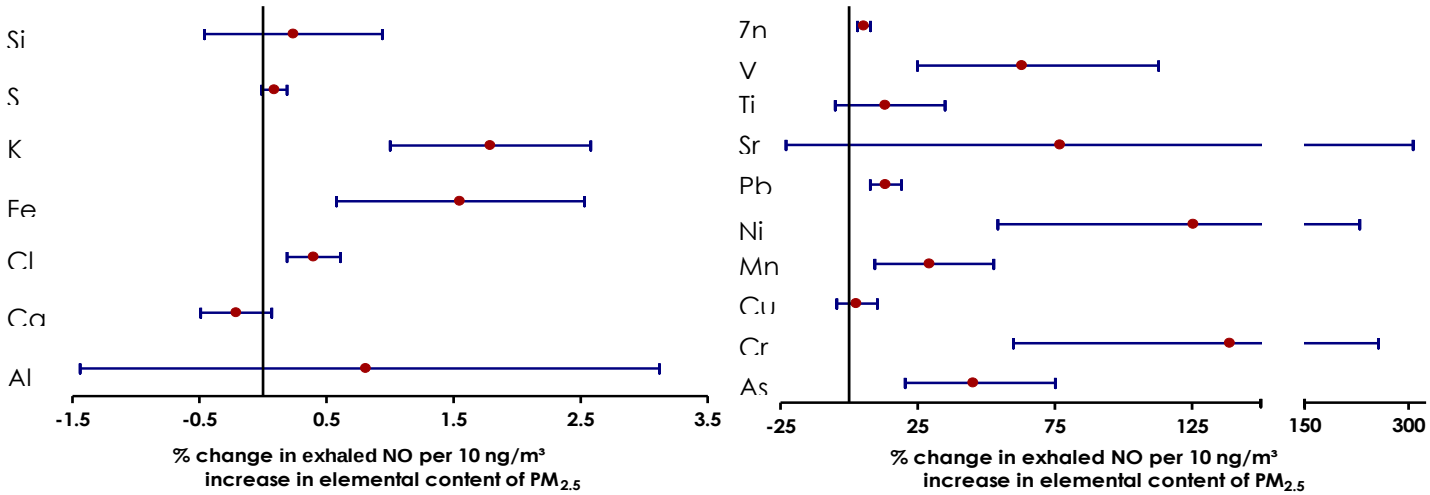

Figure 26: Point Estimates and 95% CI of the percent change in exhaled NO per 10 ng/m³ increase in elemental content of outdoor 24 hour mean $PM_{2.5}$.

III. Oxy-PAHs

Each increase in chrysene-5.6-dione with 10pg/m³ was significantly associated with an increase in exhaled NO of 14.3% (95%CI: 2.9 to 27.0 %) (Figure 27).

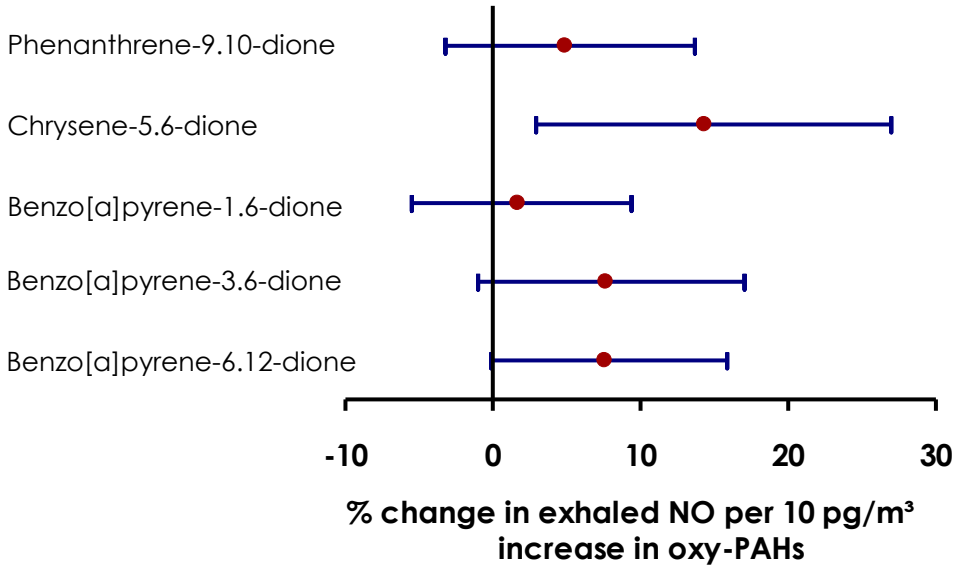

Figure 27: Point estimates and 95% CI of the percent change in exhaled NO per 10 pg/m³ increase in oxy-PAHs of outdoor 24 hour mean PM_{10}

2.3.2.3.5 Effects on Clara cell protein (CC16)

I. Gravimetric $PM_{2.5}$

No significant effect of in- or outdoor $PM_{2.5}$ concentrations on serum levels of CC16 were seen (Figure 28).

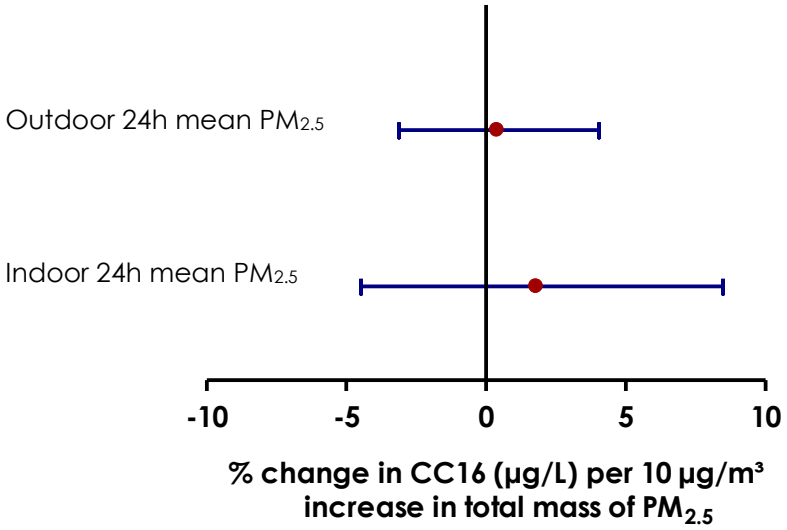

Figure 28: Point Estimates and 95% CI of the percent change in Clara cell protein (µg/L) per 10 µg/m³ increase in indoor and outdoor 24 hour mean $PM_{2.5}$

II. Metal composition of $PM_{2.5}$

Only strontium (Sr) was significantly associated with CC16. Each increase in strontium with 10 ng/m² was associated with a decrease in serum levels of CC16 with 48.9% (95%CI: -69.3 to -14.8%) (Figure 29).

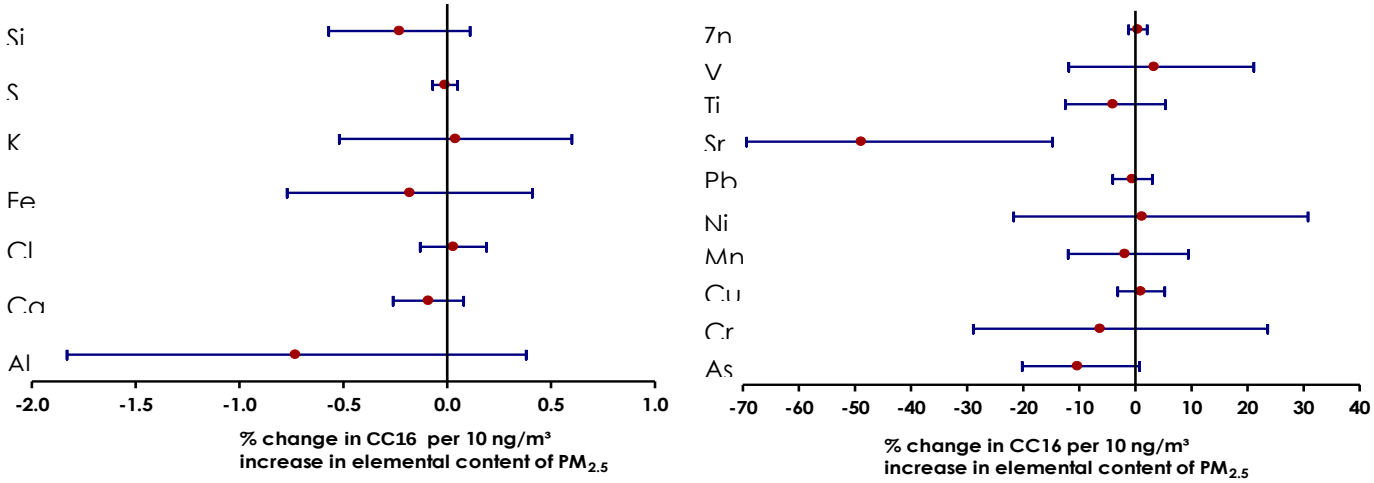

Figure 29: Point Estimates and 95% CI of the percent change in clara cell protein per 10 ng/m³ increase in elemental content of outdoor 24 hour mean $PM_{2.5}$.

III. Oxy-PAHs

None of the oxy-PAHs in PM_{10} was associated with serum levels of CC16 (Figure 30).

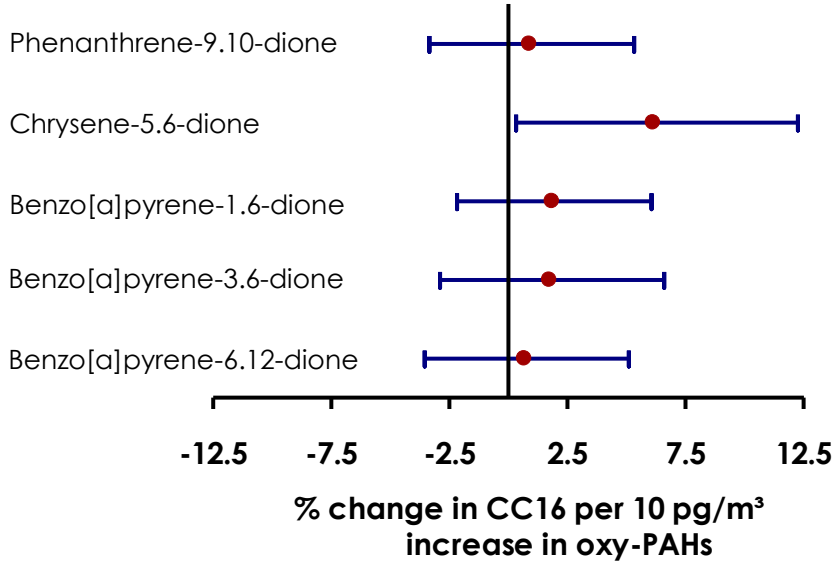

Figure 30: Point estimates and 95% CI of the percent change in CC16 per 10 pg/m³ increase in oxy-PAHs of outdoor 24 hour mean PM_{10}

2.3.2.3.6 Effects on white blood cell count

I. Gravimetric $PM_{2.5}$

No significant effect of in- or outdoor $PM_{2.5}$ concentrations on white blood cell counts were observed (Figure 31).

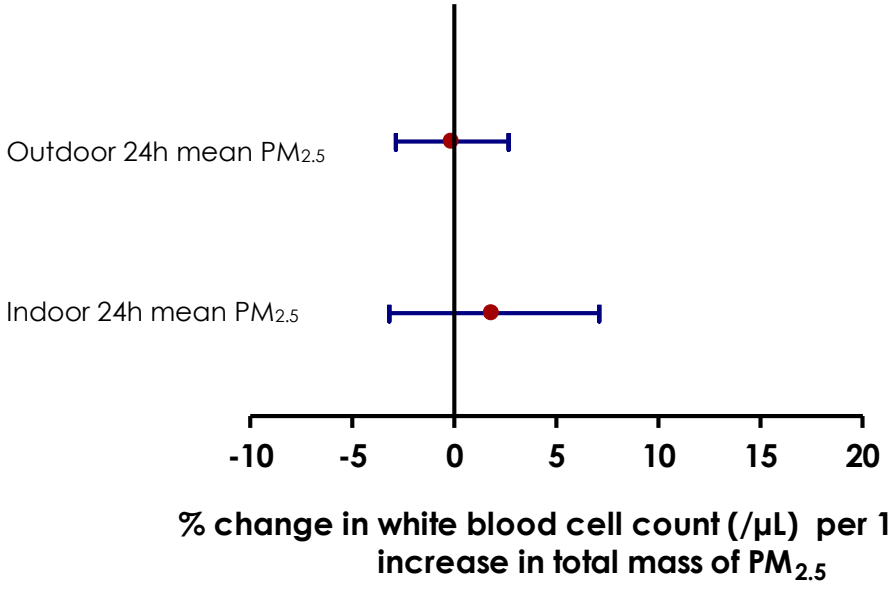

Figure 31: Point Estimates and 95% CI of the percent change in white blood cell count (/µL) per 10 µg/m³ increase in indoor and outdoor 24 hour mean $PM_{2.5}$

II. Metal composition of $PM_{2.5}$

No significant effects of metal composition of outdoor 24 h mean $PM_{2.5}$ on white blood cell counts in the circulation was observed (Figure 32).

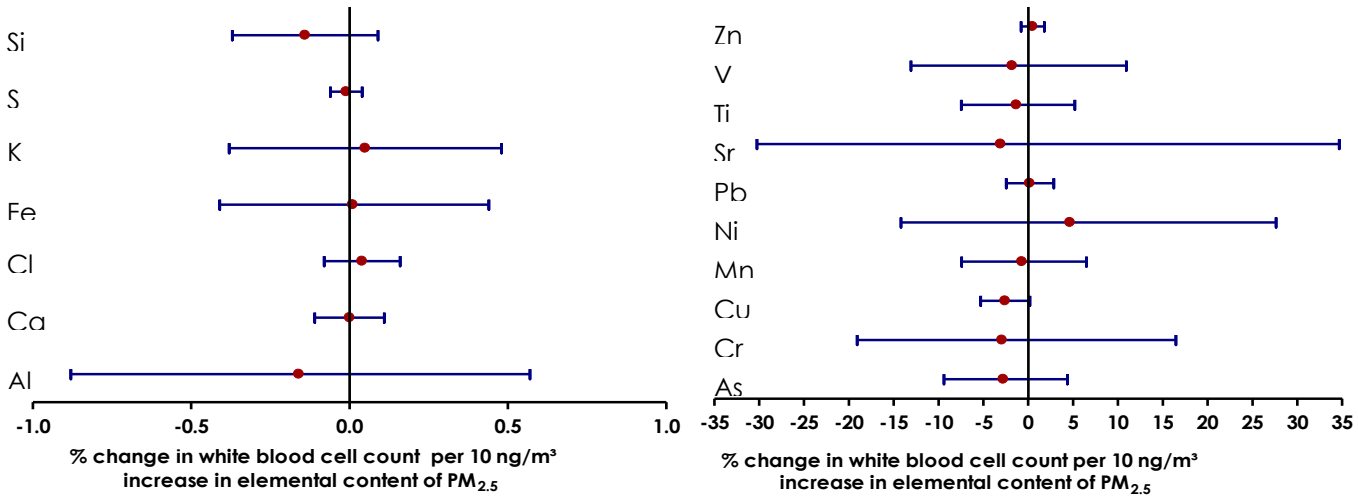

Figure 32: Point Estimates and 95% CI of the percent change in white blood cell counts per 10 ng/m³ increase in elemental content of outdoor 24 hour mean $PM_{2.5}$.

III. Oxy-PAHs

Each increase of 10pg/m³ in benzo[a]pyrene-3.6-dione and in benzo[a]pyrene-6.12-dione was associated with an increase of 3.5% and 3.4% in white blood cell counts, respectively. The other oxy-PAHs were not significantly associated with white blood cell counts (Figure 33).

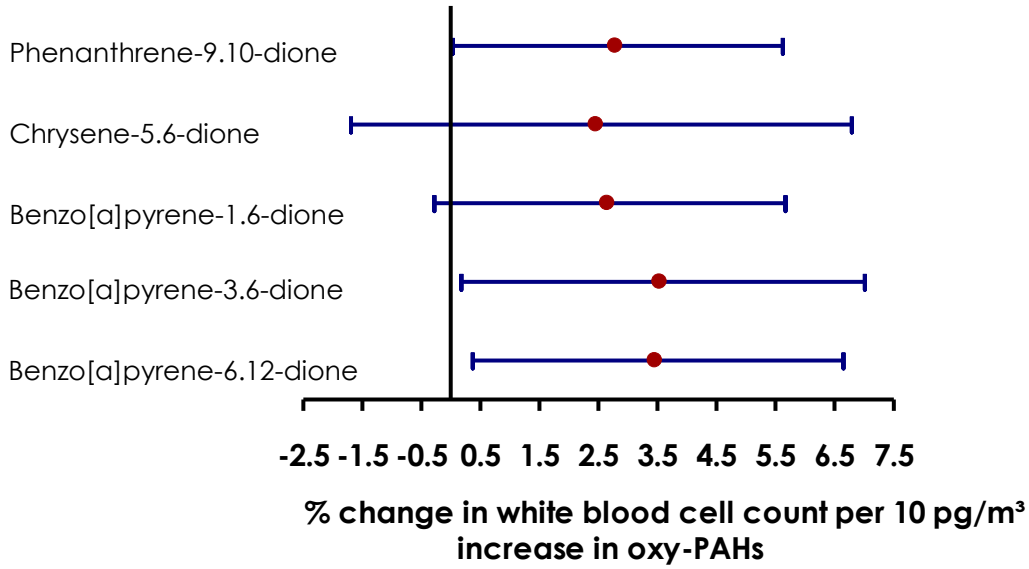

Figure 33: Point estimates and 95% CI of the percent change in white blood cell counts per 10 pg/m³ increase in oxy-PAHs of outdoor 24 hour mean PM_{10}

2.4 Study 4: Acute health effects of particulate air pollution and ozone in children [U.C.Louvain, U.Gent,U.Antwerpen, RMI/KMI, IRCEL]

The objective of this study was to assess the short-term effects of ambient air pollutants and their interactions on the health of children. All measurements are performed on summer days with elevated concentrations of ozone in ambient air or on winter days characterized by high concentrations of particulate matter. During the summer of 2007, from the 16 scouting companies who had accepted to participate to the study, we could perform experiments only on three of them because of poor weather conditions (there was no ozone peak last summer). The three camps that were studied will thus be control groups. These camps were located at Waismes (July 06), Torgny (July 25) and Ramegnies-Chin (August 05). During the winter of 2008, although 8 scouting companies had accepted to participate to second phase of the study for February and March, no campaign could be performed because of absence of pollution by particulate matter on the days the scouts were available.

2.4.1 Methodology

2.4.1.1 Ambient air monitoring (see previous chapter 2.3)

2.4.2 Results

2.4.2.1 Characteristics of the population

Almost all children are Caucasians. Mean age of the participants was 10.2 years (range: 7.5-12.5). TABLE XVIII summarizes their characteristics.

TABLE XVIII: Subjects characteristics

	Waismes	Torgny	Ramegnies-Chin
n	13	16	20
Sex (boys, %)	69.2	50	85
Caucasian (%)	92.3	100	100
Age (mean, years)	10.2	10.1	10.3
Height (mean, m)	1.40	1.41	1.41
Weight (mean, kg)	35.5	36.7	34.5
BMI (mean, kg/m²)	16.8	18.2	17.1
Variation of eNO (ppb)	-0.8	0.6	-2.6
Variation of FEV1 (%)	-0.7	-2.3	2.9

Parameters directly measured during the medical examination display no significant variations between the three groups of children.

2.4.2.2 Exposure levels

2.4.2.2.1 Ambient air pollutants monitoring summer 2007

During these three days, we measured online the ambient ozone concentration and we checked the measurements by comparison with the nearest IRCEL monitoring stations. As shown in Figure 34 the online measure was systematically lower (24 %) than the stations observations. This systematic difference persisted when comparing our measures with that of nearest German and French monitoring stations, indicating indeed that the online measurements of ozone will have to be corrected.

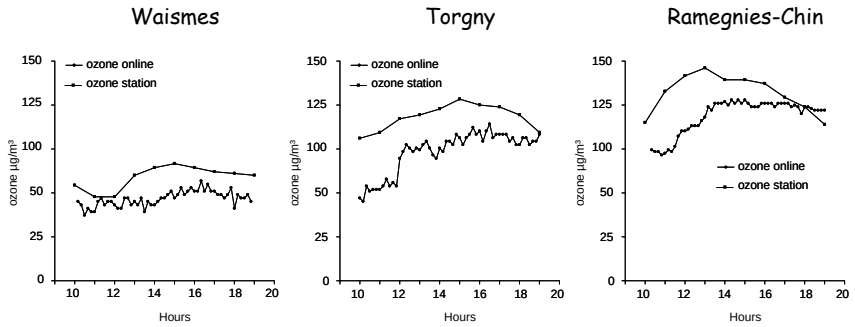

Figure 34: Ambient ozone monitoring

Figure 35 displays the ambient NOx measured by IRCEL stations and of PM concentrations measured online and by IRCEL stations. The concentrations of these pollutants are low during the three studied days.

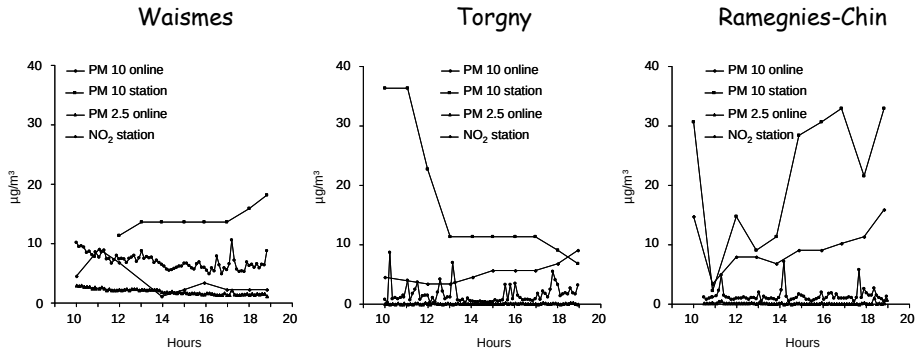

Figure 35: Ambient NOx and PM monitoring

In a cohort of 800 children living in the Southern part of Belgium (lower air pollution concentrations), Clara protein levels were measured and associated with particulate matter to study effects of PM_{10} on lung permeability at low concentrations. There was no evidence of an association between long-term exposure to ambient air pollution, as assessed at each participant's home address and the serum Clara Cell Protein concentration. However, short-term increases in particulate air pollution were associated with Clara cell protein concentration. The effects were strongest taking 7-day averages (Figure 36) of air pollution. This could not be explained by other potential important covariates including age, gender, and exposure to environmental tobacco smoke. These findings suggest that acute environmental exposures influence the integrity of the lung epithelium and lead to increased epithelial barrier permeability in the lungs of children.

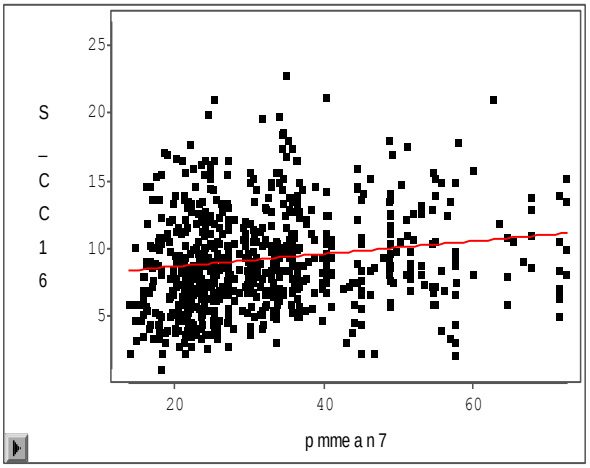

Figure 36: Correlation between Serum Clara protein and daily mean particulate matter (7 days)

2.4.2.2.2 Determination of the total mass concentration of particulate matter

In the case of summer camps TSP concentrations were the following: 11, 10 and 37 µg/m3 in Waimes-Malmedy, Torgny-Virton and Ramegnies-Chin, respectively. $PM_{2.5}$ levels were 5-7, 6-7 and 26-30 µg/m3 at the same sites.

2.4.2.2.3 Determination of the inorganic water soluble fraction of particulate matter

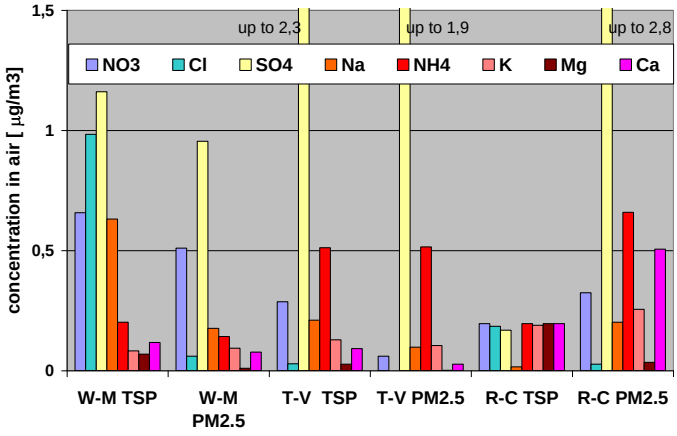

Figure 37: Concentration of water soluble ions in TSP and $PM_{2.5}$ collected in the summer camp sites

The composition of water soluble fraction in TSP and $PM_{2.5}$ collected in 3 sites of children summer camps is presented in Figure 37. Most ions were detected in low amounts, only sulphates were slightly elevated.

2.4.2.2.4 Determination of the total elemental composition in the bulk samples

Elemental composition of TSP and $PM_{2.5}$ implied mostly crustal elements such as Si, K, Al, Ca, Fe. Sulphur was present at relatively high concentrations: 0.3, 0.5 and 0.7 µg/m3 ($PM_{2.5}$) in Waimes-Malmedy, Torgny-Virton and Ramegnies-Chin, respectively. Among hazardous elements, As level was relatively low (2-3 ng/m3) at all sites, while the Pb amount varied significantly among the sites – in the first and second campaign, the Pb content did not exceed 3 ng/m3, but was elevated at the third site (15 ng/m3 in TSP, 10 ng/m3 in $PM_{2.5}$).

2.4.2.2.5 VOC concentrations at the summercamp locations

Concentration levels of the individual compounds measured during the summer camps are given in TABLE XIX as a concentration range.

TABLE XIX: Outdoor VOC concentration levels (µg/m³) measured during the summercamps in Torgney, Ramegnie-Chin and Waismes.

	Torgney (n=4)		Ramegnie-chin (n=4)		Waismes (n=3)	
	min	max	min	max	min	max
ethyl acetate	0.25	0.44	0.28	0.39	D	
benzene	0.12	0.22	0.32	0.68	0.01	0.10
trichloroethylene	ND		17.71	17.71	0.81	14.97
n-heptane	0.21	0.36	0.21	0.21	0.06	0.14
toluene	0.68	23.65	ND		ND	
hexanal	5.24	5.56	6.65	8.09	2.14	2.14
n-octane	0.20	0.39	0.22	0.41	0.08	0.17
tetrachloroethylene	0.04	0.08	0.09	0.11	0.02	0.02
ethylbenzene	0.10	0.16	0.16	0.27	0.03	0.03
p-xylene	0.20	0.41	0.28	0.55	0.05	0.07
styrene	D		0.15	0.15	0.03	0.03
phenol	0.46	1.52	1.32	2.89	0.20	0.43
heptaldehyde	2.60	2.60	2.51	3.96	1.06	1.06
n-nonane	0.52	1.23	0.65	1.05	0.13	0.28
alpha-pinene	0.34	0.59	0.13	0.19	0.14	0.20
benzaldehyde	1.18	3.06	3.14	4.24	0.82	1.27
propylbenzene	0.08	0.11	0.09	0.12	ND	
n-decane	0.72	0.98	1.38	2.03	D	
limonene	0.32	4.75	D		0.14	0.21
n-undecane	D		1.14	1.75	ND	
n-dodecaan	D		0.81	0.81	ND	

The TVOC concentration ranged between 3 and 29 µg/m³, being in the same range as the concentration levels measured in the elderly home gardens.

Box-plots of the TVOC concentration levels are given in Figure 38, showing clearly that median values of TVOC concentrations were up to a factor of six to seven higher in the indoor environment from elderly homes compared to the outdoor environment (both in the gardens of the elderly homes and on the summer camps).

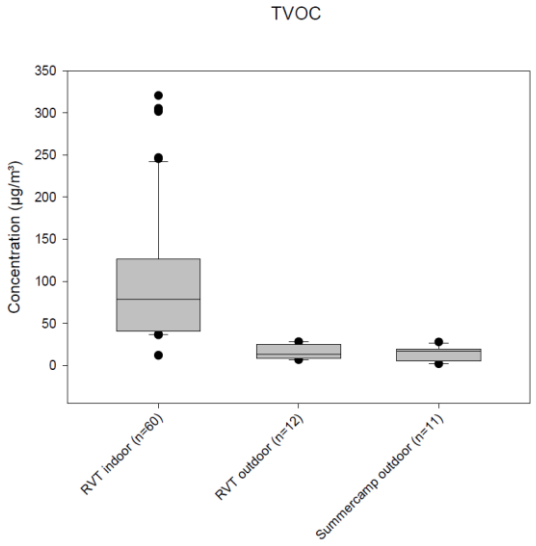

Figure 38: Box plots representing the overall TVOC-concentration levels observed in the indoor environment (elderly home, RVT) and outdoor environments (both in the garden of the elderly home and at the summer camps)

2.5 Study 5: Associations between infant mortality and air pollution in an affluent society [K.U.Leuven]

Numerous studies show associations between mortality in adults and PM_{10}. Recently, there has been growing concern about a possible association between exposure to air pollution and infant mortality as well. Infants may be particularly vulnerable to PM_{10}, since their lungs and immune system are immature.

Following a suggestion made by the referees of the interim evaluation, we collected data on infant mortality in Belgium (1998 – 2006) and we used the case-crossover design in order to investigate short-term effects of elevated levels of PM_{10} on daily infant mortality (< 1 year of age).

In addition, we assessed the shape of the association between PM_{10} and infant mortality and estimated whether the EU limit value of 50 µg/m³, which is frequently exceeded in our country, is protective.

2.5.1 Methodology

Data on mortality in infants (< 1 year of age), air pollution and daily temperatures were collected during the period 1998-2006. Mortality data were anonymous, but the following information was provided: date of death; postal code of municipality of residence; official cause of death (ICD-10 code); mature or premature at birth (with 37 weeks of gestation as cut-off value); age class (early neonatal (≤ 7 days of age), late neonatal (8 to 28 days of age) or postneonatal (29 to 365 days of age)). The administration of detailed mortality statistics is a regional affair and each region has its own policy concerning privacy. As a consequence, we were only able to obtain data on infant mortality in the Region of Flanders from the Flemish Agency for Care and Health. Whenever mortality data can be acquired from Brussels and Wallonia as well, our study can easily be extended to these regions.

Exposure to ambient PM was determined as daily average PM_{10} concentration at the infant's residence (municipality level), obtained by a land use interpolation model (Janssen et al. 2008) which uses data from the IRCEL network of measurement stations.

Temperature is a known confounder of the association between air pollution and mortality and therefore, we obtained daily average temperatures for the whole study period in Uccle from the KMI/RMI.

In the case-crossover design, each subject serves as its own control and therefore, confounding by individual characteristics is not possible. Case-crossover is now widely used for analysing short-term health effects of air pollution. Control days for each case were generated by applying a time-stratified design in order to minimise statistical bias (Janes et al. 2005) and their daily average temperature had to be within 2°C of that on the event day in order to control for confounding by variation in daily temperature (Figure 39). Conditional logistic regression was used for parameter estimation and calculation of odds ratios (OR) and confidence intervals (CI).

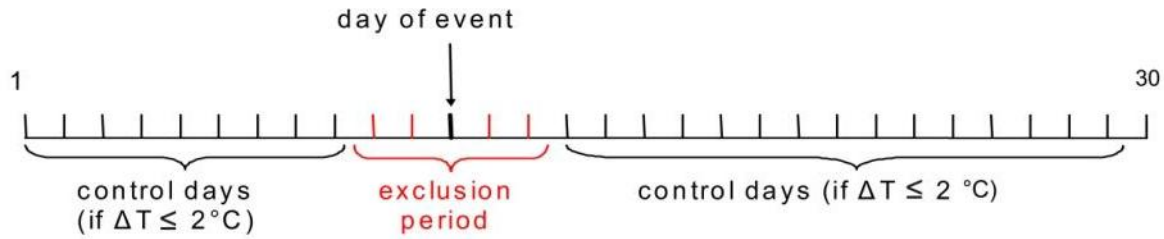

Figure 39: Time-stratified case-crossover design. Control days were taken from the same calendar month as the event day, with a ± 2 days exclusion period in order to correct for short term autocorrelation and a match on temperature between event and control.

In order to detect a possible threshold level in the exposure-response relationship, we studied the shape of the association between PM_{10} and risk of death by the use of fractional polynomials. From this family of models, the best functional form was chosen using Akaike's Information Criterion (AIC).
Possible short-term delays in the effects of exposure to PM_{10} were investigated by performing five additional case-crossover analyses with different lag structures: 1, 2 or 3 days before the day of death, or the moving average of 2 (day of event and lag day 1) or 3 (event day, lag day 1 and lag day 2) consecutive days.

We conducted stratified analyses by age class, maturity and cause of death, classified as cardiorespiratory diseases (ICD-10 I00-J99), SIDS (R95), perinatal circumstances (P00-P96), congenital and chromosomal malformations (Q00-Q99) or other. Infants who died from external causes (e.g. accidents, V00-Y98, N=73) were excluded from all analyses.

Finally, we transformed the exposure value into a binary variable (i.e. below or above the EU limit value of 50 µg/m³) and calculated the ORs for dying on days above 50 µg/m³ compared to days with PM_{10} levels below that value.
All analyses were performed in SAS, version 9.1, and all tests were two-sided with $\alpha = 0.05$

2.5.2 Results

2.5.2.1 Descriptive Data

2382 infants died from non-traumatic causes during the study period (1998-2006) and 1284 infants (54%) had been born before 37 weeks of gestation. Figures on age at death and causes of death are provided in TABLE XX.

During the study period, PM_{10} concentration averaged 31.9 µg/m³ (SD 13.8) (Figure 40A) and there were 321 days (an average of 35.7 days per year) with a mean daily concentration (population-weighted daily average for the whole region) exceeding the current EU limit value of 50 µg/m³. The EU limit value may be exceeded 35 days per year at most, so during the study period, this standard was hardly met in the northern part of the country as a whole, and not at all in a number of urban and industrial regions (orange to red coloured areas in Figure 40B)

TABLE XX. Non-traumatic causes of death in neonates in the study region (1998-2006), by age class.

Cause of death (ICD-10 code)	Early neo-natal (≤ 7 days of age)	Late neonatal (8 to 28 days of age)	Postneonatal (29 to 365 days of age)	Total
Cardiorespiratory diseases (I00 – I99)	3	3	44	50
Perinatal circum-stances (P00 – P96)	771	197	126	1094
Congenital and chromosomal abnor-malities (Q00 – Q99)	398	140	205	743
SIDS (R95)	0	0	285	285
Others	24	22	164	210
Total	1196	372	814	2382

For cases (N=2382), the average exposure was 32.6 µg/m³ (SD 15.2) and on the selected control days (N=20448) PM_{10} averaged 30.7 µg/m³ (SD 13.6). This difference suggests that there might indeed be a pattern of more cases of infant mortality on days with elevated ambient PM_{10} levels, but it has to be proven formally by a correct statistical approach, such as the case-crossover model.

2.5.2.2 Odds ratios per 10 µg/m³ increment in PM_{10} concentration

For the whole group, we found a 4% increase (95% CI: 0-8 %, P = 0.045) in the risk of death for each 10 µg/m³ increase in the concentration of PM_{10} on the event day (lag day 0) (TABLE XXI).

In the sensitivity analyses with up to three lag days or moving average concentrations, mortality tended to be positively associated with PM_{10} as well, but these associations were not significant (data not shown). Therefore, we further report only results for exposure to PM_{10} on the event day, not on the days proceeding the day of death.

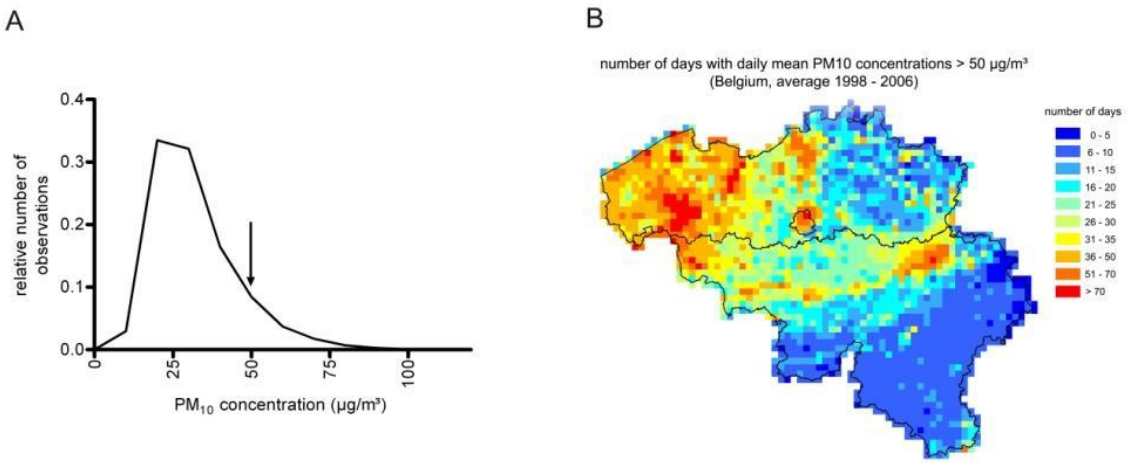

Figure 40. A) Frequency distribution of population-weighted daily mean PM_{10}-concentrations in the Region of Flanders (Belgium) during the study period (1998-2006). The arrow indicates the EU limit that may be exceeded up to 35 days per year. B) Spatial distribution of population-weighted daily mean PM_{10}-concentration, expressed as number of days with concentration > 50 µg/m³ .

Stratification by age class revealed stronger associations with deaths between two to four weeks of age (late neonates) than with deaths during other time periods. Specifically, a 10 µg/m³ increase in mean daily PM_{10} on the event day was associated with an 11% increase (1- 22%, P = 0.028) in the risk of late neonatal death. In contrast, we found no evidence of effects of PM_{10} on early neonatal or postneonatal mortality. Stratified analyses for preterm and term births revealed that odds ratios were always higher for the latter group (which was a little unexpected, since on average, term born children are considered healthier and less susceptible for air pollution effects then preterm born children), but these differences were never significant (P's for interaction ≥ 0.09, Table XXI).

Table XXI. Risk of infant death associated with a 10 µg/m³ increase in PM_{10} on the event day and with ambient PM_{10} concentrations above 50 µg/m³, stratified by age category.

Age category	All (N = 2382)	Preterm (N = 1284)	Term (N = 1086)	*P* inter-action[a]
OR for 10 µg/m³ increase in PM_{10} on event day				
All	1.04 (1.00 to 1.08)*	1.03 (0.98 to 1.08)	1.05 (0.99 to 1.11)	0.62
Early neonatal	1.04 (0.99 to 1.10)	1.03 (0.96 to 1.10)	1.07 (0.97 to 1.19)	0.49
Late neonatal	1.11 (1.01 to 1.22)*	1.10 (0.97 to 1.24)	1.13 (0.98 to 1.31)	0.77
Post neonatal	1.01 (0.95 to 1.07)	0.99 (0.88 to 1.10)	1.02 (0.94 to 1.10)	0.67
OR for days above 50 µg/m³ vs. days below 50 µg/m³ [b]				
All	1.10 (0.94 to 1.29)	0.96 (0.76 to 1.20)	1.27 (1.01 to 1.61)*	0.09
Early neonatal	0.99 (0.78 to 1.24)	0.92 (0.69 to 1.22)	1.14 (0.75 to 1.74)	0.40
Late neonatal	1.74 (1.18 to 2.58)**	1.47 (0.87 to 2.48)	2.09 (1.15 to 3.79)*	0.38
Post neonatal	1.04 (0.79 to 1.37)	0.74 (0.43 to 1.27)	1.18 (0.86 to 1.63)	0.14

Data are ORs with 95% CI. * $P \leq 0.05$ and ** $P \leq 0.01$.
[a] Interaction between exposure and maturity at birth, with preterm birth defined as born before 37 weeks of gestation
[b] Based on EU limit value.

We further stratified our analysis by cause of death (TABLE XXII), since we were particularly interested in the relation between air pollution and mortality according to cardiorespiratory diseases or SIDS. However, we found no significant results in these mortality classes. For the cardiorespiratory diseases, sample size was very low (see TABLE XX). In contrast, PM_{10} and mortality were significantly associated in cases where the cause of death was determined as 'perinatal circumstances' (age classes pooled and early neonates) or 'congenital and chromosomal abnormalities' (late neonates).

2.5.2.3 Odds Ratios for days above vs. below the EU limit value

Analyses in which the EU limit value of 50 µg/m³ was taken as a cut-off point revealed a non-significant OR for the whole group, but a highly significant result for late neonatal mortality with an OR for dying on days with PM_{10} above 50 µg/m³ of 1.74 (1.18-2.58; $P = 0.006$), compared to days below the cut-off value (Table XXI).

The corresponding Attributable Fraction (calculated as follows: AF = (OR – 1) / OR) was 43% (15-61%). Assuming causality, this means that on days above 50 µg/m³, 43% of late neonatal mortality could be triggered by an acute increase in fine particulate air pollution levels on the same day.

Table XXII. Risk of infant death associated with a 10 µg/m³ increase in PM_{10} on the event day and with ambient PM_{10} concentrations above 50 µg/m³, stratified by cause of death.

Cause of death (ICD-10)	All	Early neonatal	Late neonatal	Post-neonatal
	OR for 10 µg/m³ increase in PM_{10} on event day			
Total	1.04 (1.00 to 1.08)*	1.04 (0.99 to 1.10)	1.11 (1.01 to 1.22)*	1.01 (0.95 to 1.07)
Cardiorespiratory diseases	0.98 (0.78 to 1.25)	na	na	0.98 (0.76 to 1.26)
Perinatal circum-stances	1.06 (1.00 to 1.12)*	1.06 (1.00 to 1.14)*	1.09 (0.95 to 1.25)	1.01 (0.86 to 1.19)
Congenital and chromosomal abnormalities	1.04 0.97 to 1.12)	1.00 (0.91 to 1.11)	1.16 (1.00 to 1.35)*	1.04 (0.90 to 1.20)
SIDS	0.99 (0.89 to 1.09)	na	na	0.99 (0.89 to 1.09)
	OR for days above 50 µg/m³ vs. days below 50 µg/m³ [a]			
Total	1.10 (0.94 to 1.29)	0.99 (0.78 to 1.24)	1.74 (1.18 to 2.58)**	1.04 (0.79 to 1.37)
Cardiorespiratory diseases	0.80 (0.28 to 2.25)	na	na	0.93 (0.32 to 2.71)
Perinatal circum-stances	1.00 (0.78 to 1.28)	0.96 (0.72 to 1.29)	1.36 (0.77 to 2.38)	0.77 (0.34 to 1.72)
Congenital and chromosomal abnormalities	1.30 (0.98 to 1.74)	1.03 (0.69 to 1.54)	2.32 (1.24 to 4.34) **	1.38 (0.78 to 2.43)
SIDS	0.94 (0.60 to 1.48)	na	na	0.88 (0.55 to 1.41)

Data are ORs with 95% CI. * $P \leq 0.05$ and ** $P \leq 0.01$.
na: not applicable due to low numbers in the specified age class.
[a] Based on EU limit value.

When we stratified the analysis by cause of death, we obtained a highly significant result for 'congenital and chromosomal abnormalities' in the late neonatal group (P=0.009), but again, there was no evidence for an increased rate of SIDS-related deaths associated to air pollution (TABLE XXII). Subanalyses of congenital malformations of the circulatory or respiratory system (Q20-Q28 and Q30-Q34, respectively) revealed similar ORs as in the whole group of congenital and chromosomal abnormalities (Q00-Q99), but due to a smaller sample size, these results did not reach statistical significance.

For both approaches (continuous with 10 µg/m³ increments and binary with values below or above 50 µg/m³), we only found significant results in the age class of late neonates (8 to 28 days of age). Reasons for this might be that the most susceptible children die during the first week of life due to conditions that do not need to be triggered by air pollution, or that the measured outdoor air pollution does not reflect actual exposure during the first week of life (or during the first month for premature infants), because most of these new-borns probably would have remained in the hospital during this time. However, we had no access to data on the duration of hospitalization after birth to verify this hypothesis. The absence of a link between air pollution and postneonatal death (28 to 365 days of age) is in accordance with other recent case-crossover studies on infants (Son et al. 2008).

Nevertheless, our data suggest that air pollution may precipitate death in neonatal infants with pre-existing conditions such as chromosomal and congenital malformations (in cardiovascular, respiratory or other organs).

2.5.2.4 Shape of the association

Because the strongest associations between air pollution and mortality were found in the group of late neonates, we further analysed the shape of the association in this age class. Fractional polynomial analysis revealed that a linear model adequately describes the relationship between infant mortality and air pollution, with no evidence for a threshold or a plateau (Likelihood Ratio test for a linear model versus a null model, P = 0.030) (Figure 41). More complex fractional polynomials did not significantly improve the fit of the model, according to AIC.

The linear shape of the association suggests that the risk of death exists even below 50 µg/m³ and that more stringent EU standards on PM air pollution would proportionally reduce the number of PM-triggered deaths. Limit values do not only concern the number of exceedings per year (now 35 days), but also the maximal yearly average (now 40 µg/m³). The WHO proposes a maximum of 3 days per year above 50 µg/m³ and a yearly average below 20 µg/m³. The argument that it is difficult to meet standards in densely populated areas ignores the fact that the importance of a factor with respect to public health increases in proportion to the number of people that are exposed to it.

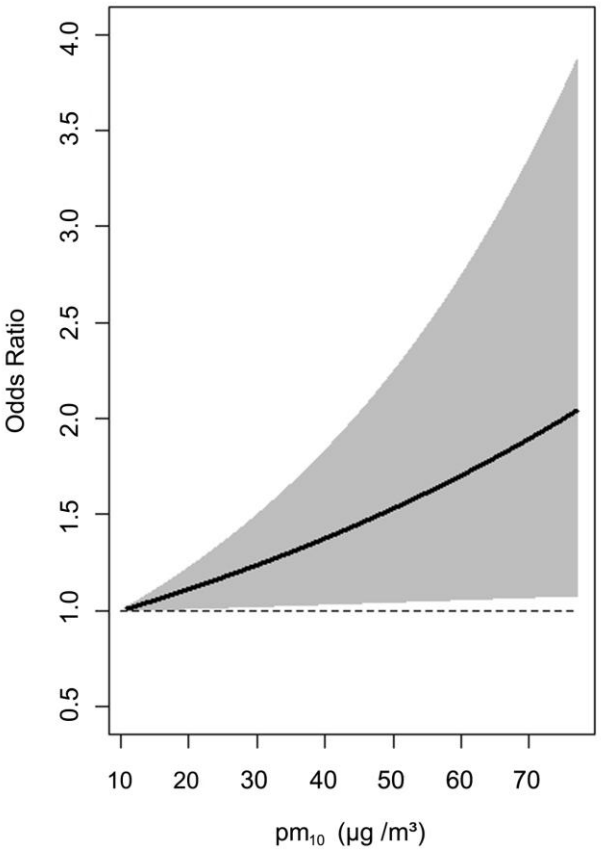

Figure 41: Shape of the association between exposure to PM_{10} and risk of mortality in late neonates, expressed as estimated OR (with 95% CI, the grey area), using fractional polynomials and 10 µg/m³ as reference; 77 µg/m³ is the 99th percentile of exposures during the study period

2.6 Study 6: Subclinical responses in healthy cyclists briefly exposed to traffic-related air pollution

Within the framework of the PM²TEN cluster project[2], members of SHAPES and PARHEALTH jointed forces to set up a field measurement campaign to investigate if cycling near a busy road would induce changes in biomarkers of pulmonary and systematic inflammation. In a controlled experiment, physically fit, non-asthmatic subjects cycled during two exposure scenarios: near a major bypass road with busy traffic (road test) and in a room with filtered air (clean room).

2.6.1 Methodology

2.6.1.1 Study population

All subjects taking part in the 'SHAPES injury surveillance system' (Int Panis L. et al. 2010) who had filled out at least two electronic diaries (n=1048), received an e-mail asking if they were willing to participate in the field tests. Briefly, these participants had been previously asked to fill out questionnaires about bicycle-related traffic accidents and also to fill out a self-reported electronic diary for one year with details about travel frequency, time spent cycling and distance travelled. The inclusion criteria were: (1) age between 18-65 years; (2) having a paid job outside the home; (3) cycling to work at least twice a week; (4) living in Belgium. Two hundred eighty-one subjects were willing to participate. Of these 281 responders, after excluding smokers and those on anti-platelet therapy, 41 were chosen in chronological order and contacted personally by phone. Of the 41 recruited subjects, 38 took part in the experiments at both locations. The Ethics Review Board of the Medical Faculty of the Vrije Universiteit Brussel (VUB) approved the study. All subjects gave a written informed consent.

2.6.1.2 Study design

Subjects performed two exercise trials during two exposure scenarios: cycling on a cycling track near a major bypass road in Antwerp (road test) and cycling in a room with filtered air (clean room), 12 to 29 days apart. The road tests were carried out over a five day period (from 4 May 2009 until 8 May 2009) with a mean of eight participants per day.

[2] http://www.belspo.be/belspo/fedra/proj.asp?l=fr&COD=PS/05

The tests in the clean room were done on 11 days (between 18 May 2009 and 3 June 2009), with a mean of three participants per day. Test were carried out from 8 am until 17 pm. Persons were asked to come by train and had to walk 1200 meters from the train station to the location in Antwerp (road test) and 700meters to the location in Brussels (clean room test).

We measured the anthropometric characteristics of the participants and administered a questionnaire to collect information about their lifestyle and medication intake. A venous blood sample was drawn and exhaled NO was measured, before the exercise. After the exercise, participants rested for 30 minutes in a seated position followed by the post-cycling examination, which included exhaled NO measurement and a venous blood sample collection.

I. Cycling near a major bypass road (road test)

Participants cycled a pre-selected route in Antwerp on a dedicated cycling path parallel to a major bypass road (a very busy 10 lane motorway with up to 200,000 vehicles per day and a major flow of heavy duty diesel vehicles). The total trajectory is 5750 meters long and mostly situated between 10 and 100 meters from the edge of the motorway. The cycling path also passes over the motorway on a dedicated bridge. Participants were asked to cycle at about the same intensity and speed as their everyday cycling to and from work. They carried a heart rate monitor (Polar X-Trainer Plus, Polar Electro OY, Kempele, Finland) and the bicycle was equipped with devices to measure exposure to particles (GRIMM and P-TRAK) and a GPS.

II. Cycling in a room with filtered air (clean room)

To create a 'clean room', three devices were used simultaneously and continuously (i.e. 24 hours a day) during the whole testing period, in a laboratory located, in Brussels, at the VUB. The Bionaire® Mini Tower air purifier (The Holmes Group, Inc., Milford, USA) was used to purify the air from particles down to one µm by HEPA filters. Fine particles were excluded from the ambient air with the MedicCleanAir® (Härі-Prolectron AG, Bronschhofen, Switserland) and the Genano® 310 (Genano OY, Espoo, Finland). The MedicCleanAir device also removes indoor gases, including ozone, from ambient air. During the cycling test, on a cycloergometer in the laboratory, a steady state heart rate equal to the individual's mean heart rate measured during the road test, was obtained.

The exercise began with an initial workload of 80 W for men and 50 W for women at a pedalling rate of 70–80 rates per minute. The load was gradually adapted to achieve the individual target heart rate during the first minute of exercise. The duration of the test was defined by the duration of the road test.Fine and ultrafine particles, present in the direct surrounding of the participant, were constantly measured, using the same GRIMM and P-TRAK devices as used during the road test (see below).

2.6.1.3 Exposure measurements

To measure personal exposure to particulate matter (PM_{10}, $PM_{2.5}$ and UFP) during the road test and during the clean room test, small portable, battery-powered, devices were used. The GRIMM 1.108 Dust monitor (Grimm Technologies Inc, USA) is a portable environment dust monitor that can simultaneously measure $PM_{1.0}$, $PM_{2.5}$, PM_{10} and total suspended particulate (TSP). Ultrafine particle counts at one-second resolution were measured using P-TRAK Ultrafine Particle Counters (TSI Model 8525, USA) for particles in the size range 0.02–1 µm (maximum 500,000 particles per cm^3). The P-TRAK is a hand-held, field instrument based on the condensation particle counting technique using isopropyl alcohol. We refer to (Berghmans et al. 2009) and (Int Panis L et al. 2010) for a detailed description of the PM and UFP measuring techniques while cycling in traffic.

2.6.1.4 Health measurements

A venous blood sample was drawn for the determination of plasma interleukin-6 (IL-6), platelet function, Clara cell protein in serum and blood cell counts and exhaled nitric oxide (NO) was measured, before the exercise. After the exercise, participants rested for 30 minutes in a seated position followed by the post-cycling examination, which included exhaled NO measurement and a venous blood sample collection.

2.6.1.5 Statistical analysis

For database management and statistical analysis, we used SAS software (version 9.1; SAS Institute Inc., Cary, NC, USA). Non-normally distributed values were log-transformed. We used linear mixed-effects models with adjustment for temperature, relative humidity and heart rate to investigate pre-cycling vs. post-cycling measurement on the studied parameters. Because within-individual repeated measures of outcomes are correlated, random effects were estimated at the subject level.

To study whether the change between pre/post-cycling measurements was different per exposure scenario, we included an interaction term between pre/post-cycling measurement and the exposure scenario (road test vs. clean room) in the model. We also ran the model with an interaction term between pre/post-cycling measurement and the UFP (or $PM_{2.5}$) concentrations during cycling.

2.6.2 Results

2.6.2.1 Participants characteristics

Thirty-eight physically fit, non-asthmatic participants (26 % women) with a mean age of 43 years (range: 28-58 years) and mean body-mass index (BMI) of 24 kg/m², participated (TABLE XXIII). Five persons reported hay fever.

TABLE XXIII Characteristics (n=38)

	Mean (SD) or number (%)
Anthropometrics	
Men/women	28/10 (74%/26%)
Age, years	43 (8.6)
BMI, kg/m²	23.7 (3.1)
Lifestyle	
Former smoker	16 (42%)
Exposure to environmental tobacco smoke	3 (8%)
Regular alcohol use	20 (53%)
Medication use	
Antiplatelet medication	0 (0%)
Lipid-lowering medication	1 (3%)
Antihypertensive medication	3 (8%)

When we compared the clinical parameters pre/post-cycling (TABLE XXIV), separately for the two exposure scenarios (road test and clean room), we observed a decrease in exhaled NO of -4.4% change from baseline (p=0.04) after the road test, but not in the clean room (-1.3% change from baseline; p = 0.63). However, the interaction term between pre/post-cycling measurement of exhaled NO and the exposure scenario was not significant (p=0.37).

Platelet function, IL-6, Clara cell protein and total leukocyte counts did not change significantly from baseline after cycling, neither during the road test, nor in the clean room. However, the percentage of blood neutrophils (though not their absolute number) increased by 3.9% after cycling in the road test (p=0.003), but not in the clean room (0.22%; p=0.83). A significant interaction (p=0.004) between percentage of neutrophils and exposure scenario was observed. In a model that included either UFP counts or $PM_{2.5}$ concentrations, the interaction terms were only significant for the percentage blood neutrophils and not for the other measured parameters (Table XXXI).

Similar results were obtained if we excluded the five persons that reported hay fever from our analysis.

TABLE XXIV: Percent change (pre/post) per exposure scenario (road test or clean room)

	Road test		Clean room		p-value for interaction		
Endpoint	Percent change (95%CI)	p-value	Percent change (95%CI)	p-value	Exposure scenario*	UFP†	$PM_{2.5}$‡
Exhaled NO	**-4.4% (-8.3% to -0.37%)**	**0.04**	-1.3% (-6.5% to 4.1%)	0.63	0.38	0.63	0.50
PFA closure time	6.5% (-1.0% to 14.5%)	0.10	5.1% (-1.0% to 11.6%)	0.11	0.76	0.60	0.59
Plasma IL-6	17.4% (-6.7% to 47.9%)	0.18	-2.9% (-19.0% to 16.4%)	0.75	0.21	0.38	0.40
Clara cell protein	1.6% (-10.8% to 15.8%)	0.82	-0.27% (-11.7% to 12.7%)	0.97	0.90	0.91	0.80
Blood leukocyte counts	1.3% (-2.0% to 4.6%)	0.44	2.5% (-1.1% to 6.0%)	0.19	0.75	0.97	0.71
Blood neutrophil counts	**4.6% (0.48% to 8.7%)**	**0.04**	2.4% (-2.3% to 7.2%)	0.32	0.36	0.35	0.20
Percentage blood neutrophils	**3.9% (1.5% to 6.2%)**	**0.003**	0.22% (-1.8% to 2.2%)	0.83	**0.004**	**0.02**	**0.01**

analysis adjusted for heart rate

* pre/post-cycling measurements and exposure scenario (road test or clean room)

† pre/post-cycling measurements and UFP concentrations

‡ pre/post-cycling measurements and $PM_{2.5}$ concentrations

In subjects free of lung and cardiovascular disease, a small, immediate (30 minutes after moderate exercise) increase in the percentage of blood neutrophils was observed in response to cycling in traffic-related exposure. Platelet function and a biomarker of lung permeability (Clara cell protein) did not show rapid changes between pre/post-cycling measurements in either exposure scenario. The change in pre/post-cycling measurement of exhaled NO did not differ significantly between the two scenarios. The health significance of this isolated change is unclear.

3 POLICY SUPPORT

PARHEALTH is a multidisciplinary project that incorporates medical, chemical and epidemiological disciplines to unravel the health consequences of air pollution in children and elderly. PARHEALTH integrates both, societal relevance, aiming protecting potential to the most susceptible group (children and children) as well as development of methods that might be relevant to monitor for specific air pollution constituents (oxy-PAH).

- In our epidemiological studies we focused to a large extend to the shape of the association to deliver policy relevant information on possible threshold levels or to provide insight to the impact of particulate air pollution on the current used air pollution standards.

- We deliver information on the shape of the association on the trigger effects of air pollution on infant mortality, showing a linear effect. Assuming causality, of our findings, the current EU limit value for PM_{10} which might be exceeded on 35 day per year, does not prevent PM_{10} from triggering mortality in late neonates.

- We provide information on the use of weather models to model air pollution constituents and its relevance as trigger of mortality at the population level

- Considering both the magnitude of the risk and the prevalence in the population, air pollution is not a trivial trigger of myocardial infarction (MI), it is of similar magnitude (explains 5 to 7% of MI in the population) as other well accepted triggers such as physical exertion, alcohol and coffee.

- At concentrations below the EU daily limit value, serum Clara protein in children, a marker of lung permeability, is associated with 7-day average particulate matter concentrations in the ambient air.

- Regarding pollutant specific compounds, we identified that iron, lead and zinc were most strongly correlated with increases in pulse pressure of elderly. For policy conclusion regarding this aspect the current findings must be interpret within the context of future studies.

4 DISSEMINATION AND VALORISATION

Presentations at congresses

- Scheers H. et al. (2008) Associations between air pollution and infant mortality in an affluent society: a case-crossover study. Poster at the International Conference on "Health Aspects of Indoor and Outdoor Air Pollution", Luxembourg (LUX), 12/11/2008.

- Scheers H. et al. (2010) Associations between air pollution and infant mortality in an affluent society. Poster at the 20th European Respiratory Society Annual Congress, Barcelona (SP), 18-22/09/2010.

- Scheers H. et al. (2010) Does air pollution trigger infant mortality in Western Europe? A case-crossover study. Poster at the conference 'From human biomonitoring to policy: a sustainable 'marriage' between health and environment', Brussels (B), 27-28/10/2010.

- Scheers H. et al. (2010) Does air pollution trigger infant mortality in Western Europe? A case-crossover study. Oral presentation by Tim Nawrot. European Federation Epidemiology (EROEPI), November 2010.

- Walgraeve, C., Demeestere, K., Dewulf, J. and Van Langenhove, H. (2010). Analysis and occurrence of oxygenated PAHs on atmospheric particulate matter. In Proceedings of the 10th Flemish Youth Conference on Chemistry (VJC10), March 1-2, 2010, Blankenberge, Belgium, 97-97 (awarded as the best poster in the field of environmental chemistry). Poster presentation

- Walgraeve, C., Demeestere, K., Dewulf, J. and Van Langenhove, H. (2010). Diffusive sampling of airborne VOCs in indoor environment: theoretical versus experimental uptake rates. In Proceedings of the 3rd EuCheMS Chemistry Congress, August 29 – September 2, 2010, Nürnberg, Germany, 1 p. (on CD-rom). Oral presentation

- A. Buczynska, Y. Makarovska, B. Horemans, E.A. Stefaniak, A. Worobiec, V. Novakovic and R. Van Grieken, Characterization of atmospheric aerosols from urban and rural areas in Belgium - at European Aerosol Conference 2008 (EAC 2008), Thessaloniki, Greece, 24-29 August 2008

- Andy Delcloo, Tim Nawrot, Lotte Jacobs and Hugo De Backer, Association between daily mortality and the composition of fine particulate matter by the use of the CTM CHIMERE, European Aerosol Conference, August 24 - 29, 2008, Thessaloniki - Greece.

- A.W. Delcloo, A. Deckmyn, R. Hamdi, H. De Backer, G. Foret and H. Van Langenhove, Coupling of the CTM CHIMERE to the high resolution LAM ALADIN for Belgium, International Technical Meeting on Air Pollution Modelling and its Application, Turino, Italy, 26 Sept - 1 Oct 2010.

5 PUBLICATIONS

- Jacobs L, Nawrot T, de GeusB, Meeusen R, Degraeuwe B, Bernard A, Sughis M, Nemery B, Int Panis L. Subclinical responses in healthy cyclists briefly exposed to traffic-related air pollution: an intervention study. *Environmental Health* 2010; 9:64

- Scheers H, Mwalili SM, Faes C, Fierens F, Nemery B, Nawrot TS. Does Air Pollution Trigger Infant Mortality in Western Europe? A Case-Crossover Study. *Environ Health Perspect* 2011:-. doi:10.1289/ehp.1002913 (Ahead of Print)

- Nawrot T, Perez L, Kunzli N, Nemery B. The Public Health Relevance of trggers of myocardial infarction: comparative risk assessment. Lancet (in press)

- Walgraeve, C., Demeestere, K., Dewulf, J., Zimmermann, R. and Van Langenhove, H. Oxygenated polycyclic aromatic hydrocarbons in atmospheric particulate matter: molecular characterization and occurrence. *Atmospheric Environment* 2010; 44: 1831-1846.

- Walgraeve, C., Demeestere, K., Dewulf, J., Van Huffel, K., Van Langenhove, H. Uptake rate behaviour of tube-type passive air samplers for volatile organic compounds under controlled laboratory conditions. Submitted to *Atmospheric Environment* 2011.

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